Maternal Alcohol Use and Sudden Infant Death Syndrome and Infant Mortality Excluding SIDS

WHAT’S KNOWN ON THIS SUBJECT: Reductions in infant mortality in the 20th century have not continued. Racial and socioeconomic inequalities in both infant mortality and sudden infant death syndrome (SIDS) persist. Rates of infant mortality in English-speaking countries are higher than the Organisation for Economic Co-operation and Development average.

WHAT THIS STUDY ADDS: At least 16.4% of SIDS and 3.4% of infant deaths not classified as SIDS are attributable to maternal alcohol use. Maternal alcohol-use disorder increases the risk of infant mortality through direct effects on the fetus and indirectly through environmental risk factors.
For many Organisation for Economic Co-operation and Development (OECD) countries, the reduction in infant mortality that occurred during the 20th century has not continued and racial and socioeconomic inequalities in infant mortality and sudden infant death syndrome (SIDS) persist. In 2007, infant mortality in English-speaking OECD countries (Australia, Canada, New Zealand, United Kingdom, and United States) was higher than the OECD average of 3.9/1000 births. Of these countries, Australia ranked 22nd of the 33 OECD countries, with an infant mortality rate of 4.2/1000 live births, followed by New Zealand and the United Kingdom, which are ranked equal at 23rd (4.8/1000 live births).

Infant mortality is considered to be an indicator of maternal health, socioeconomic conditions, and the health of a nation, so in light of the recent trends, identification of modifiable risk factors is a priority. Research has identified a number of modifiable risk factors for infant death and SIDS, including infant sleeping position, bed sharing, and maternal smoking. Rates of infant mortality have reduced after interventions addressing sleeping position and decreases in the prevalence of maternal smoking, although research indicates the latter remains a significant contributor to infant deaths. Although some research studies focusing on maternal smoking as a risk factor for infant mortality and SIDS have adjusted for maternal alcohol use during pregnancy in their analysis, many studies have overlooked the high correlation between maternal smoking and heavy postnatal alcohol consumption and have not adjusted for alcohol.

There is a high prevalence of heavy alcohol consumption by women of childbearing age and alcohol abuse has been reported to occur more frequently in mothers who have had either a stillbirth, neonatal, or infant death. The limited research is suggestive of an association between maternal alcohol use and SIDS and infant mortality. However, we are unaware of any studies examining infant deaths not classified as SIDS.

This present study examines the association between a maternal alcohol-related diagnosis recorded on health datasets, which was used as a proxy for alcohol-use disorder indicating heavy alcohol consumption, and SIDS and infant mortality not classified as SIDS (1983–2005) using linked, population-based data from the Western Australian data linkage system.

METHODS

Ethics approval for the conduct of this study was granted by the Princess Margaret Hospital Research Ethics Committee (No. 1244/EP), the Health Research Ethics Committee, Western Australia Department of Health (No. 2011/34), the Western Australia Aboriginal Health Information Ethics Committee (No. 134-04/06), and the Curtin Human Research Ethics Committee (No. 39/2010). Western Australian ethics committees do not allow researchers using linked data to report numbers where the number of cases within a stratum is <5, to prevent the likelihood of identification of individuals.

The population at-risk consists of women with a birth recorded on the Western Australian Midwives Notification System, 1983–2007, and all their offspring whose births are recorded on the Midwives Notification System. The Midwives Notification System is a statutory notification system of births with a birth weight of ≥400 g and a gestational age of ≥20 weeks that is linked with the Hospital Morbidity Dataset and the Registry of Births, Deaths, and Marriages to ensure complete ascertainment.

Exposed Cohort

An alcohol-related diagnosis was used as a proxy for alcohol-use disorder, indicating heavy maternal alcohol consumption. All mothers with an alcohol diagnosis (International Classification of Diseases Revisions 9/10 codes [ICD-9 and/or ICD-10] recorded from 10 years of age and older were identified through routinely collected administrative datasets (1983–2007) for hospital inpatient admissions (Hospital Morbidity Dataset), mental health outpatients, or alcohol nominated as a drug problem on the Perth-based, government drug and alcohol services dataset. The ICD-9 and/or ICD-10 codes included alcohol-related mental and behavioral disorders, an alcohol-related disease with a 100% attributable fraction, and other alcohol codes (Table 1). Trained coders classify the alcohol diagnosis according to what the physician has recorded in the medical notes so there will be some level of interpretation required. These women and their offspring born between 1983 and 2005 are referred to as the exposed cohort.

Comparison Cohort

The comparison cohort included a random selection of mothers with a birth recorded on the Midwives Notification System who did not have an alcohol-related diagnosis identified on the hospital morbidity dataset, mental health outpatients, or drug and alcohol services datasets described earlier. Selection of the comparison cohort also excluded women with an ICD-8 diagnostic code (alcoholic psychosis 291.0–291.3, 291.9; alcoholism 303.0–303.2, 303.9; or “accidental poisoning” E860) listed in the hospital morbidity dataset from 1970 through 1982 and the mental health outpatients dataset from 1966 through 1982. The exposed cohort was frequency-matched on maternal age within...
maternal race (non-Aboriginal, ≈3:1 unexposed; exposed; Aboriginal, ≈2:1) and year of birth of her child to mothers in the Midwives Notification System without an alcohol-related diagnosis. These women and all their children whose births are recorded on the Midwives Notification System form the comparison cohort.

Data from the administrative health datasets were linked by the Western Australian Data Linkage Branch, using probabilistic matching. These data were then linked with mortality data from the Western Australian Registry of Births, Deaths, and Marriages (1983–2007) and the Western Australian Register of Developmental Anomalies (1983–2007). After linkage, deidentified data files are provided to the researchers by using a unique identifier.

Infants born alive and with their death classified on the death registration as SIDS (7980, R95, R98, or text code of SIDS) were classified as SIDS. In Western Australia, there has been a shift away from classification of sudden unexpected deaths in infants <1 year of age as SIDS toward classifying them as “unascertainable,” and as such, we have classified codes of ‘unascertainable’ as SIDS. The classification of SIDS in Western Australia has varied across the years, with the 1989 National Institute of Child Health and Human Development Beckwith definition used until 2004. The Australian definition was changed in 2004 to the definition developed by Krous et al. for use in administrative and vital statistics. This general definition of SIDS is “the sudden unexpected death of an infant <1 year of age, with onset of the fatal episode apparently occurring during sleep, that remains unexplained after a thorough investigation, including performance of a complete autopsy and review of the circumstances of death and the clinical history.” Deaths coded as SIDS from 2004 on include SIDS categories 1 and II.

In Western Australia, all sudden and unexplained infant deaths are reported to the state or regional coroner and by law must undergo a postmortem examination and a death scene investigation before finalization of the death certificate. The majority of these are completed within 2 years. The youngest children in the cohort were born in 2005 and we had death data to the end of 2007, so it likely that very few SIDS deaths would not have the final postmortem examination and findings determined. The exception to this occurs in a small number of cases where the parents refuse the postmortem examination, primarily due to cultural reasons. Permission to forego an autopsy is determined by the coroner. However, the evidence indicates that all cases of SIDS in Western Australia undergo a postmortem examination.

The coding of the timing of the maternal alcohol-related diagnoses has been previously described. The pregnancy period was estimated by subtracting gestational age at birth from the date of birth to give the date of conception. Mothers with any alcohol diagnosis recorded during pregnancy, which may also include an alcohol-related diagnosis before and/or after pregnancy, were classified as (i) “during pregnancy” (Supplemental Table 6). For women without a diagnosis during pregnancy, the maternal alcohol-related diagnosis was coded into the timing of recording of the alcohol diagnosis in relation to pregnancy using a hierarchical coding so that diagnoses recorded within 1 year of pregnancy

### Table 1: ICD-9 and ICD-10 Alcohol Diagnosis Codes for Identifying Maternal Alcohol-Use Disorder

<table>
<thead>
<tr>
<th>Alcohol Diagnoses</th>
<th>ICD-10 Codes</th>
<th>ICD-9 Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental and behavioral disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute alcohol intoxication</td>
<td>F10.0, F10.1</td>
<td>303.0–303.03, 305.0–305.03</td>
</tr>
<tr>
<td>Alcohol dependence syndrome</td>
<td>F10.2</td>
<td>303.9–303.85</td>
</tr>
<tr>
<td>Alcohol withdrawal</td>
<td>F10.3, F10.4</td>
<td>291.0, 291.81</td>
</tr>
<tr>
<td>Alcohol psychotic disorder</td>
<td>F10.5</td>
<td>291.3, 291.5</td>
</tr>
<tr>
<td>Alcohol amnesic syndrome</td>
<td>F10.6</td>
<td>291.1</td>
</tr>
<tr>
<td>Residual and late onset alcohol psychiatric disorder</td>
<td>F10.7</td>
<td>291.2, 291.4</td>
</tr>
<tr>
<td>Other</td>
<td>F10.8, F10.9</td>
<td>291.82, 291.89, 291.9</td>
</tr>
<tr>
<td>Alcohol-related diseases</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcoholic nervous system degeneration</td>
<td>G31.2</td>
<td>331.7</td>
</tr>
<tr>
<td>Alcohol polyneuropathy</td>
<td>G62.1</td>
<td>357.5</td>
</tr>
<tr>
<td>Alcoholic myopathy</td>
<td>G72.1</td>
<td>359.4</td>
</tr>
<tr>
<td>Alcoholic cardiomyopathy</td>
<td>I42.6</td>
<td>425.5</td>
</tr>
<tr>
<td>Alcoholic gastritis</td>
<td>K29.2</td>
<td>535.30, 535.31</td>
</tr>
<tr>
<td>Alcoholic liver disease</td>
<td>K70–K70.9</td>
<td>571.0–571.3</td>
</tr>
<tr>
<td>Alcoholic pancreatitis</td>
<td>K88.0</td>
<td>577.1</td>
</tr>
<tr>
<td>“Other” alcohol disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infant damage due to alcohol</td>
<td>O35.4, P04.3, Q86.0</td>
<td>655.43, 760.71</td>
</tr>
<tr>
<td>Other</td>
<td>R78.0, T51.0, T51.8, T51.9, Y90.0–Y90.9</td>
<td>790.3, 980.0, 980.8, 980.9, E860.00</td>
</tr>
<tr>
<td>Poisoning</td>
<td>X45, X65, Y15</td>
<td>E86.09, E950.09, E98.05</td>
</tr>
<tr>
<td>Rehabilitation/history of alcohol use disorder</td>
<td>Z50.2, Z71.4, Z86.41</td>
<td>V57.89, V65.42, V11.3</td>
</tr>
<tr>
<td>Encountering health services due to alcohol problems</td>
<td>Z72.1</td>
<td>—</td>
</tr>
</tbody>
</table>

### Notes

- a Some women may have more than one diagnosis.
- b Recorded on maternal record.
were coded as (ii) \( \leq 1 \) year before pregnancy and may include exposure \( > 1 \) year before pregnancy or any exposure after pregnancy or (iii) \( \leq 1 \) year after pregnancy and may include exposure \( > 1 \) year before or after pregnancy.

Mothers with an alcohol diagnosis recorded \( > 1 \) year before or after pregnancy were grouped as (iv) \( > 1 \) year before pregnancy and may include exposure \( > 1 \) year after pregnancy or (v) \( > 1 \) year after pregnancy.

Examination of the association between a maternal alcohol-related diagnosis and SIDS was restricted to the years 1983–2005 to take into account the delay in receiving data from the coroner’s report. Deaths coded as “unascertainable” on the death registration were individually scrutinized, and only cases without an underlying possible contributing factor such as tetrology of Fallot were coded as SIDS. Infant deaths were defined as all live born infants who died within 1 year of birth between 1983 through 2005 and who did not have SIDS or “unascertainable” as the cause of death on their death record. The proportion of infant deaths and SIDS deaths were calculated per 1000 live births, stratified by alcohol exposure groups.

Hazard ratios for the risk of infant and SIDS deaths were calculated for the offspring of mothers with an alcohol-related diagnosis compared with offspring of mothers in the comparison cohort. Analyses were undertaken by using Cox regression. The proportion hazards assumption was tested and there were no significant interactions between the alcohol variables and the log of the child’s age. Results are presented as adjusted hazard ratios (aHRs), with 95% confidence intervals (CIs). We used SPSS Version 19 for the Cox regressions.

All analyses were adjusted for the factors used in frequency matching (maternal age, ethnicity, and year of birth). The results from 3 models are presented. The base model included the alcohol exposure variable plus the frequency matching variables. The second model also included maternal characteristics: maternal illicit drug use (any ICD-9-10 code for illicit drugs present on either the hospital morbidity dataset or mental health outpatients datasets, or illicit drug use recorded on the drug and alcohol services dataset), any mental health diagnoses (ICD-9-10 codes on the hospital morbidity dataset and mental health outpatients datasets) and marital status (married, never married, separated/divorced/widowed). The third model extended the second model by adjusting for maternal smoking during pregnancy. Maternal smoking during pregnancy has been collected on the Midwives Notification System since 1998 so all analyses were rerun with the binary alcohol variable for the birth years 1998–2005 with adjustment for smoking. The population-attributable fraction and 95% CIs for SIDS and infant mortality excluding SIDS in children of mothers with an alcohol diagnosis were calculated by using the method published by Natarajan et al for significant results, using whole population numbers for SIDS and infant mortality and the comparison numerators and denominators that were received from the Midwives Notification System.

**RESULTS**

There were 77,895 live births registered in the study, with 21,841 births to mothers with an alcohol diagnosis and 56,054 to comparison mothers (Table 2). Compared with comparison mothers, a higher percentage of exposed mothers were separated or never married (32.8% vs 20.9%), had an illicit drug diagnosis (31.6% vs 2.5%) or a mental health diagnosis (43.4% vs 10.0%), and smoked during pregnancy (56.7% vs 28.9%) (Table 2).

There were 303 cases of SIDS: 171 in exposed and 132 in comparison children (Table 3). The net excess proportion of SIDS for exposed children of mothers with any alcohol diagnosis was 5.5/1000 (95% CI 4.3–6.8). The highest net excess proportion of SIDS occurred when a maternal alcohol diagnosis was recorded within 1 year postpregnancy (16.5/1000; 95% CI 9.3–28.1) followed by during pregnancy (13.1/1000; 95% CI 7.3–22.2).

There were 598 infant deaths not classified as SIDS with a net excess proportion of 5.0/1000 (95% CI 3.5–6.6) for any maternal alcohol diagnosis and with the highest net excess proportion observed for children of mothers with an alcohol diagnosis recorded during pregnancy 10.2/1000 (95% CI 4.1–19.7) and within 1 year after pregnancy 9.2/1000 (95% CI 2.7–20.2) (Table 3). The net excess proportion of infant deaths not classified as SIDS was ~5.5/1000 when an alcohol diagnosis was recorded within a year before pregnancy or \( \geq 1 \) year after pregnancy but not recorded during pregnancy (Table 3).

The results for the Cox regression analyses for SIDS showed that exposed children had a 3-fold increased risk of SIDS for any maternal alcohol diagnosis (aHR 3.15; 95% CI 2.46–4.03) and an increased risk for almost every timing category (Table 4). The highest risk was when a maternal alcohol-related diagnosis was recorded within 1 year postpregnancy but not during pregnancy (aHR 8.61, 95% CI 5.04–14.69).
and there was a similar risk when a diagnosis was recorded during pregnancy (aHR 6.92, 95% CI 4.02–11.90). Adjusting for maternal smoking in the 1998–2005 model reduced the aHR for the during pregnancy exposure group to 3.85 (95% CI 1.53–9.69) but there was little change in the aHRs for the other exposure variables.

The highest population-attributable fraction for SIDS was for any maternal alcohol diagnosis (16.41%, 95% CI 9.73%–23.69%), followed by a maternal alcohol diagnosis only occurring during pregnancy (aHR 16.41%, 95% CI 9.73%–23.69%), when a diagnosis was recorded during pregnancy but was recorded within 1 year before or after pregnancy increased (Table 5). The highest risk occurred when a diagnosis was recorded during pregnancy and when a diagnosis was not recorded during pregnancy but was recorded within 1 year postpregnancy (aHR 16.41%, 95% CI 9.73%–23.69%) and when a diagnosis was not recorded during pregnancy but was recorded within 1 year prepregnancy (aHR 1.90, 95% CI 1.12–3.21). After adjustment for maternal smoking in the 1998–2005 model, the aHR for the during-pregnancy exposure group did not alter. The risk when the maternal alcohol diagnosis was recorded within 1 year before or after pregnancy was attenuated, whereas the risk when a maternal alcohol diagnosis was recorded only >1 year after pregnancy increased (Table 5).

The highest population-attributable fraction for infant mortality not classified as SIDS was for any maternal alcohol diagnosis (3.40%, 95% CI 2.28%–4.67%), followed by a maternal alcohol diagnosis only occurring >1 year postpregnancy (2.26%, 95% CI 1.42%–3.25%) (Table 5). The population attributable fraction when a maternal alcohol diagnosis was recorded during pregnancy was 2.06% (95% CI 0.48%–3.73%). The results were similar after adjustment for maternal smoking in the model restricted to 1998–2005, with the exception of any alcohol diagnosis that yielded a higher attributable fraction (25.04%, 95% CI 5.54%–48.65%) (Table 4).

The risk of infant death not classified as SIDS was increased for any alcohol exposure (aHR 1.62, 95% CI 1.36–1.93) and for each of the timing categories (Table 5). The highest risk occurred when a diagnosis was recorded during pregnancy (aHR 2.35, 95% CI 1.45–3.83) and when a diagnosis was not recorded during pregnancy but was recorded within 1 year postpregnancy (aHR 2.24, 95% CI 1.28–3.92) or within 1 year prepregnancy (aHR 1.90, 95% CI 1.12–3.21).

DISCUSSION

Maternal alcohol-use disorder increased the risk of both SIDS and infant deaths not classified as SIDS. The
presence of a maternal alcohol di-
agnosis during pregnancy, indicating heavy prenatal alcohol exposure, in-
creased the risk of SIDS by sevenfold. It has been hypothesized that some cases of SIDS may be due to abnor-
malities in the control of homeostatic functions by the developing brainstem, and experimental and human studies suggest that prenatal alcohol exposure can adversely affect brainstem development.57–59 Adjustment for maternal smoking during pregnancy in the analysis restricted to 1998–2005 at-
tenuated the results, but a fourfold risk remained. These results extend the findings of Iyasu and colleagues23 by demonstrating that the increased risk of SIDS is not restricted to Native Americans but occurs across racial groups.

The results also indicate that maternal alcohol-use disorder is acting as an environmental risk factor with an eightfold increased risk for the children of mothers who had an alcohol di-
agnosis within 1 year after pregnancy but not during pregnancy and in-
creased risk for each of the other exposure groups. These findings support other published research reporting heavy maternal alcohol use as an in-
direct or environmental risk factor for SIDS mortality in the offspring.7,8,24 We were unable to examine if the risk of SIDS occurs through bed sharing or other potentially modifiable adverse conditions, which may have contributed to the high odds ratios for children of mothers with an alcohol diagnosis recorded within 1 year postpregnancy but not during pregnancy.

Examination of infant deaths not clas-
sified as SIDS showed a similar pattern to that observed for SIDS. When a ma-
ternal alcohol diagnosis was recorded during pregnancy the risk of infant deaths not classified as SIDS more than doubled and was maintained after ad-
justment for maternal smoking in the analysis restricted to 1998–2005. The results for infant deaths not classified as SIDS add weight to the possibility that heavy prenatal alcohol exposure

### TABLE 4 Hazard Ratios for SIDS, by Alcohol Exposure

<table>
<thead>
<tr>
<th>Infant Deaths</th>
<th>Hazard Ratios</th>
<th>aHRs</th>
<th>Population-Attributable Fraction</th>
<th>aHRs</th>
<th>Population-Attributable Fraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>No alcohol diagnosis</td>
<td>1.00</td>
<td>1.00</td>
<td>Referent</td>
<td>1.00</td>
<td>Referent</td>
</tr>
<tr>
<td>Any alcohol diagnosis</td>
<td>3.13 (2.48–3.93)</td>
<td>3.15 (2.46–4.03)</td>
<td>16.41 (9.73–25.89)</td>
<td>3.41 (2.02–5.76)</td>
<td>25.04 (5.54–48.65)</td>
</tr>
<tr>
<td>Timing of exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No alcohol diagnosis</td>
<td>1.00</td>
<td>1.00</td>
<td>Referent</td>
<td>1.00</td>
<td>Referent</td>
</tr>
<tr>
<td>During pregnancy</td>
<td>7.21 (4.28–12.14)</td>
<td>6.92 (4.02–11.90)</td>
<td>2.06 (0.48–3.73)</td>
<td>3.85 (1.53–9.68)</td>
<td>3.55 (0.02–9.97)</td>
</tr>
<tr>
<td>≤ 1 y prepregnancy</td>
<td>3.04 (1.48–6.24)</td>
<td>3.10 (1.50–6.41)</td>
<td>0.77 (0.03–1.81)</td>
<td>2.81 (0.96–8.27)</td>
<td>4.87 (0.00–10.84)</td>
</tr>
<tr>
<td>≤ 1 y postpregnancy</td>
<td>8.44 (4.99–14.27)</td>
<td>8.61 (5.04–14.83)</td>
<td>2.00 (0.50–3.53)</td>
<td>8.60 (3.60–20.55)</td>
<td>3.34 (1.71–6.54)</td>
</tr>
<tr>
<td>&gt; 1 y prepregnancy</td>
<td>2.90 (1.78–4.73)</td>
<td>2.95 (1.80–4.83)</td>
<td>1.87 (0.46–3.61)</td>
<td>2.66 (1.30–5.45)</td>
<td>5.90 (0.28–14.30)</td>
</tr>
<tr>
<td>&gt; 1 y postpregnancy</td>
<td>2.68 (2.07–3.48)</td>
<td>2.73 (2.07–3.58)</td>
<td>9.17 (5.27–14.84)</td>
<td>3.34 (1.71–6.54)</td>
<td>8.27 (0.68–18.38)</td>
</tr>
</tbody>
</table>

a Adjusted for frequency matching variables maternal age, ethnicity, and year of birth.
b Adjusted for frequency matching variables and marital status, illicit drug diagnosis, and any mental health diagnosis.
c Analyses restricted to birth years 1998–2005 and adjusted for previous variables plus smoking during pregnancy.
d Population-attributable fractions not calculated for non-significant results.

### TABLE 5 Hazard Ratios for Infant Mortality Not Classified as SIDS, by Alcohol Exposure

<table>
<thead>
<tr>
<th>Infant Deaths</th>
<th>Hazard Ratios</th>
<th>aHRs</th>
<th>Population-Attributable Fraction</th>
<th>aHRs</th>
<th>Population-Attributable Fraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>No alcohol diagnosis</td>
<td>1.00</td>
<td>1.00</td>
<td>Referent</td>
<td>1.00</td>
<td>Referent</td>
</tr>
<tr>
<td>Any alcohol diagnosis</td>
<td>1.64 (1.39–1.93)</td>
<td>1.62 (1.36–1.93)</td>
<td>3.40 (2.28–4.67)</td>
<td>1.70 (1.21–2.38)</td>
<td>4.29 (1.78–7.52)</td>
</tr>
<tr>
<td>Timing of exposure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No alcohol diagnosis</td>
<td>1.00</td>
<td>1.00</td>
<td>Referent</td>
<td>1.00</td>
<td>Referent</td>
</tr>
<tr>
<td>During pregnancy</td>
<td>2.40 (1.49–3.89)</td>
<td>2.35 (1.45–3.83)</td>
<td>0.37 (0.09–0.74)</td>
<td>2.33 (1.24–4.38)</td>
<td>0.96 (0.10–2.23)</td>
</tr>
<tr>
<td>≤ 1 y prepregnancy</td>
<td>1.92 (1.14–3.23)</td>
<td>1.90 (1.12–3.21)</td>
<td>0.26 (0.05–0.56)</td>
<td>1.12 (0.45–2.79)</td>
<td>—e</td>
</tr>
<tr>
<td>≤ 1 y postpregnancy</td>
<td>2.26 (1.31–3.98)</td>
<td>2.24 (1.28–3.92)</td>
<td>0.26 (0.04–0.56)</td>
<td>1.62 (0.65–4.05)</td>
<td>—e</td>
</tr>
<tr>
<td>&gt; 1 y prepregnancy</td>
<td>1.33 (0.91–1.98)</td>
<td>1.31 (0.88–1.94)</td>
<td>—e</td>
<td>1.19 (0.70–2.01)</td>
<td>—e</td>
</tr>
<tr>
<td>&gt; 1 y postpregnancy</td>
<td>1.58 (1.32–1.92)</td>
<td>1.58 (1.30–1.93)</td>
<td>2.26 (1.42–3.25)</td>
<td>2.19 (1.43–3.34)</td>
<td>2.60 (0.83–4.87)</td>
</tr>
</tbody>
</table>

a Born alive and died within 1 y.
b Adjusted for frequency matching variables maternal age, ethnicity, and year of birth.
c Adjusted for frequency matching variables and marital status, illicit drug diagnosis, and any mental health diagnosis.
d Analyses restricted to birth years 1998–2005 and adjusted for previous variables plus smoking during pregnancy.
e Population-attributable fractions not calculated for non-significant results.
directly affects the development of the fetus, increasing the risk of death. However, the causes of infant deaths not classified as SIDS indicate that the risk from maternal alcohol-use disorder is through multiple pathways. There were a wide range of causes of death listed and many of the infant deaths had more than one cause. Almost 40% of deaths had at least one code indicating biological/direct effects of alcohol on the fetus such as prematurity, intrauterine hypoxia, growth retardation, and congenital heart disease, whereas 60% had a code indicating indirect/environmental factors such as smoke inhalation, dehydration, viral infections, neglect, and mechanical inhalation. Additional investigation of the causes of infant death is warranted. A previous study demonstrated that heavy prenatal alcohol exposure at levels of 4+ drinks/wk and binge drinking increased the risk of neonatal and postneonatal death in term infants. However, that study did not exclude SIDS from their examination of infant mortality so their results may reflect an increased risk of SIDS. To our knowledge, this is the first study to examine the relationship between maternal alcohol-use disorder during pregnancy and infant deaths not classified as SIDS.

We are confident that heavy prenatal alcohol exposure occurred when an alcohol diagnosis was recorded during pregnancy and that the women would have had overt alcohol-related problems to have the diagnosis recorded. However, underrecognition of alcohol use and alcohol-use disorders during pregnancy and the hospital setting is well documented. Health professionals in Western Australia do not routinely ask women, including pregnant women and mental health patients, about their alcohol consumption, and maternal alcohol problems may not be evident at every contact with the health services. Many mothers with an alcohol diagnosis recorded >1 year before or after pregnancy in this study may have been drinking heavily during pregnancy and/or within 1 year after pregnancy but not had this recognized or recorded in their medical records. This is likely to account for the increased risk of SIDS and infant mortality not classified as SIDS, in their children. The underrecognition of alcohol-use disorders and the high prevalence of heavy alcohol consumption by Australian women makes it likely that some of the comparison mothers were drinking heavily during pregnancy. This misclassification would have resulted in an underestimation of the risk of SIDS and infant mortality not classified as SIDS from maternal alcohol-use disorder, including the risk from prenatal alcohol exposure. This is evident by the higher population-attributable fraction obtained for any alcohol diagnosis for both types of mortality than when diagnoses were recorded during pregnancy.

The population-attributable fractions from this study indicate that between 16.4% and 25.0% of SIDS deaths and ~3% to ~4% of infant mortality not classified as SIDS are associated with maternal alcohol-use disorder and could potentially be prevented. Previous research has demonstrated that children with fetal alcohol syndrome their siblings, and their mothers also have increased risk of mortality. However, of the 115 children diagnosed with fetal alcohol syndrome in this study, fewer than 5 (<4%) of these children were classified as an infant death not classified as SIDS so we were unable to investigate this further. Maternal alcohol-use disorder should be recognized as a risk marker for familial mortality and recognized as an important issue by pediatricians and other health professionals working with young children and pregnant women.

It is also possible that information of deaths occurring outside Western Australia may not have been reported to the Western Australian Registry of Births, Deaths, and Marriages. However, migration out of Western Australia is relatively low at ~2% annually and there is no indication this would be related to alcohol use.

The strengths of this study are that it is a large, population-based cohort of high-risk mothers who have an alcohol diagnosis and all their births recorded on the Midwives Notification System, a statutory notification system. Accessing multiple data sources in the Western Australian data linkage system to identify mothers with an alcohol-related diagnosis is a valid means of identifying cases admitted to hospital for a health-related condition. This methodology overcomes loss to follow up and recall bias, enabling longitudinal follow-up of both the mothers and their offspring. Because it is a population-based sample, the results are generalizable to other similar populations.

The rate of infant mortality and SIDS has not improved since 2001 in many developed nations. These results provide evidence that maternal alcohol-use disorder is, potentially, a modifiable risk factor for SIDS and infant mortality not classified as SIDS and should be treated as a marker for risk of mortality in the offspring. Early identification of maternal alcohol-use disorders combined with brief interventions addressing alcohol use and contraception and postnatal support and follow-up have the potential to reduce infant mortality and SIDS.

**CONCLUSIONS**

The results of this study indicate that maternal alcohol-use disorder increases the risk of SIDS and infant mortality not classified as SIDS.
through direct effects on the fetus and indirectly through environmental risk factors. Additional reductions in SIDS and infant mortality not classified as SIDS could be achieved through prevention of maternal alcohol-use disorders.

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