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Authors

Woodruff, TJ
Grillo, J
Schoendorf, KC

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The Relationship between Selected Causes of Postneonatal Infant Mortality and Particulate Air Pollution in the United States

Tracey J. Woodruff,[1] Jeanne Grillo,[2] and Kenneth C. Schoendorf[2]

[1]U.S. Environmental Protection Agency, Washington, D.C. 20460 USA; [2] National Center for Health Statistics, Centers for Disease Control and Prevention, Hyattsville, MD 20782 USA

Recent studies have found associations between particulate air pollution and total and adult mortality. The relationship between particulate air pollution and mortality among infants has not been examined in the United States. This study evaluates the relationship between postneonatal infant mortality and particulate matter in the United States. Our study involved analysis of cohorts consisting of approximately 4 million infants born between 1989 and 1991 in states that report relevant covariates; this included 86 metropolitan statistical areas (MSAs) in the United States. Data from the National Center for Health Statistics-linked birth/infant death records were combined at the MSA level with measurements of particulate matter 10 μm or less (PM_{10}) from the EPA's Aerometric Database. Infants were categorized as having high, medium, or low exposures based on tertiles of PM_{10}. Total and cause-specific postneonatal mortality rates were examined using logistic regression to control for demographic and environmental factors. Overall postneonatal mortality rates were 3.1 among infants with low PM_{10} exposures, 3.5 among infants with medium PM_{10} exposures, and 3.7 among highly exposed infants. After adjustment for other covariates, the odds ratio (OR) and 95% confidence intervals (CI) for total postneonatal mortality for the high exposure versus the low exposure group was 1.10 (1.04, 1.16). In normal birth weight infants, high PM_{10} exposure was associated with respiratory causes [OR = 1.40, (1.05, 1.85)] and sudden infant death syndrome [OR = 1.26, (1.14, 1.39)]. For low birth weight babies, high PM_{10} exposure was associated, but not significantly, with mortality from respiratory causes [OR = 1.18, (0.86, 1.61)]. This study suggests that particulate matter is associated with risk of postneonatal mortality. Continued attention should be paid to air quality to ensure optimal health of infants in the United States. *Key words*: air pollution, infant mortality, particulate air pollution, postneonatal mortality. *Environ Health Perspect* 105:608–612 (1997)

Concerns about the effects of particulate matter air pollution on health date back to the historic pollution episodes in London in 1952 when a weather inversion lead to high levels of particulate matter air pollution and subsequent increases in mortality and morbidity (*1*). Since then, recent investigations of the relationship between air pollution and mortality in the United States have demonstrated an association between particulate air pollution levels and mortality at lower air pollution levels. A number of daily time–series studies have demonstrated an association between short-term exposure to particulate air pollution and mortality (*2,3*). More recent prospective cohort analyses have also demonstrated an association between long-term exposure to particulate air pollution and mortality (*4,5*). However, all the studies conducted in the United States have focused on adults. The original investigation in London observed increases in mortality for both adults over 45 years of age and children under the age of 1 year (*1*). Given this result and the number of studies showing a positive association between mortality and exposure to particulate air pollution in adults in the United States, it is reasonable to hypothesize a similar association might be observed in infants in the United States.

This hypothesis is supported by several factors. First, ecological studies in other countries have found an association between particulate air pollution and infant mortality (*6–8*); however, it is difficult to apply these results to the United States because particulate matter levels in these countries have typically been much higher than in the United States. Second, the results of previous studies among adults suggest vulnerable individuals, such as the elderly and people with preexisting cardiovascular or respiratory conditions, are more susceptible to effects from exposure to particulate air pollution than the rest of the population (*9*). Because of their high mortality rates, infants, particularly those born prematurely, may also fall into this group of susceptible individuals. Last, studies have found associations between particulate air pollution and respiratory illness in children (*10,11*).

Many studies of adults have examined the association of particulate air pollution with overall adult mortality as well as with specific causes of mortality (*9,12*). However, such an inclusive approach may not be appropriate for an analysis of air quality and mortality among infants. A majority of infant deaths are unlikely to be influenced by air pollution levels because they occur too soon after birth (*13*) or are due to causes clearly intrinsic to the infant, such as congenital anomalies (*14*). To focus only on infant deaths that may plausibly be associated with particulate air pollution, we examined the relationship between exposure to particulate air pollution and selected causes of postneonatal mortality in the United States. Postneonatal death (death of an infant over 27 days of age) is thought to be influenced more by the infant's external environment than is mortality earlier in infancy (*15*). In keeping with the previously demonstrated relationship between particulate air pollution and childhood respiratory illness (*10,11*), an association between particulate air pollution and postneonatal mortality from respiratory causes can be postulated. Similarly, because several studies have suggested that sudden infant death syndrome (SIDS) is associated with exposure to environmental tobacco smoke (*16,17*), a potential association between SIDS and particulate air pollution is plausible.

Daily time–series analyses are commonly used to evaluate the relationship between short-term exposure to particulate air pollution and adult mortality. This method is not appropriate for an analysis of postneonatal mortality and pollution because of the small numbers of infants who die in a specific location over a short period of time. In adults, cohort analyses of specific cities or areas have been used to examine associations between mortality and chronic or long-term exposure to particulate matter. While this general approach may be feasible for a similar analysis among infants, geographically limited cohort analyses will also lack power due to small numbers.

The national-linked birth/infant death records provide a unique database for the examination of particulate air pollution and infant mortality. The birth certificate portion

Address correspondence to T.J. Woodruff, U.S. Environmental Protection Agency, 401 M St. SW (2126), Washington, D.C. 20460 USA.

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of each record contains individual level information on parental risk factors for infant mortality, while the death certificate portion contains data on the cause and time of death. Thus, potentially important confounders can be controlled for in the evaluation of the relationship between particulate air pollution and infant mortality. The purpose of this study is to examine the relationship between chronic exposure to particulate air pollution and postneonatal mortality from all causes, from respiratory causes, and from SIDS in the United States.

Methods

In this study, we combined infant mortality data from the National Center for Health Statistics (NCHS) with air quality data from the EPA. Information on infant outcome and maternal and infant characteristics was obtained from the NCHS-linked birth/infant death data files for 1989–1991, the most recent period for which those data were available (*18*). Infants born in California, Indiana, Louisiana, Nebraska, New York, Oklahoma, South Dakota, and Washington were excluded from the analysis because some of those states did not report maternal education and others did not report maternal smoking status on birth certificates for the period of interest. Although those states account for 29.5% of births in the United States, previous research has demonstrated that infants born in many of those states (including California and New York, which contained the majority of excluded births) have similar infant mortality characteristics as the nation as a whole (*19*). Due to federal confidentiality constraints, NCHS data files do not contain detailed geographic information for infants born in counties with populations less than 100,000; therefore, this study was limited to infants born in counties with populations of at least 100,000. In addition, only the month and year of birth and death are recorded; the exact date of birth or death is not available from the NCHS records.

Particulate matter air pollution data were obtained from the EPA's Aerometric Database. Monitoring data were obtained for all metropolitan statistical areas (MSAs) that monitored for particulate matter of 10 μm or less (PM_{10}). The infant mortality and PM_{10} files were linked at the MSA level using 1990 Federal Information Processing Standards (FIPS) codes common to both files. One hundred and one MSAs from the linked birth/infant death file met the geographic and population inclusion criteria previously mentioned, and 86 of those MSAs were included in the EPA database.

PM_{10} exposure for each infant was based on PM_{10} levels in the infant's MSA of residence at the time of birth. For this study, an infant's exposure was considered to be the mean of the PM_{10} levels for the first 2 months of life. Two months was chosen as the exposure period because all infants in the study had PM_{10} exposure for the month of birth and at least part of the next month. The alternative, defining exposure as the average of the PM_{10} levels over an infant's entire lifetime, leads to an overestimate of the influence of PM_{10} on mortality because of declines in PM_{10} levels in the United States over time. For instance, two infants born in the same month in the same MSA will have identical exposures for the first 2 months of life. If one of the infants should die in his second month and one should survive infancy, the survivor will have a lower overall exposure because of the decreasing secular trend. Thus, the surviving infant will appear to have a lower exposure, even though the infants had identical exposure up to the first infant's death, resulting in an artificially high estimate of the association between PM_{10} and mortality.

For categorical analyses, infants were categorized as having low, medium, or high PM_{10} exposure depending on whether their 2-month mean exposure was in the bottom one-third, middle one-third, or top one-third of the range of exposures. The overall range of infant exposures was 11.9–68.8 $\mu g/m^3$. To minimize the influence of outliers, the top and bottom 1% of exposures were not used in the determination of PM_{10} exposure category. The resulting low, medium, and high exposure ranges were <28.0, 28.1–40.0, and >40.1 $\mu g/m^3$.

The outcome of interest in this study, postneonatal mortality, was defined as the death of an infant between 1 month and 1 year of age. The postneonatal mortality rate was calculated as the number of postneonatal deaths per 1,000 infants who survived the neonatal period (birth through 1 month). To focus the analysis on deaths that may plausibly be related to particulate matter, our study examined four postneonatal outcomes: 1) all postneonatal deaths; 2) normal birth weight (NBW, ≥2,500 grams) SIDS deaths; 3) NBW respiratory deaths; and 4) low birth weight (LBW, <2,500 grams) respiratory deaths. Respiratory deaths are those deaths with a reported underlying cause of death identified by the *International Classification of Diseases*, Revision 9 (ICD-9), codes 460–519. SIDS deaths are those with an ICD-9 cause of death of 798.0. Mortality rates by birth weight category were examined to test the hypothesis that particulate matter may have a stronger influence among more vulnerable populations, specifically among LBW infants. A control disease category of all other causes of death was also examined.

Postneonatal mortality rates were calculated within each of the three pollution categories for each outcome. Logistic regression was used to adjust for potential confounding variables (*20,21*). These variables were obtained from the birth certificate portion of the NCHS file and include maternal education (<12 years, ≥12 years), maternal race (black, white, other), parental marital status, and maternal smoking during pregnancy (no, yes). Separate models were run with PM_{10} levels entered as a categorical variable and as a continuous variable.

Past studies of factors associated with perinatal outcome have shown a differential effect of demographic characteristics, such as education, on outcome for black infants and white infants (*19,22*). Accordingly, interaction terms were added to the logistic regression models to assess potential effect modification. Neither substantial nor significant (all p-values >0.2) differential effect by race was observed. Infants with information missing for any of the other variables (e.g., maternal education) were excluded from the analysis. Approximately 7% of infants were excluded because of missing data.

Climate is also a potential confounder in this analysis because temperature may be related to the risk of postneonatal death (SIDS, in particular) and air quality. Monthly temperatures throughout the study for each MSA were obtained from the National Center for Climactic Research, University of Delaware. For each infant, the average temperature during the first 2 months of life (analogous to the calculation of PM_{10} exposure) was entered into the logistic models.

The date of birth may also have confounded the relationship between mortality and particulate matter in this analysis because both infant mortality and PM_{10} levels decreased during the study period. To control for this possibility, a variable accounting for each infant's month and year of birth was entered into the logistic models.

Results

A total of almost 4 million infants residing in 86 MSAs were included in this analysis (Table 1). Thirty-six percent of all infants were in the low exposure category, 15% had high PM_{10} exposure, and the remaining 49% of infants were in the middle exposure category. Infants with high exposure were the most likely to have low maternal education and unmarried parents and to have been exposed to maternal smoking during pregnancy.

Overall postneonatal mortality increased with increasing PM_{10} levels, from 3.1 in the low pollution category to 3.7 in the high category (Table 2). Postneonatal mortality increased with increasing PM_{10} exposure for each of the birth weight and cause-specific outcomes examined in this study. The postneonatal SIDS rate among normal birth weight infants with high PM_{10} exposure was 26% higher than among normal birth weight infants with low exposure. Normal birth weight infants with high PM_{10} exposure were 45% more likely to die of respiratory causes than normal birth weight infants with low exposure. Respiratory mortality among low birth weight infants was 35% higher in the high exposure group than in the low PM_{10} exposure group. Conversely, postneonatal mortality from all other causes of death did not consistently increase with increasing PM_{10} exposure and was only 8% higher among high exposure infants than among low exposure infants.

Adjustment for confounding variables weakened the associations between PM_{10} exposure and postneonatal mortality, but substantial differences between the high and low exposure groups remained. Table 3 displays the categorical and continuous odds ratios (ORs) for mortality by PM_{10} level after adjustment for maternal education, marital status, maternal race, maternal smoking during pregnancy, average temperature exposure, and year and month of birth. After adjustment, infants with high levels of PM_{10} exposure were at approximately 10% higher risk of postneonatal death than were infants with low exposure.

Further categorical analysis by cause of death showed a significant relationship between PM_{10} level and SIDS among normal birth weight infants. The adjusted ORs and 95% confidence intervals (CI) for infants with medium and high PM_{10} exposures were 1.09 (1.01–1.17) and 1.26 (1.14–1.39), respectively. Among normal birth weight infants with high PM_{10} exposure, the adjusted OR for respiratory mortality was 1.40 (1.05–1.85). The adjusted ORs for respiratory death among low birth weight infants showed an increased risk of approximately 18% for infants with high PM_{10} exposure compared with infants with low exposure, although the 95% CI includes 1.0.

The continuous ORs in Table 3 show the relative change in risk of death associated with a 10 $\mu g/m^3$ increase in PM_{10} level after adjustment for confounding variables. Overall postneonatal mortality increased by 4% for each 10 $\mu g/m^3$ rise in PM_{10}. SIDS among normal birth weight infants increased by 12% for each 10 $\mu g/m^3$ increase in PM_{10}. Postneonatal respiratory mortality among normal birth weight infants increased 20% per 10$\mu g/m^3$, while postneonatal respiratory mortality among low birth weight infants increased by 5%. No association was observed for other causes.

Table 1. Descriptive characteristics of infants by pollution exposure

Variable	All infants	Pollution category		
		Low[a]	Medium[b]	High[c]
Number of infants	3,788,079	1,374,649	1,858,649	554,781
Percent of infants	–	36	49	15
Race				
Black infants (%)	23.3	19.9	25.3	25.3
White infants (%)	73.0	75.0	71.7	72.0
Maternal education				
<12 years (%)	21.2	20.57	20.35	23.68
Parents' marital status				
Unmarried (%)	30.1	27.9	31.0	32.6
Maternal smoking				
Yes (%)	16.8	16.8	16.6	17.5
PM_{10} ($\mu g/m^3$)[d]	31.4 ± 7.8	23.6 ± 3.3	33.3 ± 3.4	44.5 ± 4.6
PM_{10} (range)	11.9–68.8	11.9–28.0	28.01–40.0	40.01–68.8

PM_{10}, particulate matter <10 μm in diameter.
[a]Low pollution: PM_{10} = 11.9–28.0 $\mu g/m^3$.
[b]Medium pollution: PM_{10} = 28.01–40.0 $\mu g/m^3$.
[c]High pollution: PM_{10} = 40.1–68.8 $\mu g/m^3$.
[d]Mean ± standard deviation.

All continuous and categorical models were also run without maternal smoking, temperature, and timing of birth. The relationships between PM_{10} exposure and postneonatal mortality in those models were virtually identical to the relationships shown in Table 3 (data not shown).

Discussion

To our knowledge, this is the first study to concentrate on the relationship between air quality and infant mortality in the United States. This analysis demonstrates an association between PM_{10} levels and several causes of postneonatal mortality. Specifically, the risk of SIDS among normal birth weight infants and respiratory mortality among normal and low birth weight infants was higher among infants with high PM_{10} exposure than among those with low exposure, though the adjusted OR for low birth weight respiratory mortality was not statistically significant. The risk of postneonatal mortality from any cause was approximately 10% higher in infants in the high PM_{10} category than in those in the low PM_{10} category, after adjustment for potential confounders. It appears that the overall elevated risk is probably due to the association between PM_{10} level and SIDS and respiratory-related mortality because there was no association with other causes of death.

Previous studies in Taiwan (*7*) and the Czech Republic (*6*) have also found an association between particulate air pollution and infant mortality. Bobak and Leon (*6*) used ecological analysis to evaluate the relationship between annual infant mortality between 1986 and 1988 and long-term exposure to several measures of pollution [PM_{10}, SO_2, and oxides of nitrogen (NO_x)] in the Czech Republic. They found a consistent positive association between PM_{10} levels and postneonatal infant mortality, even after adjusting for sociodemographic variables and the other pollutants. The associations with the other pollutants were inconsistent. Knoebel et al. (*7*) evaluated the relationship between short-term levels of particulate air pollution as measured by visibility and SIDS in Taiwan. They found positive significant associations between SIDS and visibility measured on the day before death and a stronger association with 9 days before death after adjusting for a number of covariates. The PM_{10} levels in the Czech and Taiwan studies (*6,7*) were much higher than levels in our analysis [mean of 68.5 in the Czech study (*6*); values ranged between 36 and 122 $\mu g/m^3$ in the Taiwan study (H. Knoebel, personal communication) versus 31 $\mu g/m^3$ in this analysis], yet all studies find a consistent, positive association between PM_{10} levels and postneonatal mortality.

Table 2. Birth weight and cause-specific postneonatal mortality rates by pollution exposure

Postneonatal deaths	Mortality rates per 1,000 births			
	All infants	Pollution exposure		
		Low[a]	Medium[b]	High[c]
All causes	3.39	3.11	3.50	3.70
SIDS[d], NBW	1.10	1.00	1.13	1.26
Respiratory[e], NBW	0.13	0.11	0.13	0.16
Respiratory[e], LBW	1.32	1.24	1.26	1.68
Other causes	1.88	1.77	1.96	1.91

Abbreviations: PM_{10}, particulate matter <10 μm in diameter; SIDS, sudden infant death syndrome; NBW, normal birth weight; LBW, low birth weight; ICD-9, *International Classification of Diseases,* Revision 9.
[a]Low pollution: PM_{10} = 11.9–28.0 $\mu g/m^3$.
[b]Medium pollution: PM_{10} = 28.01–40.0 $\mu g/m^3$.
[c]High pollution: PM_{10} = 40.01–68.8 $\mu g/m^3$.
[d]SIDS: ICD-9 = 798.0
[e]Respiratory: ICD-9 = 460.0–519.9.

Table 3. Adjusted odds ratios[a] and 95% confidence intervals for cause-specific postneonatal mortality by pollution exposure

Cause of postneonatal death	Pollution category			Continuous
	Low[b]	Medium[c]	High[d]	10 μg/m³ change
All causes	Ref	1.05 (1.01–1.09)	1.10 (1.04–1.16)	1.04 (1.02–1.07)
SIDS,[e] NBW	Ref	1.09 (1.01–1.17)	1.26 (1.14–1.39)	1.12 (1.07–1.17)
Respiratory death,[f] NBW	Ref	1.08 (0.87–1.33)	1.40 (1.05–1.85)	1.20 (1.06–1.36)
Respiratory death,[f] LBW	Ref	0.93 (0.73–1.18)	1.18 (0.86–1.61)	1.05 (0.91–1.22)
All other causes	Ref	1.03 (0.97–1.08)	0.97 (0.90–1.04)	1.00 (0.99–1.00)

Abbreviations: Ref, reference; SIDS, sudden infant death syndrome; NBW, normal birth weight; LBW, low birth weight; PM_{10}, particulate matter <10 μm in diameter; MSA, metropolitan statistical areas; ICD-9, *International Classification of Diseases*, Revision 9.

[a]Adjusted for maternal race, maternal education, marital status, month of birth, maternal smoking during pregnancy, and average MSA temperature for the first 2 months of life.

[b]Low pollution: PM_{10} = 11.90–28.00 μg/m³.

[c]Medium pollution: PM_{10} = 28.01–40.00 μg/m³.

[d]High pollution: PM_{10} = 40.01–68.80 μg/m³.

[e]SIDS: ICD-9 = 798.0.

[f]Respiratory: ICD-9 = 460.0–519.9.

While no other study in the United States has examined mortality effects in children, this study is consistent with two similarly designed prospective cohort studies of adult mortality evaluating the effects of long-term exposure to particulate air pollution: the Harvard Six City study (*4*) and the American Cancer Society study (*5*). In the Harvard Six City study, Dockery et al. (*4*) analyzed survival of 8,111 adults followed for 14 years in six cities in the eastern United States. After adjusting for sociodemographic risk factors, the authors found a 27% increased risk for mortality between the cities with the highest and lowest levels of inhalable particulate matter. Similarly, Pope et al. (*5*) used 7-year survival data between 1982 and 1988 for over half a million adults in 151 U.S. cities, using the American Cancer Society database. After adjusting for sociodemographic risk factors, they found fine particle concentrations measured in the beginning of the study, represented by both sulfates and particles less than 2.5 μm, were associated with a 15% and 17% increase in mortality, respectively. Using the results from the logistic model employing a continuous representation of PM_{10}, this study estimated a 25% increase in risk of postneonatal death between infants exposed to the highest and lowest levels of PM_{10}, after adjustment for potential confounding factors. This increased risk is similar to the relative risk estimates observed in the previous two prospective cohort studies. In addition, the Harvard Six City study (*4*) and the American Cancer Society study (*5*) found higher associations with cardiopulmonary related causes, which is similar to this study.

The results of this study are also consistent with the current literature on the mortality and morbidity effects associated with particulate matter. In addition to the prospective cohort studies, numerous studies have observed associations between mortality and short-term exposure to PM_{10} in adults (*2,3,12*). Analysis by cause of death finds a stronger relationship between PM_{10} and respiratory-related causes (*2,3,12*). Also, a number of studies have reported positive associations between respiratory-related morbidity and particulate matter (*23–25*). In particular, several studies report an association between respiratory effects in children and both short-term and long-term exposure to PM_{10} (*10,11,26*).

An intriguing finding of this study is the association between high PM_{10} levels and SIDS. An association between SIDS and environmental tobacco smoke has been demonstrated previously, and tobacco smoke contains a substantial proportion of particulate matter (*27*). Further exploration is needed to elucidate the observed associations and clarify clues they might give as to the etiology and prevention of SIDS.

In this study, the strength of association between PM_{10} exposure and mortality decreased after control for demographic factors and maternal smoking during pregnancy. The logistic models used in the analysis were parsimonious, but included the most important variables associated with infant mortality that are available from the linked birth/infant death file. Similar studies among adults have considered other risk factors such as occupational exposures, body mass index, and alcohol consumption. While those specific factors may not be relevant to an examination of infant mortality, it is possible that additional characteristics of the infant, the parents, or the environment may be confounding the relationships between particulate matter and postneonatal mortality observed in this study. It is important to note, however, that in previous prospective cohort studies, adjustment for additional factors did not significantly alter the observed relationships between particulate air pollution and adult mortality (*4,5*).

One difference between particulate matter studies among adults and similar studies among children is that the particulate matter levels are more likely to represent a consistent chronic exposure among infants than among adults, who are more liable to have moved during their lifetime. However, this study has the limitation, as do other epidemiological studies of PM_{10}, of only using measured levels to represent ambient concentrations, which do not necessarily represent exposure to an individual. A limited number of studies have been conducted to assess the relationship between monitored levels of PM_{10} and personal exposures. Studies have found that monitored PM_{10} levels are highly correlated with levels at specific locations (e.g., between a central monitor and one in the backyard of a home) within the monitored area (*2,28,29*). Also, PM_{10} levels are different, but correlated, between indoors and outside (*2,28–30*). By limiting this study to postneonatal mortality, we hoped to minimize the influence of mortality among infants who never leave the hospital between birth and death and therefore may not have been exposed to the monitored levels of PM_{10}. The inclusion of SIDS should further assure a population of infants that is exposed to the ambient environment because the diagnosis of SIDS is limited to essentially healthy infants who have no medical reason to be confined to the indoors.

There is a high correlation between indoor and outdoor levels of fine particulate matter, defined as particles of 2.5 μm or less ($PM_{2.5}$) (*28,29*). The relationship between $PM_{2.5}$ and adult mortality was examined in both the Harvard Six City study (*4*) and the American Cancer Society study (*5*), both of which found a significant association between mortality and chronic exposure to fine particles and sulfates. It is possible that PM_{10} levels in this analysis are a surrogate for fine particles, which includes all particles less than 2.5 μm in size that are components of PM_{10}. There are few $PM_{2.5}$ and sulfate monitoring data available for 1989–1991 so it is not possible to determine if specific components of PM_{10} account for the associations found in this study.

A number of previous studies have demonstrated a positive association between particulate matter and mortality among adults in the United States. This analysis indicates that particulate matter may also influence an infant's chance of survival. While further examination of

this association is needed, the results of this study, combined with results from studies examining air pollution and morbidity among children, suggest that continued attention must be paid to the nation's air quality to ensure the optimal health of infants and children in the United States.

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