

Alcohol as a risk factor for SIDS

David P. Phillips¹, PhD

Kimberly M. Brewer², BA

Paul Wadensweiler³

(1) Department of Sociology, University of California at San Diego

(2) Department of Sociology, University of California at San Diego

(3) Department of Human Biology, University of California at San Diego

Corresponding Author: Professor David P. Phillips

University of California at San Diego, Sociology Dept. 0533,

9500 Gilman Drive, La Jolla, CA 92093-0533

Telephone: 858-534-0482 Fax: 858-534-4753

Email: dphillips@ucsd.edu

Running Head: Alcohol as a risk factor for SIDS

Word Count: 3,493

Conflict of Interest: David P. Phillips, Kimberly M. Brewer, and Paul Wadensweiler have no conflict of interest to disclose. Research was funded by the Marian E. Smith Foundation. No member of the Marian E. Smith Foundation participated in the production of this study.

Abstract

Aim

To test whether alcohol is a risk factor for SIDS

Design/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 comparative measures were used: the expected number of deaths on New Year versus the observed number (expected values were determined using LOESS), 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 comparison 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 SIDS, 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 SIDS; in addition, future research should further explore possible connections between SIDS and alcohol.

INTRODUCTION

In the US, 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 significantly increase the risk of SIDS, 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., over 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 judgment^{7,8} and worsen pediatric care.^{9,10} However, within this broad literature, studies focusing on 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 like 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 on small, geographically non-representative 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 nationwide, official US datasets 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) for three decades (1973-2006) to test whether SIDS increases when caretakers are affected by alcohol.

METHODS

We examined daily mortality from three official, nationwide, exhaustive US datasets:

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 dataset 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 1973-1994; this information is available only sporadically after 1994.

Second, we analyzed the linked birth and infant death dataset,²¹ which links information from birth and death certificates for decedents under 1. This dataset is available online from 1995-2005 and records information about the infant and the infant's mother, e.g., whether she consumed alcohol.

Using these datasets, we focused on 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

Third, 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: 8PM to 3:59AM. 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.^{26,27} 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.²⁷ 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 properly seatbelted in the presence of alcohol-impaired adults. When analyzing FARS, we defined “children” as persons under 9 because: (1) This age group requires special seating arrangements, according to official regulations;²⁸ we assumed that this vulnerable age group is comparable to the SIDS age group examined in our other datasets. (2) It is undesirable to restrict FARS analyses to infants under 1 because this restriction yields a very small sample size (n=243).

To determine whether SIDS spikes on New Year, we followed Phillips et al.²⁹ and fitted a locally weighted polynomial regression (LOESS) line to daily mortality, from 1/1/1973—12/31/2004. Phillips et al. determined that a six-week "bandwidth" was appropriate for their LOESS analysis; we also used this bandwidth. This standard non-parametric procedure³⁰⁻³² makes minimal distributional assumptions and corrects for seasonality and trend. Thus, LOESS corrects for the fact that SIDS increases in winter³³ and has decreased over time.⁵

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

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

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

We calculated NY for each New Year between 7/1/1973 and 6/30/2004, with two exceptions: ICD

codes were revised on 1/1/1979 (when ICD-9 replaced ICD-8) and on 1/1/1999 (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: 7/1/1978—6/30/1979 and 7/1/1998—6/30/1999.

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.

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

Following official recommendations³⁴ and our previous practice,^{29,35-39} we calculated standard errors^{34,40} and significance levels, even though we examined complete counts, not samples. As in our previous work,^{29,35-39} 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. Nationwide hourly alcohol consumption is not directly available. However, an indirect, proxy nationwide 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 July 4, we examine the number of persons

involved in alcohol-related crashes occurring in the eight hours between 8PM of July 3 and 3:59AM of July 4.

Figure 1 reveals an extreme spike of 1,016 (984 to 1,048) persons involved in alcohol-related crashes in the last 4 hours of December 31 and the first 4 hours of January 1. 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 to 662) persons in the last 4 hours of July 4 and the first 4 hours of July 5 (Grubbs statistic=4.42; $P < 0.01$). For Figure 1, the average is 372.46 persons, and thus there is no spike in the eight hours centered on the beginning of Christmas: 385 (365 to 404) persons. Henceforth, we focus on New Year because it shows evidence of extreme alcohol consumption during the high-risk period. Given the AS hypothesis, SIDS should also show an extreme spike on New Year (and possibly a smaller spike on July 5).

As predicted, Figure 2 shows a 33% (22% to 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, Figure 2 also shows an increase in SIDS on July 5: 14% (0% to 28%). However, this spike is not a statistical outlier in either sense mentioned above.

Figure 2 also shows an 18% (7% to 31%) spike just after April 20th, a US counterculture holiday devoted to the celebration of cannabis consumption.⁴¹ This spike was not predicted and, like July 5, is not a statistical outlier in either sense mentioned above. In addition, there is no spike in SIDS on July 4 [0% (-13% to 13%)] or on December 25 [-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%) (Figure 3). We return to this finding in the Discussion.

Day of Week

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

In the high-risk period, the number of persons involved in alcohol-related crashes is much higher on Saturday and Sunday than on any other day of the week (Figure 4). Consequently, given the AS hypothesis, SIDS should increase more markedly on weekends than on weekdays.

As predicted, Figure 5A shows that the number of SIDS cases is significantly higher on weekends. By contrast, non-SIDS infant mortality actually *decreases* on weekends (Figure 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 datasets:

1) Linked Birth and Infant Death Dataset

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 to 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 to 2.02) times the death rate for children of non-alcohol-consuming mothers. Thus, alcohol-impaired mothers are less able to protect their children.

2) FARS dataset

One corollary of the AS hypothesis is that children should be less likely to be properly seatbelted in the presence of alcohol-impaired adults. This expectation is consistent with FARS data: In crashed vehicles containing at least one alcohol-impaired adult ($BAC \geq .08$), there were 1.20 children

improperly seatbelted for each child properly seatbelted [1576/1309=1.20 (1.12 to 1.29)]. In all other crashed vehicles, this ratio was significantly lower, only 0.51 [4259/8325=0.51 (0.49 to 0.53)]. Thus, this dataset 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 mostly European settings, we used three very large, exhaustive US-wide datasets. We uncovered several findings consistent with the AS hypothesis:

- (1) Alcohol consumption and SIDS spike far more around New Year than at any other time.
- (2) Alcohol consumption and SIDS rise significantly on weekends.
- (3) The SIDS death rate is abnormally high for children of alcohol-consuming mothers.
- (4) Alcohol-impaired caretakers are less likely to properly seatbelt children 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 Figure 3 does not in fact undermine the AS hypothesis. One assumption underlying this hypothesis is that *vulnerable* children should be the most affected 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 Figure 3 should reveal a larger New Year spike for males. As predicted: male

NY=13% (4% to 23%); female NY= -4% (-14% to 6%). These NY values differ significantly ($P<0.05$, two-tailed test).⁴⁰

Alternative Explanations

All the above findings are consistent with the AS hypothesis but may also be consistent with competing explanations:

1. The evidence from the linked birth and infant death dataset is consistent with intrauterine effects of alcohol. However, this “intrauterine” hypothesis cannot easily explain why a spike in *postneonatal* SIDS coincides with a spike in drinking at New Year. Similarly, this hypothesis cannot easily explain 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 on one particular New Year, rather than from a general tendency for SIDS to spike on *each* New Year. However, SIDS spikes on 26 of 29 New Years examined ($P<0.0001$; binomial test).
3. On 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 one hour has been added to the day. However, on this day: (1) SIDS increases nonsignificantly: 5% (-6% to 16%); (2) 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 on 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 inpatient, 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.

(B) Inside medical settings, the average daily number of SIDS cases is *smaller* on weekends than on weekdays: -7% (-9% to -4%). In marked contrast, *outside* medical settings, the average daily number of SIDS cases is larger on weekends than on weekdays: 11% (7% to 15%).

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

5. On 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 on Christmas and July 4, 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 January 1st. 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% to 52%); non-autopsied cases: 28% (-4% to 66%)]. In our dataset, 87% of SIDS cases were autopsied.

7. The New Year spike might be an artifact of LOESS. However, this spike remains when a different technique is used: We used the number of SIDS cases 14 days before and 14 days after New Year to estimate the number expected on 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% to 54%) on New Year.⁴⁰ Thus, LOESS *and* an alternative technique reveal a SIDS spike on New Year.

Some of these alternative explanations might partially account 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: (1) uncover a previously unknown phenomenon: a 33% spike in SIDS on New Year; (2) generalize our findings to the entire country and to many decades; (3) correct for seasonality, trend, and random fluctuations; (4) take advantage of very large, pre-collected, electronic datasets.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: (1) an independent risk factor for SIDS; (2) a risk factor only in conjunction with other risk factors, like co-sleeping; (3) 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: (A) degradation of medical care on New Year, (B) careless assignment of cause of death on New Year, (C) a putative tendency for death registrars to record unknown dates of death as January 1st. (See alternative explanations 5, 6, 7.) Our evidence also indicates that other potential risk factors associated with holidays (like 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: 1) effects of alcohol on health, and 2) 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 deemphasize cultural risk factors.⁴² 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.

ACKNOWLEDGMENTS

Funding: Marian E. Smith Foundation

REFERENCES

1. Centers for Disease Control and Prevention, Sudden infant death syndrome (SIDS) and sudden unexpected infant death (SUID) <http://www.cdc.gov/nchs/hdi.htm> (accessed August 16, 2010).
2. Moon RY, Horne RSC, Hauck FR. Sudden infant death syndrome. *Lancet* 2007; **370**: 1578-87.
3. Blair PS, Sidebotham P, Berry PJ, Evans M, Fleming PJ. Major epidemiological changes in sudden infant death syndrome: a 20-year population-based study in the UK. *Lancet* 2006; **367**: 314-9.
4. Kinney HC, Thatch BT. The sudden infant death syndrome. *N Engl J Med* 2009; **361**: 795-805.
5. National Institute of Child Health and Human Development, SIDS: "Back to Sleep" campaign <http://www.nichd.nih.gov/SIDS/> (accessed August 16, 2010).
6. Kinney HC, Paterson DS. Sudden infant death syndrome. In: Golden JA, Harding B, eds. *Pathology and Genetics: Acquired and Inherited Diseases of the Developing Nervous System*, ISN Neuropathology Press, Basel (2004), 194-203.
7. Cherpitel CJ. Alcohol and violence-related injuries: an emergency room study. *Addiction* 2006; **88**: 79-88.

8. Haggård-Grann U, Hallqvist J, Långström N, Möller J. Alcohol consumption in the new millennium – weighing up the risks and benefits for our health. *Addiction* 2005; **101**: 100-8.
9. Suchman NE, Luthar SS. Maternal addiction, child maladjustment and socio-demographic risks: implications for parenting behaviors. *Addiction* 2000; **95**: 1417-28.
10. Cohen LR, Hien DA, Batchelder S. The impact of cumulative maternal trauma and diagnosis on parenting behavior. *Child Maltreatment* 2008; **13**: 27-38.
11. Scragg R, Mitchell EA, Taylor BJ, Stewart, Ford RP, Thompson JM, Allen EM, Becroft DM. Bed sharing, smoking, and alcohol in the sudden infant death syndrome. New Zealand Cot Death Study Group. *BMJ* 1993; **307**: 1312-8.
12. Hunt CE, Hauck FR. Sudden infant death syndrome. *CMAJ* 2006; **174**.
13. Blair PS, Sidebotham P, Evason-Coombe C, Edmonds M, Heckstall-Smith EMA, Fleming P. Hazardous cosleeping environments and risk factors amenable to change: case-control study of SIDS in south west England. *BMJ* 2009; **339**: b3666.
14. Webb RT, Wicks S, Dalman C, Pickles AR, Appleby L, Mortensen PB, Haglund B, Abel KM. Influence of Environmental Factors in Higher Risk Sudden Infant Death Syndrome Linked With Parental Mental Illness. *Arch Gen Psychiatry* 2010; **67**: 69-77.

15. King-Hele SA, Abel KM, Webb RT, Mortensen PB, Appleby L, Pickles AR. Risk of Sudden Infant Death Syndrome With Parental Mental Illness. *Arch Gen Psychiatry* 2007; **64**: 1323-30.
16. Fleming P, Blair P, McKenna J. New knowledge, new insights, and new recommendations. *Arch Dis Child* 2006; **91**: 799-801.
17. McGarvey C, McDonnell M, Hamilton K, O'Regan M, Matthews T. An 8 year study of risk factors for SIDS: bed-sharing versus non-bed-sharing. *Arch Dis Child* 2006; **91**: 318-23.
18. L'Hoir MP, Engelberts AC, Well GTJ. Case-control study of current validity of previously described risk factors for SIDS in the Netherlands. *Arch Dis Child* 1998; **79**: 386-93.
19. Carpenter RG, Irgens LM, Blair PS, England PD, Fleming P, Huber J, Jorch G, Schreuder P. Sudden unexplained infant death in 20 regions in Europe: case control study. *Lancet* 2004; **363**: 185-91.
20. National Center for Health Statistics, Mortality data from the national vital statistics system <http://www.cdc.gov/nchs/nvss.htm> (accessed August 16, 2010).
21. National Center for Health Statistics, Period linked birth-infant death data files http://www.cdc.gov/nchs/data_access/Vitalstatsonline.htm (accessed August 16, 2010).

22. National Highway Traffic Safety Administration, Fatal Accident Reporting System Encyclopedia 1994-2008, US Department of Education, Washington, DC (2010) <http://www-fars.nhtsa.dot.gov> (accessed August 16, 2010).
23. US Department of Health Education and Welfare, National Center for Health Statistics, *Manual of the International Classification of Diseases, Injuries, and Causes of Death: Adapted for Use in the United States, Eight Revision*, Public Health Service, Washington, DC (1967).
24. Public Health Division, NSW Health, *Annotated International Classification of Diseases: Ninth Revision, Clinical Modification: Fifth Edition*, (October 1995) <http://www.health.nsw.gov.au/public-health/icd/icd9.htm>. (accessed August 16, 2010).
25. World Health Organization, *International Statistical Classification of Disease-Related Health Problems, 10th Revision*, <http://apps.who.int/classifications/apps/icd/icd10online/> (accessed August 16, 2010).
26. Task Force on Infant Positioning and SIDS. Does Bed Sharing Affect the Risk of SIDS? *Pediatrics* 1997; **100**: 272.
27. Fleming P, Blair P, McKenna J. New knowledge, new insights, and new recommendations. *Arch Dis Child* 2006; **91**: 799-801.

28. National Highway and Traffic Safety Administration, NHTSA Child Passenger Safety
<http://www.nhtsa.gov/Safety/Booster-Seats> (accessed August 16, 2010).

29. Phillips DP, Jarvinen JR, Abramson IS, Phillips RR. Cardiac mortality is higher around Christmas and New Year's than at any other time: the holidays as a risk factor for death. *Circulation*. 2004; **110**: 3781-8.

30. Simonoff JS, *Smoothing Methods in Statistics*, Springer-Verlag, New York, NY (1996).

31. Ruckstuhl AF, Jacobson MP, Field RW, Dodd JA. Baseline subtraction using robust local regression estimation. *J Quant Spectrosc Radiat Transf*. 2001; **68**: 179-93.

32. Dataplot [database online]. National Institutes of Standards and Technology, Gaithersburg, MD (2001).

33. Byard RW, Krous HF. Sudden infant death syndrome: overview and update. *Pediatr Dev Pathol* 2003; **6**: 112-27.

34. National Center for Health Statistics, *Vital Statistics of the United States 1990. Mortality Part A. Vol 2, Sect 7, Technical Appendix*. US Department of Health and Human Services, Hyattsville, MD (1994).

35. Phillips DP, Liu GC, Kwok K, Jarvinen JR, Zhang W and Abramson IS. The Hound of the Baskervilles effect: natural experiment on the influence of psychological stress on the timing of death. *BMJ* 2001; **323**: 1443-6.
36. Phillips DP, Christenfeld N and Ryan NM. An increase in the number of deaths in the United States in the first week of the month—an association with substance abuse and other causes of death. *N Engl J Med.* 1999; **341**: 93-8.
37. Phillips DP, Christenfeld N and Ryan NM. Increase in US medication-error deaths between 1983 and 1993. *Lancet* 1999; **351**: 643-4.
38. Phillips DP, Ruth TE and Wagner LM. Psychology and survival. *Lancet* 1993; **342**: 1142-5.
39. Phillips DP and Paight DJ. The impact of televised movies about suicides: a replicative study. *N Engl J Med.* 1987; **317**: 809-11.
40. Gardner MJ, Altman DG, eds, *Statistics with Confidence: Confidence Intervals and Statistical Guidelines, 2nd ed*, BMJ Books, London, England, (2000).
41. Grim R, What 420 Means: The True Story Behind Stoners' Favorite Number
http://www.huffingtonpost.com/2009/04/20/what-420-means-the-true-s_n_188320.html (accessed August 16, 2010).

42. National Library of Medicine, Pub Med <http://www.ncbi.nlm.nih.gov/pubmed> (accessed August 16, 2010).

Figure 1. Number of Persons Involved in Alcohol-Related Crashes, by Day of the Year, 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, Figure 1 focuses on crashes occurring during the eight hours centered on the beginning of each day. Thus, e.g., for New Year, we examine the number of persons involved in alcohol-related crashes occurring between 8PM of December 31 and 3:59AM of January 1.

In Figure 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 on 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 precisely identify holidays with movable dates (like Thanksgiving).

Source of data: the Fatality Analysis Reporting System.²²

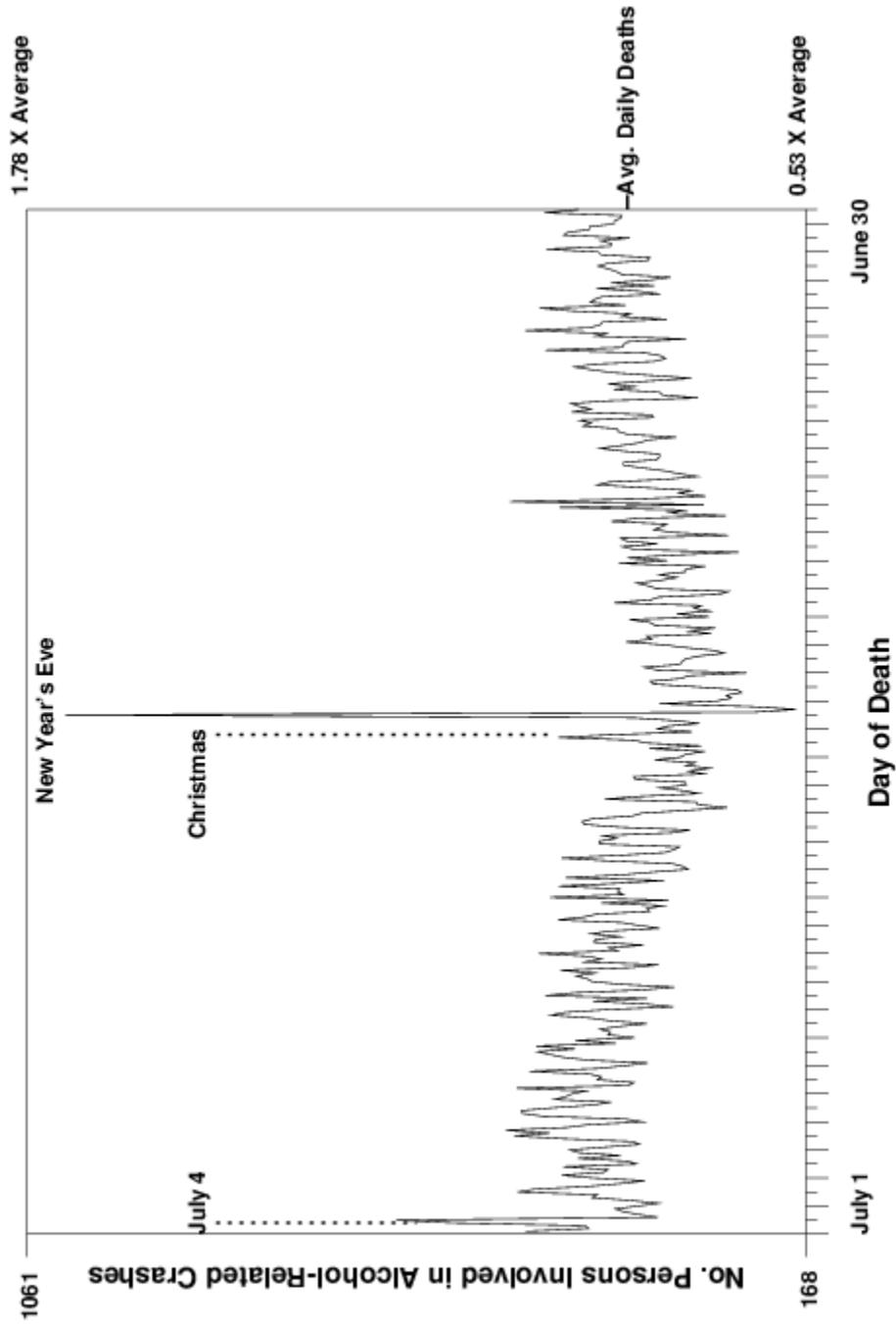


Figure 2. Observed Versus Expected SIDS Deaths by Day of Year, for Postneonatal Infants, United States, 1973-2004.

The expected number of deaths (indicated by the broken line) was calculated using 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

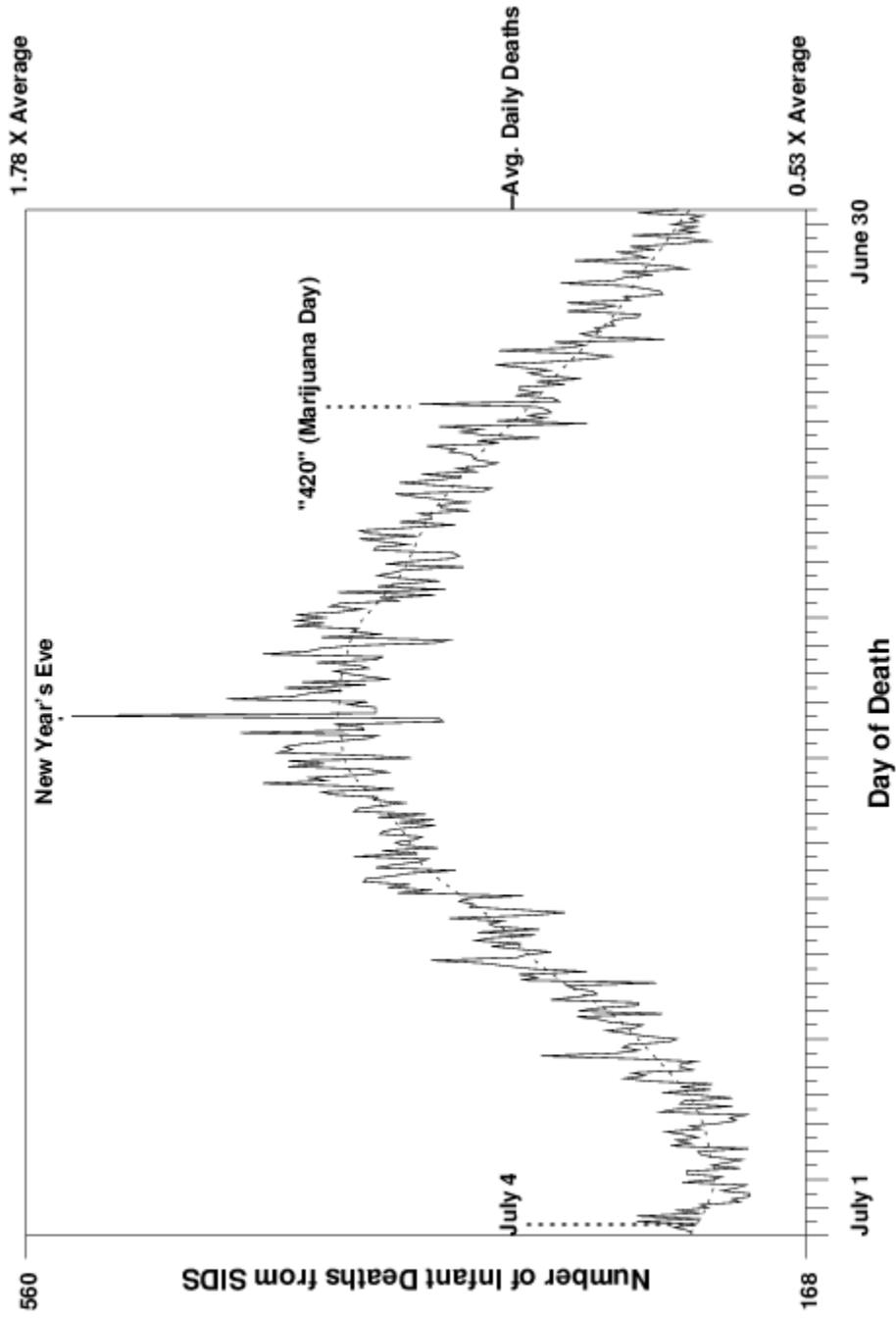


Figure 3. Observed Versus Expected Infant Deaths from All Causes except SIDS, by Day of Year, for Postneonatal Infants, United States, 1973-2004.

The expected number of deaths (indicated by the broken line) was calculated using 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

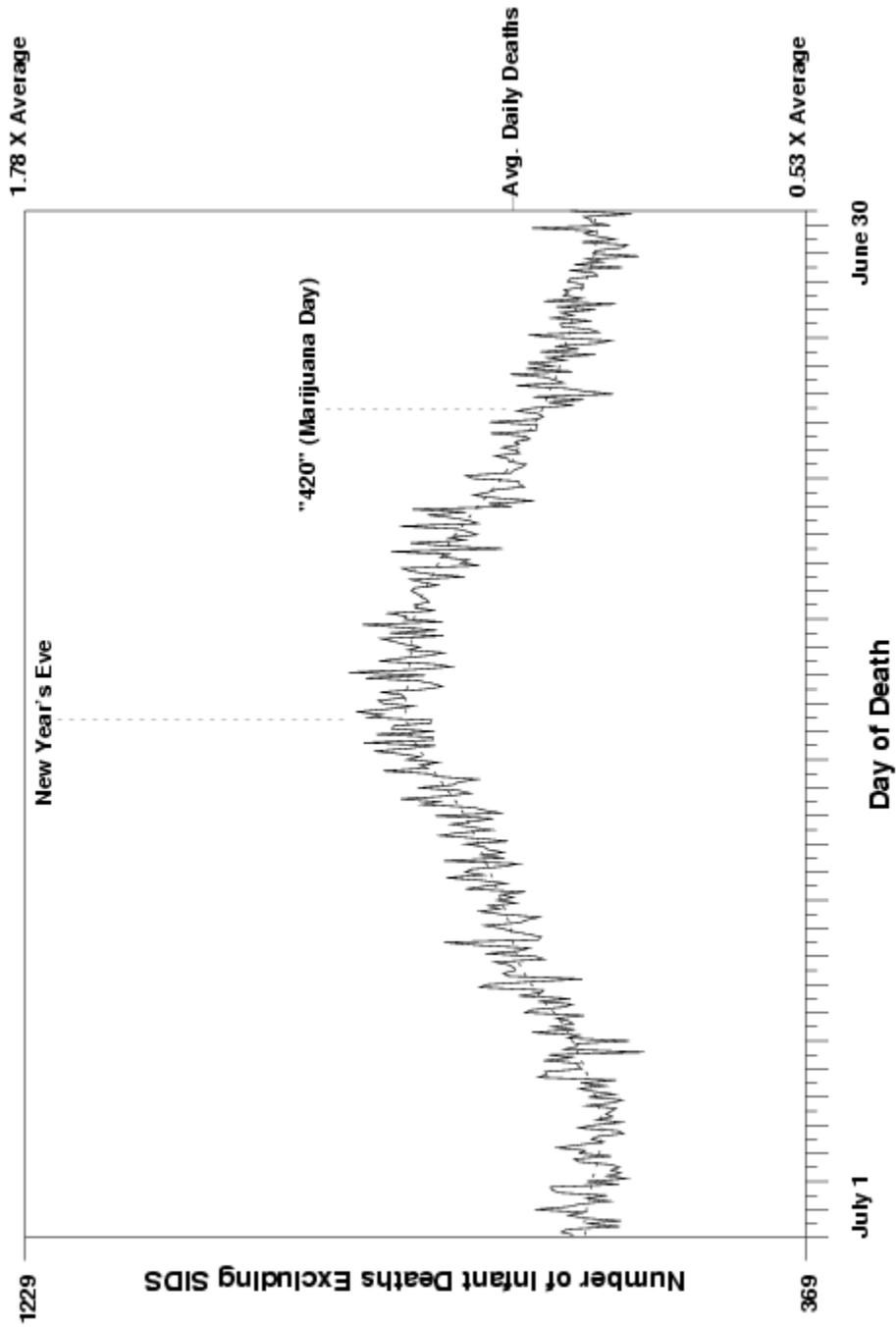


Figure 4. Number of Persons Involved in Alcohol-Related Crashes, by Day of the Week, United States, 1994-2008.

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, Figure 4 focuses on crashes occurring during the eight hours centered on the beginning of each day. Thus, e.g., for Saturday, we examine the number of persons involved in alcohol-related crashes occurring between 8PM of Friday and 3:59AM of Saturday. We ensured that there were an equal number of Mondays, Tuesdays, etc. by omitting from the analysis the last 5 days from December of 2008.

Source of data: the Fatality Analysis Reporting System.²²

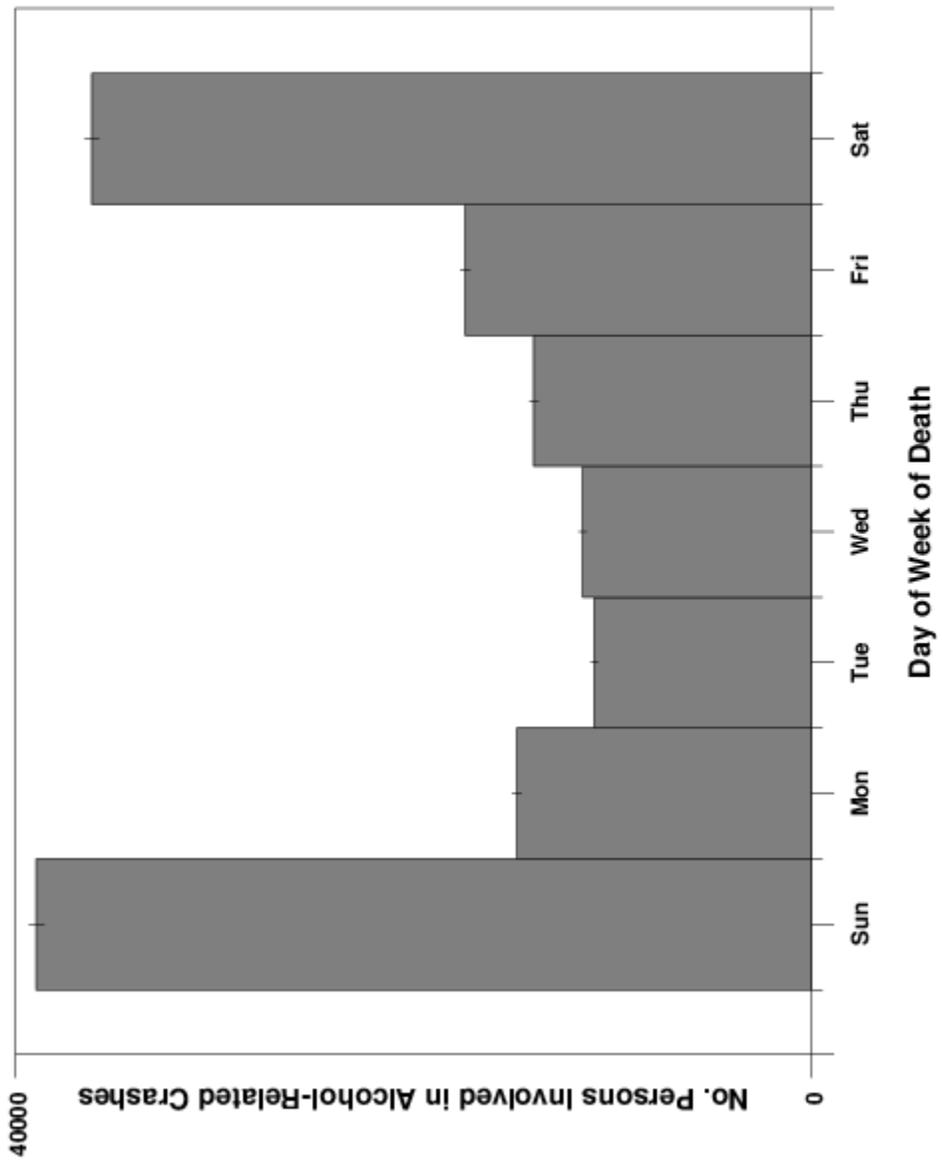


Figure 5. Number of Postneonatal Infant Deaths by Day of Week, for Infants Dying From (A) SIDS and from (B) Other causes, United States, 1973-2006.

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, Panels A and B are plotted to the same scale. See Methods for further details.

Source of data: computerized US death certificates.²⁰

