Heat Stress and Sudden Infant Death Syndrome Incidence: A United States Population Epidemiologic Study

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ABSTRACT. Objectives. To determine the role of heat stress in sudden infant death syndrome (SIDS) by examining the SIDS rates during periods of extreme environmental temperatures in a period when most infants were placed prone for sleep.

Design. A retrospective study of SIDS rates and mortality rates attributable to excessive environmental heat in relationship to climatologic temperature was performed. Data were collected for each of 454 counties in 4 states (Arkansas, Georgia, Kansas, and Missouri) from May 1 to September 30, 1980, and were then summed to yield the mortality rates for each 5°F (2.8°C) temperature range.

Results. χ² analyses revealed significant relationships for heat-related mortality rates and both the maximal daily temperature and mean daily temperature but demonstrated no such relationships for SIDS rates. No association between SIDS rates and heat-related mortality rates was found with Spearman’s ranked correlation, for either the maximal daily temperature or the mean daily temperature.

Conclusions. On the basis of our findings of no significant association between SIDS and either measure of temperature during periods of high heat stress-related death rates, it seems unlikely that the heat stress associated with the combination of prone sleep positions and elevated environmental temperatures has a significant role in the development of SIDS. Pediatrics 2004; 113:e586–e592. URL: http://www.pediatrics.org/cgi/content/full/113/6/e586; SIDS, thermal stress, bedding, overwrapping, sleep position.

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ABBREVIATIONS. SIDS, sudden infant death syndrome; \( T_{\text{max}} \), maximal daily temperature; \( T_{\text{avg}} \), average daily temperature.

The thermal stress theory for sudden infant death syndrome (SIDS) states that many SIDS deaths occur as a result of increased demands on thermoregulatory mechanisms attributable to a prone sleep position, increased ambient temperature, and/or a decreased ability to eliminate body heat as a result of excessive clothing covering the infant.² Although the relationship between climatic temperature and SIDS incidence has been examined carefully, the studies were performed predominantly in nations without extreme temperature variations, such as Great Britain, Tasmania, and New Zealand.²⁻⁵ Such research has consistently associated increased SIDS incidence with colder outdoor temperatures. It has been suggested that some parents may respond to such cold waves by overwrapping infants or excessively increasing room temperatures.³ In contrast, a cross-cultural study found comparatively weak relationships between climatic temperature and SIDS incidence in communities in the United States.⁶ Therefore, it is possible that seasonal overwrapping of infants may not be a major cause of SIDS in the United States.

Sections of the United States, particularly the Midwest, are subject to periodic heat waves, which can lead to severe epidemics of deaths attributable to exposure to excessive heat.⁷ Such conditions present an opportunity to examine the effect of increased environmental temperature on SIDS incidence. We reasoned that, if thermal stress increases the risk of SIDS, then an increase in the number of SIDS deaths should be observed when there is an increase in the number of heat-related deaths in the general population.

METHODS

Population

This study concentrated on the heat waves of the summer of 1980. In that year, excessive heat from all sources was listed as the underlying cause of death for a total of 1700 deaths across the United States,⁸ which was notably greater than the median of 274 deaths per year from 1979 to 1998 and was the largest number of heat-related deaths during that period.⁷ To avoid a false-negative finding attributable to seasonal variations in the SIDS rate, the period was restricted to May 1 through September 30, 1980. All deaths attributable to SIDS and exposure to excessive heat resulting from weather conditions in each of the counties of the 4 states with the greatest numbers of heat-related deaths, namely, Arkansas, Georgia, Kansas, and Missouri, during this period were considered.

Data Sources

SIDS mortality data were defined as all records from the Mortality Detail Files, 1979–1980,¹⁰ that fit the aforementioned criteria and for which an International Classification of Diseases, 9th revision, code of 900.0 was assigned for the underlying cause of death. Heat-mortality data were defined as all records for which an International Classification of Diseases, 9th revision, code of 900.0 (“death due to excessive heat due to weather conditions”) was assigned. Population data for each county were obtained from the Census of Population and Housing, 1980 (United States): County Population by Age, Sex, Race, and Spanish Origin,¹¹ with the populations being defined as the entire population of the county for heat-related deaths and the population <1 year of age for SIDS deaths.

Data on the maximal daily temperature (\( T_{\text{max}} \)) and minimal daily temperature were determined for each county of the 4 states.
studied from the *Record of Climatological Observations.* Temperatures for the counties were recorded from a single weather station within each county. For counties that were missing data, temperature data were calculated as the average of values from 2 weather stations in 2 bordering counties. If original data were missing for all bordering counties, then data for the next closest counties were used. Average daily temperatures ($T_{avg}$) in each location then were calculated as the average of $T_{max}$ and the minimal daily temperature. All temperature data were rounded to the nearest integer.

**Statistical Analyses**

For each 5°F temperature range (for example, 60–64°F [15.6–17.8°C]), the population of each county was multiplied by the number of days the county experienced that temperature range. These values then were summed for all counties, to yield the total population at risk for each temperature range. The total number of deaths that occurred in that temperature range then was divided by the total population at risk to yield the mortality rate for each temperature range.

$\chi^2$ tests for trend were performed to determine the association between the SIDS rate and the heat-related death rate for $T_{avg}$ and $T_{max}$. To determine the association between the SIDS rate and the heat-related death rate, Spearman’s rank correlation coefficient was used. A P value of < .05 was considered statistically significant. These analyses were repeated after recoding of any infant deaths attributable to exposure to excessive heat as deaths attributable to SIDS. In these cases, the population associated with heat-related deaths was equal to the entire population of the county minus the population < 1 year of age.

Because of the relatively few cases (111) of SIDS recorded during the study period, we used a simulation to calculate the empirical power to detect a linear increase in the SIDS rate with increasing temperature. With the available SIDS deaths, the power was calculated to be 0.78 if the SIDS rate at the highest temperature was 2.7 times that at the lowest temperature. The power was 0.70 if the SIDS rate at the highest temperature was 1.8 times that at the lowest temperature. If the SIDS rate at the highest temperature was 2.7 times that at the lowest temperature, the power was calculated to be 0.63 (Table 1).

**RESULTS**

Between May 1 and September 30, 1980, in the states of Arkansas, Georgia, Kansas, and Missouri, a total of 111 SIDS deaths and 402 deaths attributable to excessive heat resulting from weather conditions were recorded. $\chi^2$ tests demonstrated increased heat-related mortality rates with increasing $T_{max}$ values ($P < .0001$) but indicated no such relationship for SIDS rates ($P = .605$) (Fig 3). Heat-related mortality rates increased with $T_{avg}$ values ($P < .0001$), but again there was no association between SIDS rates and $T_{avg}$ ($P = .945$) (Fig 4). In addition, analysis of the recoded data with Spearman’s ranked correlation indicated no association for either $T_{max}$ ($P = .9572$) or $T_{avg}$ ($P = .4175$). Finally, we considered the possibility that there could be a threshold for heat stress, increasing SIDS risks at higher temperature ranges. Therefore, we analyzed the dichotomized $T_{avg}$ variable. The rate for $T_{avg}$ of < $85°F$ was 3.0 cases per 1 000 000 population, whereas the rate for ≥ $85°F$ was 3.4 cases per 1 000 000. This difference was not statistically significant ($P = .568$).

**DISCUSSION**

In this study, we failed to find a relationship between elevated environmental temperatures and the incidence of SIDS during a period in which deaths attributable to excessive environmental temperatures in the general population were increased to epidemic levels. Physiologic and epidemiologic research suggests increased risks attributable to heat stress among prone-sleeping infants, because their ability to eliminate heat may be reduced in this position. Therefore, US infants as a group should have been unusually susceptible to heat stress.

The power calculation indicated that the number of SIDS deaths studied was sufficient to allow detection of an increase in SIDS rates of 1.8-fold between the highest and lowest temperatures. Smaller increases would have been undetectable. Increases of this magnitude are relatively small, considering that the differences between summer and winter SIDS rates during the 1980s in New Zealand, for example, were five- to sevenfold. Therefore, if the large seasonal variations in SIDS rates observed in the past were primarily attributable to heat stress, as suggested previously, then we would expect to have observed changes of this magnitude among heat-stressed infants. Furthermore, the present study indicated that heat-related death rates in the general population we studied were much higher at the highest temperature, compared with the lowest. If SIDS infants are to be considered at high risk for death attributable to heat stress, then an increase in death rates of a magnitude comparable to that for deaths attributable to heat stress in the general population would be expected. The statistical power was more than adequate to detect such an increase.

These findings suggest that the heat stress theory...
Fig 1. SIDS rates versus heat-related mortality rates for $T_{\text{max}}$. Error bars indicate ±SEM. It should be noted that, because of the repeated exposure of the populations in this study to the various temperature ranges, resulting in an extremely large population at risk, these daily rates per temperature range have little relationship to annual rates.

Fig 2. SIDS rates versus heat-related mortality rates for $T_{\text{avg}}$. 

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for SIDS should be reevaluated. There are confounding factors in interpreting the insulation data, arguably the most important findings supporting this theory. Materials that are high in thermal insulation value (as measured in tog units) also contribute to death through other mechanisms such as rebreathing.
of expired air causing asphyxiation. Studies have found that infant bedding items such as quilts, comforters, and blankets have a high potential for causing rebreathing. Fleming et al found that infants who died as a result of SIDS were wrapped in clothing or covered by bedding with a significantly greater tog value than were control infants. The increased SIDS risk associated with increased body insulation was highest among prone-sleeping infants. Because prone sleeping and increased insulation can interact to reduce an infant’s ability to eliminate heat, the investigators attributed the increased SIDS risk in the prone position to thermal stress. However, those investigators did not include bulky bedding items (such as duvets, quilts, or comforters) covering infants in their multivariate analysis. These items not only are very substantial components of the tog estimate but also place infants at increased risk for rebreathing. In fact, Ponsonby et al found that the increase in the risk of SIDS resulting from quilt or comforter use was evident only for supine-sleeping infants, suggesting that heat stress may not be the primary mechanism for the increased risk associated with these items.

The thermal stress theory of SIDS originated with premortem and postmortem observations of elevated body temperature or excessive sweating among infants with SIDS. The observations of overwrapping or elevated room temperature on the day of death provided additional support for this theory. It is important to note that the thermal stress theory draws a sharp distinction between SIDS resulting from heat stress and death attributable to extreme hyperthermia leading to heat stroke. Therefore, it has been suggested that thermal stress, with or without moderate increases in body temperature, can result in fatal disruption of vital systems among infants predisposed to SIDS. It has been proposed that congenital or acquired anatomic abnormalities in brainstem centers regulating respiratory or cardiovascular stability could represent the underlying defects that render SIDS infants susceptible to death resulting from heat stress.

Our reasoning in assuming that a substantial number of infants at risk for SIDS experienced significant heat stress is as follows. First, several factors indicate that young infants can be expected to experience more heat stress than older children or adults when environmental temperatures increase. The full spectrum of heat-related illnesses among infants must be considered, because more cases of nonlethal environmental heat-related illnesses (eg, heat prostration and water-deficiency heat exhaustion) than actual heat stroke occur among infants. The actual death rates for infants, compared with the elderly, during heat waves are less likely to reflect the true incidence of heat stress, because death seems to be less frequent among infants diagnosed with heat stroke than in the older population. The risk for all heat-related illnesses is thought to be increased among infants, compared with older children or adults. This is attributed to increased metabolic rate, increased surface area/body mass ratio, and increased tendency for emesis as well as refusal to accept orally administered fluids when overheated, which make infants more susceptible to heat stress and related dehydration. Therefore, for any given thermal exposure, infants may be expected to experience symptoms of heat stress more rapidly than other age groups.

Second, it might be expected that most parents would pay particular attention to keeping infants cool in hot weather. Evidence suggests that this is not the case. A study of heat-stressed infants hospitalized for treatment found that 68% of the infants’ caretakers were unaware of the need for increased fluid intake by their infants during hot weather and had seriously underestimated the seriousness of the infants’ initial symptoms. Other studies also indicated a lack of parental perception regarding infant heat stress. It has been reported that the practice of leaving sleeping infants in parked cars while parents run errands is a frequent cause of heat-related morbidity and death. It is noteworthy that 24% of mothers admit to this practice, again indicating serious underestimation by caretakers of their infants’ ability to cope with increased environmental temperatures.

Third, it is unlikely that many infants kept indoors during the 1980 heat wave received adequate protection as a result of access to home air conditioning or fans. A government-sponsored study of the availability of home air conditioners in the 4 states we studied, spanning 1980 to 1984, indicated that most households (51%) had inadequate access to air conditioners sufficient to reduce the risk of heat stroke. Even more relevant were the findings of a 1980 study that found that >80% of the households in the lower one-third socioeconomic group did not have air conditioning. Fans in the home were not found to decrease the risk of heat stroke. Therefore, it can be inferred that the majority of the infants, including those at highest risk for SIDS (ie, infants in low-income households), did not have access to environments adequate to eliminate heat stress.

Fourth, any consideration of the possible relationship of heat stress to SIDS in the United States must also take into account the fact that the population at highest risk for one is also that at highest risk for the other. It has been well established in several studies, including an analysis of the 1980 heat wave in Missouri, that low-income, urban, and African American populations are at highest risk for heat stroke because of the combination of an urban “heat-island” effect that increases both indoor and outdoor temperatures, substandard housing, poor ventilation, and decreased access to air conditioning. When infant physiologic factors, the failure of caregivers to perform appropriate interventions, and populations at greatest risk for both heat stress and SIDS are taken into account, it might reasonably be predicted that, if heat stress per se is a significant cause of SIDS, SIDS incidence should increase significantly during summer heat waves, when the number of deaths attributable to overheating in the general population increases.

Heat stress can be defined as a deviation from a
thermoneutral environment that requires increased use of the body’s mechanisms for cooling. In contrast, heat stroke, defined as a body temperature of \(\geq 105^\circ F (\geq 40.6^\circ C)\) and altered mental status, leads to cardiac arrhythmias, seizures, or hypovolemic shock, with tachycardia and eventual circulatory collapse. Depression of central nervous system thermoregulatory mechanisms is thought to be involved in such deaths. Because it is difficult to diagnose heat stroke after death among both infants and adults, because of the lack of specific pathologic findings, the most important information for the post-mortem diagnosis comes from body-temperature measurements and death-scene investigations. In this study, there were 3 infant deaths among the 402 deaths attributable to excessive heat exposure. Two of those deaths occurred on consecutive days in Jackson County, Missouri (which includes Kansas City). During the summer heat wave, 5 deaths diagnosed as SIDS occurred in Jackson County, which suggests that the medical examiner for that jurisdiction was accustomed to distinguishing infant deaths attributable to SIDS from heat stroke deaths. Even if all 3 heat-related deaths had been attributed to SIDS, the relationship of SIDS incidence to environmental temperature would have remained insignificant.

Finally, it must be noted that either genetic or acquired abnormalities involving sweat gland function or the thermoregulatory centers of the brain may render individuals vulnerable to heat stress, which can be fatal. It is also clear that the numbers of deaths primarily attributable to respiratory or cardiovascular disease increase during periods of environmental heat stress. This has been attributed to the fact that accommodation to increased environmental temperatures results in increased demands on the heart and lungs. Accordingly, it would be expected that, if infants with SIDS have congenital or acquired abnormalities of thermoregulation or of respiratory or cardiovascular function, then the numbers of SIDS deaths would increase during periods of dangerously elevated environmental temperatures. This did not occur; therefore, it seems unlikely that a congenital or acquired defect in thermoregulation is an underlying cause of a significant number of infant deaths attributed to SIDS.

The present findings do not exclude the possibility that a combination of factors, including increased body insulation or febrile illnesses associated with the winter months, are causal in certain SIDS cases. More research is needed on potential interactions between these factors. However, given that the large majority of infants were placed prone for sleep during the period of this study, it seems that heat stress produced by increased environmental temperatures and prone positions alone does not have a substantial causal role in SIDS.

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Neither the collectors of the original data nor the Consortium bear any responsibility for the analyses or interpretations presented here.

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