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Ambient Temperature and Sudden Infant Death Syndrome in the United States

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Abstract

Background—Sudden infant death syndrome (SIDS) is a leading cause of infant mortality in the United States. While thermal stress is implicated in many risk factors for SIDS, the association between ambient temperature and SIDS remains unclear.

Methods—We obtained daily individual-level infant mortality data and outdoor temperature data from 1972 to 2006 for 210 United States cities. We applied a time-stratified case-crossover analysis to determine the effect of ambient temperature on the risk of SIDS by season. We stratified the analysis by race, infant age, and climate.

Results—There were a total of 60,364 SIDS cases during our study period. A 5.6°C (10°F) higher daily temperature on the same day was associated with an increased SIDS risk of 8.6% (95% confidence interval (CI): 3.6%, 13.8%) in the summer, compared to a 3.1% decrease (95% CI: -5.0%, -1.3%) in the winter. Summer risks were greater among Black infants (18.5%, 95% CI: 9.3%, 28.5%) than White infants (3.6%, 95% CI: -2.3%, 9.9%), and among infants 3–11 months old (16.9%, 95% CI: 8.9%, 25.5%) than infants 0–2 months old (2.7%, 95% CI: -3.5%, 9.2%). The temperature-SIDS association was stronger in climate clusters in the Midwest and surrounding northern regions.

Conclusions—Temperature increases were associated with an elevated risk of SIDS in the summer, particularly among infants who were Black, 3 months and older, and living in the Midwest and surrounding northern regions.

Keywords

Sudden infant death syndrome; temperature; case-crossover

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Data availability: Because the mortality dataset was used under a Data User Agreement from each state, the dataset and code are not available for replication.

Conflicts of interest: The authors declare no conflict of interest.

Introduction

Sudden infant death syndrome (SIDS) is the third leading cause of overall infant mortality and the leading cause of postneonatal infant mortality (1 month to 1 year of age) in the United States.^{1–3} SIDS typically occurs during sleep and is defined as the sudden death of an infant under one year of age that remains unexplained even after a thorough case investigation, including an autopsy.⁴ While the incidence of SIDS decreased drastically following initiatives that promoted supine sleeping positions in the early 1990s, rates have remained stagnant for the past 15 years.³

The cause of SIDS is unknown. However, challenges to the thermoregulatory capacity of infants (i.e., thermal stress) have been implicated in many of the well-known risk factors, such as prone sleeping position, overwrapping infants, and elevated room temperature.^{3,5–8} For infants in the critical developmental period for homeostatic control, thermal stress can increase demand on the thermoregulatory mechanism during sleep and can impair arousal mechanisms, respiratory drive, cerebral oxygenation, and cardiac responses.⁹ Studies have found that bedroom heating increases SIDS risk,¹⁰ whereas well-ventilated bedrooms and use of a fan is associated with decreased risk of SIDS.^{11,12} These findings suggest that indoor heat is an important risk factor for SIDS. In addition, indoor and outdoor temperatures have been shown to correlate strongly, particularly in the warm season.^{13–15} Therefore, outdoor heat is a plausible risk factor for SIDS, both directly from the thermal stress of outdoor heat exposure and indirectly through its effect on indoor temperature.

Despite numerous studies investigating risk factors related to thermal stress, there is limited literature on the role of outdoor heat as a risk factor for SIDS. The majority of studies on SIDS and outdoor temperature date to the 1990s that focus on outdoor cold and report a negative association between SIDS and temperature.¹⁶ It was generally concluded that hypothermia was not a direct cause, but rather that low temperature was a marker for parental behavioral changes (e.g., the use of excessive infant clothing and bedding) and seasonal increases in respiratory infection.^{17,18} Since then, there has been a paucity of studies investigating the association between outdoor temperature and SIDS. Among the few recent studies, one study reported that heat exposure is a risk factor for SIDS¹⁹ while others reported a null association.^{20–22}

As climate change may lead to greater frequency and intensity of heat waves, it is critical to evaluate temperature as a potential risk factor for SIDS. In this study, we conducted a large national study using individual-level infant mortality data to assess the effect of ambient temperature on SIDS risk over a 35-year period in 210 United States cities.

Methods

Infant Mortality Data

We obtained daily infant mortality data collected between 1972 and 2006 for 210 United States cities from the National Center for Health Statistics (NCHS). Infant deaths were defined as deaths of infants less than 1 year of age. SIDS cases were identified using

International Classification of Diseases 8th Revision (ICD-8), 9th Revision (ICD-9), and 10th Revision (ICD-10) codes 795.0, 798.0, and R95.0, respectively.

Environmental Data

We obtained daily 24-hr average outdoor air and dew-point temperatures measured at airport weather stations from the National Oceanic and Atmospheric Administration (NOAA). Relative humidity was calculated with the following formula:

$$\text{Relative humidity (rhum)} = 100\% \times \left(\frac{112 - 0.1T_{\text{air}} + T_{\text{dewpt}}}{112 + 0.9T_{\text{air}}} \right)$$

where T_{air} and T_{dewpt} denote 24-hr average air temperature and dew-point temperature, respectively. The closest weather station was matched to each city. For missing measurements, values from the nearest station within 60 kilometers were used as in previous studies.^{23,24}

As one study found an association between ozone and SIDS,²⁵ we obtained ozone (daily max 8-h mean) data from the United States Environmental Protection Agency's Air Quality System Technology Transfer Network. Daily max 8-h mean ozone data were available for 1985-2006 in 202 of the 210 cities that we examined.

Statistical Analyses

We used a time-stratified case–crossover analysis to assess the association between same-day air temperature and SIDS. The case–crossover design is ideal for assessing associations between short-term exposures and rare outcomes such as SIDS,²⁶ and has been used in previous studies assessing heat exposure and SIDS.^{19,22} The case–crossover design compares each subject's exposure in a time period just prior to a case-defining event with that subject's exposure at other times. Since each subject is his or her own control, all measured or unmeasured time-invariant subject characteristics (e.g., sex, race, socioeconomic status, year/season of birth) are inherently adjusted for, such that there is no confounding by these characteristics. We chose control days as every three days preceding and/or following the date of death within the same month.

We investigated the association between daily average air temperature and SIDS using a conditional logistic regression model (PROC PHREG in SAS, version 9.4, SAS Institute, Cary, NC). An estimate for each season was obtained through an interaction term between temperature and season (defined as spring, Mar-May; summer, Jun-Aug; fall, Sept-Nov; winter, Dec-Feb). We used a continuous variable for air temperature and adjusted for relative humidity as a continuous linear variable and day of the week with indicator variables. We scaled the results to represent a percent excess SIDS risk per 5.6°C (10°F) difference in temperature. We examined the effect of same-day temperature exposure (lag 0) as well as exposures on previous days (*i.e.*, lag 1, lag 2). In addition, we stratified the analysis age, race, sex, and climate (described below).

Climate Clusters

Given the variability in climate across the United States, we stratified our analysis by different climate characteristics. To do so, we categorized the 210 cities into eight climate clusters (Figure 1). The clusters were created through an agglomerative hierarchical approach to minimize within-cluster heterogeneity and maximize between-cluster heterogeneity based on the mean and standard deviation of temperature and relative humidity for all four seasons in each city (more details in Nordio *et al.*, 2015).²⁴ While geographic proximity was not a factor in the clustering algorithm, cities were grouped in a manner in which a predominant geographic pattern can be identified (e.g., West Coast, Northeast, Gulf Coast, etc.).

Sensitivity Analyses

We conducted several sensitivity analyses to examine the effects of the Back to Sleep (BTS) campaign, air conditioning prevalence, and an alternative temperature exposure metric on the temperature-SIDS association. As the incidence of SIDS drastically declined after the introduction of the BTS campaign in the early 1990s, we stratified our analysis by time period (i.e., 1972-1993 and 1994-2006). In addition, air conditioning has been shown to modify the association between temperature and adult mortality.^{27,28} We calculated the percentage of households in the city with any air conditioning (central or room) based on the Census Bureau's American Housing Survey (more details in Reid *et al.* 2009).²⁸ We assessed the effect of AC on temperature-related SIDS risk by stratifying the analysis by level of air conditioning prevalence (i.e., lower, middle, and upper tertiles). Lastly, as apparent temperature has been used in previous studies examining temperature-related mortality,^{22,29} we compared the risk estimates derived from apparent temperature and air temperature. Apparent temperature is defined as an individual's perceived air temperature at a given humidity level. Due to greater completeness of air temperature data, the apparent temperature data was only used for sensitivity analysis. Apparent temperature (*AT*) was calculated with the following formula:

$$AT = -2.653 + (0.994 \times T_{\text{air}}) + (0.0153 \times (T_{\text{dewpt}})^2)$$

Results

There were 554,644 infant deaths between 1972 and 2006, of which 60,364 (10.9%) were attributed to SIDS (Table). SIDS incidence was higher among males (60.0%), and was highest in the winter (30.7%) and lowest in the summer (18.9%). The study population consisted of 63.7% White, 33.0% Black, 2.2% Asian/Pacific Islander, and 1.0% Native American infants. More than half of SIDS cases occurred at 0-2 months of age. The number of SIDS cases were greatest in clusters 1, 2, and 5, which in aggregate represented more than 60% of total cases. The eight climate clusters exhibited distinct combinations of temperature and relative humidity profiles (eFigure 1, eTable 1). More detailed descriptions of each climate cluster can be found in Nordio *et al.* 2015.²⁴

We estimated the association between SIDS and temperature for each season. For a 5.6°C (10°F) increase in temperature, the risk of SIDS increased by 8.6% (95% CI: 3.6%, 13.8%) in the summer (Jun-Aug) and decreased by 3.1% (95% CI: -5.0%, -1.3%) in the winter (Dec-Feb) (Figure 2). There was no association between SIDS and temperature in the fall and spring. We also assessed the effect of temperature exposure from 1 and 2 days prior to the day of death. In the winter, protective associations were also observed for warmer temperatures 1 and 2 days prior that were similar in magnitude to same-day temperature effects. In the summer, there was an 8.0% (95% CI: 3.2%, 13.1%) increased risk of SIDS for a 5.6°C increase in the previous day's temperature, but there was no association with temperature 2 days prior. When the same day and previous day summer temperatures were included in the model simultaneously, the cumulative risk did not explain more risk than using either single temperature model. The results were robust to the adjustment of ozone (eTable 2), as well as the use of apparent temperature (i.e., perceived air temperature at a given humidity level; see Methods section) instead of air temperature (eFigure 2). There was no indication that apparent temperature was a better predictor than air temperature. In addition, there was no evidence of nonlinearity in the relationship between SIDS and temperature in the summer (eFigure 3).

Temperature-related risk of SIDS varied by race and infant age. The summertime risk was greater for Black infants than for White infants (Figure 3A). For a 5.6°C increase in temperature, the excess risk of SIDS in the summer was 18.5% (95% CI: 9.3%, 28.5%) for Black infants compared to 3.6% (95% CI: -2.3%, 9.9%) for White infants. There were not enough SIDS cases to observe a statistically significant association in other race categories (eTable 3).

Risk of SIDS was higher among infants 3-11 months of age compared to those 0-2 months of age (Figure 3B, eTable 4). For a 5.6°C increase in temperature, the excess risk of SIDS in the summer was 16.9% (95% CI: 8.9%, 25.5%) for infants 3-11 months old compared to 2.7% (95% CI: -3.5%, 9.2%) for infants 0-2 months old. Summertime temperature-related SIDS risk was greatest among 4 month old infants (20.9%, 95% CI: 4.3%, 40.2%). Furthermore, Black infants 3-11 months old had a 24.3% (95% CI: 9.8%, 40.7%) increased risk of SIDS per 5.6°C increase in temperature during the summer. There were no differences in SIDS risk observed between male and female infants (eFigure 4).

The incidence of SIDS declined drastically following the BTS campaign in the early 1990s. To assess whether changes in infant sleep practices modified the association between temperature and SIDS, we stratified our analysis by pre- and post-BTS periods: 1972-1993 (22 years) and 1994-2006 (13 years) (Figure 4). The summer and winter risk estimates in the post-BTS period were similar in magnitude to those in the pre-BTS period, although with wider confidence intervals because of the substantial reduction in SIDS cases. When we stratified the analysis further by age group, SIDS risk among 3-11 month old infants was the same between the pre-BTS period (16.8%, 95% CI: 7.4%, 27.0%) and post-BTS period (16.6%, 95% CI: 2.0%, 33.4%). This suggests that temperature-related SIDS risk was not modified by any BTS-related changes in infant sleeping conditions.

Differences in temperature-associated SIDS risk were observed between climate clusters (Figure 5). In the summer, temperature increases were associated with an elevation in SIDS risk in cluster 2 (Upper Midwest/Northeast) by 11.5% (95% CI: 2.8%, 21.0%) and in cluster 3 (North Midland) by 18.4% (95% CI: 2.4%, 36.8%). In addition, a borderline significant positive association was observed in cluster 5 (West Coast) by 11.6% (95% CI: -1.5%, 26.4%; $p = 0.08$). We observed generally higher risks among clusters with cooler summers, although this trend was not statistically significant ($p=0.14$) (eFigure 5). Cities in clusters 2 and 3 had the coldest winters and are located predominantly in the Upper and Industrial Midwest (eTable 1). In addition, clusters 2 and 5 had the lowest air conditioning prevalence among all the clusters (eFigure 6). While air conditioning prevalence did not modify the association between temperature and SIDS, the temperature-SIDS association was most robust among cities with moderate AC prevalence (77.4-95.7% prevalence) (eFigure 7). In the winter, an increase in temperature was associated with a decreased risk of SIDS in cluster 5, which included cities on the West Coast. In all other clusters, the associations were also negative but not robust in the winter.

Discussion

In a large, national study of 60,364 SIDS cases, acute (i.e., same or previous day) exposure to increased ambient temperature was associated with an increased risk of SIDS in the summer. This increased risk was greater among Black infants and infants 3 months or older. The risk varied geographically by climate characteristics, with more pronounced heat effects estimated in clusters with cooler summers. Our findings suggest that heat exposure is an important risk factor for SIDS. Subsequently, projected increases in the frequency and intensity of heat waves related to climate change may pose a substantial risk to infants.

Our results are consistent with a recent case-crossover study in Montreal, Canada of 196 SIDS cases, which found a nearly three-fold increased risk of SIDS on higher temperature days ($>29^{\circ}\text{C}$ relative to 20°C) during the summer.¹⁹ In addition, risks were much greater among infants 3 months or older. This is consistent with the more than six-fold risk we found among infants 3-11 months old compared with infants 0-2 months old. In contrast, null associations have been reported in a case-crossover study (861 SIDS cases) conducted in California³⁰ and in an ecologic analysis of SIDS rates (111 SIDS cases) conducted in Arkansas, Georgia, Kansas, and Missouri.²¹ A study in Taiwan of 1,671 SIDS cases reported a protective effect of higher temperature days ($>26.4^{\circ}\text{C}$ relative to $21.9 - <23.3^{\circ}\text{C}$ in daily maximum temperature).²⁰ Aside from these studies, however, the majority of the literature on outdoor temperature and SIDS date to the 1990s and focus on the effects of colder outdoor temperatures but not heat exposure.^{16,18,31} Furthermore, given the relatively rare incidence of SIDS, prior studies have been limited by sample size and geographic distribution. To date, this present study represents the largest analysis on SIDS and outdoor temperature.

It is hypothesized that for a vulnerable infant in a critical development period, exposure to external stressors (e.g., prone sleeping) can overwhelm cardiorespiratory autonomic control and sleep arousal mechanisms.⁷ Many of the well-known risk factors for SIDS (e.g., prone sleeping, infant overwrapping, bedroom heating, bed sharing) are associated with thermal

stress.³ Conversely, a well-ventilated bedroom and the use of a fan reduce the risk of SIDS.^{11,12} Thermal stress can lead to infant death by disrupting the respiratory drive, laryngeal closure reflex, and arousal mechanisms.^{9,21,32}

We found greater heat-related SIDS risk among infants 3-11 months old than infants 0-2 months old. Overwrapping, room heating, and profuse night sweating have been more strongly associated with SIDS among infants older than 2 months of age.^{32,33} In addition, prone sleeping alters cardiorespiratory autonomic control and results in decreased cerebral oxygenation during sleep, particularly at 2 to 3 months of age.³⁴ Other studies have also suggested that there are temporary protective mechanisms among neonates that disappear as infants develop,³⁵ and that changes in breastfeeding rates may contribute to age-related differences in the susceptibility to SIDS.² Therefore, multiple factors may contribute to the elevation in temperature-related SIDS risk among infants 3 months and older.

Our results suggest that estimated differential effects of outdoor heat contribute to racial disparities in SIDS rates, given that for the same temperature increase, Black infants had more than a five-fold risk compared with White infants. Race/ethnicity may serve as a proxy for socioeconomic status in the United States, specifically for heat-related health effects.³⁶ However, SIDS rates among infants who are African American, Native American, or Alaskan Native are at least twice the national average, regardless of socioeconomic status.^{1,7} Therefore, our results may reflect biologic and/or cultural differences that increase susceptibility to SIDS with heat exposure. Studies have investigated the role of serotonergic brainstem abnormalities to explain increased vulnerability of male infants to SIDS,³⁷ but to our knowledge, physiologic differences by race/ethnicity have not been identified. While racial and ethnic differences in sleeping environments (e.g., supine positioning, bed sharing, use of soft bedding) contribute to disparities in the incidence of SIDS,^{3,38-40} we found that temperature-related SIDS risk ratios remained the same after the BTS campaign drastically reduced SIDS rates in the mid-1990s. This suggests that changes in infant sleeping conditions resulting from the BTS campaign did not modify the association of outdoor temperature exposure with SIDS risk. In other words, while the BTS campaign was associated with reduced incidence of SIDS, infant susceptibility to SIDS resulting from outdoor heat exposure remained unaffected.

We also found important differences among regions with different climate characteristics. Among the eight climate clusters, summertime temperature-related SIDS risk was more pronounced in clusters in the Upper/Industrial Midwest and the surrounding region. We observed a general, albeit not statistically significant, trend of higher risks in clusters with cooler summers. This is consistent with observations that individuals living in cities with low summer temperatures have higher mortality risks during periods of high temperature, after controlling for air conditioning.²⁴ Among adults, there is evidence of acclimatization to local weather conditions and that temperature variability is a key driver of geographic differences in heat exposure effects.⁴¹ However, little is known about physiological adaptation to heat among infants. The role of air conditioning in mitigating heat effects is also well established.²⁷ While we found differences in air conditioning prevalence between climate clusters, we did not find evidence that air conditioning prevalence modified the temperature-related SIDS risk. Nonetheless, geographic differences in behavioral adaptation

to heat (e.g., avoidance of outdoor exposure, air conditioning use), urban design (e.g., green space, shading on street surfaces), or building characteristics (e.g., building heat penetration) could contribute to differences in susceptibility between the climate clusters. These potential modifying factors and their roles in the geographic and racial/ethnic disparities in SIDS risk would be important topics for future SIDS research.

Behavioral effects are also important for understanding the negative wintertime temperature-SIDS relationship. Consistent with older studies that report increased SIDS incidence with colder temperatures,^{16,18} we found that warmer temperatures during the winter were protective against SIDS. Studies have shown that the increased SIDS risk associated with colder temperatures in the winter can be explained by excessive clothing and overwrapping of infants.⁷ Therefore, the winter effects reflect parental behaviors in response to cold temperature rather than the effects of actual exposure to colder temperatures.

Many of the limitations of our study stem from data availability. We utilized monitored outdoor temperatures and not personal temperature exposure data, which can lead to exposure misclassification. We did not have individual mortality data after 2006, as these were no longer available from NCHS. We also did not have additional race/ethnicity categories (e.g., Hispanic) to fully assess racial/ethnic disparities. Nonetheless, we found important differences that warrant targeted public health interventions. While the case-crossover design was ideal for adjusting for time-invariant subject-level confounders that were not available in the data, there were important assumptions. We assumed that the temperature-SIDS relationship was similar across multiple cities. To address this, we stratified our analysis by climate clusters, but there may be important differences within clusters. While a city-level analysis would be ideal, this was not possible due to the limited number of SIDS cases per city. In addition, we were not able to evaluate the role of potential modifiers, such as preterm birth and maternal tobacco use, however, these are time-invariant individual risk factors that are inherently adjusted for in the case-crossover design.

In summary, we identified outdoor temperature as an important risk factor for SIDS, especially among Black infants, infants 3 months and older, and infants living in the Midwest and surrounding northern regions in the United States. Our results suggest that future increases in frequency and intensity of heat waves related to climate change may have important implications for infant health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
Location of 210 cities by climate cluster. Honolulu, Hawaii (cluster 7) and Anchorage, Alaska (cluster 2) are not shown.

Figure 2. Percent increase in SIDS for a 5.6 °C (10 °F) increase in same-day temperature (lag 0), prior day temperature (lag 1), and 2 days prior temperature (lag 2) by season across 210 cities (1972-2006). 95% confidence intervals are shown.

Figure 3. Percent increase in SIDS for a 5.6 °C (10 °F) increase in same-day temperature by season across 210 cities (1972-2006), stratified by race (A) and age (0-2 months vs. 3-11 months) (B). 95% confidence intervals are shown.

Figure 4. Percent increase in SIDS for a 5.6 °C (10 °F) increase in same-day temperature by season across 210 cities, stratified by time period (1972-1993 vs. 1994-2006) and age (0-2 months vs. 3-11 months).

Figure 5. Percent increase in SIDS for a 5.6 °C (10 °F) increase in same-day temperature by season and climate cluster (1972-2006).

Table
SIDS cases (n=60,364) in 210 United States cities during 1972-2006

Season	n	(%)
Spring	15,460	(25.6)
Summer	11,413	(18.9)
Fall	14,952	(24.8)
Winter	18,539	(30.7)
Male	36,237	(60.0)
Race		
White	38,456	(63.7)
Black	19,923	(33.0)
Asian/Pacific Islander	1,327	(2.2)
Native American	608	(1.0)
Other	50	(0.1)
Climate cluster^a		
Cluster 1 (Northeast)	9,672	(16.0)
Cluster 2 (Upper Midwest + AK)	14,481	(24.0)
Cluster 3 (North Midland)	4,842	(8.0)
Cluster 4 (South Midland)	6,630	(11.0)
Cluster 5 (West Coast)	13,592	(22.5)
Cluster 6 (Gulf Coast)	4,489	(7.4)
Cluster 7 (FL, bordering Mexico + HI)	3,197	(5.3)
Cluster 8 (Between CA and NM)	3,461	(5.7)
Age		
0-2 months	34,101	(56.0)
3-11 months	26,263	(44.0)

^aClimate clusters were created based on a statistical approach that grouped cities based on their temperature and relative humidity profiles (see Methods). The regions listed in this table reflect the predominant geographic distribution of each cluster.

SIDS: sudden infant death syndrome. AK: Alaska. FL: Florida. HI: Hawaii. CA: California. NM: New Mexico.