Sudden Infant Death Syndrome and Air Pollution: A Selected Annotated Bibliography


BACKGROUND: There is growing concern that moderate levels of outdoor air pollution may be associated with infant mortality, representing substantial loss of life-years. To date, there has been no investigation of the effects of outdoor pollution on infant mortality in the UK. METHODS: Daily time-series data of air pollution and all infant deaths between 1990 and 2000 in 10 major cities of England: Birmingham, Bristol, Leeds, Liverpool, London, Manchester, Middlesbrough, Newcastle, Nottingham and Sheffield, were analysed. City-specific estimates were pooled across cities in a fixed-effects meta-regression to provide a mean estimate. RESULTS: Few associations were observed between infant deaths and most pollutants studied. The exception was sulphur dioxide (SO2), of which a 10 mug/m(3) increase was associated with a RR of 1.02 (95% CI 1.01 to 1.04) in all infant deaths. The effect was present in both neonatal and postneonatal deaths. CONCLUSIONS: Continuing reductions in SO2 levels in the UK may yield additional health benefits for infants.

Full-text available at: [http://jech.bmj.com/](http://jech.bmj.com/) (not a U.S. government site)

Anderson ME, Bogdan GM. *Environments, indoor air quality, and children.* Pediatr Clin North Am. 2007 Apr;54(2):295-307. This article addresses air-quality science in the indoor environments in which children and adolescents find themselves, including the home, the school, and other environments such as work and recreational situations. The home arena is covered extensively, presenting an analysis of the usual exposures such as environmental tobacco smoke and bioaerosols and also touching on discrete issues such as sudden infant death syndrome, carbon monoxide, and public housing. Recreation and work environments are covered as well.

Full text available: [www.pediatric.theclinics.com](http://www.pediatric.theclinics.com) (not a U.S. Government site)


7/25/07
Objective: We evaluated the influence of outdoor air pollution on infant death in the South Coast Air Basin of California, an area characterized by some of the worst air quality in the United States. Methods: Linking birth and death certificates for infants who died between 1989 and 2000, we identified all infant deaths, matched 10 living control subjects to each case subject, and assigned the nearest air monitoring station to each birth address. For all subjects, we calculated average carbon monoxide, nitrogen dioxide, ozone, and particulate matter < 10 microm in aerodynamic diameter exposures experienced during the 2-week, 1-month, 2-month, and 6-month periods before a case subject's death. Results: The risk of respiratory death increased from 20% to 36% per 1-ppm increase in average carbon monoxide levels 2 weeks before death in early infancy (age: 28 days to 3 months). We also estimated 7% to 12% risk increases for respiratory deaths per 10-microg/m3 increase in particulate matter < 10 microm in aerodynamic diameter exposure experienced 2 weeks before death for infants 4 to 12 months of age. Risk of respiratory death more than doubled for infants 7 to 12 months of age who were exposed to high average levels of particulates in the previous 6 months. Furthermore, the risk of dying as a result of sudden infant death syndrome increased 15% to 19% per 1-part per hundred million increase in average nitrogen dioxide levels 2 months before death. Low birth weight and preterm infants seemed to be more susceptible to air pollution-related death resulting from these causes; however, we lacked statistical power to confirm this heterogeneity with formal testing. Conclusions: Our results add to the growing body of literature implicating air pollution in infant death from respiratory causes and sudden infant death syndrome and provide additional information for future risk assessment.

Full-text available at: http://www.pediatrics.org (not a U.S. Government site)

Woodruff TJ, Parker JD, Schoendorf KC. Fine particulate matter (PM2.5) air pollution and selected causes of postneonatal infant mortality in California. Environ Health Perspect. 2006 May; 114(5):786-90.

Studies suggest that airborne particulate matter (PM) may be associated with postneonatal infant mortality, particularly with respiratory causes and sudden infant death syndrome (SIDS). To further explore this issue, we examined the relationship between long-term exposure to fine PM air pollution and postneonatal infant mortality in California. We linked monitoring data for PM<or=2.5 microm in aerodynamic diameter (PM2.5) to infants born in California in 1999 and 2000 using maternal addresses for mothers who lived within 5 miles of a PM2.5 monitor. We matched each postneonatal infant death to four infants surviving to 1 year of age, by birth weight category and date of birth (within 2 weeks). For each matched set, we calculated exposure as the average PM2.5 concentration over the period of life for the infant who died. We used conditional logistic regression to estimate the odds of postneonatal all-cause, respiratory-related, SIDS, and external-cause (a control category) mortality by exposure to PM2.5, controlling for the matched sets and maternal demographic factors. We matched 788 postneonatal infant deaths to 3,089 infant survivors, with 51 and 120 postneonatal deaths
due to respiratory causes and SIDS, respectively. We found an adjusted odds ratio for a 10-microg/m3 increase in PM2.5 of 1.07 [95% confidence interval (CI), 0.93-1.24] for overall postneonatal mortality, 2.13 (95% CI, 1.12-4.05) for respiratory-related postneonatal mortality, 0.82 (95% CI, 0.55-1.23) for SIDS, and 0.83 (95% CI, 0.50-1.39) for external causes. The California findings add further evidence of a PM air pollution effect on respiratory-related postneonatal infant mortality.


The toxicology of antimony and its compounds is known from three sources: its medicinal use over centuries, studies of process workers in more recent times, and more recent still, studies of its presence in modern city environments and in domestic environments. Gross exposure to antimony compounds over long periods, usually the sulfide (SbS3) or the oxide (Sb2O3) has occurred in antimony miners and in antimony process workers. There have been relatively few of these, and few studies of possible symptoms have been made. Antimony sulfide imported from, at different times, China, South Africa, and South America was processed in the North-East of England from about 1870 to 2003. The process workers in North-East England have been studied at different times, notably by Sir Thomas Oliver in 1933, and by the Newcastle upon Tyne University Department of Occupational Medicine on later occasions. Studies which have been made of the working environment, and in particular of the risk of lung cancer in process workers, have underlined the high levels of exposure to antimony compounds and to other toxic materials. However, the working conditions in antimony processing have improved markedly over the last 30 years, and the workforce had been much reduced in numbers following automation of the process. Prior to the cessation of the industry in the UK it had become a 'white coat' operation with relatively few people exposed to high concentrations of antimony. Antimony, which is normally present in domestic environments, has also been studied as a possible cause of cot death syndrome (SIDS) but extensive investigations have not confirmed this. The full importance of environmental antimony has still to be determined, and evidence of specific effects has not yet been presented.

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Environmental tobacco smoke (ETS), containing the developmental neurotoxicant,
nicotine, is a prevalent component of indoor air pollution. Despite a strong association with active maternal smoking and sudden infant death syndrome (SIDS), information on the risk of SIDS due to prenatal and postnatal ETS exposure is relatively inconsistent. This literature review begins with a discussion and critique of existing epidemiologic data pertaining to ETS and SIDS. It then explores the biologic plausibility of this association, with comparison of the known association between active maternal smoking and SIDS, by examining metabolic and placental transfer issues associated with nicotine, and the biologic responses and mechanisms that may follow exposure to nicotine. Evidence indicates that prenatal and postnatal exposures to nicotine do occur from ETS exposure, but that the level of exposure is often substantially less than levels induced by active maternal smoking. Nicotine also has the capacity to concentrate in the fetus, regardless of exposure source. Experimental animal studies show that various doses of nicotine are capable of affecting a neonate's response to hypoxic conditions, a process thought to be related to SIDS outcomes. Mechanisms contributing to deficient hypoxia response include the ability of nicotine to act as a cholinergic stimulant through nicotinic acetylcholine receptor (nAChR) binding. The need for future research to investigate nicotine exposure and effects from non-maternal tobacco smoke sources in mid to late gestation is emphasized, along with a need to discourage smoking around both pregnant women and infants.


Nevas M, Lindstrom M, Virtanen A, Hielm S, Kuusi M, Arnon SS, Vuor Korkeala H. Infant botulism acquired from household dust presenting as sudden infant death syndrome

Clostridium botulinum type B was detected by multiplex PCR in the intestinal contents of a suddenly deceased 11-week-old infant and in vacuum cleaner dust from the patient's household. C. botulinum was also isolated from the deceased infant's intestinal contents and from the household dust. The genetic similarity of the two isolates was demonstrated by pulsed-field gel electrophoresis and randomly amplified polymorphic DNA analysis, thereby confirming that dust may act as a vehicle for infant botulism that results in sudden death.


Centers for Disease Control and Prevention (CDC). Indoor air quality in hospitality venues before and after implementation of a clean indoor air law—Western New York, 2003.

Secondhand smoke (SHS) contains more than 50 carcinogens. SHS exposure is responsible for an estimated 3,000 lung cancer deaths and more than 35,000 coronary heart disease deaths among never smokers in the United States each year, and for lower respiratory infections, asthma, sudden infant death syndrome, and chronic ear infections.
among children. Even short-term exposures to SHS, such as those that might be experienced by a patron in a restaurant or bar that allows smoking, can increase the risk of experiencing an acute cardiovascular event. Although population-based data indicate declining SHS exposure in the United States over time, SHS exposure remains a common but preventable public health hazard. Policies requiring smoke-free environments are the most effective method of reducing SHS exposure. Effective July 24, 2003, New York implemented a comprehensive state law requiring almost all indoor workplaces and public places (e.g., restaurants, bars, and other hospitality venues) to be smoke-free. This report describes an assessment of changes in indoor air quality that occurred in 20 hospitality venues in western New York where smoking or indirect SHS exposure from an adjoining room was observed at baseline. The findings indicate that, on average, levels of respirable suspended particles (RSPs), an accepted marker for SHS levels, decreased 84% in these venues after the law took effect. Comprehensive clean indoor air policies can rapidly and effectively reduce SHS exposure in hospitality venues.

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There is now substantial evidence that both short- and long-term increases in ambient air pollution are associated with increased mortality and morbidity in adults and children. Children's health is particularly vulnerable to environmental pollution, and infant mortality is still a major contributor to childhood mortality. In this systematic review we summarize and evaluate the current level of epidemiologic evidence of an association between particulate air pollution and infant mortality. We identified relevant publications using database searches with a comprehensive list of search terms and other established search methods. We included articles in the review according to specified inclusion criteria. Fifteen studies met our inclusion criteria. Evidence of an association between particulate air pollution and infant mortality in general was inconsistent, being reported from locations with largely comparable pollution levels. There was some evidence that the strength of association with particulate matter differed by subgroups of infant mortality. It was more consistent for postneonatal mortality due to respiratory causes and sudden infant death syndrome. Differential findings for various mortality subgroups within studies suggest a stronger association of particulate air pollution with some causes of infant death. Research is needed to confirm and clarify these links, using the most appropriate methodologies for exposure assessment and control of confounders. Key words: infant mortality, particulate air pollution, postneonatal respiratory mortality, sudden infant death syndrome, and systematic review.

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Although the rate of sudden infant death syndrome (SIDS) has been reduced with the 'Back to Sleep' campaign, SIDS is still a common cause of death in infancy. A range of environmental factors may interact to contribute to the adverse health conditions conducive to SIDS. Nine studies have evaluated the association between exposure to air pollution and the incidence of SIDS. The available evidence is inadequate to come to any conclusion about a relationship between air pollution and SIDS, although the body of evidence appears to suggest that air pollution (especially particles and some gaseous pollutants) may play a certain role in the occurrence of SIDS. We suggest that future studies should focus on the research design, role of indoor air quality and the effect of smaller particles, particularly those in the ultrafine range.

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Chong et al. examined risk factors for sudden infant death syndrome (SIDS) before and after the start of the Swedish campaign to reduce the risk of SIDS. They found that maternal smoking was the strongest risk factor for SIDS in the post-campaign compared to the pre-campaign period. Conclusion: After successful results of the SIDS campaigns to prevent prone sleeping, strong efforts need to be undertaken to eliminate maternal smoking during pregnancy altogether without replacing cigarette smoking with other nicotine delivery devices such as snuff, gum or patches.

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Background: Sudden infant death syndrome (SIDS) affects 1 in 1000 live births and is the most common cause of infant death after the perinatal period. Objective: To determine the influence of air pollution on the incidence of SIDS. Methods: Time-series analyses were performed to compare the daily mortality rates for SIDS and the daily air pollution concentrations in each of 12 Canadian cities during the period of 1984-1999. Serial autocorrelation was controlled for by city, and then the city-specific estimates were pooled. Increased daily rates of SIDS were associated with increases, on the previous day, in the levels of sulfur dioxide (SO2), nitrogen dioxide (NO2), and carbon monoxide but not ozone or fine particles measured every sixth day. Effects persisted despite adjustments for season alone or the combination of daily mean temperature, relative humidity, and changes in barometric pressure for NO2 and SO2 but not carbon.
monoxide. Results: Increases in both SO2 and NO2, equivalent to their interquartile ranges, were associated with a 17.72% increase in SIDS incidence. Conclusion: Ambient SO2 and NO2 may be important risk factors for SIDS


Air Pollution attributable Postneonatal Infant Mortality in U.S. metropolitan areas: A risk assessment study.

Background: The impact of outdoor air pollution on infant mortality has not been quantified. Methods: Based on exposure-response functions from a U.S. cohort study, we assessed the attributable risk of postneonatal infant mortality in 23 U.S. metropolitan areas related to particulate matter <10 mum in diameter (PM10) as a surrogate of total air pollution. Results: The estimated proportion of all cause mortality, sudden infant death syndrome (normal birth weight infants only) and respiratory disease mortality (normal birth weight) attributable to PM10 above a chosen reference value of 12.0 mug/m3 PM10 was 6% (95% confidence interval 3-11%), 16% (95% confidence interval 9-23%) and 24% (95% confidence interval 7-44%), respectively. The expected number of infant deaths per year in the selected areas was 106 (95% confidence interval 53-185), 79 (95% confidence interval 46-111) and 15 (95% confidence interval 5-27), respectively. Approximately 75% of cases were from areas where the current levels are at or below the new U.S. PM2.5 standard of 15 mug/m3 (equivalent to 25 mug/m3 PM10). In a country where infant mortality rates and air pollution levels are relatively low, ambient air pollution as measured by particulate matter contributes to a substantial fraction of infant death, especially for those due to sudden infant death syndrome and respiratory disease. Even if all counties would comply with the new PM2.5 standard, the majority of the estimated burden would remain. Conclusion: Given the inherent limitations of risk assessments, further studies are needed to support and quantify the relationship between infant mortality and air pollution.

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Ferng, SF, Lee LW.
Indoor air quality assessment of daycare facilities with carbon dioxide, temperature, and humidity as indicators.

Poor indoor air quality (IAQ) in daycare facilities affects both attending children and care providers. Incident rates of upper-respiratory-tract infections have been reported to be higher in children who attend daycare. Excessive carbon dioxide (CO2) exposure can cause several health effects and even sudden infant death. For this study, 26 facilities were randomly selected in a Midwestern county of the United States. CO2, room temperature, and relative humidity were used as indicators for IAQ and comfort levels.
These IAQ parameters were continuously monitored for eight hours at each facility by a direct-reading instrument that was calibrated before each measurement. More than 50 percent of the facilities had an average CO2 level over the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) standard of 1,000 parts per million (ppm). For temperature and relative humidity, respectively, 42.3 percent and 15.4 percent of facilities were outside of the ASHRAE-recommended comfort zones. The nap-time average CO2 level was about 117 ppm higher than the non-nap-time level. The increment of the nap-time CO2 level in the sleeping-only room over the level in multipurpose rooms was statistically significant (p < .05). According to stepwise multiple regression analysis, nap-time CO2 level was predicted by CO2 level before occupancy, nap-time average temperature, carbon monoxide, and child density (R2 = .83). It is recommended that an appropriate IAQ standard for daycare facilities be established and that children should not be placed in a completely isolated room during nap time.

Incidence and geographical distribution of Sudden Infant Death Syndrome in relation to content of nitrate in drinking water and groundwater levels.  

Previous studies indicate that the enteral bacterial urease is inhibited in victims of sudden infant death syndrome (SIDS). One possible inhibitor of this bacterial activity is nitrate. If ambient pollution by nitrate is involved in the etiology of SIDS, only a fraction of the nitrate concentration not infrequently found in drinking water would be enough for this inhibition. Occurrence of SIDS (n=636) in Sweden during the period 1990 through 1996 were analyzed regarding geographical and seasonal distribution in relation to the nitrate concentration in drinking water and changes in the groundwater level. Both the birth rate and the incidence of SIDS decreased during the study period. One quarter of the municipalities constituting 11 percent of the population had no cases, the maximum incidence when the rest of the country had its lowest incidence, and the occurrence of individual deaths was associated with the recharge of groundwater, which increases its nitrate content. The local incidence of SIDS was correlated (rs=0.34-0.87) to maximally recorded concentrations of nitrate in drinking water. The seasonal distribution of SIDS was widely different from the south to the north of the country and seems to be associated with differences in the groundwater level changes subsequent to precipitation, frost penetration, and melting of snow. Use of drinking water with high peak concentrations or great variations in nitrate concentration was correlated to the incidence of SIDS.

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Tutka P, Wielosz M, Zatonski W.  
Exposure to environmental tobacco smoke and children health.  

This paper reviews the investigations of the effects of pre- and/or postnatal exposure to environmental tobacco smoke (ETS) on children health reported in the literature. The evidence from epidemiological studies demonstrates that children's exposure to ETS is a
risk factor for a variety of diseases, including respiratory disorders and middle ear disease. However, the current research base on the ETS-associated risks is still inadequate to fully support strategies, programs and policy development in this area. For example, it is not definitively determined what methods should be used for assessing ETS exposure and predicting potential health risks of exposed children. Based on the available data, we tried to find out which methods seem to be most desirable for quantifying ETS exposure in children. It is our opinion that among all biomarkers, the measurements of blood, saliva or urinary cotinine and hair nicotine are, as for today, the most specific and sensitive methods for an objective assessment of ETS exposure in children. A combination of the measurement of body fluids cotinine and hair nicotine with the questionnaire and interview-derived information seems to be the optimal method for assessing ETS exposure in children.

Quinn JB.  
**Baby's bedding: Is it creating toxic nerve gases?**  
Midwifery Today Int Midwife 2002 Spr; 61:21-22.

The author discusses the research done by Barry Richardson in Great Britain and T.J. Sprott in New Zealand on chemicals used in the manufacture of baby mattresses. There are three substances of concern: phosphorus, arsenic, and antimony. Their theory is that a common household fungus, Scopularis brevicaulis, establishes itself in the mattresses and by consuming the three substances creates three nerve gases: phosphine, arsine, and stibine. When the baby sleeps on the mattress, warming it to body temperature, the gases are released from the mattress, and the baby breathes in these gases. It is suggested that to prevent this from occurring, mattresses should be wrapped in a gas-impermeable plastic, and only cotton bedding should be used.

Lipfert FW, Zhang J, Wyzga RE.  
**Infant mortality and air pollution: A comprehensive analysis of U.S. data for 1990.**  

This paper uses U.S. linked birth and death records to explore associations between infant mortality and environmental factors, based on spatial relationships. The analysis considers a range of infant mortality end points, regression models, and environmental and socioeconomic variables. The basic analysis involves logistic regression modeling of individuals; the cohort comprises all infants born in the United States in 1990 for whom the required data are available from the matched birth and death records. These individual data include sex, race, month of birth, and birth weight of the infant, and personal data on the mother, including age, adequacy of prenatal care, and smoking and education in most instances. Ecological variables from Census and other sources are matched on the county of usual residence and include ambient air quality, elevation above sea level, climate, number of physicians per capita, median income, racial and ethnic distribution, unemployment, and population density. The air quality variables considered were 1990 annual averages of PM10, CO, SO2, SO4(2-), and "non-sulfate PM10" (NSPM10--obtained by subtracting the estimated SO4(2-) mass from PM10). Because all variables were not available for all counties (especially maternal smoking), it was necessary to
consider various subsets of the total cohort. We examined all infant deaths and deaths by age (neonatal and postneonatal), by birth weight (normal and low [< 2500 g]), and by specific causes within these categories. Special attention was given to sudden infant death syndrome (SIDS). For comparable modeling assumptions, the results for PM10 agreed with previously published estimates; however, the associations with PM10 were not specific to probable exposures or causes of death and were not robust to changes in the model and/or the locations considered. Significant negative mortality associations were found for SO4 (2-). There was no indication of a role for outdoor PM2.5, but possible contributions from indoor air pollution sources cannot be ruled out, given higher SIDS rates in winter, in the north and west, and outside of large cities.

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