Air Pollution and Sudden Infant Death Syndrome

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ABSTRACT. *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 (SO_2), nitrogen dioxide (NO_2), 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 NO_2 and SO_2 but not carbon monoxide.**

Results. **Increases in both SO_2 and NO_2, equivalent to their interquartile ranges, were associated with a 17.72% increase in SIDS incidence.**

Conclusion. **Ambient SO_2 and NO_2 may be important risk factors for SIDS.** *Pediatrics* **2004;113:e628–e631. URL: http://www.pediatrics.org/cgi/content/full/113/6/e628;** ***air pollution, epidemiology, SIDS.***

ABBREVIATIONS. SIDS, sudden infant death syndrome; PM_{10}, particulate matter with a median aerodynamic diameter of <10 μm.

The rate of sudden infant death syndrome (SIDS) in the mid-1990s was ~1 in 1000 live births.[1,2] In Canada, the incidence of SIDS decreased from 1.09 in 1000 live births in 1984 to 0.49 in 1000 live births in 1999. Many risk factors have been identified and publicized, including the winter season,[2,3] lower social status of parents,[4] environmental tobacco smoke,[1,4] and the prone position.[2] The role of ambient air pollution has received little attention, is controversial,[3,5,6] and is not often considered in epidemiologic studies and reviews of SIDS.[1,2,4] One reason why the influence of air pollution on SIDS has received relatively little attention is related to the challenging methodologic issues involved. Similar to the studies that documented the currently accepted risk factors for SIDS, air pollution studies must be observational and not interventional. Large populations must be studied because of the relatively infrequent event rate and the expected modest effect size of air pollution. Administrative database studies comparing regional mortality statistics and regional air pollution concentrations and case-control designs are efficient for the study of infrequent events but are prone to confounding. If the rates of SIDS in areas with high levels of pollution were compared with the rates in areas with lower levels, then it would be necessary to control for the many other potential personal risk factors for SIDS that may differ according to region to avoid potential confounding.

To investigate the influence of ambient air pollution on SIDS, we conducted a time-series analysis comparing daily rates of SIDS and daily concentrations of air pollution during a 16-year period in Canada. A large, unbiased population sample was studied. Social status and the many other risk factors mentioned above required no adjustment, because we compared mortality rates on higher– and lower–air pollution days for the same population. To bias the results, a variable would need to be associated with daily changes in air pollution and also be a risk factor for SIDS, making confounding unlikely.

METHODS

The Canadian cities studied were Halifax, Saint John, Quebec, Montreal, Ottawa, Toronto, Hamilton, Windsor, Winnipeg, Edmonton, Calgary, and Vancouver. The total population for these 12 cities is 10 310 309 (1996 Census). These cities were chosen on the basis of the availability of air pollution data. SIDS data were extracted from Statistics Canada Vital Statistics, which records all births and deaths in Canada. SIDS is defined as a sudden, unexplained death of a child <1 year of age for which a clinical investigation and autopsy fail to reveal a cause of death. The offices of the Chief Coroner/Chief Medical Examiner must investigate every case. Autopsies have been mandatory since the 1990s, but whether this was the case in all jurisdictions in the 1980s could not be determined. Data were available for the period of January 1, 1984, to December 31, 1999.

Air pollution data were obtained from the National Air Pollution Surveillance system for carbon monoxide (CO), nitrogen dioxide (NO_2), ozone, sulfur dioxide (SO_2), and particulate matter with a median aerodynamic diameter of <10 μm (PM_{10}) or <2.5 μm. Daily data were available for pollutant gases, whereas particulate matter levels were measured every sixth day. When data were available from >1 monitoring site, they were averaged. Daily weather data obtained from the Environment Canada meteorology archive included 24-hour mean temperatures and relative humidity levels and changes in barometric pressure between 8 AM on the day of observation and 8 AM the preceding day.

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Received for publication Aug 29, 2003; accepted Jan 12, 2004.

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Daily variations in the number of SIDS cases were correlated with daily variations in ambient concentrations of air pollutants by using a random-effects regression model for count data.[7] A linear association between air pollution and the incidence of SIDS was assumed on the logarithmic scale, with the association varying at random among cities. The SIDS frequency is known to vary with time, with higher death rates in winter than in summer. Air pollution also varies seasonally. To adjust for temporal trends that might confound the SIDS-air pollution association, we used piecewise constant functions in time that varied by 3, 6, or 12 months throughout the study period. For each city, we selected the temporal model that maximized the evidence that the model residuals did not display any type of structure, including serial correlation, using Bartlett's test[8] for each city separately. Time lags of 0 to 5 days were examined in addition to multiple-day lags of 2 to 6 days. The time-lagged association between air pollution and SIDS incidence with the largest ratio of effect estimate to its SE (*t* ratio), positive or negative, was selected for each city. The results for each city were pooled, and the estimates of the pooled effect size and the SE were calculated by using a fixed- or random-effects model. A random-effects model was used whenever the between-city variance was >0, even if the heterogeneity among effect-size estimates for individual cities was not statistically significant. The presence of statistical heterogeneity in effect size between cities was assessed by using the *Q* statistic.[9] Several analyses were conducted to examine the sensitivity of the SIDS-air pollution association to a number of other variables including weather factors (daily mean temperature, daily mean relative humidity, and maximum change in barometric pressure, all measured on the day of death), length of time-period adjustment (all cities used 3 months), seasonal indicator variables, and size-fractionated particulate mass. Pooled estimates were expressed as the percentage increase in SIDS incidence associated with an increase in the concentration of air pollutants equivalent to the interquartile range among all cities.

RESULTS

The number of cases of SIDS in the 16 years was 1556, varying by city from 0 to 34 cases annually (Table 1). The 24-hour average pollutant concentrations, averaged for the period of observation (from 1984 to 1999), are presented in Table 2. Air-quality measures improved somewhat between 1984 and 1999, with the exception of ozone. The concentrations of air pollutants in 1984 and 1999 were as follows: 31 and 34 ppb for ozone, 23 and 19 ppb for NO_2, 7 and 5 ppb for SO_2, 1.1 and 0.7 ppm for CO, and 30 and 21 $\mu g/m^3$ for PM_{10}, respectively.

On the basis of Bartlett's test, the time factor selected was 12 months for Toronto and Quebec and 6 months for all other cities except Edmonton, for which no time was used. On the basis of the *Q* statistic, heterogeneity among the 11 cities was not statistically significant for any of the pollutants (SO_2, $P = .41$; NO_2, $P = .67$; CO, $P = .45$).

With adjustment for time, the strongest association between SO_2 levels and SIDS incidence was found with the SO_2 concentration on the previous day (Table 3). There was an 8.49% increase (95% confidence interval: 2.15%–15.22%) in SIDS incidence for an interquartile increase in SO_2 levels ($P = .0079$). The strongest associations for NO_2 and CO were with the average levels in the 3 days preceding the death. For an interquartile increase in NO_2 levels, there was a 15.23% increase (95% confidence interval: 6.74%–24.39%) in SIDS incidence ($P = .003$); for CO, the increase was 10.73% (95% confidence interval: 5.02%–16.75%) ($P = .0002$). No significant effect was observed for ozone or particulate air pollution.

To assess the influence of the analytic techniques on the findings, the relationship between air pollution and SIDS incidence was tested with several different models (Table 4). CO effects were not robust. They remained significant when results were adjusted for each climate variable individually but not when all climate variables were entered simultaneously in the model and not when a 3-month factor or season was included in the model. In contrast, SO_2 and NO_2 effects remained significant in the model using a 3-month factor and also in the full model adjusting for time, temperature, relative humidity, and barometric pressure. There was no significant effect modification according to season for NO_2, CO, or SO_2.

The combined effects of pollutants, adjusted for time, are presented in Table 5. Ozone effects were negatively correlated with CO, NO_2, and SO_2 effects. Pearson correlations with NO_2 were 0.22 for SO_2 and 0.55 for CO. The correlation between SO_2 and CO effects was 0.21. All correlations were significant at $P < .0001$. SO_2 and NO_2 had independent effects, but the effects of CO disappeared when either SO_2 or NO_2 was considered.

DISCUSSION

In the largest cities in Canada, we found that SIDS was associated with air pollution, with the effects of SO_2 and NO_2 seeming to be independent of sociodemographic factors, temporal trends, and weather.

TABLE 1. Number of SIDS Cases in Canada, According to City and Year, 1984–1999

City	No. of Cases							
	1984–1985	1986–1987	1988–1989	1990–1991	1992–1993	1994–1995	1996–1997	1998–1999
Calgary	24	35	26	26	14	14	13	16
Edmonton	26	46	39	35	19	25	12	15
Halifax	8	10	10	7	4	1	4	4
Hamilton	10	14	11	4	10	6	7	4
Montreal	13	21	43	34	31	27	15	10
Ottawa/Hull	16	15	12	17	21	17	8	5
Quebec	4	6	6	7	5	7	6	5
Saint John	4	2	4	2	3	5	2	1
Toronto	51	52	59	24	24	21	19	16
Vancouver	33	58	55	46	38	23	8	9
Windsor	6	8	8	10	10	12	3	2
Winnipeg	11	15	9	13	12	10	10	9
Total	206	281	282	225	191	168	107	96

TABLE 2. Mean 24-Hour Concentrations and Interquartile Ranges of Air Pollutants, According to City, Averaged for the Period of Observation, (1984–1999)

City	Population	Mean 24-h Pollutant Concentration (Interquartile Range)						
		Ozone (ppb)	CO (ppm)	NO_2 (ppb)	SO_2 (ppb)	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$* ($\mu g/m^3$)	$PM_{2.5–10}$ ($\mu g/m^3$)
Calgary	768 082	33.45 (15.50)	1.02 (0.57)	25.81 (11.50)	3.42 (3.00)	25.37 (17.62)	10.42 (6.61)	14.94 (12.55)
Edmonton	616 306	31.37 (18.00)	1.12 (0.75)	24.70 (13.00)	2.47 (3.00)	23.84 (16.39)	10.31 (6.86)	14.21 (13.15)
Halifax	179 539	30.16 (13.00)	0.88 (0.50)	15.45 (11.50)	9.93 (7.83)	19.21 (13.00)	11.12 (7.45)	8.22 (5.87)
Hamilton	322 352	35.14 (21.75)	0.93 (0.40)	21.32 (9.50)	8.81 (6.75)	28.15 (22.21)	16.47 (13.76)	11.66 (8.44)
Montreal	1 775 846	28.59 (16.96)	0.65 (0.46)	21.76 (10.43)	5.81 (4.83)	27.73 (17.21)	15.11 (11.12)	12.73 (8.76)
Ottawa	938 745	29.09 (15.50)	0.81 (0.53)	19.63 (13.34)	4.21 (4.00)	19.60 (7.51)	11.36 (9.15)	8.76 (6.43)
Quebec	504 605	29.30 (15.00)	0.60 (0.50)	19.52 (14.00)	4.90 (5.67)	22.50 (14.96)	11.28 (8.01)	11.22 (8.89)
Saint John	72 494	34.95 (13.67)	0.60 (0.60)	9.56 (7.50)	8.53 (9.25)	13.51 (10.59)	8.07 (6.55)	5.46 (4.59)
Toronto	2 385 421	34.79 (19.74)	1.19 (0.50)	25.55 (10.45)	4.63 (4.25)	26.16 (17.16)	15.13 (12.36)	10.99 (6.97)
Vancouver	1 831 665	26.99 (14.77)	1.04 (0.54)	20.36 (7.54)	4.76 (3.25)	20.34 (12.88)	12.09 (8.14)	8.38 (6.29)
Windsor	197 694	36.87 (28.00)	0.78 (0.60)	25.47 (12.00)	7.69 (5.67)	29.61 (18.92)	16.67 (12.14)	12.94 (8.76)
Winnipeg	688 477	30.49 (16.00)	0.58 (0.30)	14.92 (9.50)	0.96 (1.50)	25.08 (18.22)	9.26 (5.63)	15.88 (14.47)
Total	10 281 226	31.77 (17.32)	0.85 (0.52)	20.34 (10.86)	5.51 (4.92)	23.43 (15.56)	12.27 (8.98)	11.28 (8.76)

* $PM_{2.5}$, particulate matter with a diameter of <2.5 μm.

TABLE 3. Increases in SIDS Incidence Associated With Increases in Levels of Individual Air Pollutants Equivalent to Their Interquartile Ranges, in Several Canadian Cities, 1984–1999*

Single-Pollutant Model	Metric	Estimate	*P* Value	Increase in SIDS Incidence (%)
CO	3-d average	0.1958	.0002	10.73
NO_2	3-d average	0.0131	.0003	15.23
SO_2	1-d lag	0.0166	.0079	8.49

* Values are adjusted for time.

The effects of CO disappeared when climate effects were considered, whereas the effects of ozone and particles were not significant. The latter finding might have been attributable in part to the reduction in observations, with measurements being available every sixth day.

Woodruff et al[10] studied the cross-sectional association between elevated PM_{10} levels and SIDS incidence among 4 million infants born between 1989 and 1991. The unit of observation was a metropolitan statistical area. Among normal birth weight infants, levels of air pollution in 86 metropolitan statistical areas were associated with the death rates in the metropolitan statistical areas, with an odds ratio of 1.26 (95% confidence interval: 1.14%–1.39) between the areas of highest and lowest exposure, estimated as the mean for the first 2 months of life (44.5 and 23.6 $\mu g/m^3$, respectively). The odds ratio was adjusted for maternal ethnicity, education, smoking during pregnancy, marital status, month of birth, and average ambient temperature during the first 2 months of life. SIDS rates were not adjusted for birth weight or income, and gaseous pollutants were not considered in the study. Another cross-sectional study, by Lipfert et al,[5] found an association between levels of particulates, but not gases, and SIDS incidence, with an unadjusted odds ratio of 1.42 (no confidence interval given) between metropolitan statistical areas with low versus high levels of PM_{10} pollution (27.5 and 50.9 $\mu g/m^3$, respectively). This association persisted after adjustment for several factors including gender, ethnic background, birth weight, mother's aid, adequacy of prenatal care, smoking, and education. The database cohort included all infants born in the United States in 1990. Air pollution exposure was estimated at the county level and included PM_{10}, CO, sulfate oxide, and sulfate. Positive associations were found also with nonsulfate PM_{10}, and negative mortality associations were observed for sulfates. SIDS incidences were higher in the winter, in the northern and western areas of the United States, and outside large cities. One earlier study, which lacked modern statistical techniques for time-series analyses, reported increased visibility and decreased PM_{10} levels before the occurrence of SIDS.[11] A second study found no increase in daily SO_2 levels on days of SIDS, compared with 1 and 2 weeks before and after,[12] whereas a third study reported a correlation between SIDS and elevated levels of gas pollutants (SO_2, NO_2, CO, and hydrocarbons) 7 weeks before the event.[13]

Lipfert et al,[5] in reviewing their results and those of others, cautioned against attributing differences in SIDS rates to air pollution when there may be other observable but as yet unexplained differences between regions that influence mortality rates. The present report makes a significant contribution by confirming an adverse effect of air pollution in a large population study using a study design that is not confounded by differences among populations or geographic areas (a potential weakness of previous studies that compared regions). One weakness of the present study is the lack of daily data on particulate material of <2.5 μm, which reduces the power to detect an effect. However, the observed SO_2 and NO_2 effects were unlikely to be confounded by particulate material, because no independent effect was observed. Another weakness is the lack of data on personal exposure to air pollution. However, personal exposure monitoring would necessitate a prospective study, which is not feasible given that the incidence of SIDs is only 1 per 1000 live births. Furthermore, the inaccuracy of area exposure data, through random misclassification, would be expected to reduce the observed effect estimate, suggesting that the observed SO_2 and NO_2 effects might be even larger than reported.

It can be argued that air pollution may be a causal

TABLE 4. Association Between SIDS and Gaseous Air Pollutants (SO_2, NO_2, and CO) With Different Statistical Models to Adjust for Time, Climate, and Particulate Air Pollution

Covariates in Model with Air Pollution	CO (3-d Average)		NO_2 (3-d Average)		SO_2 (1-d Average)	
	β	*P* Value	β	*P* Value	β	*P* Value
None	.27552	<.0001	.01581	.0001	.0147	.1276
3-mo time factor for all cities	.02236	.7922	.01020	.0121	.01663	.0018
Time factor + season (summer/winter)	.12102	.0784	.00805	.0608	.01259	.0343
Time factor + temperature + relative humidity + barometric pressure	.11367	.1603	.01011	.0144	.01371	.0136
Time factor + PM_{10}	.20454	.2247	−.01268	.3789	−.04041	.3628
Time factor + $PM_{2.5}$*	−.25694	.1251	−.01060	.4635	−.04152	.3212
Time factor + PM_{10} + $PM_{2.5}$	−.09669	.6549	−.00858	.4685	−.03028	.4474

* $PM_{2.5}$, particulate matter with a diameter of <2.5 μm.

TABLE 5. Increases in SIDS Incidence Associated With Increases in Levels of Air Pollutants Equivalent to Their Interquartile Ranges, in Several Canadian Cities, 1984–1999*

Multipollutant Model	Metric	Estimate	*P* Value	Increase in SIDS Incidence (%)
SO_2	1-d lag	0.0120	.0311	11.67
+ NO_2	3-d average	0.0102	.0184	6.05
CO	3-d average	0.1268	.0763	6.82
+ SO_2	1-d lag	0.0156	.0128	7.97
CO	3-d average	0.0619	.5592	3.27
+ NO_2	3-d average	0.0076	.1768	8.59
CO	3-d average	0.0394	.7344	2.07
+ SO_2	1-d lag	0.0113	.0662	5.71
+ NO_2	3-d average	0.0058	.4085	6.52

* Individual air pollutant effects are adjusted for copollutants. Values are adjusted for time.

risk factor for SIDS. First, the association is consistent with the following observations: air pollution has been associated with increased morbidity and mortality rates for both cardiac and respiratory diseases, and SIDS manifests as a cardiopulmonary arrest. Second, one indoor air pollutant, namely, environmental tobacco smoke, has been associated with an increased risk of SIDS.[1,4] The gaseous air pollutants SO_2 and CO, but not particulates, have been associated with low birth weight, which is in turn a risk factor for infant death.[4,14,15] Finally, there is a biologically plausible explanation for the findings, at least for SO_2. SO_2 is a highly water-soluble gas that is absorbed into and irritates the upper respiratory tract. At 0.5 ppm, it can provoke bronchoconstriction among individuals with asthma.[16] The acute effects of NO_2, an oxidant, are more controversial, but NO_2 may increase respiratory symptoms and bronchial responsiveness.[17–19] Irritation and edema of the nasal mucosa or pharynx would increase total pulmonary resistance and the work of breathing, which may be particularly detrimental for infants, who have been said to be obligate nose breathers[20] and at least have a preference for the nasal airway.[21] Irritation of the larynx also may provoke vagally mediated apnea among infants.[22] SIDS is thought to occur among infants with impaired ventilatory control. It can be speculated that gaseous air pollutants may predispose subjects to episodes of apnea and/or increase the work of breathing, which may be particularly dangerous for infants with disordered ventilatory control, who may be unable to generate a sufficient compensatory response.

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