Addiction

RESEARCH REPORT

doi:10.1111/j.1360-0443.2010.03199.x

Alcohol as a risk factor for sudden infant death syndrome (SIDS)

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ABSTRACT

Aim To test whether alcohol is a risk factor for sudden infant death syndrome (SIDS). **Design and setting** US epidemiological study using computerized death certificates, linked birth and infant death dataset, and Fatality Analysis Reporting System. **Participants** All SIDS cases ($n = 129\,090$) and other infant deaths ($n = 295\,151$) from 1973–2006; all persons involved in late-night alcohol-related crashes ($n = 135\,946$) from 1994–2008. **Measurements** Three measures were used: the expected number of deaths on New Year versus the observed number (expected values were determined using a locally weighted scatterplot smoothing polynomial), the average number of weekend deaths versus the average number of weekday deaths, and the SIDS death rate for children of alcohol-consuming versus non-alcohol-consuming mothers. **Findings** These measures indicate that the largest spikes in alcohol consumption and in SIDS (33%) occur on New Year, alcohol consumption and SIDS increase significantly on weekends, and children of alcohol-consuming mothers are much more likely to die from SIDS than are children of non-alcohol-consuming mothers. **Conclusions** Alcohol consumption appears to be a risk factor for sudden infant death syndrome, although it is unclear whether alcohol is an independent risk factor, a risk factor only in conjunction with other known risk factors (like co-sleeping), or a proxy for other risk factors associated with occasions when alcohol consumption increases (like smoking). Our findings suggest that caretakers and authorities should be informed that alcohol impairs parental capacity and might be a risk factor for sudden infant death syndrome; in addition, future research should further explore possible connections between sudden infant death syndrome and alcohol.

Keywords Alcohol, holidays, New Year, pediatric accidents, SIDS, weekends.

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Submitted 26 April 2010; initial review completed 7 July 2010; final version accepted 8 September 2010

INTRODUCTION

In the United States, sudden infant death syndrome (SIDS) is the leading cause of postneonatal infant deaths [1]. Much evidence suggests that caretaker decisions about the infant's sleeping environment can increase the risk of SIDS significantly, e.g. decisions about the infant's sleeping position [2–4]. This evidence has prompted vigorous efforts to promote safe sleeping environments [2–4], e.g. the 'Back to Sleep' campaign [5].

Despite these efforts, many SIDS cases still involve unsafe sleeping environments (e.g. more than 30% of US SIDS cases involve prone infants) [6]. Residual caretaker ignorance may explain some of these unsafe practices, but there may be an additional, rarely considered, risk factor: alcohol-impaired caretakers.

In general, alcohol is known to impair judgement [7,8] and worsen pediatric care [9,10]. However, within this broad literature, studies focusing upon SIDS provide mixed evidence on the effects of alcohol consumption [11–19]. Some studies concluded that alcohol is not a risk factor [11,12]. Other studies concluded that alcohol is a risk factor only when combined with factors such as bed sharing (i.e. co-sleeping) [13–17]. Still other studies concluded that alcohol has an independent effect on the risk of SIDS [18,19]. Perhaps the best evidence linking alcohol and SIDS comes from two non-US studies [18,19]. Both showed that the number of SIDS cases increased within 24 hours of caretaker consumption of alcohol.

All these earlier studies show similar weaknesses: they are based upon small, geographically non-representative

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samples. Taken together, these investigations raise, but do not resolve, an important hypothesis: alcohol-impaired caretakers are less able to protect vulnerable infants. Henceforth, we term this the 'alcohol–SIDS hypothesis', or the AS hypothesis.

Unlike earlier investigations, our study uses three nation-wide, official US data sets to assess the AS hypothesis [20–22]. In what appears to be the first large-scale study of this topic, we examined all SIDS cases ($n = 129\,090$) and other infant deaths ($n = 295\,151$) for three decades (1973–2006) to test whether SIDS increases when caretakers are affected by alcohol.

METHODS

We examined daily mortality from three official, nation-wide, exhaustive US data sets: first, we analyzed all US computerized death certificates [20], which provide day of week of death for 1973–2006, and exact date of death for 1973–2004. Our analyses of this data set generally examined these periods. However, a few analyses examined shorter periods because the necessary information is missing for some years: when analyzing setting of death (e.g. home), we examined 1989 onwards. When analyzing secondary causes of death, we examined 1983 onwards. When comparing autopsied versus non-autopsied decedents, we examined 1973–94; this information is available only sporadically after 1994.

Secondly, we analyzed the linked birth and infant death data set [21], which links information from birth and death certificates for decedents under 1 year. This data set is available online from 1995 to 2005 and records information about the infant and the infant's mother, e.g. whether or not she consumed alcohol.

Using these data sets, we focused upon postneonatal SIDS, i.e. decedents aged 28–364 days. SIDS is coded 795.0 in the 8th Revision of the International Classification of Diseases (ICD-8) [23], 798.0 in ICD-9 [24] and R95 in ICD-10 [25].

Thirdly, we analyzed the Fatality Analysis Reporting System (FARS) [22]. This is available online from 1994–2008 and provides information on all US motor vehicle accidents involving at least one fatality.

We used FARS for two purposes:

1. To discover occasions when alcohol consumption spiked most dramatically during evening and early morning: 8 p.m.–3:59 a.m. We assumed that this is a high-risk period for SIDS because alcohol consumption during these nocturnal hours would be particularly likely to affect the infant's sleeping environment [16,26]. For example, during these nocturnal hours, an alcohol-impaired caretaker might be less likely to place an infant in the safe supine position and/or monitor the infant [16]. Henceforth, we term these nocturnal hours 'the high-risk period'. We found two statistically independent cyclical occasions when alcohol consumption spikes during the high-risk period: New Year (occurring on a yearly cycle) and the weekend (occurring on a weekly cycle). These two cycles are independent, because New Year is equally likely to occur on each day of the week (unlike holidays with movable dates, e.g. Labor Day and Thanksgiving).
2. Most generally, the AS hypothesis states that alcohol-impaired caretakers are less able to protect vulnerable children. We used FARS to test one corollary of this hypothesis: children are less likely to be seat-belted properly in the presence of alcohol-impaired adults. When analyzing FARS, we defined 'children' as people aged under 9 years because: (i) this age group requires special seating arrangements, according to official regulations [27]; we assumed that this vulnerable age group is comparable to the SIDS age group examined in our other data sets. (ii) It is undesirable to restrict FARS analyses to infants under 1 year because this restriction yields a very small sample size ($n = 243$).

To determine whether SIDS spikes at New Year, we followed Phillips *et al.* [28] and fitted a locally weighted scatterplot smoothing polynomial (LOESS) line to daily mortality, from 1 January 1973 to 31 December 2004. Phillips *et al.* determined that a 6-week 'bandwidth' was appropriate for their LOESS analysis; we also used this bandwidth. This standard non-parametric procedure [29–31] makes minimal distributional assumptions and corrects for seasonality and trend. Thus, LOESS corrects for the fact that SIDS increases in winter [32] and has decreased over time [5].

LOESS enabled us to estimate the expected number of deaths at New Year (and indeed on any other day). We defined the New Year effect as:

$$\text{NY} = \frac{\text{observed number of deaths on 1 January}}{\text{expected number of deaths on 1 January}}$$

where the expected number was determined by LOESS. Thus, e.g. NY = 1.30 indicates that there are 30% more deaths on 1 January than would be expected after correction for seasonality and trend.

We calculated NY for each New Year between 1 January 1973 and 30 June 2004, with two exceptions: ICD codes were revised on 1 January 1979 (when ICD-9 replaced ICD-8) and on 1 January 1999 (when ICD-10 replaced ICD-9). To avoid distortions potentially arising from these changes, we did not examine New Year mortality during the ICD transition periods: 1 July 1978–30 June 1979 and 1 July 1998–30 June 1999.

To ensure that our findings were not an artifact of LOESS, we also used a different procedure to test the statistical significance of the New Year effect. We specify this procedure later in the paper.

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In addition to the New Year analysis, we determined whether SIDS increases at weekends. In weekend analyses, we ensured that the study period contained an equal number of each day of the week.

Following official recommendations [33] and our previous practice [28,34–38], we calculated standard errors [33,39] and significance levels, even though we examined complete counts, not samples. As in our previous work [28,34–38], our study design allowed examination of numbers of cases, rather than rates.

RESULTS

Day of year

Figure 1 examines evidence of alcohol consumption in the high-risk period for each day of the year. Nation-wide hourly alcohol consumption is not directly available. However, an indirect, proxy nation-wide measure is available: FARS provides hourly data on alcohol-related motor vehicle crashes [22]. For each day of the year, we used this information to identify the date(s) for which alcohol consumption spikes in the high-risk period. Thus, e.g. for 4 July, we examine the number of people involved in alcohol-related crashes occurring during the 8 hours between 8 p.m. July 3 and 3:59 a.m. July 4.

Figure 1 reveals an extreme spike of 1016 (984–1048) people involved in alcohol-related crashes in the last 4 hours of 31 December and the first 4 hours of 1 January. This spike is far larger than the spike for any other day of the year and is a statistical outlier (Grubbs statistic = 9.32; $P < 0.01$). There is only one other outlier in the year: a significantly smaller spike of 637 (612–662) people in the last 4 hours of 4 July and the first 4 hours of 5 July (Grubbs statistic = 4.42; $P < 0.01$). For Fig. 1 the average is 372.46 people, and thus there is no spike in the 8 hours centered on the beginning of Christmas: 385 (365–404) people. Henceforth, we focus upon New Year because it shows evidence of extreme alcohol consumption during the high-risk period. Given the AS

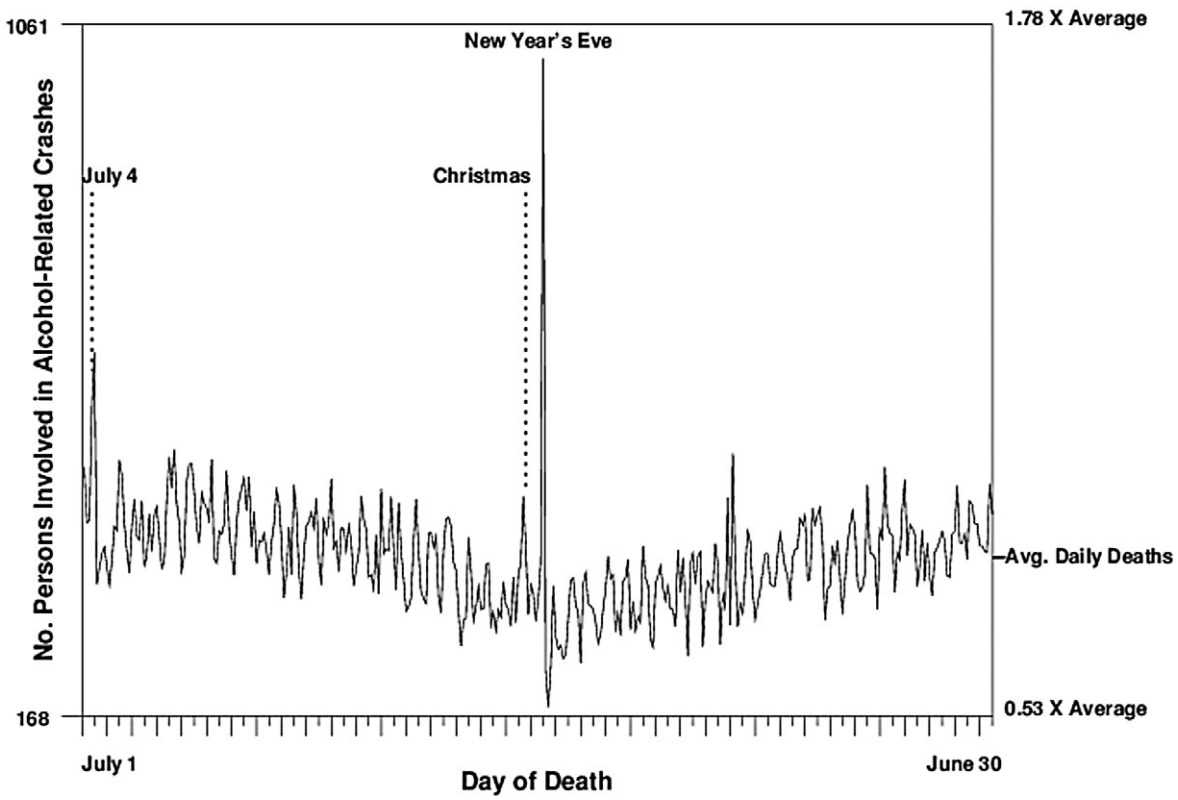

Figure 1 Number of people involved in alcohol-related crashes, by day of the year, United States, 1994–2008 (n = 135 946). We define alcohol-related crashes as crashed vehicles containing at least one person with a blood alcohol content of 0.08% or higher. For reasons specified in Methods, Fig. 1 focuses upon crashes occurring during the 8 hours centered on the beginning of each day. Thus, e.g. for New Year, we examine the number of people involved in alcohol-related crashes occurring between 8 p.m. 31 December and 3:59 a.m. 1 January. In Fig. 1, there is a large number of cases for each day; hence the standard error of this number [sqrt(n)] is relatively small. Thus, the observed number of cases at New Year is many standard errors above the number expected under the null hypothesis that crash frequency is unaffected by New Year. The figure also identifies the other major holidays with fixed dates, but cannot identify holidays precisely with movable dates (such as Thanksgiving). Source of data: the Fatality Analysis Reporting System [22]

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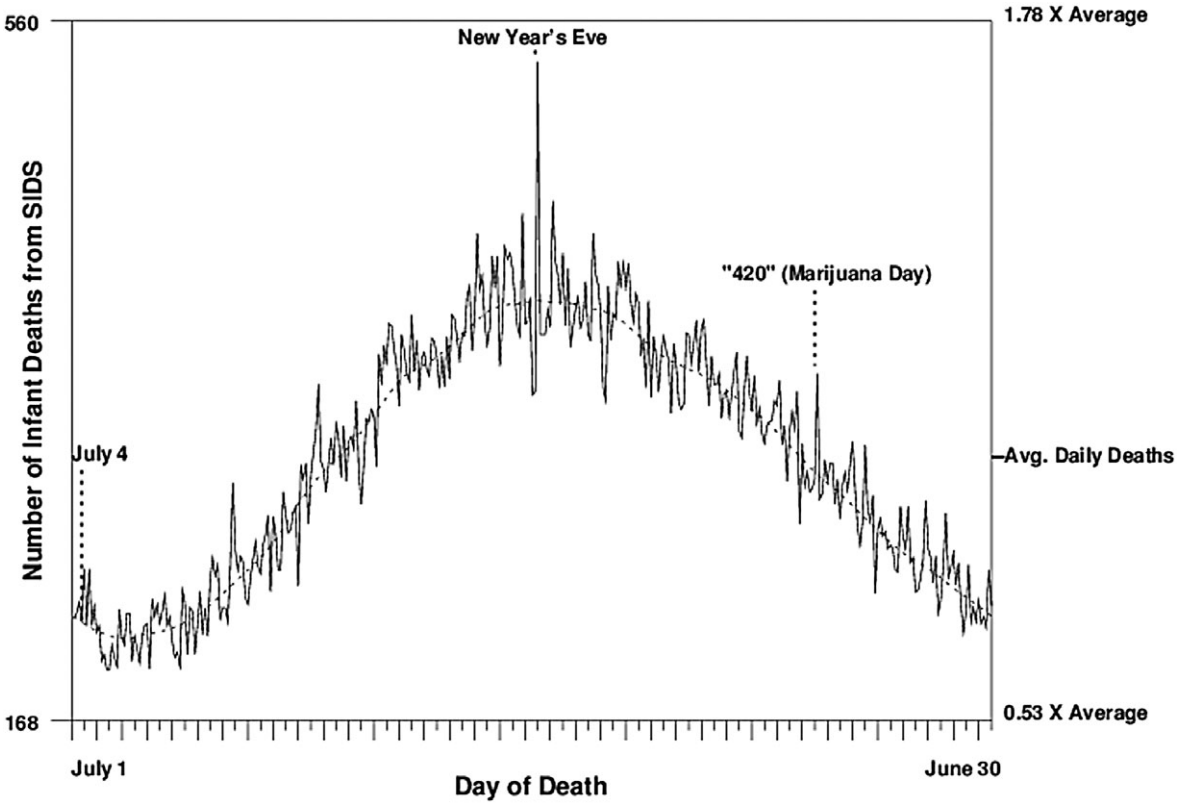

Figure 2 Observed versus expected sudden infant death syndrome (SIDS) deaths by day of year, for postneonatal infants, United States, 1973–2004. The expected number of deaths (indicated by the broken line) was calculated using locally weighted scatterplot smoothing polynomial (LOESS) regression; this line indicates the number of deaths that would be expected if mortality was affected by seasonal fluctuations and by trend, but not by holidays. See Methods for details. Postneonatal infant decedents are those dying 28–364 days after birth. SIDS deaths are those classified as 795.0 (ICD-8) [23]; 798.0 (ICD-9) [24]; and R95 (ICD-10) [25]. Source of data: computerized US death certificates [20]

hypothesis, SIDS should also show an extreme spike at New Year (and possibly a smaller spike on 5 July).

As predicted, Fig. 2 shows a 33% (22–45%) spike in SIDS on New Year above and beyond the normal winter increase in SIDS. This spike is far larger than the spike for any other day of the year, and is a statistical outlier (Grubbs statistic = 5.37; $P < 0.01$). The SIDS spike is evident not only when all 29 years are combined, but also when these years are considered separately. For 26 of these years, the observed number of SIDS cases exceeds the number expected. This number of positive residuals (26) is far larger than the number of positive residuals for any other day of the year and is a statistical outlier (Grubbs statistic = 4.16; $P < 0.01$). Thus, two types of evidence indicate that the New Year spike in SIDS is exceptional: evidence for all years combined and for each year separately.

As predicted, Fig. 2 also shows an increase in SIDS on 5 July: 14% (0–28%). However, this spike is not a statistical outlier in either sense mentioned above.

Figure 2 also shows an 18% (7–31%) spike just after 20 April, a US counterculture holiday devoted to the celebration of cannabis consumption [40]. This spike was not predicted and, like 5 July, is not a statistical outlier in either sense mentioned above. In addition, there is no spike in SIDS on 4 July [0% (–13% to 13%)] or on 25 December [–5% (–14% to 5%)].

Unlike infants dying from SIDS, infants dying from non-SIDS causes do not display a statistically significant New Year spike: 5% (–2% to 12%) (Fig. 3). We return to this finding in the Discussion.

Day of week

Alcohol consumption increases not only at New Year but also at weekends. For each day of the week, Fig. 4 displays the number of people involved in alcohol-related crashes in the high-risk period. For example, for Monday, we examine crashes occurring between 8 p.m. on Sunday and 3:59 a.m. on Monday.

In the high-risk period, the number of peoples involved in alcohol-related crashes is much higher on Saturday and Sunday than on any other day of the week

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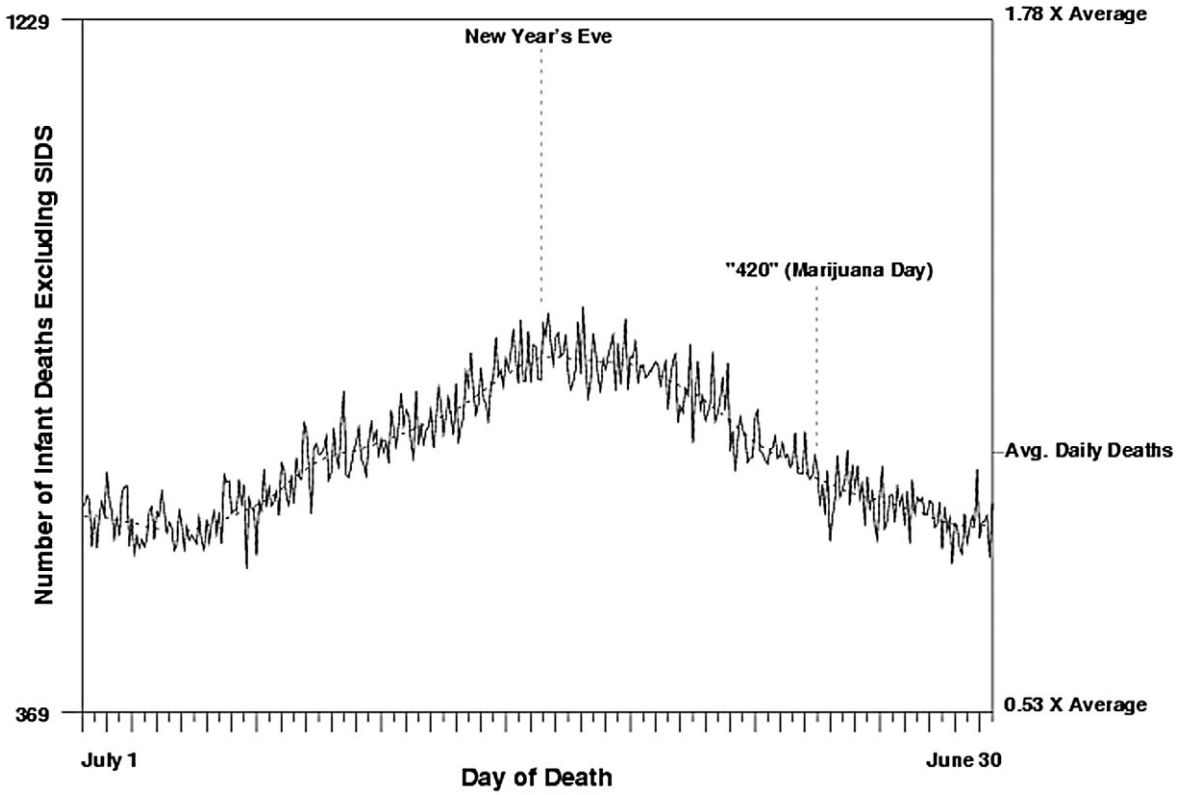

Figure 3 Observed versus expected infant deaths from all causes except sudden infant death syndrome (SIDS), by day of year, for postneonatal infants, United States, 1973–2004. The expected number of deaths (indicated by the broken line) was calculated using locally weighted scatterplot smoothing polynomial (LOESS) regression; this line indicates the number of deaths that would be expected if mortality was affected by seasonal fluctuations and by trend, but not by holidays. See Methods for details. Figures 2 and 3 are plotted to the same ratio scale: the maximum *y*-value is 1.78 times the daily average number of deaths throughout the year; the minimum *y*-value is 0.53 times the daily average number of deaths throughout the year. Source of data: computerized US death certificates [20]

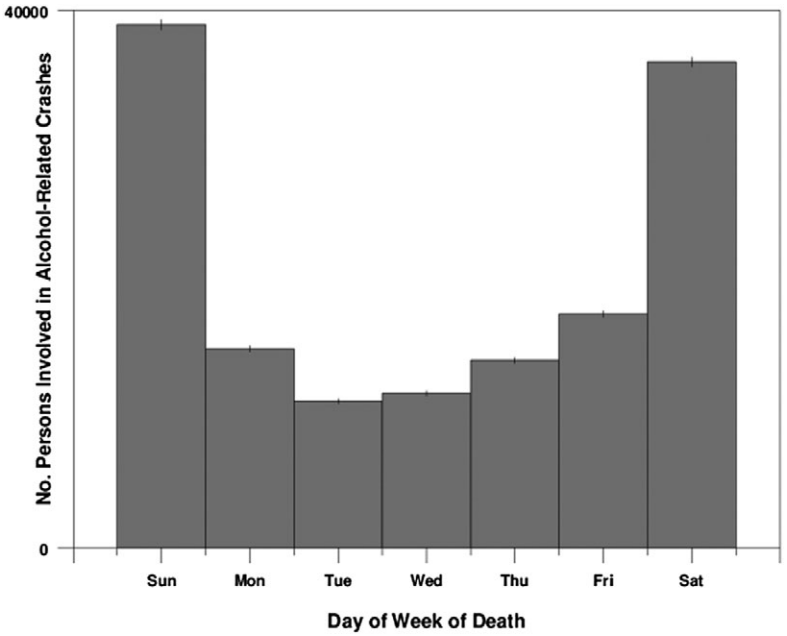

Figure 4 Number of people involved in alcohol-related crashes, by day of the week, United States, 1994–2008. We define alcohol-related crashes as crashed vehicles containing at least one person with a blood alcohol content of 0.08% or higher. For reasons specified in Methods, Fig. 4 focuses upon crashes occurring during the 8 hours centered on the beginning of each day. Thus, e.g. for Saturday, we examine the number of people involved in alcohol-related crashes occurring between 8 p.m. Friday and 3:59 a.m. Saturday. We ensured that there were an equal number of Mondays, Tuesdays, etc. by omitting from the analysis the last 5 days from December 2008. Source of data: the Fatality Analysis Reporting System [22]

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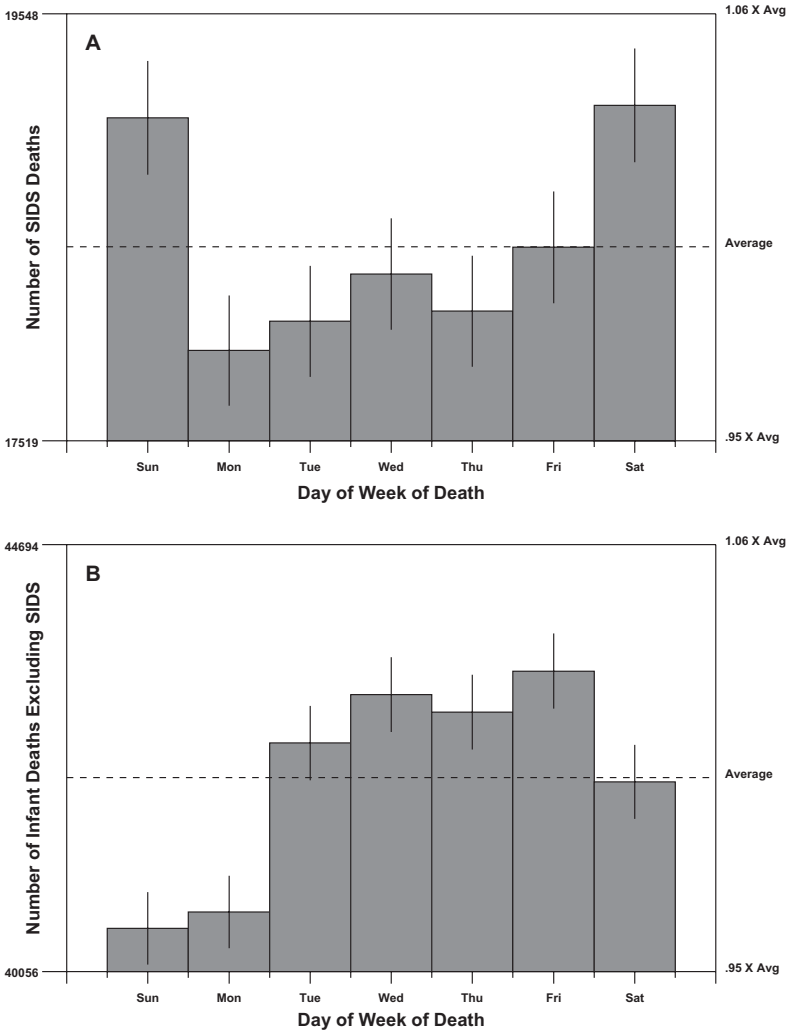

Figure 5 Number of postneonatal infant deaths by day of week, for infants dying from (a) sudden infant death syndrome (SIDS) and (b) other causes, United States, 1973–2006. In this study period, there is an equal number of Mondays, Tuesdays, etc. SIDS deaths are those classified as 795.0 (ICD-8) [23]; 798.0 (ICD-9) [24]; and R95 (ICD-10) [25]. To facilitate comparison, (a) and (b) are plotted to the same scale. See Methods for further details. Source of data: computerized US death certificates [20]

(Fig. 4). Consequently, given the AS hypothesis, SIDS should increase more markedly on weekends than on weekdays.

As predicted, Fig. 5a shows that the number of SIDS cases is significantly higher at weekends. By contrast, non-SIDS infant mortality actually decreases at weekends (Fig. 5b).

Additional evidence bearing on the AS hypothesis

Most generally, the AS hypothesis states that alcohol-impaired caretakers are less able to protect vulnerable children. We tested two corollaries of this hypothesis with the aid of two additional data sets, as follows.

Linked birth and infant death data set

Given the AS hypothesis, infants born to mothers who consumed alcohol during pregnancy should be particularly likely to die from SIDS. The SIDS death rate for children of alcohol-consuming mothers is 2.64 (2.39–2.89) times the SIDS death rate for children of non-alcohol-consuming mothers. Similar, but less extreme, results hold for infants dying from non-SIDS causes. For these causes, the death rate for children of alcohol-consuming mothers is 1.89 (1.76–2.02) times the death rate for children of non-alcohol-consuming mothers. Thus, alcohol-impaired mothers are less able to protect their children.

FARS data set

One corollary of the AS hypothesis is that children should be less likely to be seat-belted properly in the presence of alcohol-impaired adults. This expectation is consistent with FARS data: in crashed vehicles containing at least one alcohol-impaired adult (BAC >= 0.08), there were 1.20 children seat-belted improperly for each child seat-belted properly [1576 of 1309 = 1.20 (1.12–1.29)]. In all other crashed vehicles, this ratio was significantly lower, only 0.51 [4259 of 8325 = 0.51 (0.49 to 0.53)].

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Thus, this data set also indicates that alcohol-impaired adults are less likely to protect vulnerable children.

DISCUSSION

This appears to be the first, large-scale study to test the AS hypothesis: alcohol-impaired caretakers are less able to protect vulnerable infants. Unlike earlier studies [11–19], which examined small, geographically non-representative samples in mainly European settings, we used three very large, exhaustive, US-wide data sets. We uncovered several findings consistent with the AS hypothesis, as follows.

1 Alcohol consumption and SIDS spike far more around New Year than at any other time.
2 Alcohol consumption and SIDS rise significantly at weekends.
3 The SIDS death rate is abnormally high for children of alcohol-consuming mothers.
4 Alcohol-impaired caretakers are less likely to seat-belt children properly in their vehicles.

One finding appears to undermine the AS hypothesis. Given this hypothesis, vulnerable infants dying from causes other than SIDS should also show a New Year spike. Figure 3 appears to undermine this expectation: NY = 5% (−2% to 12%). However, the apparent absence of a New Year spike in Fig. 3 does not, in fact, undermine the AS hypothesis. One assumption underlying this hypothesis is that vulnerable children should be affected the most by alcohol-impaired caretakers. Males are more vulnerable than females—evident from higher male infant mortality [20]. Thus, separate male and female analyses of the data in Fig. 3 should reveal a larger New Year spike for males. As predicted: male NY = 13% (4–23%); female NY = −4% (−14% to 6%). These NY values differ significantly ($P < 0.05$, two-tailed test) [39].

Alternative explanations

All the above findings are consistent with the AS hypothesis but may also be consistent with competing explanations, as follows.

1. The evidence from the linked birth and infant death data set is consistent with intrauterine effects of alcohol. However, this 'intrauterine' hypothesis cannot explain easily why a spike in postneonatal SIDS coincides with a spike in drinking at New Year. Similarly, this hypothesis cannot explain easily why a weekend increase in postneonatal SIDS coincides with a weekend increase in drinking.
2. The New Year spike in SIDS might result from an extraordinarily large spike at one particular New Year, rather than from a general tendency for SIDS to spike at each New Year. However, SIDS spikes on 26 of 29 New Years examined ($P < 0.0001$; binomial test).
3. At New Year and weekends, caretakers may sleep longer and leave infants unsupervised for longer intervals. Given this 'sleeping in' hypothesis, SIDS should also spike during other occasions when caretakers sleep longer. During the Autumn shift to daylight savings, many sleep later because 1 hour has been added to the day. However, on this day: (i) SIDS increases non-significantly: 5% (−6% to 16%); and (ii) SIDS increases for only 15 of 29 years ($P = 0.5$; binomial test). This analysis corrected for the 25-hour day when Autumn daylight savings begins—we multiplied the expected value by 25/24.
4. Medical care might worsen at New Year and weekends, perhaps because holiday/weekend personnel might be reduced or less skilled on these occasions. Given this 'worsened care' hypothesis, the New Year and weekend effects inside medical settings should exceed these effects outside medical settings.

Infants are particularly likely to encounter medical personnel in in-patient, emergency department and ambulatory care settings; they are less likely to encounter these personnel at home. Contrary to what is expected under the worsened care hypothesis:

(a) the New Year effect is smaller, and statistically insignificant, inside medical settings: NY is 16% (−7% to 42%) inside medical settings, versus 49% (14% to 93%) outside medical settings; and
(b) inside medical settings, the average daily number of SIDS cases is smaller at weekends than on weekdays: −7% (−9% to −4%). In marked contrast, outside medical settings, the average daily number of SIDS cases is larger at weekends than on weekdays: 11% (7% to 15%).

Additional evidence undermines the worsened care hypothesis: if holidays degrade care, SIDS should also spike at Christmas and 4 July, but no such spikes are evident.

5. At New Year and weekends, death registrars might be less precise when determining cause of death. Careless classification could generate additional SIDS cases, because SIDS is an ill-defined cause. Given this hypothesis, SIDS should also spike at Christmas and 4 July, but no such spikes are evident.
6. The New Year spike in SIDS might result from a putative tendency to record unknown dates of death as 1 January. Date of death is more often known for autopsied cases. Thus, given this 'date misclassification' hypothesis, NY for SIDS should be smaller for autopsied than for non-autopsied cases. However, this is not so [autopsied cases: 38% (24–52%); non-autopsied cases: 28% (−4% to 66%)]. In our data set, 87% of SIDS cases were autopsied.
7. The New Year spike might be an artifact of LOESS. However, this spike remains when a different tech-

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nique is used: we used the number of SIDS cases 14 days before and 14 days after New Year to estimate the number expected at New Year. This technique corrects for seasonality, trend and the possible influence of Christmas (which occurs 7 days before New Year). This alternative technique reveals a SIDS spike of 38% (24–54%) at New Year [39]. Thus, LOESS and an alternative technique reveal a SIDS spike at New Year.

Some of these alternative explanations might account partially for our findings, although we found no evidence supporting this view. In fact, our evidence undermines each alternative explanation. In contrast, all evidence presented is consistent with the AS hypothesis. Thus, at present, this hypothesis seems to be the most comprehensive and plausible explanation for our findings.

Strengths and limitations

Our study seems to be the first large-scale investigation of the AS hypothesis, and has several important advantages. Using this design, we could: (i) uncover a previously unknown phenomenon: a 33% spike in SIDS at New Year; (ii) generalize our findings to the entire country and to many decades; (iii) correct for seasonality, trend and random fluctuations; and (iv) take advantage of very large, pre-collected, electronic data sets [20–22].

However, our study design also has important disadvantages: our very large samples had little information per case. Consequently, we could not specify the mechanisms connecting alcohol with SIDS. Thus, we cannot determine whether alcohol is: (i) an independent risk factor for SIDS; (ii) a risk factor only in conjunction with other risk factors, such as co-sleeping; and (iii) a proxy for other risk factors associated with occasions when alcohol consumption increases (i.e. the ecological fallacy).

However, our evidence indicates that these risk factors cannot plausibly include: (i) degradation of medical care at New Year; (ii) careless assignment of cause of death at New Year; and (iii) a putative tendency for death registrars to record unknown dates of death as 1 January (see Alternative explanations 5, 6 and 7). Our evidence also indicates that other potential risk factors associated with holidays (such as altered patterns in socializing) are implausible because they are characteristic, not only of New Year, but of other major holidays. However, there is no SIDS spike on these other holidays.

Research implications

Despite these limitations, our findings have potentially significant implications for two substantial literatures: (i) effects of alcohol on health and (ii) factors affecting SIDS. Historically, these literatures have been considered largely distinct; our findings suggest a potentially important relationship between these literatures: alcohol may be a risk factor for SIDS.

Current SIDS investigations de-emphasize cultural risk factors [41]. However, our findings suggest that cultural risk factors (e.g. bed sharing) [13–17], mediated by alcohol consumption, play a significant role. Thus, it seems advisable to increase the emphasis on cultural factors in future SIDS investigations.

Policy implications

Our findings seem to have noteworthy policy implications.

1. When SIDS is suspected, investigators should enquire about recent alcohol history of the infant's caretakers.
2. The 'Back to Sleep' campaign was largely successful [5]. A similar campaign might now be implemented: there should be increased efforts to inform caretakers that alcohol impairs parental capacity and might be a risk factor for SIDS.

CONCLUSION

Despite persistent, long-term efforts to understand and reduce the incidence of SIDS, it remains by far the most frequent cause of postneonatal infant mortality. Thus, it is important to identify and control risk factors for SIDS. Our study is the first to provide large-scale US evidence identifying alcohol consumption as a possible risk factor. This implies that two areas of research (on SIDS and on alcohol), previously considered largely distinct, may actually be linked. This and other implications are potentially significant and warrant further investigation.

Declarations of interest

David P. Phillips, Kimberly M. Brewer, and Paul Wadensweiler have no conflicts of interest to disclose.

Acknowledgements

This research was funded by the Marian E. Smith Foundation. No member of the Marian E. Smith Foundation participated in the production of this study.

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