Maternal Alcohol-Use Disorder and Child Outcomes

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abstract

OBJECTIVES: Investigate the relationship between maternal alcohol-use disorder and multiple biological and social child outcomes, including birth outcomes, child protection, justice contact, and academic outcomes for both Indigenous and non-Indigenous children.

METHODS: Women with a birth recorded on the Western Australian Midwives Notification System (1983–2007) and their offspring were in scope. The exposed cohort were mothers with an alcohol-related diagnosis (*International Classification of Diseases, Ninth Revision* and *International Classification of Diseases, 10th Revision*) recorded in an administrative data set and their offspring (non-Indigenous: n = 13 969; Indigenous: n = 9635). The exposed cohort was frequency matched with mothers with no record of an alcohol-related diagnosis and their offspring (comparison cohort; non-Indigenous: n = 40 302; Indigenous: n = 20 533).

RESULTS: Over half of exposed non-Indigenous children (55%) and 84% of exposed Indigenous children experienced ≥1 negative outcome. The likelihood of any negative outcome was significantly higher for the exposed than the comparison cohort (non-Indigenous: odds ratio [OR] = 2.67 [95% confidence interval (CI) = 2.56–2.78]; Indigenous: OR = 2.67 [95% CI = 2.50–2.85]). The odds were greatest for children whose mothers received a diagnosis during pregnancy (non-Indigenous: OR = 4.65 [95% CI = 3.87–5.59]; Indigenous: OR = 5.18 [95% CI = 4.10–6.55]); however, numbers were small.

CONCLUSIONS: The effects of maternal alcohol-use disorder are experienced by the majority of exposed children rather than a vulnerable subgroup of this population. These findings highlight the need for universal prevention strategies to reduce harmful alcohol use and targeted interventions to support at-risk women and children.

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Dr O'Leary conceptualized and designed the original cohort study, acquired the data, obtained funding, contributed to the design of this study and interpretation of results, and drafted the initial manuscript; Dr Lawrence contributed to the design of this study and provided expert statistical advice on the analysis and interpretation of results; Ms Hafekost contributed to the design of this study, undertook the statistical analysis, contributed to the interpretation of results, and drafted the initial manuscript; Dr Zubrick contributed to the study design of this study and interpretation of results; Dr Bower conceptualized and designed the original cohort study, obtained funding, and contributed to the design of this study and interpretation of results; and all authors revised and approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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WHAT'S KNOWN ON THIS SUBJECT: There is a substantial body of literature demonstrating the negative impact maternal alcohol-use disorder can have on children through prenatal alcohol exposure and/or exposure to negative environmental factors associated with alcohol-use disorder during childhood.

WHAT THIS STUDY ADDS: This study uses linked administrative data to demonstrate the high proportion of non-Indigenous and Indigenous offspring of mothers with an alcohol-use disorder who have the cooccurrence of ≥1 negative biological and/or social outcome.

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A substantial body of literature identifies the increased risk of poor child outcomes that are associated with heavy maternal alcohol use.[1,2] These poor outcomes have been attributed to both the biological effects of exposure to alcohol in utero[3,4] and the social or environmental factors, which cluster with parental alcohol problems.[5–7] Prenatal alcohol exposure is associated with an increased risk of a range of neurodevelopmental problems, which are referred to as fetal alcohol spectrum disorders (FASDs).[2–4] Although these neurodevelopmental disorders can adversely impact children's development, children of mothers with alcohol-use disorders are also exposed to a range of environmental risk factors, including risk of parental mental health problems and other substance use, economic disadvantage, family dysfunction and instability, poor parenting skills, and social isolation.[5–8] Previous studies have demonstrated that children of mothers with maternal alcohol-use disorders are at an increased risk of a range of adverse developmental outcomes, including poor academic achievement, behavioral problems, and contact with police and the justice system.[1–3,9–13]

A number of studies using the same population cohort of Western Australian mothers with an alcohol-related diagnosis and their children considered in this present article have demonstrated an increased likelihood of a range of negative biological and social outcomes for children of mothers with an alcohol-related diagnosis.[14–22] These previous studies revealed that the highest risk of intellectual disability,[20] prenatally or perinatally acquired cerebral palsy,[18] being small for gestational age, being born very preterm,[21] or alcohol-related birth defects[19] occurred in children whose mothers received an alcohol-related diagnosis during pregnancy, indicating that maternal alcohol-use disorder is a direct cause of these biological outcomes. In addition, there were significantly increased odds of stillbirth,[22] sudden infant death syndrome, and other infant mortality[23] for children of mothers with an alcohol-related diagnosis recorded prepregnancy and/or postpregnancy.

In contrast to the studies on biological outcomes, the timing of the diagnosis made little difference in the strength of association in the analyses of educational outcomes, including failing to meet minimum academic standards,[16] having poor attendance,[14] and having child contact with the justice system.[15] The authors suggested that social or environmental factors may drive this relationship. Increased odds of a substantiated child protection allegation or an out-of-home period have also been reported, with the risk highest being when the mother had an alcohol diagnosis recorded during pregnancy or within 1 year prepregnancy or postpregnancy.[17] The increased risk was maintained over time and was significantly higher for children with a FASD, an intellectual disability, and low birth weight, which may indicate an interplay of biological and social or environmental factors on the risk of child protection contact.

The existing work from this Western Australian cohort study has not established what proportion of exposed children experience multiple adverse effects. This study combines the outcome data used in these existing analyses to examine the relationship between maternal alcohol-use disorder and the co-occurrence of multiple negative biological and social outcomes.

METHODS

This project made use of routinely linked administrative data from Western Australia to identify preterm birth, being small for gestational age, cerebral palsy, intellectual disability, birth defects, education outcomes, and contact with the justice and child protection systems in the offspring of women with and without a record of an alcohol-related diagnosis. The linkage of these data sets has previously been described in detail.[19]

Cohorts

The cohort was selected from women who had a live birth recorded in the Western Australia Midwives Notification System (MNS) between 1983 and 2007.[18,19] The exposed cohort was defined as mothers who had an alcohol-related diagnosis, which is a proxy for heavy maternal alcohol use (recorded on ≥1 of the following: hospital morbidity, mental health inpatient or outpatient, or drug and alcohol services data set) and their offspring. An alcohol-related diagnosis (or alcohol diagnosis) was based on *International Classification of Diseases, Ninth Revision* (ICD-9) or *International Classification of Diseases, 10th Revision* (ICD-10) codes and included alcohol-related mental and behavioral disorders that could be entirely attributed to alcohol and "other" alcohol codes.[18]

The timing of an alcohol diagnosis was hierarchically coded, with those recorded during pregnancy given the highest priority.[18] The following categories were used: (1) any alcohol diagnosis during pregnancy (this included women who may also have had an alcohol diagnosis at another time point), (2) an alcohol diagnosis ≤1 year before pregnancy (this group included women who may have also had an alcohol diagnosis recorded >1 year before pregnancy or after pregnancy), (3) an alcohol diagnosis ≤1 year after pregnancy (this included women who may have had an alcohol diagnosis >1 year before or after pregnancy), (4) an alcohol diagnosis >1 year before pregnancy (this could include those with a diagnosis >1 year after pregnancy),

and (5) an alcohol diagnosis >1 year after pregnancy.

The comparison cohort included mothers with no alcohol-related diagnosis recorded on any of the relevant linked administrative data sets and their offspring. The exposed cohort was frequency matched with comparison mothers on maternal age within the Indigenous status and the year of the child's birth at a ratio of 1:3 for non-Indigenous and 1:2 for Indigenous mothers. The final population cohort included 84 439 births. The exposed cohort included 13 969 non-Indigenous and 9635 Indigenous offspring and the comparison cohort included 40 302 non-Indigenous and 20 533 Indigenous offspring.

Outcome Data Sources

Births were identified through the MNS, a population-based surveillance system, which provided data on maternal demographic and pregnancy factors and data on small-for-gestational-age (bottom 10th percentile of optimal birth weight)[24] and preterm (<37 weeks' gestation) births. The Western Australian Register of Developmental Anomalies provided data on birth defects, which are coded by using the British International Classification of Diseases System (ninth revision).[25] Defects due to a chromosomal cause were excluded. The alcohol-related birth defect codes included in this study and additional information about the registry have been previously published.[19] Intellectual disability data were obtained from the Intellectual Disability Exploring Answers Database.[26] For this analysis, a binary outcome (presence or absence) of intellectual disability, defined as an IQ of <70, identified from either source was used. The Western Australian Cerebral Palsy Register[27] provided data on all cases of prenatal, perinatal, and postnatal cerebral palsy.[18]

Educational data were obtained from the Western Australia Department of Education for government public schools; details have been published previously.[16] Briefly, achievement data were obtained from the Western Australia Literacy and Numeracy Assessment for the years 1999 through 2007, which included assessments for numeracy, reading, writing, and spelling for children in years 3, 5, and 7. From 2005 to 2007, children in year 9 were also assessed in reading, writing, and numeracy. In addition, National Assessment Program: Literacy and Numeracy results were obtained from 2008. The primary outcome of interest was failure to meet the minimum standards on any testing domain in any of the recorded school years with valid records for 45 382 children.[16] Attendance was classified as 1 of the following: regular (90%–100% attendance), indicated (80%–89% attendance), moderate (60%–79% attendance), and severe (<60% attendance). The small number of students who had an attendance of <30% were excluded because there are some cases in which students change schools, sometimes enrolling under a different name, without informing the previous school.[14] For the purpose of this analysis, we focused on those children who fell into the severe attendance category (equating to 13 486 children), referred to in this article as "having poor school attendance."

Department of Corrective Services (now called Department of Justice) custodial and community corrections data for juveniles and adults from 1985 to 2011 were available for 68 598 children. In Western Australia, the minimum age for such an offense to be recorded on this data set is 10 years, so children born after 2002 were not included. A justice contact, for the purposes of this analysis, was defined as a custodial or community corrections record within the justice system.

The Department of Communities, Child Protection and Family Support data set included cases of substantiated maltreatment (substantiation) and out-of-home care from 1990 to 2007.[17] Contact with child protection was defined as either a substantiated maltreatment allegation or a record of a period of out-of-home care for ≥1 day.

Ethics approval for the conduct of the study was granted by the Princess Margaret Hospital Human Research Ethics Committee (no. 1244/EP), the Western Australia Department of Health Human Research Ethics Committee (no. 2011/34), and the Western Australian Aboriginal Health Ethics Committee (no. 134-04/06).

Statistical Analysis

All analysis was conducted by using SAS 9.3 (SAS Institute, Inc, Cary, NC). All outcomes were considered binary (presence or absence).

To identify if academic outcomes, justice outcomes, and child protection outcomes predominantly co-occurred in the same group of children or individually affecting a larger group of children, we cross-tabulated these outcomes.

To identify cumulative impacts, the number of negative outcomes were summed, and results were categorized as 1, 2, or ≥3. Because some outcome variables were likely to be negatively associated and not captured in a summed scale, the ϕ coefficient was calculated to estimate the association between outcome variables included in the analysis. This was displayed graphically to demonstrate both positive and negative associations between the outcome variables of interest in the study.

Logistic regression was used to examine the relationship between an alcohol diagnosis, the timing of diagnosis relative to pregnancy, and the odds of a negative outcome. Multinomial logistic regression was

TABLE 1 Sample Characteristics by Indigenous Status and Alcohol Exposure

	Non-Indigenous			Indigenous		
	Comparison	Exposed	Unadjusted OR (95% CI)	Comparison	Exposed	Unadjusted OR (95% CI)
	n (%)	*n* (%)		*n* (%)	*n* (%)	
Maternal age group, y						
<20	4790 (11.9)	1709 (12.2)	1.02 (0.95–1.09)	5400 (26.3)	2558 (26.6)	1.02 (0.96–1.08)
20–<25	11 829 (29.4)	4141 (29.6)	Reference	6855 (33.4)	3190 (33.1)	Reference
25–<30	11 969 (29.7)	4103 (29.4)	0.98 (0.98–1.03)	4727 (23.0)	2208 (22.9)	1.00 (0.94–1.07)
30–<35	7890 (19.6)	2714 (19.4)	0.98 (0.93–1.04)	2505 (12.2)	1185 (12.3)	1.02 (0.94–1.10)
35–<40	3219 (8.0)	1095 (7.8)	0.97 (0.9–1.05)	929 (4.5)	442 (4.6)	1.02 (0.91–1.15)
40+	605 (1.5)	207 (1.5)	0.98 (0.83–1.15)	117 (0.6)	52 (0.5)	0.96 (0.69–1.33)
Maternal mental health record						
No	36 616 (90.9)	6599 (47.2)	Reference	18 103 (88.2)	6792 (70.5)	Reference
Yes	3686 (9.2)	7370 (52.8)	11.09 (10.6–11.6)	2430 (11.8)	2843 (29.5)	3.12 (2.93–3.31)
Maternal illicit drug record						
No	39 690 (98.5)	8626 (61.8)	Reference	19 573 (95.3)	7477 (77.6)	Reference
Yes	612 (1.5)	5343 (38.3)	40.17 (36.8–43.8)	960 (4.7)	2158 (22.4)	5.89 (5.44–6.39)
Marital status						
Married	34 812 (86.4)	10 273 (73.5)	Reference	13 374 (65.1)	5593 (58.1)	Reference
Never married	5098 (12.7)	3117 (22.3)	2.07 (1.97–2.18)	6697 (32.6)	3767 (39.1)	1.35 (1.28–1.42)
Separated, widowed, or divorced	357 (0.9)	525 (3.8)	4.98 (4.35–5.71)	355 (1.7)	209 (2.2)	1.14 (1.18–1.67)
Unknown	35 (0.1)	54 (0.4)	5.23 (3.42–8.00)	107 (0.5)	66 (0.7)	1.47 (1.08–2.01)
Socioeconomic status, %						
<10 (most advantaged)	2712 (6.7)	559 (4.0)	Reference	107 (0.5)	22 (0.2)	Reference
10–<25	5079 (12.6)	1218 (8.7)	1.16 (1.04–1.30)	353 (1.7)	81 (0.8)	1.12 (0.67–1.87)
25–<50	8642 (21.4)	2525 (18.1)	1.42 (1.28–1.57)	1389 (6.8)	454 (4.7)	1.59 (0.99–2.55)
50–<75	9490 (23.6)	3402 (24.4)	1.74 (158–1.92)	3379 (16.5)	1249 (13.0)	1.80 (1.13–2.86)
75–<90	6033 (15.0)	2630 (18.8)	2.11 (1.91–2.34)	4418 (21.5)	1801 (18.7)	1.98 (1.25–3.15)
10 (most disadvantaged)	3516 (8.7)	2028 (14.5)	2.80 (2.52–3.11)	6078 (29.6)	2860 (29.7)	2.29 (1.44–3.63)
Unknown	4830 (12.0)	1607 (11.5)	1.61 (1.45–1.80)	4809 (23.4)	3168 (32.9)	3.20 (2.02–5.08)

used to quantify the risk of multiple negative outcomes for children of mothers with an alcohol diagnosis.

All models included the frequency-matching variables of maternal age and child's year of birth, and non-Indigenous and Indigenous data were analyzed separately. The Indigenous status of the mother is self-reported and recorded in the MNS. Multivariate models were adjusted for other maternal and child characteristics associated with adverse child outcomes. These covariates included the mother's marital status at the time of birth, maternal mental health or illicit drug record, socioeconomic status, health region (metropolitan or rural), child's sex, and parity. To produce the most parsimonious model, covariates were eliminated from the multivariate model if they were not significantly associated with the outcome (α =.05).

The attributable fraction refers to the risk that can be attributed to exposure, whereas the population attributable fraction (PAF) takes into account the prevalence of exposure within the population. The attributable fraction and PAF were calculated to allow for estimation of the impact of heavy maternal alcohol use on any of the child outcomes included in the analyses. The SAS STDRATE procedure was used to calculate the PAF and attributable fraction.

RESULTS

Exposed non-Indigenous mothers were more likely than comparison mothers to be unmarried (26.5% vs 13.6%), be in the lowest 10% of socioeconomic status (14.5% vs 8.7%), have a mental health diagnosis (52.8% vs 9.2%), and have an illicit drug diagnosis (38.3% vs 1.5%) (Table 1). In the Indigenous mother cohort, there was little difference between exposed and comparison mothers in the distribution across socioeconomic groups. However, a greater proportion of exposed than of comparison mothers had a mental health diagnosis (29.5% vs 11.8%) or an illicit drug diagnosis (22.4% vs 4.7%).

A greater proportion of Indigenous children than of non-Indigenous children had a record of any negative outcome irrespective of exposure status (Table 2). A greater proportion of children of mothers with an alcohol diagnosis had 1 or more negative outcomes (non-Indigenous: 53.4%; Indigenous: 84.0%) than did comparison children (non-Indigenous: 31.2%; Indigenous: 68.0%). The relationship between the number of negative outcomes identified for both non-Indigenous and Indigenous comparison and exposed children is displayed in Figs 1 and 2.

TABLE 2 Outcomes by Indigenous Status and Alcohol Exposure

	Non-Indigenous			Indigenous		
	Comparison	Exposed	Unadjusted OR (95% CI)	Comparison	Exposed	Unadjusted OR (95% CI)
	n (%)	*n* (%)		*n* (%)	*n* (%)	
Fetal alcohol syndrome						
No	40 302 (100)	13 960 (99.9)	Reference	20 513 (99.9)	9540 (99.0)	Reference
Yes	0 (0)	9 (0.1)	N/A	20 (0.1)	95 (1.0)	10.21 (6.3–16.55)
Intellectual disability						
No	39 763 (98.7)	13 649 (97.7)	Reference	20 040 (97.6)	9254 (96.1)	Reference
Yes	539 (1.3)	320 (2.3)	1.73 (1.50–1.99)	493 (2.4)	381 (4.0)	1.67 (1.46–1.92)
Birth defect						
No	39 567 (98.2)	13 700 (98.1)	Reference	20 250 (98.6)	9429 (97.9)	Reference
Yes	735 (1.8)	269 (1.9)	1.06 (0.92–1.22)	283 (1.4)	206 (2.1)	1.56 (1.30–1.87)
Cerebral palsy						
No	40 206 (99.8)	13 910 (99.6)	Reference	20 435 (99.5)	9596 (99.6)	Reference
Yes	96 (0.2)	59 (0.4)	1.78 (1.28–2.46)	98 (0.5)	39 (0.4)	0.85 (0.58–1.23)
Small for gestational age						
No	36 871 (91.5)	11 782 (84.3)	Reference	17 416 (84.8)	6969 (72.3)	Reference
Yes	3319 (8.2)	2121 (15.2)	2.0 (1.89–2.12)	2644 (12.9)	2222 (23.1)	2.10 (1.97–2.24)
Missing	112 (0.3)	66 (0.5)	N/A	473 (2.3)	444 (4.6)	N/A
Preterm (<37 wk)						
No	37 271 (92.5)	12 368 (88.5)	Reference	17 505 (85.3)	7508 (77.9)	Reference
Yes	2919 (7.2)	1540 (11.0)	1.26 (1.49–1.70)	2556 (12.5)	1683 (17.5)	1.54 (1.44–1.64)
Missing	112 (0.3)	61 (0.4)	N/A	472 (2.3)	444 (4.6)	N/A
Child protection[a]						
No	39 203 (97.3)	11 748 (84.1)	Reference	18 553 (90.4)	6725 (69.8)	Reference
Yes	961 (2.4)	2140 (15.3)	7.43 (6.87–8.04)	1834 (8.9)	2783 (28.9)	4.19 (3.92–4.47)
Out of scope	138 (0.3)	81 (0.6)	N/A	146 (0.7)	127 (1.30)	N/A
School failure[b]						
No	17 947 (44.5)	5565 (39.8)	Reference	4198 (20.5)	1219 (12.7)	Reference
Yes	4130 (10.3)	2206 (15.8)	1.72 (1.62–183)	6569 (32.0)	3548 (36.8)	1.86 (1.72–2.01)
Out of scope	18 225 (45.2)	6198 (44.4)	N/A	9766 (47.6)	4868 (50.5)	N/A
Poor school attendance[c]						
No	12 076 (30.0)	4129 (29.6)	Reference	3065 (14.9)	972 (10.1)	Reference
Yes	2726 (6.8)	1998 (14.3)	2.14 (2.00–229)	5880 (28.6)	2882 (29.9)	1.55 (1.42–1.68)
Out of scope	25 500 (63.3)	7842 (56.1)	N/A	11 588 (56.4)	5781 (60.0)	N/A
Justice contact[d]						
No	31 712 (78.7)	10 427 (74.6)	Reference	13 833 (67.4)	5890 (61.1)	Reference
Yes	930 (2.3)	860 (6.2)	2.81 (2.56–3.09)	2784 (13.6)	2162 (22.4)	1.82 (1.71–1.94)
Out of scope	7660 (19.0)	2682 (19.2)	N/A	3916 (19.1)	1583 (16.4)	N/A
Negative outcomes						
0	27 723 (68.8)	6513 (46.6)	Reference	6579 (32.0)	1539 (16.0)	Reference
1	9457 (23.5)	4480 (32.1)	2.02 (1.93–2.11)	7053 (34.4)	2964 (30.8)	1.80 (1.68–1.93)
2	2554 (6.3)	2077 (14.9)	3.46 (3.25–3.69)	4980 (24.3)	3024 (31.4)	2.60 (2.42–2.79)
3	568 (1.4)	899 (6.4)	6.74 (6.05–7.51)	1921 (9.4)	2108 (21.9)	4.69 (4.32–5.10)

N/A, not applicable.

[a] Children who died before 1990 are not included in the child protection data set.

[b] Children born before 1989 are not included in the academic outcomes data set.

[c] Children born before 1992 are not included in the attendance data set.

[d] Children born after 2002 are not eligible to be in the justice data set.

The proportion of children with poor social outcomes was higher in the exposed than in the comparison cohort, with pronounced differences observed for child protection contact (non-Indigenous 15.3% vs 2.4% and Indigenous 28.9% vs 8.9%, respectively), academic outcomes (non-Indigenous 15.8% vs 10.3% and Indigenous 36.8% vs 32.0%, respectively), and contact with the justice system (non-Indigenous 6.2% vs 2.3% and Indigenous 22.4% vs 13.6%, respectively; Table 2). The same pattern was observed for each of the biological outcomes with the exception of cerebral palsy in Indigenous children, in which the proportion was lower for the exposed than for the comparison cohort (0.4% vs 0.5%, respectively).

The proportion of children having a justice contact who also failed to reach academic benchmarks is shown in Table 3. Approximately 35% of exposed and comparison non-Indigenous children in contact with the justice system failed to reach minimum academic standards (Table 3). Failure to reach minimum benchmarks was highest for Indigenous children in contact with

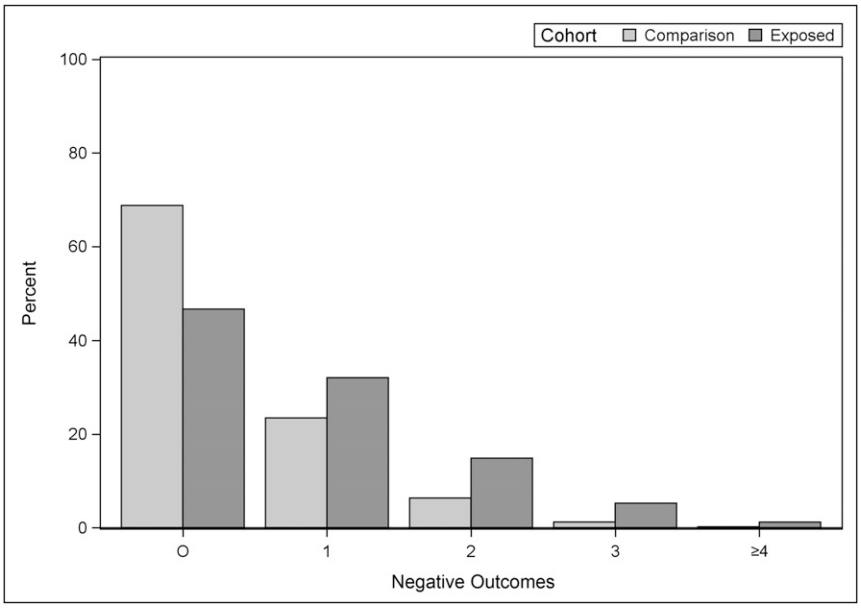

FIGURE 1
Number of negative outcomes identified in non-Indigenous children by maternal alcohol-use diagnosis.

the justice system, with 80% of the exposed and 71% of the comparison cohort failing to reach a minimum academic standard. For non-Indigenous exposed children in contact with the justice system, school attendance was also poorer (63.6%) than for comparison children (52.9%). However, this was not evident for Indigenous children, with the proportion being lower for exposed (81.4%) than for comparison children (89.6%; Table 4). Of note, numbers are small, so the results should be interpreted within this context.

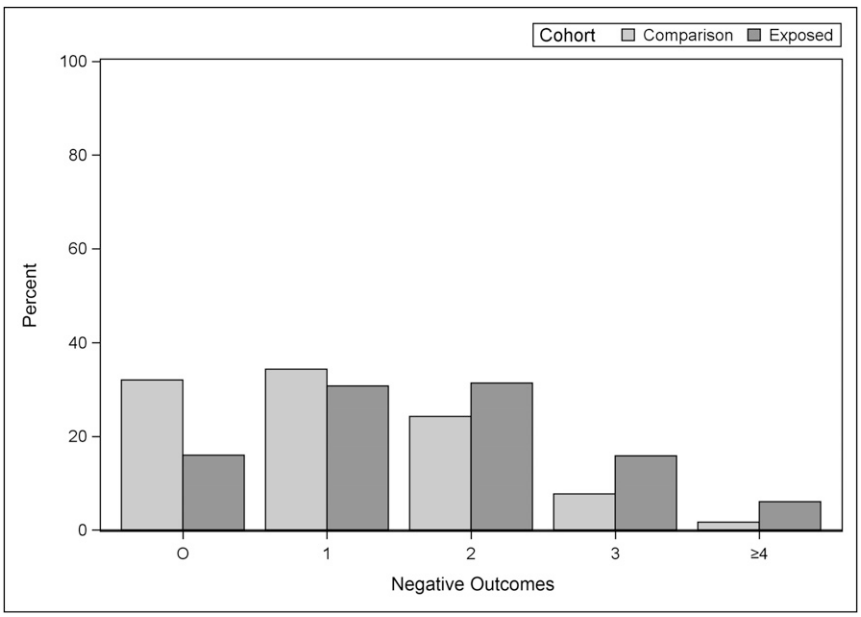

FIGURE 2
Number of negative outcomes identified in Indigenous children by maternal alcohol-use diagnosis.

The percentage of children failing to meet academic standards was higher in Indigenous than in non-Indigenous children irrespective of child protection contact. For children who had child protection contact, the proportion failing to meet minimum academic standards was similar between exposed and comparison cohorts (non-Indigenous 40.4% vs 40.8% and Indigenous 70.5% vs 75.2%, respectively; Table 5). The percentage of exposed and comparison children with poor school attendance showed a pattern similar to the results for academic failure (non-Indigenous 39.7% vs 41.9% and Indigenous 66.0% vs 72.9%, respectively; Table 6).

The adjusted odds of a child whose mother had an alcohol-related diagnosis having any negative outcome was >2.5 times greater than for the comparison cohort (non-Indigenous: odds ratio [OR] = 2.67 [95% confidence interval (CI) = 2.56–2.78]; Indigenous: OR = 2.67 [95% CI = 2.50–2.85]; Table 7). The highest odds were observed when an alcohol-related diagnosis was recorded during pregnancy (non-Indigenous: OR = 4.65 [95% CI = 3.87–5.59]; Indigenous: OR = 5.18 [95% CI = 4.10–6.55]).

The association between each of the outcome variables for children in the exposed cohort is shown in Supplemental Figs 3 and 4. These figures indicate that there are both positive and negative associations between the outcomes included in the study. For example, there were positive associations between child protection contact, school failure, poor school attendance, and justice contact. These outcomes were negatively associated with the biological outcomes. Given the negative associations between some outcomes, analysis that makes use of

TABLE 3 Failure To Meet Minimum Academic Standards by Justice Contact and Exposure to a Maternal Alcohol-Use Disorder

	Justice Contact											
	Non-Indigenous						Indigenous					
	Comparison		Exposed		OR (95% CI)		Comparison		Exposed		OR (95% CI)	
	No	Yes	No	Yes	Justice Contact	Failure To Reach Minimum Academic Standard	No	Yes	No	Yes	Justice Contact	Failure To Reach Minimum Academic Standard
Failure to reach minimum academic standard												
No	81.5	65.7	72.1	63.1	3.1 (2.5–3.8)	—	39.8	29.1	26.5	19.8	2.0 (1.6–2.5)	—
Yes	18.5	34.3	27.9	36.9	2.0 (1.5–2.7)	—	60.2	70.9	73.5	80.2	1.8 (1.6–2.1)	—
Total	21 144	289	7262	282	—	—	9581	927	3895	746	—	—
Justice contact												
No	—	—	—	—	—	1.7 (1.6–1.8)	—	—	—	—	—	1.8 (1.7–2.0)
Yes	—	—	—	—	—	1.1 (0.8–1.6)	—	—	—	—	—	1.7 (1.3–2.1)

—, not applicable.

summed outcomes should be interpreted within this context.

The adjusted odds of exposed children having 1, 2, or ≥3 negative outcomes by the timing of an alcohol diagnosis in relation to pregnancy are shown in Table 8. The odds of exposed non-Indigenous children having ≥3 negative outcomes were 3.79 (95% CI = 3.3–4.35) and were 4.47 for Indigenous children (95% CI = 4.07–4.92) for "any" alcohol-related diagnosis. The highest odds were observed when an alcohol-related diagnosis was recorded during pregnancy (non-Indigenous: OR = 9.62 [95% CI = 6.78–13.64]; Indigenous: OR = 14.12 [95% CI = 10.67–18.70]).

The PAF for any alcohol-related diagnosis in the non-Indigenous cohort was 1.2% (95% CI = 1.1%–1.3%) and was 4.9% for the Indigenous cohort (95% CI = 4.5%–5.2%; Table 9).

DISCUSSION

This study adds new and important information about the magnitude and breadth of the negative impact of maternal alcohol-use disorders on children. Over half (55%) of non-Indigenous and 84% of Indigenous children whose mothers had an alcohol-related diagnosis had ≥1 of the negative outcomes examined in this study. Both Indigenous and non-Indigenous children whose mothers had an alcohol-related diagnosis were 2.67 times more likely than children in the respective comparison cohorts to have a negative outcome (non-Indigenous: OR = 2.67 [95% CI = 2.56–2.78]; Indigenous: OR = 2.67 [95% CI = 2.50–2.85]).

Of particular concern is the likelihood of exposed children having ≥3 negative outcomes. The odds of experiencing ≥3 negative outcomes when any alcohol-related diagnosis was the exposure variable were 3.8 for non-Indigenous and 4.5 for Indigenous children. When a mother had an alcohol-related diagnosis recorded during pregnancy, the ORs increased to 9.6 and 14.1, respectively.

The findings of this study revealed that children of mothers with an

TABLE 4 Poor Attendance at School by Justice Contact and Exposure to a Maternal Alcohol-Use Disorder Exposure

	Justice Contact											
	Non-Indigenous						Indigenous					
	Comparison		Exposed		OR (95% CI)		Comparison		Exposed		OR (95% CI)	
	No	Yes	No	Yes	Justice Contact	Poor Attendance at School	No	Yes	No	Yes	Justice Contact	Poor Attendance at School
Poor attendance at school												
No	78.7	47.1	63.1	36.4	2.2 (1.0–5.1)	—	31.4	10.5	21.9	18.6	6.3 (3.1–13.1)	—
Yes	21.3	52.9	36.9	63.6	1.6 (0.8–3.2)	—	68.6	89.6	78.1	81.4	2.0 (1.5–2.6)	—
Total	11 178	34	4656	33	—	—	6738	134	2859	140	—	—
Justice contact												
No	—	—	—	—	—	2.2 (2.0–2.3)	—	—	—	—	—	1.6 (1.5–1.8)
Yes	—	—	—	—	—	1.6 (0.5–4.6)	—	—	—	—	—	0.5 (0.2–1.1)

—, not applicable.

TABLE 5 Failure To Reach Minimum Academic Standards by Child Protection Contact and Exposure to a Maternal Alcohol-Use Disorder

	Child Protection Contact											
	Non-Indigenous						Indigenous					
	Comparison		Exposed		OR (95% CI)		Comparison		Exposed		OR (95% CI)	
	No	Yes	No	Yes	Child Protection Contact	Failure To Reach Minimum Academic Standard	No	Yes	No	Yes	Child Protection Contact	Failure To Reach Minimum Academic Standard
Failure to reach minimum academic standard												
No	81.9	59.2	74.0	59.6	8.0 (7.0–9.2)	—	40.0	29.5	26.0	24.8	5.9 (4.9–7.0)	—
Yes	18.1	40.8	26.0	40.4	5.0 (4.2–5.9)	—	60.0	70.5	74.0	75.2	3.9 (3.5–4.4)	—
Total	21 496	581	6502	1269	—	—	9723	1044	3207	1560	—	—
Child protection contact												
No	—	—	—	—	—	1.6 (1.5–1.7)	—	—	—	—	—	1.9 (1.7–2.1)
Yes	—	—	—	—	—	1.0 (0.8–1.2)	—	—	—	—	—	1.3 (1.1–1.5)

—, not applicable.

alcohol-related diagnosis are at risk of having multiple negative social outcomes, which supports the findings of previous research.[28,29] A higher proportion of children in contact with the justice system and whose mothers had an alcohol-related diagnosis had poor school attendance than comparison children did. In contrast, the proportion of children in contact with the justice system who failed to meet academic standards was similar between exposed and comparison cohorts for both non-Indigenous and Indigenous children, a finding that was replicated for school outcomes in children with a child protection contact. This may indicate that the relationship between exposure and multiple outcomes is not driven by school factors alone. These results highlight that multiple agencies need to work together to provide a coordinated and collaborative program of prevention and early intervention strategies to improve long-term outcomes for children of mothers with an alcohol-related diagnosis.

In this study, Indigenous children were more likely than non-Indigenous children to experience negative outcomes, particularly social outcomes, and this was more pronounced in the exposed cohort. A higher proportion of Indigenous Australians have deep and persistent disadvantage, unemployment, housing overcrowding, imprisonment, poor health outcomes, and early mortality than non-Indigenous Australians do,[30,31] and reducing this disparity is an ongoing government priority.[32] Comprehensive Indigenous-specific programs designed to address maternal alcohol-use disorder, prevent prenatal alcohol exposure, and improve the biological and social outcomes of Indigenous children need to be well funded and

TABLE 6 Poor Attendance at School by Child Protection Contact and Exposure to a Maternal Alcohol-Use Disorder

	Child Protection Contact											
	Non-Indigenous						Indigenous					
	Comparison		Exposed		OR (95% CI)		Comparison		Exposed		OR (95% CI)	
	No	Yes	No	Yes	Child Protection Contact	Poor Attendance at School	No	Yes	No	Yes	Child Protection Contact	Poor Attendance at School
Poor attendance at school												
No	82.3	58.1	68.9	60.3	8.7 (7.5–10.3)	—	35.0	27.1	20.9	34.0	10.2 (8.4–12.3)	—
Yes	17.7	41.9	31.1	39.7	3.8 (3.1–4.6)	—	65.0	72.9	79.1	66.0	3.6 (3.2–4.1)	—
Total	14 372	430	5063	1064	—	—	8122	823	2582	1272	—	—
Child protection contact												
No	—	—	—	—	—	2.1 (1.9–2.3)	—	—	—	—	—	2.0 (1.8–2.3)
Yes	—	—	—	—	—	0.9 (0.7–1.2)	—	—	—	—	—	0.7 (0.6–0.9)

—, not applicable.

TABLE 7 Odds of Any Negative Outcome by Indigenous Status and Timing of Alcohol Diagnosis Relative to Pregnancy

	Non-Indigenous		Indigenous	
	OR[a]	95% CI	OR[a]	95% CI
No diagnosis	Reference	—	Reference	—
Alcohol-related diagnosis at any time	2.67	2.56–2.78	2.67	2.50–2.85
Timing of alcohol-related diagnosis				
During pregnancy	4.65	3.87–5.59	5.18	4.10–6.55
>1 y postpregnancy	2.63	2.50–2.76	2.39	2.21–2.59
>1 y prepregnancy	2.39	2.22–2.56	2.73	2.37–3.14
≤1 y postpregnancy	4.15	3.41–5.04	3.50	2.63–4.64
≤1 y prepregnancy	2.85	2.48–3.28	2.83	2.25–3.57

—, not applicable.
[a] Adjusted for infant year of birth and maternal age group (frequency-matching variables).

delivered consistently across time to improve the long-term outcomes of Indigenous people.

Over half of the exposed children had ≥1 negative outcome, highlighting that preventing and reducing the prevalence of maternal alcohol-use disorder should be a public health priority. The low PAF reflects the relatively low prevalence of mothers with an alcohol-related diagnosis recorded in health and mental health data sets, particularly among non-Indigenous women. In Western Australia, asking women who are pregnant and women of childbearing age about their alcohol consumption is not routinely done and urgently needs to be implemented and accompanied by brief interventions and treatment when required.[33]

TABLE 8 Odds of Negative Outcomes by Indigenous Status and Timing of Alcohol-Related Diagnosis Relative to Pregnancy

No. Outcomes	Non-Indigenous		Indigenous	
	OR[a]	95% CI	OR[b]	95% CI
No diagnosis	Reference	—	—	—
Alcohol-related diagnosis at any time				
≥3	3.79	3.30–4.35	4.47	4.07–4.92
2	2.38	2.19–2.58	2.55	2.35–2.76
1	1.68	1.59–1.77	1.69	1.57–1.83
During pregnancy				
≥3	9.62	6.78–13.64	14.12	10.67–18.70
2	3.66	2.78–4.81	4.84	3.69–6.35
1	2.54	2.06–3.14	2.69	2.07–3.50
>1 y postpregnancy				
≥3	3.17	2.71–3.71	3.62	3.24–4.06
2	2.19	1.99–2.42	2.31	2.10–2.54
1	1.65	1.54–1.76	1.61	1.47–1.76
>1 y prepregnancy				
≥3	3.33	2.75–4.03	4.68	3.87–5.66
2	2.08	1.84–2.36	2.66	2.24–3.15
1	1.51	1.39–1.65	1.72	1.47–2.02
≤1 y postpregnancy				
≥3	5.27	3.60–7.72	6.67	4.74–9.39
2	3.71	2.84–4.85	3.37	2.44–4.65
1	2.02	1.61–2.54	1.72	1.24–2.38
≤1 y prepregnancy				
≥3	4.20	3.13–5.63	4.91	3.68–6.56
2	2.25	1.81–2.78	2.66	2.03–3.47
1	1.61	1.37–1.90	1.63	1.26–2.11

—, not applicable.
[a] Adjusted for infant year of birth, maternal age group, marital status, maternal mental health record, maternal illicit drug record, socioeconomic status, child sex, and parity.
[b] Adjusted for infant year of birth, maternal age group, marital status, maternal mental health record, maternal illicit drug record, socioeconomic status, child sex, parity, and health region (metropolitan or rural).

The use of linked, population-based, administrative data sets providing maternal and child data from the child's birth through their life span is a major strength of this study and enables examination of a range of biological and social outcomes, overcoming many of the issues around longitudinal follow-up of high-risk populations.

An alcohol-related diagnosis was used as a proxy for heavy maternal alcohol use. The Western Australia data-linkage system is a valid means of identifying patients who had been admitted to hospitals for health-related conditions; however, to ensure that these are given the correct ICD-9 or ICD-10 classification by the clinical coders, it is reliant on medical officers accurately recording the alcohol diagnosis in the medical records.[21] A limitation of this methodology is that it does not allow for direct estimation of the quantity, frequency, and timing of the mother's drinking.

Although we are confident that mothers in the exposed cohort were consuming significant amounts of alcohol, we have previously identified that some women in the comparison cohort had a child diagnosed with FASD and were therefore drinking but did not have an alcohol diagnosis recorded.[19] The outcomes in this study are based on administrative data sets and can be biased in reflecting the use of services. However, these limitations are likely to bias the results toward the null.

Information on attendance and achievement at school was available only for students attending public schools, which represents approximately two-thirds of Western Australia school students and 84% of Aboriginal students. Therefore, the results for educational outcomes may

TABLE 9 Attributable Risk and PAF of Any Negative Outcome Associated With Any Alcohol Diagnosis and Indigenous Status

	Non-Indigenous		Indigenous	
	Estimate	95% CI	Estimate	95% CI
Attributable risk	0.415	0.406–0.424	0.191	0.184–0.198
PAF, %	1.2	1.1–1.3	4.9	4.5–5.2

not be generalizable to students attending private schools.

CONCLUSIONS

The majority of children of mothers with an alcohol-related diagnosis are at risk of poor outcomes, which are due to both direct biological and indirect social and environmental effects of maternal alcohol misuse. Prevention, early identification, and treatment of alcohol-related problems in women of childbearing age or who are pregnant and follow-up of their children is required to prevent fetal and child harm. This is a public health priority requiring a coordinated, interagency approach to improve the outcomes for children of mothers with an alcohol-use disorder across their life course.

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ABBREVIATIONS

CI: confidence interval
FASD: fetal alcohol spectrum disorder
ICD-9: *International Classification of Diseases, Ninth Revision*
ICD-10: *International Classification of Diseases, 10th Revision*
MNS: Midwives Notification System
OR: odds ratio
PAF: population attributable fraction

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