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OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

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*Pediatrics* 2006;118:493-502

DOI: 10.1542/peds.2006-0027

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American Academy of Pediatrics

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# Air Pollution and Infant Death in Southern California, 1989–2000

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The authors have indicated they have no financial relationships relevant to this article to disclose.

## ABSTRACT

**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  $\mu\text{m}$  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- $\mu\text{g}/\text{m}^3$  increase in particulate matter <10  $\mu\text{m}$  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.

www.pediatrics.org/cgi/doi/10.1542/peds.2006-0027

doi:10.1542/peds.2006-0027

### Key Words

air pollution, infant death, postneonatal death, epidemiology, respiratory death, sudden infant death syndrome, mortality

### Abbreviations

PM<sub>10</sub>—particulate matter <10  $\mu\text{m}$  in aerodynamic diameter  
SoCAB—South Coast Air Basin  
CI—confidence interval  
OR—odds ratio  
SIDS—sudden infant death syndrome  
ICD-9—*International Classification of Diseases, 9th Revision*  
ICD-10—*International Classification of Diseases, 10th Revision*  
pphm—parts per hundred million  
O<sub>3</sub>—ozone  
NO<sub>2</sub>—nitrogen dioxide  
CO—carbon monoxide  
LBW—low birth weight  
SO<sub>2</sub>—sulfur dioxide  
PM<sub>2.5</sub>—particulate matter <2.5  $\mu\text{m}$  in aerodynamic diameter

Accepted for publication Feb 21, 2006

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**W**E EVALUATED THE influence of outdoor air pollution on infant death in the South Coast Air Basin (SoCAB) of southern California during 1989–2000. This region, encompassing 6745 square miles of nondesert area in Los Angeles, San Bernardino, Riverside, and Orange Counties, is home to ~15 million people. We examined both short-term (weeks before death) and longer-term (1–6 months before death) exposures to criteria air pollutants. Previous US studies of air pollution and infant death relied solely on large-scale regional exposure assessments and comparisons across counties or metropolitan statistical areas.<sup>1–3</sup> In the SoCAB, a region known for high air pollution levels but also large geographic and temporal pollution variations, we assigned subjects to individual air monitors on the basis of residential location.

Within the first year of life, infants develop rapidly; therefore, their vulnerability may change within weeks or months. In contrast to previous studies, we used a risk set approach, or use that matched on infant developmental age. Rather than relying on annual average exposure contrasts<sup>2–5</sup> or averages for the first months of life,<sup>1</sup> we created exposure windows of different lengths before death. Therefore, we capitalized on both spatial and temporal (seasonal) variations in air pollution concentrations in this region while holding constant the developmental age of the infants at the time of exposure.

## METHODS

### Subject Selection

We identified all infant deaths (deaths during the first year of life) among infants born alive, in 1989–2000, to mothers residing in 389 SoCAB zip codes located within ~16 kilometers (10 miles) of a South Coast Air Quality Management monitoring station, from electronic files assembled by the California Department of Health Services. A total of 2 975 059 births and 19 664 infant deaths were recorded. Infant death data were available through the end of 2000; therefore, we included births through December 31, 1999, which allowed us to monitor all infants through their first full year of life. We matched infant death certificates with California electronic birth certificate data by relying on combinations of infants' full name, date of birth, gender, and zip code. We matched 12 019 infant deaths (61%) with respect to all variables and another 4842 (25%) with respect to name, date of birth, and gender but not zip code (2025 were matched according to the full name, 2817 the infant's first or last name only, and 265 the infant's first and last name and the mother's surname). We excluded 2538 infant deaths (13%) for which we were not able to identify California birth certificates and to create exposure estimates, because we could not assign the infants an air monitoring station according to their zip codes at birth.

For each infant death, we selected randomly 10 control subjects from among all children who were born in the same year as the case subject, were alive at the age at which the case subject's death occurred, and were residing within the same set of 389 zip codes. We excluded multiple births, deaths attributable to external causes (*International Classification of Diseases, 9th Revision* [ICD-9], codes 800–999 and *International Classification of Diseases, 10th Revision* [ICD-10], codes V01–Y89), and subjects with insufficient air monitoring data, namely, <60, <20, <10, or <5 days of measurements available for carbon monoxide (CO), nitrogen dioxide (NO<sub>2</sub>), and ozone (O<sub>3</sub>) during a given 6-month, 2-month, 1-month, or 2-week period, respectively, and <12, <4, <2, or <1 days of measurements available for particulate matter <10 μm in aerodynamic diameter (PM<sub>10</sub>) during a given 6-month, 2-month, 1-month, or 2-week period, respectively. Additional study subjects were excluded from analyses because of missing birth certificate data for individual-level covariates such as maternal age. Because the number of these exclusions varied according to averaging period and model, final sample sizes are reported with the results.

### Outcomes

We evaluated total infant deaths during the first year of life, as well as all respiratory causes of death (ICD-9 codes 460–519, 769, 770.4, 770.7, 770.8, and 770.9 and ICD-10 codes J00–J98, P22.0, P22.9, P27.1, P27.9, P28.0, P28.4, P28.5, and P28.9) and sudden infant death syndrome (SIDS) (ICD-9 code 798.0 and ICD-10 code R95). Furthermore, we examined neonatal deaths (during the first 27 days of life) and postneonatal deaths (from 28 days to the first birthday) separately.

### Exposure Assessment

Each infant was assigned to the nearest, "best," air monitoring station on the basis of the zip code at birth, taking distance, geographic features, and wind flow patterns into account. Exposure averages for each air pollutant were calculated for 2-week, 1-month, 2-month, and 6-month periods before death for case subjects and before the age of death of the matched case subjects for control subjects, relying on CO, NO<sub>2</sub>, O<sub>3</sub>, and PM<sub>10</sub> measurements collected at a maximum of 31 stations during 1989–2000. Therefore, the start and end dates of each control subject's exposure period were based on the case subject's age at death. For example, if an infant death occurred at 75 days of age, then the 1-month exposure before death spanned days 44 to 74 of life for the case subject and the control subjects; we matched subjects with respect to developmental stage but not birth or death season, because an eligible control subject could have been born on any day in the same year. Air pollution concentration averages measured at assigned monitoring stations were based on hourly measure-

ments for gaseous pollutants (CO, NO<sub>2</sub>, and O<sub>3</sub>) and 24-hour average measurements taken every 6 days for PM<sub>10</sub>.

### Statistical Methods

We used conditional logistic regression analysis to derive estimates of air pollution effects on infant deaths for age-matched risk sets. We modeled air pollutants as continuous and categorical measures, relying on percentiles of pollutant distributions for this population. Exposures below the 25th percentile represented the reference category for each pollutant. We evaluated 6-month exposure windows before death only for infants who died in the second half of their first year.

We adjusted for all risk factors available on birth and/or death certificates, including maternal age, race/ethnicity, and education, level of prenatal care, infant gender, parity, birth county, and death season. In stratified analyses, we also evaluated whether estimated effects differed for low birth weight (LBW) children (<2500 g) and/or preterm children (<37 completed weeks of gestation), compared with children who were born at term and weighed  $\geq 2500$  g, to assess whether either subpopulation might be more susceptible to dying during infancy when exposed to air pollutants after birth.

### RESULTS

A greater proportion of case subjects than control subjects were born premature and/or LBW (Table 1). In a multivariate model, the risk of infant death was associated positively with receiving no prenatal care, low and high parity, and younger (<20 years) and older ( $\geq 35$  years) maternal ages. Infants born to mothers of black or other race were at higher risk of death than were those born to white, Hispanic, or Asian women. Female infants were less likely to die within the first year of life than were male infants, as were children born to more highly educated mothers. Risks of infant death were higher in spring and winter than in summer and autumn and were higher in San Bernardino County but lower in Orange County, compared with Los Angeles County.

CO and NO<sub>2</sub> exposures were strongly correlated positively, whereas both were correlated inversely with O<sub>3</sub> exposure (Table 2) and were correlated moderately with PM<sub>10</sub> averages, as expected from well-known seasonal and geographic pollutant patterns in the SoCAB. We observed positive associations between CO, NO<sub>2</sub>, and PM<sub>10</sub> concentrations 2 months before death and the risk of postneonatal infant death from all causes (Table 3), specifically, an 11% (odds ratio [OR]: 1.11; 95% confidence interval [CI]: 1.06–1.16) increase in risk of death per 1-ppm increase in CO concentration, an 8% (OR: 1.08; 95% CI: 1.04–1.11) increase per 1-part per hundred million (pphm) increase in NO<sub>2</sub> concentration, and a 4% (OR: 1.04; 95% CI: 1.01–1.06) increase per 10-

$\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> concentration. In multipollutant models including all pollutants, estimated effect sizes decreased by  $\sim 50\%$  and CIs widened. Positive associations were also observed for 2-week and 1-month averages before death for CO and NO<sub>2</sub>, but the estimated effect sizes were slightly smaller (results not shown).

We also observed positive associations between CO, PM<sub>10</sub>, and NO<sub>2</sub> exposures 2 months before death and the risk of SIDS during the postneonatal period (Table 3). In multipollutant models, however, only the association with 2-month NO<sub>2</sub> exposure persisted, suggesting an 8% increase in the risk of SIDS per 1-pphm increase in NO<sub>2</sub> concentration. The estimated effect sizes were again slightly smaller for shorter averaging periods (results not shown). Negative associations between O<sub>3</sub> concentrations and SIDS risk in single-pollutant models were attenuated with adjustment for other pollutants.

For respiratory causes of postneonatal death, CO concentrations 2 weeks and 1 month before death resulted in the strongest associations, whereas associations for NO<sub>2</sub> exposures disappeared in multipollutant models and none was observed for O<sub>3</sub> exposures. Furthermore, the strongest associations were observed during the early postneonatal period of 28 days to 3 months of life (20%–36% increases in single and multipollutant models per 1-ppm increase in 2-week average CO concentration) (Table 4); results for 1-month averages were very similar (results not shown). Associations observed for particulate exposures seemed to strengthen with infant age. Although we estimated 7% to 12% increases in risk per 10- $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> concentration 2 weeks before death for deaths occurring between 4 and 12 months of age, the same risk increases were 12% to 16% at 7 to 12 months of age (single-pollutant OR: 1.12; 95% CI: 1.02–1.23; multipollutant OR: 1.16; 95% CI: 1.03–1.31). For postneonatal deaths resulting from bronchopulmonary dysplasia (ICD-9 code 770.7 and ICD-10 code P27.1), we observed strong associations for high CO and PM<sub>10</sub> exposures; however, the precision of estimates derived from multipollutant models was low, because of the small number of deaths in this category (for CO exposures of  $\geq 2.17$  ppm 1 month before death: single-pollutant OR: 1.96; 95% CI: 1.01–3.80; multipollutant OR: 1.84; 95% CI: 0.78–4.36; for PM<sub>10</sub> exposures of  $\geq 59.4$   $\mu\text{g}/\text{m}^3$  1 month before death: single-pollutant OR: 2.47; 95% CI: 1.34–4.54; multipollutant OR: 2.05; 95% CI: 0.99–4.25).

We did not observe associations for infant deaths from all causes during the neonatal period (0–27 days of age; results not shown). The numbers of SIDS and respiratory deaths occurring during the neonatal period (0–27 days) were too small (<100 deaths) for assessment of associations. For infant deaths occurring in the second half of the first year of life (7–12 months), we did not observe associations for exposures averaged over a 6-month period, except for an association between PM<sub>10</sub>

**TABLE 1 Demographic Characteristics of Study Subjects**

Parameter	Mean or Proportion		Adjusted OR (95% CI)
	Case Subjects (n = 13 146)	Control Subjects (n = 151 015)	
Gestational age, mean ± SD, d	234.1 ± 66.6	278.0 ± 28.8	
Birth weight, mean ± SD, g	2184.6 ± 1165.9	3375.4 ± 537.8	
Preterm (<37 completed wk), % (n)	53.0 (6972)	9.2 (13 932)	
LBW (<2500 g), % (n)	46.0 (6045)	4.8 (7278)	
Infant gender, % (n)			
Male	56.3 (7395)	51.0 (77 000)	1.00
Female	43.7 (5751)	49.0 (74 015)	0.81 (0.78–0.84)
Prenatal care, % (n)			
None	5.1 (672)	1.4 (2055)	3.39 (3.09–3.73)
During first trimester	72.8 (9570)	76.1 (114 983)	1.00
After first trimester	22.1 (2904)	22.5 (33 977)	0.95 (0.91–1.00)
Parity, % (n)			
First birth	38.1 (5010)	38.9 (58 718)	1.04 (1.00–1.09)
Second or third birth	44.8 (5883)	47.8 (72 201)	1.00
Fourth or subsequent birth	17.1 (2253)	13.3 (20 096)	1.21 (1.15–1.28)
Maternal race/ethnicity, % (n)			
White	22.5 (2953)	26.0 (39 269)	1.00
Hispanic	52.7 (6926)	56.3 (84 992)	0.96 (0.91–1.01)
Black	16.8 (2210)	8.5 (12 822)	2.04 (1.92–2.17)
Asian	4.5 (591)	5.8 (8807)	0.94 (0.86–1.03)
Other	3.5 (466)	3.4 (5125)	1.23 (1.11–1.36)
Maternal education, % (n)			
<9 y	18.0 (2371)	19.0 (28 621)	0.98 (0.92–1.04)
9–11 y	23.9 (3144)	20.9 (31 570)	1.08 (1.03–1.14)
12 y	30.5 (4009)	28.4 (42 924)	1.00
13–15 y	17.2 (2254)	17.3 (26 173)	0.93 (0.88–0.98)
≥16 y	10.4 (1368)	14.4 (21 727)	0.71 (0.66–0.76)
Maternal age, % (n)			
<20 y	15.2 (1991)	12.0 (18 113)	1.22 (1.15–1.29)
20–29 y	51.3 (6737)	54.4 (82 163)	1.00
30–34 y	19.8 (2606)	21.7 (32 814)	1.03 (0.98–1.08)
35–39 y	10.7 (1407)	9.8 (14 825)	1.23 (1.16–1.32)
≥40 y	3.1 (405)	2.1 (3100)	1.70 (1.52–1.90)
Death season, % (n)			
Spring	25.0 (3291)	24.5 (37 052)	1.06 (1.01–1.12)
Summer	24.2 (3185)	25.4 (38 367)	1.00
Autumn	23.7 (3120)	25.7 (38 827)	0.97 (0.92–1.03)
Winter	27.0 (3550)	24.4 (36 769)	1.16 (1.10–1.22)
Birth county, % (n)			
Los Angeles	69.0 (9067)	69.8 (105 466)	1.00
Orange	14.3 (1885)	17.0 (25 733)	0.94 (0.89–0.99)
San Bernardino	11.4 (1501)	8.2 (12 337)	1.36 (1.28–1.44)
Riverside	5.3 (693)	5.0 (7479)	1.06 (0.98–1.16)

The total sample sizes reflected in each row are slightly smaller than the total sample sizes of  $n = 17\,126$  case subjects and  $171\,260$  control subjects because of missing data for some parameters. Therefore, the sample sizes available for the adjusted ORs shown are  $n = 13\,146$  case subjects and  $151\,015$  control subjects. The adjusted ORs are from a model including all of the parameters in the table, that is, infant gender, level of prenatal care, parity, maternal race/ethnicity, education, and age, death season, and birth county.

exposures and respiratory deaths (for  $PM_{10}$  exposures of  $37.6$  to  $<52.6 \mu\text{g}/\text{m}^3$ : multipollutant OR: 1.55; 95% CI: 0.88–2.73; for  $PM_{10}$  exposures of  $\geq 52.6 \mu\text{g}/\text{m}^3$ : multipollutant OR: 2.42; 95% CI: 1.15–5.10). Similar models for children who died as a result of congenital malformations showed no effects for any of the air pollutants examined.

When we grouped infants according to those born with LBW and/or preterm and those born with normal weight and at term, associations seemed stronger for the

former group; however, we lacked statistical power to confirm this heterogeneity with formal testing (Table 5). For respiratory deaths, point estimates for 2-week CO averages were much higher for preterm/LBW infants, although 95% CIs were wide because of the small number of cases. This pattern was not observed for 1-month exposure averages. There was an approximately twofold increase in the risk of respiratory death among preterm/LBW infants in the highest  $PM_{10}$  exposure quartile, compared with a 56% increase in risk experienced by term

**TABLE 2** Pollutant Levels and Pearson Correlation Coefficients According to Exposure Period

Pollutant	Mean (Range)	Pearson Correlation Coefficients			
		CO	NO <sub>2</sub>	O <sub>3</sub>	PM <sub>10</sub>
Two weeks before death					
CO, ppm	1.63 (0.38–3.44)	1.00			
NO <sub>2</sub> , pphm	3.84 (1.92–6.15)	0.72	1.00		
O <sub>3</sub> , pphm	2.19 (0.46–4.12)	−0.57	−0.31	1.00	
PM <sub>10</sub> , μg/m <sup>3</sup>	46.2 (21.0–83.5)	0.33	0.48	0.12	1.00
One month before death					
CO, ppm	1.63 (0.42–3.35)	1.00			
NO <sub>2</sub> , pphm	3.84 (2.02–5.88)	0.71	1.00		
O <sub>3</sub> , pphm	2.19 (0.50–4.06)	−0.61	−0.35	1.00	
PM <sub>10</sub> , μg/m <sup>3</sup>	46.3 (25.0–77.2)	0.33	0.48	0.12	1.00
Two months before death					
CO, ppm	1.63 (0.45–3.23)	1.00			
NO <sub>2</sub> , pphm	3.83 (2.11–5.73)	0.72	1.00		
O <sub>3</sub> , pphm	2.20 (0.56–4.00)	−0.63	−0.38	1.00	
PM <sub>10</sub> , μg/m <sup>3</sup>	46.3 (27.6–74.2)	0.32	0.48	0.12	1.00
Six months before death					
CO, ppm	1.63 (0.67–2.86)	1.00			
NO <sub>2</sub> , pphm	3.84 (2.33–5.36)	0.71	1.00		
O <sub>3</sub> , pphm	2.21 (1.02–3.63)	−0.61	−0.39	1.00	
PM <sub>10</sub> , μg/m <sup>3</sup>	46.3 (31.3–69.5)	0.29	0.44	0.16	1.00

infants of normal weight. Similarly, associations between CO, NO<sub>2</sub>, and PM<sub>10</sub> concentrations and risks of SIDS seemed stronger for preterm/LBW infants; however, 95% CIs were wide and overlapped greatly in multipollutant models.

## DISCUSSION

We found CO and NO<sub>2</sub> exposures 2 months before death to be associated with increased risk of postneonatal death for infants residing in the SoCAB. For particulates, both short-term and longer-term (6-month) PM<sub>10</sub> averages had influences on infant death. Similar to previous studies,<sup>1,2,5</sup> associations seemed to be attributable mostly to deaths resulting from respiratory causes and SIDS. LBW and preterm infants seemed to be more susceptible to air pollution-related effects, but these subgroups of infants were small and our estimates were imprecise.

We examined gaseous and particulate exposures experienced during different time periods before death (2 weeks to 6 months). Previous studies focusing on long-term exposures relied almost exclusively on spatial comparisons. A Czech study<sup>5</sup> matched control and case subjects according to date of birth; therefore, the calculated measures of average lifetime air pollution varied according to study district but not time, because they spanned the same seasons. Those authors reported that risk of postneonatal death resulting from respiratory mortality causes increased by 95% (OR: 1.95; 95% CI: 1.09–3.50) per 50-μg/m<sup>3</sup> increase in total suspended particulate concentration, but no associations were observed during the neonatal period or for other causes of death, including SIDS. For the first US study of postneonatal death in 86 metropolitan statistical areas,<sup>1</sup> researchers relied on

air pollution concentrations averaged over the first 2 months of life, assuming that this early infancy period provided an adequate ranking of exposure experiences during the 3-year study period. That study reported exposures to particulates to be associated with postneonatal death attributable to respiratory causes (OR: 1.20; 95% CI: 1.06–1.36 per 10-μg/m<sup>3</sup> increase in PM<sub>10</sub> exposure) and SIDS (OR: 1.12; 95% CI: 1.07–1.17 per 10-μg/m<sup>3</sup> increase in PM<sub>10</sub> exposure). Surprisingly, PM<sub>10</sub> exposure seemed to affect risk of respiratory death for LBW infants less strongly (OR: 1.05; 95% CI: 0.91–1.22 per 10-μg/m<sup>3</sup> increase in PM<sub>10</sub> exposure); no risk estimates were reported for SIDS among LBW infants. A second US study, focusing on one of the years included previously but expanding geographically to 180 counties, confirmed the particulate associations for respiratory infant deaths and SIDS but found no difference between LBW and normal birth weight or between neonatal and postneonatal deaths.<sup>2</sup> The authors also reported a possible 10% (OR: 1.10; 95% CI: 0.99–1.23) increase in SIDS risk per 1-ppm increase in CO exposure. That study relied on countywide annual average exposure estimates; therefore, seasonal air pollution fluctuations and the possible influence of exposures experienced immediately before death were not evaluated.

Previous short-term exposure studies examining pollution increases within days before death provided some evidence for possible associations between gaseous pollutants and respiratory death for infants and young children. Several time-series studies reported positive associations between the gaseous pollutants CO and nitrogen oxides and respiratory infant deaths.<sup>6–10</sup> Studies focusing on children <5 years of age in Sao Paulo, Brazil (a city in

**TABLE 3 Association Between Air Pollution and All-Cause Postneonatal Death and SIDS for Infants Born Between 1989 and 2000 to Mothers Living in 389 SoCAB Zip Codes**

Pollutant/Exposure Period	OR (95% CI)	
	Single-Pollutant Models	Multiple-Pollutant Models
<b>All-cause death</b>		
CO/2 mo before death	4915 case subjects, 52 491 control subjects	4616 case subjects, 48 986 control subjects
Per 1 ppm	1.11 (1.06–1.16)	1.06 (1.00–1.13)
1.06 to <2.05 ppm <sup>a</sup>	1.07 (0.99–1.16)	1.05 (0.95–1.16)
≥2.05 ppm	1.25 (1.12–1.39)	1.15 (1.00–1.33)
NO <sub>2</sub> /2 mo before death	4820 case subjects, 50 998 control subjects	4616 case subjects, 48 986 control subjects
Per 1 pphm	1.08 (1.04–1.11)	1.04 (0.99–1.09)
3.03 to <4.60 pphm	1.08 (0.99–1.17)	1.04 (0.94–1.15)
≥4.60 pphm	1.21 (1.09–1.34)	1.10 (0.97–1.25)
PM <sub>10</sub> /2 mo before death	4855 case subjects, 51 947 control subjects	4616 case subjects, 48 986 control subjects
Per 10 μg/m <sup>3</sup>	1.04 (1.01–1.06)	1.02 (0.99–1.05)
35.2 to <54.1 μg/m <sup>3</sup>	0.96 (0.89–1.04)	0.92 (0.84–1.00)
≥54.1 μg/m <sup>3</sup>	1.14 (1.03–1.27)	1.07 (0.95–1.20)
O <sub>3</sub> /2 mo before death	4962 case subjects, 52 967 control subjects	4616 case subjects, 48 986 control subjects
Per 1 pphm	0.93 (0.89–0.97)	0.96 (0.91–1.01)
1.33 to <2.95 pphm	0.85 (0.78–0.93)	0.91 (0.82–1.01)
≥2.95 pphm	0.80 (0.71–0.91)	0.85 (0.74–0.97)
<b>SIDS</b>		
CO/2 mo before death	1487 case subjects, 14 624 control subjects	1381 case subjects, 13 627 control subjects
Per 1 ppm	1.19 (1.10–1.28)	1.09 (0.97–1.22)
1.08 to <2.12 ppm	1.08 (0.92–1.26)	0.97 (0.81–1.17)
≥2.12 ppm	1.43 (1.17–1.75)	1.11 (0.86–1.44)
NO <sub>2</sub> /2 mo before death	1450 case subjects, 14 226 control subjects	1381 case subjects, 13 627 control subjects
Per 1 pphm	1.15 (1.08–1.23)	1.08 (0.99–1.18)
3.07 to <4.65 pphm	1.36 (1.16–1.60)	1.31 (1.08–1.59)
≥4.65 pphm	1.55 (1.27–1.88)	1.44 (1.13–1.85)
PM <sub>10</sub> /2 mo before death	1454 case subjects, 14 400 control subjects	1381 case subjects, 13 627 control subjects
Per 10 μg/m <sup>3</sup>	1.03 (0.99–1.08)	1.01 (0.95–1.07)
35.8 to <55.7 μg/m <sup>3</sup>	0.94 (0.81–1.08)	0.90 (0.76–1.06)
≥55.7 μg/m <sup>3</sup>	1.13 (0.93–1.36)	0.99 (0.80–1.24)
O <sub>3</sub> /2 mo before death	1495 case subjects, 14 754 control subjects	1381 case subjects, 13 627 control subjects
Per 1 pphm	0.89 (0.82–0.96)	0.93 (0.84–1.02)
1.30 to <2.95 pphm	0.74 (0.63–0.87)	0.88 (0.73–1.07)
≥2.95 pphm	0.73 (0.58–0.92)	0.86 (0.66–1.11)

The following variables were included in all models: infant gender, maternal age, race, and education, level of prenatal care, death season, birth county, and parity. Multiple-pollutant models included those variables plus averages for all 4 pollutants (CO, NO<sub>2</sub>, PM<sub>10</sub>, and O<sub>3</sub>).

<sup>a</sup> Values listed are the 25th to <75th and ≥75th percentile cutoff points from single-pollutant models.

which air pollution is dominated by motor vehicle emissions, similar to Los Angeles), reported a 3% increase in respiratory mortality risk per 1-ppm increase in CO levels (greatest 8-hour moving average measured 2 days before death) and a 9% (95% CI: 0.95–1.24) increase in mortality risk when same-day CO levels were >90th percentile.<sup>7</sup> Risk of death resulting from respiratory causes was also associated with nitrogen oxides (on the basis of a 3-day moving average).<sup>8</sup> A small increase in infant deaths was observed in Mexico City when NO<sub>2</sub> levels increased (1.4% per 10-ppb increase), but this association was reduced in size when a measure of fine particles (particulate matter <2.5 μm in aerodynamic diameter [PM<sub>2.5</sub>]) was entered into the model.<sup>9</sup> In Seoul, South Korea, a 39% increase in postneonatal respiratory death was observed per 5.7-ppm increase in same-day CO levels (OR: 1.39; 95% CI: 1.01–1.91) in single-pollutant models.<sup>10</sup> Because particles were also found to

contribute strongly to these outcomes, it is not clear which pollutant was more important. Studies focusing on short-term exposures and SIDS also reported associations for gaseous pollutants. Hoppenbrouwers et al,<sup>11</sup> relying on daily average concentrations of CO, NO<sub>2</sub>, O<sub>3</sub>, and hydrocarbons in Los Angeles County in the late 1970s, found temporal correlations between daily SIDS rates and levels of CO, sulfur dioxide (SO<sub>2</sub>), and hydrocarbons ( $r > 0.80$ ) and also NO<sub>2</sub> ( $r = 0.74$ ), lagging pollutant concentrations by as much as 7 weeks. SIDS mortality rates peaked a few weeks after air pollution during the middle of winter; however, that study did not control for other time-varying factors or winter respiratory infection epidemics. Knobel et al<sup>12</sup> reported an approximately threefold increase in the daily SIDS rate for infants who lived in regions of Taiwan with the lowest measure of visibility on the day of death. They reported that aerosols that reduce visibility the most are in the

**TABLE 4 Association Between Air Pollution and Respiratory Death for Infants Born Between 1989 and 2000 to Mothers Living in 389 SoCAB Zip Codes**

Pollutant/Exposure Period	OR (95% CI)					
	Postneonatal Deaths (28 d to 1 y)		Postneonatal Deaths (28 d to 3 mo)		Postneonatal Deaths (4–12 mo)	
	Single-Pollutant Models	Multiple-Pollutant Models	Single-Pollutant Models	Multiple-Pollutant Models	Single-Pollutant Models	Multiple-Pollutant Models
CO/2 wk before death	650 case subjects, 6567 control subjects	603 case subjects, 6020 control subjects	266 case subjects, 2660 control subjects	248 case subjects, 2430 control subjects	384 case subjects, 3907 control subjects	355 case subjects, 3590 control subjects
Per 1 ppm	1.14 (1.03–1.25)	1.16 (1.00–1.35)	1.20 (1.02–1.40)	1.36 (1.09–1.70)	1.10 (0.96–1.24)	1.04 (0.86–1.27)
1.02 to <2.08 ppm <sup>a</sup>	1.18 (0.93–1.50)	1.23 (0.93–1.62)	1.41 (0.95–2.07)	1.67 (1.05–2.65)	1.18 (0.87–1.59)	1.17 (0.82–1.68)
≥2.08 ppm	1.42 (1.05–1.92)	1.62 (1.09–2.40)	1.97 (1.21–3.22)	2.81 (1.47–5.36)	1.16 (0.78–1.71)	1.06 (0.63–1.79)
NO <sub>2</sub> /2 wk before death	643 case subjects, 6351 control subjects	603 case subjects, 6020 control subjects	261 case subjects, 2573 control subjects	248 case subjects, 2430 control subjects	382 case subjects, 3778 control subjects	355 case subjects, 3590 control subjects
Per 1 pphm	1.06 (0.98–1.14)	0.96 (0.86–1.07)	1.05 (0.93–1.18)	0.91 (0.77–1.07)	1.04 (0.95–1.15)	0.98 (0.85–1.13)
2.92 to <4.68 pphm	1.14 (0.91–1.43)	0.99 (0.76–1.30)	1.18 (0.82–1.69)	0.88 (0.57–1.37)	1.03 (0.77–1.38)	0.96 (0.67–1.35)
≥4.68 pphm	1.20 (0.91–1.58)	0.85 (0.59–1.22)	1.25 (0.80–1.93)	0.71 (0.40–1.27)	1.06 (0.74–1.51)	0.95 (0.60–1.52)
PM <sub>10</sub> /2 wk before death	636 case subjects, 6414 control subjects	603 case subjects, 6020 control subjects	265 case subjects, 2579 control subjects	248 case subjects, 2430 control subjects	371 case subjects, 3835 control subjects	355 case subjects, 3590 control subjects
Per 10 μg/m <sup>3</sup>	1.05 (1.01–1.10)	1.04 (0.98–1.09)	1.01 (0.95–1.08)	1.00 (0.92–1.09)	1.12 (1.02–1.23)	1.07 (1.00–1.15)
33.0 to <56.0 μg/m <sup>3</sup>	1.13 (0.91–1.41)	1.09 (0.86–1.38)	1.16 (0.82–1.63)	0.97 (0.67–1.42)	1.08 (0.81–1.44)	1.02 (0.75–1.40)
≥56.0 μg/m <sup>3</sup>	1.46 (1.13–1.88)	1.40 (1.03–1.89)	1.44 (0.96–2.17)	1.23 (0.76–2.00)	1.41 (1.02–1.96)	1.36 (0.92–2.01)
O <sub>3</sub> /2 wk before death	661 case subjects, 6634 control subjects	603 case subjects, 6020 control subjects	269 case subjects, 2680 control subjects	248 case subjects, 2430 control subjects	392 case subjects, 3954 control subjects	355 case subjects, 3590 control subjects
Per 1 pphm	1.03 (0.93–1.14)	1.03 (0.91–1.16)	0.99 (0.85–1.16)	1.06 (0.89–1.28)	1.06 (0.93–1.21)	1.00 (0.84–1.18)
1.22 to <2.98 pphm	0.83 (0.65–1.07)	1.06 (0.79–1.41)	0.95 (0.63–1.42)	1.21 (0.76–1.91)	0.85 (0.61–1.17)	0.80 (0.54–1.17)
≥2.98 pphm	0.89 (0.64–1.25)	1.08 (0.73–1.59)	0.89 (0.51–1.53)	1.23 (0.66–2.28)	0.96 (0.62–1.48)	0.80 (0.49–1.33)

The following variables were included in the models: infant gender, maternal age, race, and education, level of prenatal care, death season, birth county, and parity. Multiple-pollutant models included those variables plus averages for all 4 pollutants (CO, NO<sub>2</sub>, PM<sub>10</sub>, and O<sub>3</sub>). Respiratory causes of death included ICD-9 codes 460 to 519, 769, 770.4, 770.7, 770.8, and 770.9 and ICD-10 codes J00 to J98, P22.0, P22.9, P27.1, P27.9, P28.0, P28.4, P28.5, and P28.9.

<sup>a</sup> Values listed are the 25th to <75th and ≥75th percentile cutoff points for infant deaths at ages 28 days to 1 year from single-pollutant models. The 25th to 75th percentile values for the other age categories were as follows: 28 days to 3 months: CO: 1.06–2.14 ppm; NO<sub>2</sub>: 2.97–4.76 pphm; PM<sub>10</sub>: 34.0–57.0 μg/m<sup>3</sup>; O<sub>3</sub>: 1.18–3.00 pphm; 4 to 12 months: CO: 0.99–2.03 ppm; NO<sub>2</sub>: 2.88–4.62 pphm; PM<sub>10</sub>: 32.5–55.0 μg/m<sup>3</sup>; O<sub>3</sub>: 1.24–2.96 pphm.

**TABLE 5 Association Between Air Pollution and Postneonatal Death (28 Days to 1 Year of age), Stratified According to Birth Weight and Gestational Age**

Pollutant/Exposure Period	OR (95% CI)	
	Single-Pollutant Models	Multiple-Pollutant Models
<b>Term/normal-weight infants</b>		
All-cause death		
CO/2 mo before death	2723 case subjects, 44 425 control subjects	2545 case subjects, 41 404 control subjects
Per 1 ppm	1.12 (1.05–1.19)	1.07 (0.98–1.17)
1.06 to <2.05 ppm <sup>a</sup>	0.99 (0.89–1.11)	0.95 (0.83–1.09)
≥2.05 ppm	1.25 (1.08–1.45)	1.08 (0.89–1.30)
PM <sub>10</sub> /2 mo before death	2679 case subjects, 43 971 control subjects	2545 case subjects, 41 404 control subjects
Per 10 μg/m <sup>3</sup>	1.03 (0.99–1.06)	1.02 (0.98–1.06)
35.3 to <54.1 μg/m <sup>3</sup>	0.97 (0.87–1.08)	0.95 (0.85–1.07)
≥54.1 μg/m <sup>3</sup>	1.11 (0.96–1.27)	1.05 (0.90–1.23)
<b>SIDS</b>		
CO/2 mo before death	1045 case subjects, 12 425 control subjects	968 case subjects, 11 578 control subjects
Per 1 ppm	1.17 (1.07–1.29)	1.09 (0.95–1.25)
1.08 to <2.12 ppm	1.06 (0.88–1.28)	0.96 (0.77–1.20)
≥2.12 ppm	1.40 (1.10–1.78)	1.10 (0.81–1.50)
NO <sub>2</sub> /2 mo before death	1018 case subjects, 12 073 control subjects	968 case subjects, 11 578 control subjects
Per 1 pphm	1.16 (1.08–1.25)	1.09 (0.98–1.21)
3.08 to <4.65 pphm	1.37 (1.12–1.66)	1.35 (1.07–1.70)
≥4.65 pphm	1.63 (1.28–2.06)	1.55 (1.15–2.10)
PM <sub>10</sub> /2 mo before death	1021 case subjects, 12 240 control subjects	968 case subjects, 11 578 control subjects
Per 10 μg/m <sup>3</sup>	1.01 (0.96–1.07)	0.99 (0.93–1.06)
35.8 to <55.7 μg/m <sup>3</sup>	0.97 (0.81–1.16)	0.93 (0.76–1.13)
≥55.7 μg/m <sup>3</sup>	1.06 (0.84–1.34)	0.93 (0.71–1.22)
<b>Respiratory death</b>		
CO/2 wk before death	248 case subjects, 5550 control subjects	228 case subjects, 5079 control subjects
Per 1 ppm	1.14 (0.95–1.36)	1.26 (0.98–1.62)
1.02 to <2.07 ppm	0.76 (0.51–1.15)	0.90 (0.54–1.49)
≥2.07 ppm	0.98 (0.59–1.62)	1.18 (0.59–2.37)
PM <sub>10</sub> /2 wk before death	241 case subjects, 5426 control subjects	228 case subjects, 5079 control subjects
Per 10 μg/m <sup>3</sup>	1.06 (0.99–1.14)	1.05 (0.96–1.14)
33.0 to <55.5 μg/m <sup>3</sup>	0.99 (0.69–1.44)	1.04 (0.69–1.56)
≥55.5 μg/m <sup>3</sup>	1.46 (0.95–2.26)	1.56 (0.94–2.60)
<b>Preterm and/or LBW infants</b>		
All-cause death		
CO/2 mo before death	2012 case subjects, 5963 control subjects	1900 case subjects, 5619 control subjects
Per 1 ppm	1.12 (1.01–1.25)	1.01 (0.87–1.19)
1.09 to <2.12 ppm	1.20 (0.99–1.47)	1.28 (1.01–1.63)
≥2.12 ppm	1.30 (1.00–1.69)	1.29 (0.91–1.84)
PM <sub>10</sub> /2 mo before death	1999 case subjects, 5904 control subjects	1900 case subjects, 5619 control subjects
Per 10 μg/m <sup>3</sup>	1.06 (1.00–1.13)	1.04 (0.96–1.12)
35.4–54.7 μg/m <sup>3</sup>	0.92 (0.77–1.11)	0.80 (0.65–0.99)
≥54.7 μg/m <sup>3</sup>	1.24 (0.97–1.57)	1.11 (0.83–1.48)
<b>SIDS</b>		
CO/2 mo before death	376 case subjects, 1627 control subjects	352 case subjects, 1521 control subjects
Per 1 ppm	1.46 (1.09–1.94)	1.35 (0.88–2.06)
1.12 to <2.18 ppm	1.35 (0.78–2.34)	1.27 (0.61–2.62)
≥2.18 ppm	1.84 (0.89–3.83)	1.45 (0.51–4.10)
NO <sub>2</sub> /2 mo before death	368 case subjects, 1590 control subjects	352 case subjects, 1521 control subjects
Per 1 pphm	1.40 (1.09–1.79)	1.22 (0.86–1.72)
3.11 to <4.72 pphm	1.80 (0.97–3.33)	1.83 (0.84–4.01)
≥4.72 pphm	2.34 (1.13–4.86)	2.10 (0.75–5.87)
PM <sub>10</sub> /2 mo before death	369 case subjects, 1600 control subjects	352 case subjects, 1521 control subjects
Per 10 μg/m <sup>3</sup>	1.26 (1.06–1.50)	1.11 (0.89–1.39)
36.2 to <56.6 μg/m <sup>3</sup>	0.80 (0.47–1.37)	0.64 (0.34–1.18)
≥56.6 μg/m <sup>3</sup>	1.95 (0.96–3.99)	1.29 (0.53–3.12)
<b>Respiratory death</b>		
CO/2 wk before death	382 case subjects, 760 control subjects	355 case subjects, 704 control subjects
Per 1 ppm	1.03 (0.83–1.27)	1.01 (0.73–1.40)
1.08 to <2.20 ppm	1.03 (0.65–1.64)	0.87 (0.47–1.61)
≥2.20 ppm	1.44 (0.76–2.73)	1.61 (0.62–4.19)
PM <sub>10</sub> /2 wk before death	375 case subjects, 737 control subjects	355 case subjects, 704 control subjects
Per 10 μg/m <sup>3</sup>	1.08 (0.98–1.20)	1.10 (0.96–1.26)
33.5 to <58.0 μg/m <sup>3</sup>	1.26 (0.81–1.97)	1.08 (0.66–1.75)
≥58.0 μg/m <sup>3</sup>	1.78 (1.02–3.10)	2.04 (1.00–4.18)

The following variables were included in the models: infant gender, maternal age, race, and education, level of prenatal care, death season, birth county, and parity. Multiple-pollutant models included those variables plus averages for all 4 pollutants (CO, NO<sub>2</sub>, PM<sub>10</sub>, and O<sub>3</sub>).

<sup>a</sup> Values listed are the 25th to <75th and ≥75th percentile cutoff points from single-pollutant models.

0.2- $\mu\text{m}$  to 2- $\mu\text{m}$  size range and thus include fine and ultrafine particles. Measurements of  $\text{PM}_{10}$ ,  $\text{SO}_2$ , and CO in some of the regions studied indicated that levels of all of these pollutants were correlated negatively with visibility ( $r = -0.59$ ,  $r = -0.40$ , and  $r = -0.46$ , respectively). More recently, on the basis of data for 12 Canadian cities, Dales et al<sup>13</sup> reported SIDS to be associated with increases in  $\text{SO}_2$  and  $\text{NO}_2$  levels ( $\sim 6\%$  per interquartile increase in pollutant concentration, with a 1-day lag for  $\text{SO}_2$  and a 3-day average before death for  $\text{NO}_2$ ).

Therefore, time-series studies linked short-term exposure to pollutants from motor vehicle exhaust to respiratory and SIDS deaths among infants, corroborating our findings of associations between 2-week, 1-month, and 2-month exposures to CO and  $\text{NO}_2$  and respiratory deaths and SIDS. CO is emitted directly in vehicle exhaust and does not react readily in the atmosphere to form other compounds, whereas  $\text{NO}_2$  is formed adjacent to roadways through reactions between nitrogen oxide (released directly in exhaust) and  $\text{O}_3$ .<sup>14</sup> Therefore, CO and  $\text{NO}_2$  are mostly markers for vehicle-related combustion and additional toxic components in vehicle exhaust, including ultrafine particles. A number of the previous studies also reported associations with  $\text{SO}_2$  and  $\text{PM}_{10}$  levels. We did not evaluate  $\text{SO}_2$  levels in our study because concentrations of this pollutant are low in the Los Angeles Basin. This pollutant may be more important in areas with a significant presence of coal-fired electricity generation or use of coal for domestic heating and cooking and in areas where there is still a relatively high concentration of sulfur in diesel fuel.  $\text{PM}_{10}$  encompasses primary particles emitted directly from motor vehicles and secondary particles formed through atmospheric reactions and crustal elements from windblown dust. In the SoCAB, the contribution of each of these components to the overall particle mass concentration varies according to geographic location and season.<sup>15,16</sup> Generally, it is difficult to compare  $\text{PM}_{10}$ -related findings across studies, because the composition of particles in this size range varies significantly depending on geographic region and time period studied. Because the SoCAB  $\text{PM}_{2.5}$  monitoring network was not built until 1999, we had insufficient sample size available (infant deaths for 1999–2000 only) to evaluate associations with this pollutant.

Unlike previous studies, we not only evaluated neonatal (0–27 days) and postneonatal (28 days to 1 year) deaths separately but also distinguished between different postneonatal age ranges (28 days to 3 months, 4–6 months, and 7–12 months), to examine whether specific periods during infancy exhibit more susceptibility to air pollution effects. Associations between CO levels and respiratory causes of death seemed isolated to the early postneonatal period of 28 days to 3 months, whereas associations for  $\text{PM}_{10}$  levels seemed stronger in later

infancy (4–12 months). Currently, there is a lack of mechanistic information regarding why this might be the case. For SIDS, no pattern according to postneonatal age seemed to exist.

The most important source of bias in this study was exposure misclassification, especially for pollutants known to have substantial spatial concentration gradients (such as CO), because we relied on existing government monitoring stations, which we linked to residential addresses reported on birth certificates for assessment of personal air pollution exposures. The average distance between zip code geometric centroids and assigned stations for the gaseous pollutants was 7.4 km (4.6 miles), whereas for  $\text{PM}_{10}$  the average distance was 10.5 km (6.5 miles). We used residential zip codes reported on birth certificates to assign exposures because we did not have residential information for control subjects at the time of the case subjects' deaths. Therefore, exposure misclassification might have resulted for families that moved during the infant's first year of life, but 70% of the case subjects who could be matched back to a birth certificate lived at the same zip code at birth and death. It seems reasonable to assume that any exposure misclassification resulting from residential mobility was not different between case and control subjects. Other important sources of exposure misclassification were discussed previously.<sup>17–20</sup>

Because we relied on birth and death registry data for this study, we did not have information on a number of potentially important confounding variables, such as maternal active and passive smoking and infant sleeping position. However, a major strength of this study is that our exposure measures took into account both temporal and spatial variations in air pollution in the SoCAB. Confounding attributable to variables that may vary spatially (for example, smoking, because of its correlation with socioeconomic status) may be less influential when estimated effects are attributable to factors that also have strong temporal variations, as observed in the SoCAB for these pollutants. Also, data from our recent postnatal survey of  $\sim 2500$  Los Angeles County women who delivered during 2003 suggested a very low smoking rate among women during pregnancy (4%) (unpublished data). Finally, some of these factors are correlated with variables for which we adjusted in our analyses (maternal age, race/ethnicity, and education); therefore, we might have adjusted for these factors at least partially and indirectly.

Toxicologic studies may be able to determine mechanisms through which air pollutants contribute to infant death. For example, rats reared for prolonged periods in the downtown areas of Sao Paulo, Brazil, had inflammatory alterations of the airways and pulmonary parenchyma that resulted in diminished pulmonary defenses and increased death attributable to respiratory causes.<sup>21,22</sup> Maternal cigarette smoking is a widely accepted

risk factor for SIDS. Postulated mechanisms include abnormalities in brain development that result in a tendency toward central apnea and disturbances in respiratory control mechanisms, abnormal pulmonary development in neonates, and the promotion of respiratory infections.<sup>23</sup> Exposure to tobacco smoke has also been linked to an increased risk of acute lower respiratory illness, including bronchiolitis, reduced lung function, and asthma, in infancy.<sup>23</sup> Because constituents of ambient air pollution and tobacco smoke overlap (eg, CO and particles), it is conceivable that air pollution may act through similar pathways. However, to date the evidence is insufficient to determine whether the concentrations found in ambient air are sufficient to induce effects similar to those of cigarette smoke.

## CONCLUSIONS

Our findings corroborate previous studies in and outside the United States. On the basis of previous reports,<sup>1</sup> Kaiser et al<sup>24</sup> estimated that the proportions of all-cause, SIDS-related, and respiratory disease-related deaths attributable to PM<sub>10</sub> levels of >12.0 µg/m<sup>3</sup> were 6% (95% CI: 3%–11%), 16% (95% CI: 9%–23%), and 24% (95% CI: 7%–44%), respectively. They noted that, even if all US counties studied were in compliance with the new US PM<sub>2.5</sub> standard, the majority of the estimated burden would remain. A novelty and major strength of this study is our use of both temporal and spatial variations to assess the risk of infant death attributable to air pollution in the SoCAB. The results reported in this article add to the growing body of literature implicating air pollution in infant death and provide additional information for future risk assessment.

## ACKNOWLEDGMENTS

This work was supported by National Institute of Environmental Health Sciences grant R01 ES010960-01.

We thank Curt Miller of the South Coast Air Quality Management District for providing air monitoring data.

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*Pediatrics* 2006;118:493-502

DOI: 10.1542/peds.2006-0027

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