Journal of Health Economics 28 (2009) 688–703

Contents lists available at ScienceDirect

Journal of Health Economics

journal homepage: www.elsevier.com/locate/econbase

Air pollution and infant health: Lessons from New Jersey☆

Janet Currie*, Matthew Neidell, Johannes F. Schmieder

Columbia University, Department of Economics, International Affairs Building, 420 W. 118th Street, New York, NY 10027, United States

ARTICLE INFO

Article history:
Received 22 July 2008
Received in revised form 25 January 2009
Accepted 11 February 2009
Available online 27 February 2009

JEL classification:
I18
Q53

Keywords:
Air pollution
Infant health
Carbon monoxide
Birth weight
Infant mortality

ABSTRACT

We examine the impact of three "criteria" air pollutants on infant health in New Jersey in the 1990s by combining information about mother's residential location from birth certificates with information from air quality monitors. Our work offers three important innovations. First, we use the exact addresses of mothers to select those closest to air monitors to improve the accuracy of air quality exposure. Second, we include maternal fixed effects to control for unobserved characteristics of mothers. Third, we examine interactions of air pollution with smoking and other risk factors for poor infant health outcomes. We find consistently negative effects of exposure to carbon monoxide (CO), both during and after birth, with effects considerably larger for smokers and older mothers. Since automobiles are the main source of carbon monoxide emissions, our results have important implications for regulation of automobile emissions.

The primary goal of pollution abatement is to protect human health, but there is still much debate about the specific health effects. This paper addresses this issue by examining the impact of air pollution on infant health in New Jersey over the 1990s. Policy makers and the public are highly motivated to protect these most vulnerable members of society. There is increasing evidence of long-term effects of poor infant health on future outcomes; for example, low birth weight has been linked to future health problems and lower educational attainment (see Currie (2008) for a summary of this research). Studying infants also overcomes several empirical challenges because, unlike adult diseases that may reflect pollution exposure that occurred many years ago, the link between cause and effect is more immediate.

Our analysis improves upon much of the previous research by improving the assignment of pollution exposure from air quality monitors to individuals. Most observational analyses that assess the impact of air pollution on health assign exposure to pollution by either approximating the individual's location as the centroid of a geographic area or computing average pollution levels within the geographic area. In our data we know the exact addresses of mothers, enabling us to improve on the assignment of pollution exposure.

Despite this improvement in pollution measurement, we must still confront the problem that air pollution is not randomly assigned, making potential confounding a major concern. Since air quality is capitalized into housing prices (Chay and Greenstone, 2003a,b) families with higher incomes or preferences for cleaner air are likely to sort into locations with better air quality, and failure to account for this will lead to overestimates of the effects of pollution. Alternatively, pollution levels are higher in urban areas where there are often more educated individuals with better access to health care, which can cause underestimates of the effects of pollution. Our data permits us to follow mothers over time, so we include both pollution monitor and maternal fixed effects to capture all time-invariant characteristics of the neighborhood and mother. In our richest specification, the effects of pollution are identified using variation in pollution exposure between children in the same families, after controlling flexibly for time trends, seasonal patterns, weather, pollution monitor locations, and several observed characteristics of the mother and child.

Infants at higher risk of poor outcomes may be differentially affected by pollution, so we also examine whether pollution has a differential impact on infant health depending on maternal characteristics, such as whether the mother smoked during pregnancy and older maternal age. Previous research has suggested that smoking

☆ We are grateful for funding under NIH grant R21 HD055613-01. All opinions and any errors are our own. We would also like to thank Katherine Hempstead and Matthew Weinberg of the New Jersey Department of Health for facilitating our access to the data. Seminar participants at Tilburg University provided helpful comments.

* Corresponding author. Tel.: +1 212 854 4520; fax: +1 212 854 8059.
E-mail address: jc2663@columbia.edu (J. Currie).

0167-6296/$ – see front matter © 2009 Elsevier B.V. All rights reserved.
doi:10.1016/j.jhealeco.2009.02.001

might exacerbate the effect of air pollution by increasing inflammatory responses and airway reactivity (Xu and Wang, 1998). Alternatively, since cigarette smoke contains high levels of pollutants, including carbon monoxide (CO), infants may already be exposed to high levels so that the marginal impact may be smaller in smokers than in non-smokers if the effects of pollutants are non-linear. Previous work has also suggested that infants of older mothers might be more susceptible to problems related to smoking (Cnattingius, 1997), so it is also possible that these infants are more vulnerable to the effects of pollution. To our knowledge, this is the first study to ask whether there are such differential effects.

Our estimates confirm that carbon monoxide has a significant effect on fetal health even at the relatively low levels of pollution experienced in New Jersey in recent years, and that it has further effects on infant mortality conditional on measures of health at birth. In particular, we estimate that a one unit change in mean CO during the last trimester of pregnancy increases the risk of low birth weight by 8%. Furthermore, a one unit change in mean CO during the first 2 weeks after birth increases the risk of infant mortality by 2.5% relative to baseline levels. These findings for CO are robust to many different specifications. We also find that the effects of CO on infant health at birth are two to six times larger for smokers and for mothers over age 35. Since the major source of CO in urban areas is automobile exhaust, these findings have implications for regulations of automobile emissions.

The rest of the paper is laid out as follows. Section 1 provides necessary background about the ways in which pollution may affect infant health and the previous literature. Section 2 describes our methods, while data are described in Section 3. Section 4 presents our results, and Section 5 details our conclusions.

1. Background

A link between air pollution and infant health has long been suspected although the exact biological mechanisms through which it occurs are not well understood. Carbon monoxide is an odorless, colorless gas that primarily comes from transportation sources, with as much as 90% of CO in cities coming from motor vehicle exhaust (Environmental Protection Agency, January 1993, 2003). CO bonds with hemoglobin more easily than oxygen, reducing the body's ability to deliver oxygen to organs and tissues. While CO is poisonous to healthy adults at high levels, infants are particularly susceptible because they are smaller and often have existing respiratory problems. In pregnant women, exposure to CO reduces the availability of oxygen to be transported to the fetus. Moreover, carbon monoxide readily crosses the placenta and binds to fetal haemoglobin more readily than to maternal haemoglobin and is cleared from fetal blood more slowly than from maternal blood, leading to concentrations that may be 10–15% higher in the fetus's blood than in the mother's. Indeed, much of the negative effect of smoking on infant health is believed to be due to the CO contained in cigarette smoke (World Health Organisation, 2000).

Particulate matter can take many forms, including ash and dust, and motor vehicle exhaust is a major source. The smallest particles are widely believed to cause the most damage since they are inhaled deep into the lungs and can possibly enter the bloodstream (Environmental Protection Agency, 2003). The mechanisms through which particles harm health are controversial, with a leading theory being that they cause an inflammatory response that weakens the immune system (Seaton et al., 1995). Since particles cannot cross the placenta, they would have to damage the fetus indirectly by provoking inflammation in the mother.

Ozone (the major component of smog) is formed through reactions between nitrogen oxides and volatile organic compounds (which are found in auto emissions, among other sources) in heat and sunlight. Ozone is a highly reactive compound that damages tissue, reduces lung function, and sensitizes the lungs to other irritants. For example, exposure to ozone during exercise reduces lung functioning in adults and causes symptoms such as chest pain, coughing, and pulmonary congestion. It is not clear why ozone would affect the fetus, though like PM10 it might indirectly affect the infant by compromising the mother's health.

The discussion suggests that one might well expect CO to have larger effects than other pollutants because of its ability to cross the placenta and accumulate in the blood of the fetus. However, pollution exposure could indirectly affect the fetus through the health of the mother by, for example, weakening her immune system. Moreover, all three pollutants can directly affect infants after birth.[1] Although the available research points towards potential impacts, it provides little guidance about the necessary levels of pollution to induce negative effects or when fetuses or infants are most vulnerable.

Many epidemiological studies have demonstrated links between very severe pollution episodes and increased mortality of infants and others. One of the most famous focused on a "killer fog" in London, England and found dramatic increases in cardiopulmonary mortality (Logan and Glasg, 1953). It has been less clear whether levels of air pollution that are common in the U.S. today have effects on infant health.

Previous epidemiological research on the effects of moderate pollution levels on prenatal health suggest negative effects but have produced inconsistent results. Chart 1 provides a list of previous studies examining this relationship, limiting our review to developing countries that are likely to have comparable levels of pollutions to New Jersey For example, Ritz and Yu (1999) report that CO exposure in the last trimester of pregnancy increased the incidence of low birth weight (defined as birth weight less than 2500 g), while Ritz et al. (2000) report that CO exposure in the 6 weeks before birth is correlated with gestation in some regions of southern California but not in others. Ritz et al. (2000) report that PM10 exposure 6 weeks before birth increases preterm birth, while Maisonet et al. (2001) find that PM10 has no effect on low birth weight.

Studies of the effects of pollution on infant mortality also yield mixed results. For example, Woodruff et al. (1997) report that infants with high exposure to PM10 are more likely to die in the post neonatal period. But Lipfert et al. (2000) find that although they can reproduce some earlier results showing effects of county-level pollution measures on infant mortality, the results are not robust to including controls for maternal characteristics.

An important limitation of these studies is that the observed relationships could reflect unobserved factors correlated with both air pollution and child outcomes. Many of the studies in Basu et al., 2004; Bell et al., 2007; Brauer et al., 2008; Chen et al., 2002; Dugandzic et al., 2006; Friedman et al., 2001; Huynh et al., 2006; Lee et al., 2008; Liu et al., 2003; Liu et al., 2007; Parker et al., 2008; Parker and Woodruff, 2008; Parker et al., 2005; Ritz et al., 2007; Ritz et al., 2006; Rogers and Dunlop, 2006; Rogers et al., 2000; Sagiv et al., 2005; Salam et al., 2005; Wilhelm and Ritz, 2005; Chart 1 have very minimal (if any) controls for potential confounders. Families with higher incomes or greater preferences for cleaner air may be

[1] Alternatively, since motor vehicle exhaust is a major contributor of CO and PM10, these pollutants may themselves be markers for other components of exhaust which injure infants. Components such as polycyclic aromatic hydrocarbons (PAHs), acetonitrile, benzene, butadiene, and cyanide (see http://www.epa.gov/ttn/atw/hapindex.html) have been shown to have effects on developing fetuses in animal studies, such as retarded growth. Studies in humans have shown elevated levels of an enzyme induced by PAHs in women about to have preterm deliveries (Huel et al., 1993).

Study	Location	Years	Outcomes	Pollutants	Effects
Lipfert et al. (2000)	All of U.S.	1990	infant mortality	PM10, CP, SO2, SO4, PM2.5	County level annual avg. pollution measures did not have robust relationships to pollutants when maternal variables were controlled.
Liu et al. (2003)	Vancouver, Canada	1985-1998	low BW, preterm birth, growth retard.	CO, NO2, SO2, O3	SO2 in 1st month increases LBW. SO2 and CO in last month increases preterm birth. Growth retardation associated with CO, NO2, SO2 in 1st month.
Mainsonet et al. (2001)	Northeastern U.S.	1994-96	low BW	CO, PM10, SO2	CO in last trimester and SO2 in 2nd trimester increase LBW. No effect of PM10.
Ritz et al. (2000)	Los Angeles	1989-1993	preterm birth	CO, NO2, O3, PM10	PM10 exposure 6 weeks before birth increases preterm birth. CO exposure in same interval has effects only in some areas.
Ritz and Yu (1999)	Los Angeles	1989-1993	low BW	CO, NO2, O3, PM10	CO exposure in last trimester increased incidence of low BW.
Woodruff et al. (1997)	86 U.S. MSAs	1989-91	infant mortality	PM10	Infants with high exposure more likely to die in postneonatal period.
Bell et al. (2007)	MA, CT	1999-2002	low BW	PM10&2.5, SO2, NO2, CO	All pollutants at various trimesters related to birthweight
Brauer et al. (2008)	Vancouver, BC	1999-2002	low BW, gestation	PM10&2.5, SO2, NO2, CO, O3	gestation associated with all but O3, birthweight associated with PM2.5
Chen et al (2002)	Washoe County, NV	1991-99	low BW	PM10, CO, O3	no associations after adjusting for confounding
Huynh et al. (2006)	California	1999-2000	gestation	PM2.5, CO	Exposure to PM2.5 but not CO associated with preterm birth
Lee et al. (2008)	London, UK	1988-2000	gestation	PM10, O3	no association
Liu et al. (2007)	Calgary, Edmonton, Montreal	1985-2000	IUGR	SO2, NO2, CO, O3, PM2.5	CO, NO2 & PM2.5 associated with IUGR
Parker et al. (2008)	Utah	1984-90	low BW, gestation	Steel mill closure	association with gestation but not birthweight
Parker et al. (2005)	California	2000	low BW	PM2.5, CO	Exposure to PM2.5 but not CO associated with birthweight
Parker & Woodruff (2008)	US	2001-03	low BW	PM10&2.5	Association with PM10 but not PM2.5; varied by region
Ritz et al. (2007)	LA County, CA	2003	gestation	CO, NO2, O3, PM2.5	CO & PM2.5 assocaited with preterm birth
Ritz et al. (2006)	LA County, CA	1989-2000	mortality	CO, NO2, O3, PM10	CO and PM10 associated with mortality
Sagiv et al. (2005)	4 Pennsylvania counties	1997-2001	gestation	PM10, SO2	Both associated with preterm birth
Salam et al. (2005)	California	1975-87	low BW	CO, PM10, O3, NO2	O3 during 2nd and 3rd trimester, CO during first
Woodruff et al. (2008)	US counties > 250k	1999-2002	mortality (by cause)	PM2.5, PM10, CO, O3, SO2	PM10 associated with respiratory; O3 associated with SIDS
Rogers & Dunlop (2006)	Rural counties in Georgia	1986-1988	very low BW	PM10	PM10 associated with very low BW
Rogers et al. (2000)	Rural counties in Georgia	1986-1988	very low BW	SO2, TSP	Above median exposure associated with increase in very low BW
Dugandzic et al. (2006)	Nova Scotia, CA	1988-2000	low BW	PM10, SO2, O3	No relationship after adjusting for confounders
Basu et al. (2004)	California	2000	low BW	PM2.5	PM2.5 associated with lower birthweight; fairly comparable across methods
Wilhelm & Ritz (2005)	LA County, CA	1994-2000	low BW, gestation	PM10, PM2.5, CO	CO and PM10 associated with both if within 1 mile

Notes: BW = birth weight

Chart 1. Selected epidemiological studies of effects of pollution on infant health, developed countries.

more likely to sort into neighborhoods with better air quality. These families are also likely to provide other investments in their children, so that fetuses and infants exposed to lower levels of pollution also receive more family inputs, such as better quality prenatal care. If these factors are unaccounted for, this would lead to an upward bias in estimates. Alternatively, pollution emission sources tend to be located in urban areas, and individuals in urban areas may be more educated and have better access to health care, factors that may improve health. Omitting these factors would lead to a downward bias, suggesting the overall direction of bias from confounding is unclear.

Two studies by Chay and Greenstone (2003a,b) deal with the problem of omitted confounders by focusing on "natural experiments" provided by the implementation of the Clean Air Act of 1970 and the recession of the early 1980s.[2] Both the Clean Air Act and the recession induced sharper reductions in particulates in some counties than in others, and they use this exogenous variation in levels of pollution at the county-year level to identify its effects. They estimate that a one unit decline in particulates caused by the implementation of the Clean Air Act (recession) led to between five and eight (four and seven) fewer infant deaths per 100,000 live births. They also find some evidence that the decline in TSPs led to reductions in the incidence of low birth weight. However, the levels of particulates studied by Chay and Greenstone are much higher than those prevalent today; for example, PM10 levels have fallen by nearly 50% from 1980 to 2000. Furthermore, only TSPs were measured during the time period they examine, which eliminates their ability to examine other pollutants that are correlated with particulates emissions.

Currie and Neidell (2005) extend this line of research by examining the effect of more recent levels of pollution on infant health, and by examining other pollutants in addition to particulates. Using within-zip code variation in pollution levels, they find that a one unit reduction in carbon monoxide over the 1990s in California saved 18 infant lives per 100,000 live births. However, they were unable to find any consistent evidence of pollution effects on health at birth. This paper improves on Currie and Neidell (2005) by using more accurate measures of pollution exposure, controlling for mother fixed effects, and investigating the interaction of air pollution with smoking and other risk factors.[3]

2. Methods

As discussed in the previous section, air pollution may affect infants differently before and after birth. Before birth, pollution may affect infants either because it crosses the protective barrier of the placenta or because it has a systemic effect on the health of the mother. After birth, infants are directly exposed to inhaled pollutants. Hence, our analysis proceeds in two parts: First we examine the effects of pollution on health at birth as measured by birth weight and gestation. Second, we examine the effect of pollution on infant mortality conditional on health at birth.

2.1. Modeling birth outcomes

In order to examine the effect of pollution on health at birth, we restrict the sample to women who lived within 10 km (about 6.2 miles) of a monitor and estimate baseline models of the following form:

$$O_{ijmt} = \sum_{s=1}^{3} (P_{mt}^{s}\beta^{s} + w_{mt}^{s}\gamma^{s}) + x_{ijmt}\delta + Y_t + \varepsilon_{ijmt} \quad (1)$$

where O is a birth outcome, i indexes the individual, j indexes the mother, m indexes the nearest monitor, and t indexes time periods. The vector P_{mt} contains measures of ambient pollution levels in each of the first, second, and third trimesters of the mother's pregnancy, denoted by s, using the monitor closest to the mother's residence. We construct the trimester measures by taking the average pollution measure over the trimester,[4] so β^s reflects the effect from a change in mean pollution levels for trimester s.[5] The w_{mt} represents daily precipitation and daily minimum and maximum temperature averaged over each trimester of the pregnancy. We control for weather in the vector w because it may have independent effects on birth outcomes and is correlated with ambient pollution levels (Samet et al., 1997).

The vector x_{ijmt} includes mother and child specific characteristics taken from the birth certificate that are widely believed to be significant determinants of birth outcomes. These characteristics include dummy variables for the mother's age (19–24, 25–34, 35+), mother's education (12, 13–15, or 16+ years), and birth order (2nd, 3rd, 4th or higher), an indicator for whether it is a multiple birth, whether the mother is married, whether the child is male, whether the mother is African-American, Hispanic, and other or unknown race, and whether the mother smokes, and the number of cigarettes if she smokes. Since these variables are all categorical, to preserve sample size we control for missing values by including an additional "missing" category for each variable. Appendix Table 1 shows the complete specification for one of our models that includes the coefficients on the dummy variables for missing controls. Given that family income is not included on the birth certificate, we also include a measure of median family income and the fraction of poor households in 1989 in the mother's census block group as a proxy. The vector Y_t includes month and year dummy variables to capture seasonal effects (pollution is strongly seasonal and birth outcomes may also be) as well as trends over time, such as improvements in health care.

As previously mentioned, a limitation of model (1) is that pollution exposure is likely to be correlated with omitted characteristics of families that are related to infant health. In order to control for omitted characteristics of neighborhoods and for differential seasonal effects in these characteristics (for example, coastal areas experience less economic activity in winter than in summer relative

[2] These studies are similar in spirit to a sequence of papers by C. Arden Pope, who investigated the health effects of the temporary closing of a Utah steel mill (Pope, 1989; Ransom and Pope, 1992; Pope et al., 1992) and to Friedman et al. (2001) who examine the effect of changes in traffic patterns in Atlanta due to the 1996 Olympic games. However, these studies did not look specifically at infants.

[3] Smoking data was not available in the California data used by Currie and Neidell (2005). An additional issue is that this paper (like the others discussed above) examines the effect of outdoor air quality measured using monitor in fixed locations. Actual personal exposures are affected by ambient air quality, indoor air quality, and the time the individual spends indoors and outdoors. One might expect, for example, that infants spend little time outdoors so that outdoor air quality might not be relevant. Research on the relationship between indoor and outdoor air quality (Spengler et al., 2000; Wilson et al., 2000) suggests that much of what is outdoors comes indoors. Furthermore, although the cross-sectional correlation between ambient air quality and personal exposure is low (between .2 and .6 in most studies of PM10 for e.g.), the time-series correlation is higher. This is because for a given individual indoor sources of air pollution may be relatively constant and uncorrelated with outdoor air quality. So for a given individual much of the variation in air quality comes from variation in ambient pollution levels.

[4] We describe these trimester measures in more detail in the following section.

[5] While this measure captures high ambient levels sustained over a period of time, we also estimated models using the maximum daily value of pollution over the same intervals, but found that it was not statistically significant in any of our models.

to inland areas), we estimate models of the form:

$$O_{ijmt} = \sum_{s=1}^{3}(P_{mt}^{s}\beta^{s} + w_{mt}^{s}\gamma^{s}) + x_{ijmt}\delta + Y_t + \varphi_{mt} * Q_t + \varepsilon_{ijmt} \quad (2)$$

where now φ_{mt} is a fixed effect for the closest air pollution monitor and $\varphi_{mt}{}^{*}Q_t$ is an interaction between the monitor effect and the quarter of the year. In this specification, we compare the outcomes of children who live in close proximity to each other and are born in the same quarter to capture average neighborhood characteristics within a season.

Model (2) may still suffer from omitted variables bias. In particular, unobserved characteristics of mothers, such as her regard for her own health, may be important for her infant's health and may also be correlated with her choice of neighborhoods. Hence, in our richest specification we estimate:

$$O_{ijmt} = \sum_{s=1}^{3}(P_{mt}^{s}\beta^{s} + w_{mt}^{s}\gamma^{s}) + x_{ijmt}\delta + Y_t + \varphi_{mt} * Q_t + \varsigma_j + \varepsilon_{ijmt} \quad (3)$$

where ζ_j is a mother-specific fixed effect. These models control for time-invariant characteristics of both neighborhoods and mothers, so that the effects of pollution are identified by variation in pollution at a particular monitor between pregnancies. Much of this variation is driven by changes in pollution levels over time, due to air quality regulations, and within the year, due to seasonal patterns in pollution and unpredictable variations in human activity.

A necessary condition to identify the impact of pollution is that variation in infants' pollution exposure is uncorrelated with other characteristics of the infant or the infant's families that may affect infant health. It would be a problem, for example, if first children were more likely to be low birth weight and mothers systematically moved to cleaner environments between the first and second births because their incomes increased. In order to check that the variation in pollution is uncorrelated with mobility, we performed the following exercise. We first estimated the actual "within family" variation in each pollutant. We then estimated what the within family variation would have been if each mother had stayed in the location in which she was first observed. The within family variances were virtually identical: the actual and simulated within standard deviations for ozone are 0.939 and 0.947, respectively, for CO are 0.301 and 0.271, respectively, and for PM10 are 0.410 and 0.407, respectively, for ozone. This suggests that mothers do not appear to be systematically moving to cleaner or dirtier areas between births.

2.2. Model for infant mortality

In order to examine infant mortality conditional on health at birth, we modify the birth outcomes model to capture the fact that birth outcomes are a one-time occurrence but mortality is a continuously updated outcome. For example, the risk of death is highest in the first week or two of life and drops sharply thereafter. Therefore, we estimate a weekly hazard model with time-varying covariates to account for a varying probability of survival and levels of pollution over the infants' first year of life. To do this, we treat an infant who lived for n weeks as if they contributed n person-week observations to the sample. The dependent variable is coded as 1 in the period the infant dies, and 0 in all other periods. Each time-invariant covariate (such as birth parity) is repeated for every period, while the time-varying covariates (such as pollution and weather) are updated each period.

Based on this data structure, we estimate a model in which the probability of death D_{ijmt} is specified as

$$D_{ijmt} = \alpha(t) + \sum_{\tau=1}^{4}(\theta^{\tau}P_{mt}\beta^{\tau} + w_{mt}^{s}\gamma^{s}) + x_{ijmt}\delta + O_{ijmt}\pi + Y_t + \varphi_{mt} * Q_t + \varsigma_j + \varepsilon_{ijmt} \quad (4)$$

where $\alpha(t)$ is a measure of duration dependence, specified as a linear spline function in the weeks since the infant's birth. We choose break points after 1, 2, 4, 8, 12, 20, and 32 weeks to capture the shape of the actual empirical hazard. P_{mt} measures exposure to the three pollutants in a given week. Since the infant death hazard varies greatly with time since birth, it is likely that an effect of pollution on infant death, if it exists, would also vary with the baseline hazard. We allow for such differential effects by interacting the weekly pollution measure P_{mt} with 4 dummy variables θ^{τ} indicating time since birth. θ^{1} equals one if time since birth is between 0 and 2 weeks, θ^{τ} between 2 and 4 weeks, θ^{3} between 4 and 6 weeks, and θ^{4} for over 6 weeks. Thus the effect of pollution as measured by β^{τ} can differ arbitrarily over these four intervals.

Because infant death might be affected by pollution before birth as well as by pollution after birth, we add birth weight as a measure of infant health outcomes at birth (O_{ijmt}) to the list of independent variables. We control for birth weight flexibly by including a series of dummy variables (<1500 g, 1500–2500 g, 2500–3500 g, and over 3500 g).[6] To the extent that birth weight is a sufficient statistic for health at birth, β^{τ} from Eq. (4) will capture the independent effect of pollution after birth conditional on health at birth.

This model can be thought of as a flexible, discrete-time, hazard model that allows for time-varying covariates, non-parametric duration dependence, monitor-specific quarter effects and mother fixed effects. Allison (1982) shows that estimates from models of this type converge to those obtained from continuous time models.

This procedure yields a very large number of observations since most infants survive all 52 weeks of their first year. In order to reduce the number of observations, we limit this part of the analysis to mothers who lost at least one child. In terms of observable characteristics, families with a death are more likely to have mothers who are African American (30% vs. 19% overall), unmarried (62% vs. 72% overall) and who are smokers (13% vs. 9.5% overall). However, mean ozone, CO, and PM10 measures in the trimester before birth are virtually identical in families with deaths and those without.[7]

One way to think about these estimates is in terms of underlying heterogeneity in the vulnerability of infants. Although the average family with a death is different than the average family without one, we are concerned about the impacts of pollution on the infant at the life/death margin. If the characteristics of the marginal infant who dies because of an increase in pollution is similar to the characteristics of the marginal infant who survives the same increase in pollution, then our results will tell us about the effects of variations in pollution for the range of pollution we observe.

3. Data

Detailed data on atmospheric pollution come from the New Jersey Department of environmental protection Bureau of Air Monitoring, accessed from the technology transfer network air quality system database maintained by the U.S. Environmental Protection

[6] Our results are, however, insensitive to including birth weight as a continuous variable.

[7] To the extent these conditions are not met, we will instead identify a local average treatment effect.

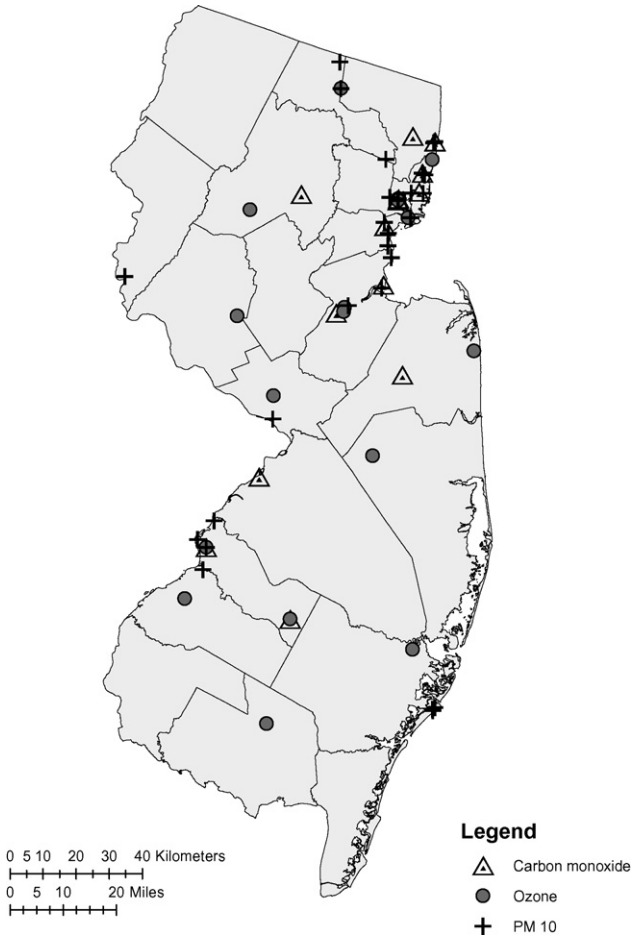

Fig. 1. Location of air monitors in New Jersey.

Agency (EPA).[8] The location of each of 57 monitors and what each one measures is shown in Fig. 1. Unfortunately, it is more the exception than the rule for a monitor location to measure all three of the pollutants that we study. PM10 is the most frequently monitored pollutant, followed by O3 and CO. Because of this limitation of the data, we will examine the impact of each pollutant in separate models (and samples), though we will also show one specification that includes both CO and O3, the two pollutants that have the largest effects individually. Fig. 1 demonstrates that monitors are heavily clustered in the most populated areas of the state, which lie along the transportation corridor between New York and Philadelphia.

For each monitor, we construct measures of pollution by taking the mean of the daily values either over the three trimesters before birth (for the birth outcomes models) or for each week after birth (for the infant mortality model). For the pollutants of interest, the daily measures we use are the 8-h maximums of CO and O3 and the 24-h average of PM10, which correspond with national ambient air quality standards.[9] County level weather data come from the Surface Summary of the Day (TD3200) from the National Climatic Data Center.[10]

Data on infant births and deaths come from the New Jersey Department of Health birth and infant death files for 1989 to 2003. Vital Statistics records are a very rich source of data that cover all births and deaths in New Jersey. Birth records have both detailed information about health at birth and background information about the mother, such as race, education, and marital status. We traveled to Trenton, New Jersey to use a confidential version of the data with the mother's address, name, and birth date. The use of this data allows us to more precisely match mothers to pollution monitors and to identify siblings born to the same mother. Births were linked to the air pollution measures taken from the closest monitor by using the mother's exact address and the latitude and longitude of the monitors. It was also possible to link birth and death records to identify infants who died in the first year of life.

Descriptive statistics for infant health outcomes, pollution measures, and control variables are shown in Table 1. The first four columns show means for all births in New Jersey, the sample of births with residential address that were successfully geocoded, the sample of births within 10 km of an ozone monitor, and the sample of births to smoking mothers within 10 km of an ozone monitor. Because different monitors measure different pollutants, the subsamples used in the regression models are slightly different.[11] Of the 1.75 million births in New Jersey over our sample period, 36% were successfully geocoded and within 10 km of an ozone monitor, with roughly 10% of these births to mothers who smoked. Column 5 restricts the sample further to children with a sibling within the sample, which is the final sample we use in our analysis. Almost 20% of the total births are in the sibling sample and within 10 km of a monitor. Finally, column 6 further restricts the final sample to the subset of mothers who smoked at both births, with the sample becoming much smaller but still sizable at 21,099 births.

A comparison of columns 1 and 2 shows no differences in maternal characteristics between successfully and unsuccessfully geocoded mothers. A comparison of columns 2, 3, and 4 of Panel A shows that infant health is worse in the population closer to monitors, and much worse in the sample of smokers. For example, the death rate is 6.9 per 1000 births overall, 7.7 in the sample closer to monitors, and 9.9 among the smokers. Comparing column 3 to column 5 or column 4 to column 6 suggests, however, that infants with siblings in the sample do not differ systematically from those without, which improves our ability to generalize results from the sibling regression models.

Panels B and C give means of the pollution measures for the subsets of the geocoded sample. A comparison of columns 3 and 4 suggests no systematic difference in air quality between the areas where smokers and nonsmokers live. Similarly, mothers with more than one birth over the sample period are exposed to comparable levels of air quality as mothers with a single birth.[12]

[8] The data is available at: <http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm>.

[9] The 8-hour maximum corresponds to taking the maximum 8-period moving average within a 24 h period. Although we choose these measures because they are based on air quality standards, the measures are highly correlated with other common measures of short-term spikes in pollutants. For example, the correlation between the maximum 8 hour reading for CO with the maximum 1 hour average for CO and daily mean for CO is 0.91 and 0.94, respectively. Comparable correlations for ozone are 0.98 and 0.93. These correlations are even higher within monitor, and our models incorporate monitor fixed effects. Since PM10 is not measured every day, the weekly mean for PM10 may be noisier than those for other pollutants.

[10] This data is available at http://www4.ncdc.noaa.gov/cgi-win/wwcgi.dll?wwAW~MP#MR. If weather data was not available for a county and date, we interpolated using data from surrounding counties. Our tests of this procedure (using counties with weather data) indicated that it was highly accurate.

[11] Sample sizes also vary slightly for different outcomes because of missing values for the outcomes.

[12] Although these mean pollution levels are far below air quality standards, the standards are based on daily maximum concentrations. For determining compliance with air quality standards for CO, the EPA calculates 8 h moving average values, and then asks whether the daily maximum of this moving average ever exceeds 9 ppm during the year. For ozone, the 3-year moving average of the fourth-highest daily maximum 8-hour average ozone concentrations must be less than .08 ppm. For

Table 1
Sample means.

	[1] All	[2] Geocoded	[3] <10 km monitor	[4] <10 km monitor and smoking	[5] Like (3) but ≥1 sibling	[6] Like (4) but ≥1 sibling
Number of observations	1,754,861	1,502,205	628,874	61,996	283,393	21,099
Panel A: outcomes						
Birth weight in grams	3320.2	3319.8	3267.3	3054.6	3236.4	2937.4
	[617.4]	[615.4]	[630.6]	[656.1]	[660.6]	[682.2]
Infant death	0.0073	0.0069	0.0078	0.0099	0.0086	0.0128
Gestation	38.86	38.83	38.71	38.28	38.55	37.84
	[2.672]	[2.302]	[2.475]	[2.892]	[2.643]	[3.212]
Low birth weight	0.077	0.076	0.089	0.157	0.107	0.210
Panel B: pollution measures last trimester before birth						
Ozone (8 h moving average in 0.01 ppm)		3.73	3.60	3.61	3.60	3.57
		[1.498]	[1.492]	[1.524]	[1.503]	[1.528]
CO (8 h moving average in ppm)		1.59	1.64	1.55	1.60	1.51
		[0.703]	[0.792]	[0.772]	[0.758]	[0.732]
PM10 (24 h moving average in 10 μg/m^3)		2.97	2.99	2.99	2.97	3.01
		[0.746]	[0.737]	[0.744]	[0.739]	[0.748]
Panel C: pollution measures 1 week after birth						
Ozone (8 h moving average in 0.01 ppm)		3.74	3.60	3.60	3.62	3.55
		[1.800]	[1.791]	[1.822]	[1.805]	[1.825]
CO (8 h moving average in ppm)		1.58	1.64	1.55	1.60	1.51
		[0.796]	[0.881]	[0.862]	[0.848]	[0.817]
PM10 (24 h moving average in 10 μg/m3)		2.96	2.98	2.97	2.95	2.99
		[1.507]	[1.495]	[1.491]	[1.480]	[1.504]
Panel D: control variables						
Mother age in years	28.72	29.22	28.25	27.44	27.75	26.92
	[5.938]	[5.995]	[6.164]	[5.992]	[6.003]	[5.645]
Mother African American	0.187	0.19	0.30	0.41	0.35	0.54
Mother Hispanic	0.172	0.18	0.23	0.14	0.20	0.10
Mother years of education	13.35	13.27	12.79	11.77	12.74	11.46
	[2.600]	[2.632]	[2.681]	[1.946]	[2.565]	[1.843]
Multiple birth	0.0338	0.032	0.029	0.026	0.060	0.069
Mother married	0.725	0.72	0.61	0.36	0.59	0.29
Birth parity	1.956	1.98	2.00	2.46	2.44	3.33
	[1.145]	[1.148]	[1.186]	[1.615]	[1.288]	[1.856]
Child male	0.512	0.51	0.51	0.52	0.51	0.51
Mother smoking	0.129	0.09	0.10	1.00	0.12	1.00
Number of cigarettes per day	1.035	1.01	1.03	10.06	1.16	10.35
	[3.971]	[3.903]	[3.911]	[7.625]	[4.105]	[7.571]
Median family income census		4.66	4.05	3.53	3.97	3.25
tract 1989 ($10,000)		[1.766]	[1.584]	[1.375]	[1.621]	[1.307]
Fraction poor in census tract 1989		0.09	0.13	0.17	0.14	0.20
		[0.103]	[0.120]	[0.137]	[0.129]	[0.143]
Mean precipitation in previous		13.02	13.05	13.11	12.98	13.03
90 days		[4.211]	[4.149]	[4.158]	[4.080]	[4.074]
Mean of daily max temperature		63.70	64.09	64.42	64.10	64.67
previous 90 days		[14.65]	[14.74]	[14.70]	[14.74]	[14.74]
Mean of daily min temperature		21.34	22.04	22.26	21.87	22.43
previous 90 days		[15.18]	[15.15]	[15.11]	[15.18]	[15.12]

Notes: Standard deviations in brackets. Column [6] contains births where the mother smoked during the pregnancy for at least one sibling.

It is also important to note that the means in Table 1 mask considerable variation in pollution levels both across monitors and over time. In the most polluted areas, mean CO levels started at 4 ppm at the beginning of the sample period, but declined to roughly 1 ppm by 2005. Figs. 2–4 plot pollution levels at one particular pollution monitor (the Camden Lab monitor in Camden) over time and residual pollution levels after controlling for the time and monitor effects and the weather variables included in our regression models.[13] The "a" series plot 3 month moving averages (corresponding to the measures of pollution we use in birth outcome models), while the "b" series plot 7 day moving averages (corresponding to the measures of pollution we use in the infant mortality models). These plots show that although adjusting for these factors accounts for seasonal and annual trends, there is still considerable variation left to identify the effects of pollution.[14] Panel D of Table 1 shows means of the control variables available in the Vital Statistics data, the decennial census, and the weather data.

PM10, the 24 h average must not exceed 150 μg/m^3 more than once per year on average over three years (see http://www.epa.gov/air/criteria.html). For the period of our sample, several CO monitors experienced AQS violations in the period (e.g. 4 out of 13 monitors in 1989) but none after 1995; there were 2 ozone monitors in violation (1995 and 1998); and no PM10 monitors in violation.

[13] The patterns, not shown here, are very similar for the other monitors. The time period for these graphs (1994 to 1998) is restricted to improve exposition.

[14] While these figures are on the monitor level, we also checked how much of the variation in pollution is absorbed by our regression controls on the mother level. For example for CO the standard deviation is 0.7 in the full sample. After taking out the controls in equation (1), this is reduced to 0.5. Taking out monitor * quarter fixed effects and mother fixed effects reduces the standard deviation to 0.21 and 0.17, respectively. As a group the controls account for a significant part of the variation in pollution, mostly because of the inclusion of seasonal controls and monitor dummies, but there is a substantial amount of variation remaining to identify health effects.

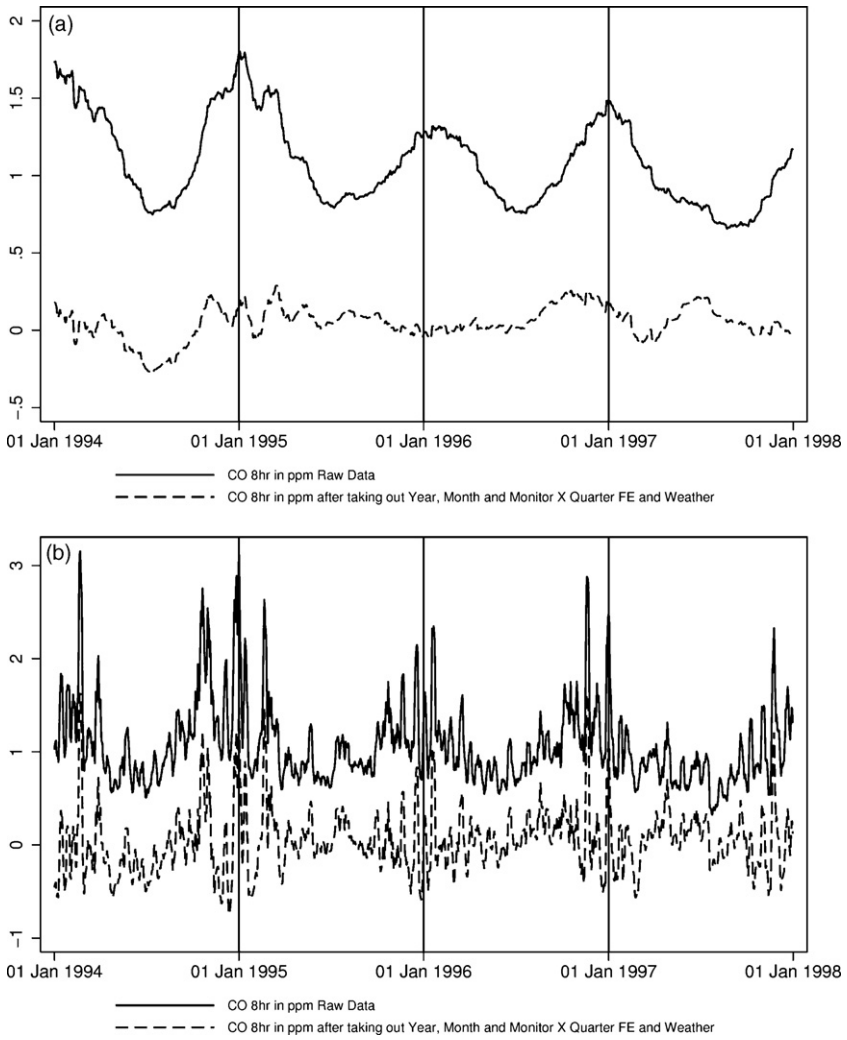

Fig. 2. (a) Air quality at Camden lab monitor, 90 day moving average of CO. (b) Air quality at Camden lab monitor, 7 day moving average of CO.

Mothers within 10 km of a monitor are almost a year younger on average than the sample mean. It is striking that mothers within 10 km of a monitor are also much more likely to be African American or Hispanic and have half a year less education on average compared to the full sample. They are also less likely to be married, but only slightly more likely to smoke than mothers who live further away from monitors. Furthermore, census tracts near monitors are lower income and have a higher fraction of poor inhabitants than further census tracts. These patterns are consistent with residential sorting based on air quality: monitors are generally located in more polluted areas, and the characteristics of those closer to the monitors are generally worse than those farther from the monitors.

The pattern of relative disadvantage is even more pronounced for the population of mothers who smoke. These mothers are much more likely to be African-American (though less likely to be Hispanic), have a year less education, are much less likely to be married, and live in the poorest census tracts compared to non-smoking mothers who live within 10 km of a monitor. In contrast, mothers with more than one birth in the sample look quite similar to mothers observed to have had only one birth.

These systematic differences demonstrate the importance of adequately controlling for characteristics of neighborhoods and families, as we do in our specifications.

4. Results

Estimates of the effects of pollution on all mothers within 10 km of a monitor are shown in Table 2. Each group of 3 columns shows estimates of Eqs (1)–(3) for a different pollutant. The mother fixed effects model, Eq. (3), is only identified from mothers with at least 2 children in the sample. To assure that the differences between the models are not driven by changes in the sample composition, the sample for estimating all three equations is restricted to children with at least one sibling in the sample (corresponding to column (5) of Table 1). In all models we cluster standard errors at the census tract level to allow for common shocks to mother's exposed to comparable levels of pollution.

Table 1 suggests that the models that do not adequately control for characteristics of the mother's location and for her own characteristics can be misleading. For example, although urban mothers are typically exposed to higher levels of pollution, they are also wealthier and more educated in our data and may have better access to health care. Failure to control for these factors could yield estimated coefficients that are biased down and possibly even wrong-signed. Few of the pollution measures in columns (1), (4), and (7) are statistically significant, and when they are, they are as likely to suggest positive effects on birth weight and gestation as negative ones.

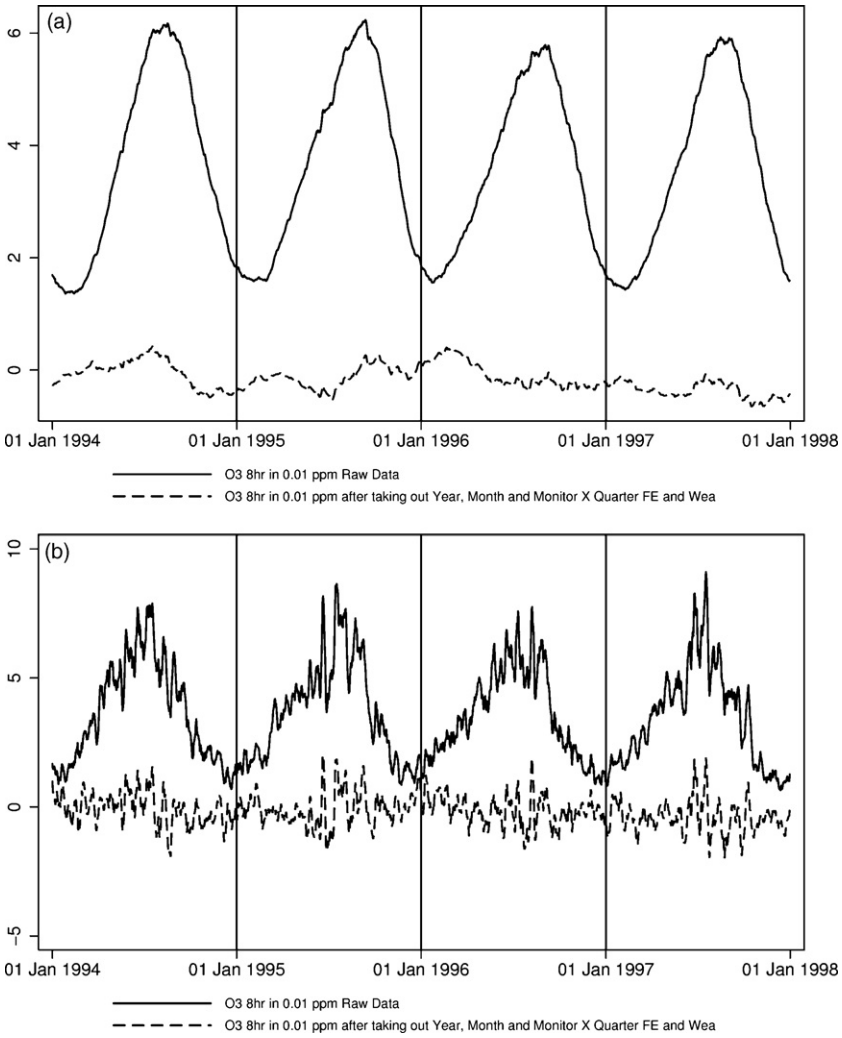

Fig. 3. (a) Air quality at Camden lab monitor, 90 day moving average of OZ. (b) Air quality at Camden lab monitor, 7 day moving average of OZ.

However, once we include monitor*quarter fixed effects (as in columns (2), (5), and (8)) the estimates suggest that CO in the last trimester of the pregnancy reduces birth weight, increases the probability of low birth weight, and shortens gestation. Now the only wrong-signed coefficient suggests that increases in PM10 in the first trimester of pregnancy increase gestation.

Finally, when we control for mother fixed effects in columns (3), (6), and (9), the estimates for CO become even larger. Ozone in the second trimester now has a statistically significant negative effect at the 10% level on birth weight and gestation. For PM10 the first trimester in the low birth weight regression is statistically significant at the 10% level. This pattern of results across specifications suggests the importance of controlling for both maternal and neighborhood fixed effects to account for confounding factors. It also suggests that in New Jersey, conditional on other observable characteristics of mothers, mothers in more polluted areas have unobserved characteristics that make them more likely to have healthy infants.

To summarize: third trimester CO has statistically significant, negative effects on infant health in all of our specifications, with the estimated effect gradually increasing as we control more thoroughly for potential confounders. In contrast, the estimated effects of PM10 and ozone are inconsistent across specifications, with none statistically significant at the 95% level in the models that control for mother fixed effects. The estimates in Table 2 imply that a one unit increase in the mean level of CO during the last trimester (where the mean is 1.64 and standard deviation is 0.79) would reduce average birth weight by 16.65 g (from a base of 3236 g)—a reduction of about a half a percent. The proportional effects are greater for low birth weight where a one unit change in mean CO would lead to an increase in low birth weight of 0.0083 (from a base of 0.106)—an 8% increase in the incidence of low birth weight. The greater effect for low birth weight than for mean birth weight suggests that infants at risk of low birth weight are most likely to be affected by pollution, an observation that we explore further below by examining infants with various risk factors. Additionally, a one unit change in mean CO is estimated to reduce gestation by 0.074 week (from a base of 38.55 weeks)—a reduction in mean gestation of 0.2%.

One way to put these estimates into perspective is to compare them to the effects of smoking. The coefficients on smoking and number of cigarettes from the models for CO are shown in Table 3 (the estimated effects of smoking in models for other pollutants are very similar but are not shown). In models that do not include maternal fixed effects, smoking is estimated to have extremely negative effects on infant health, consistent with much of the prior literature. For example, being a smoker is estimated to reduce birth weight by 162 g in models that include monitor fixed effects, and each additional cigarette smoked reduces birth weight by 5 g, for a total reduction of approximately 212 g at the mean of 10 cigarettes

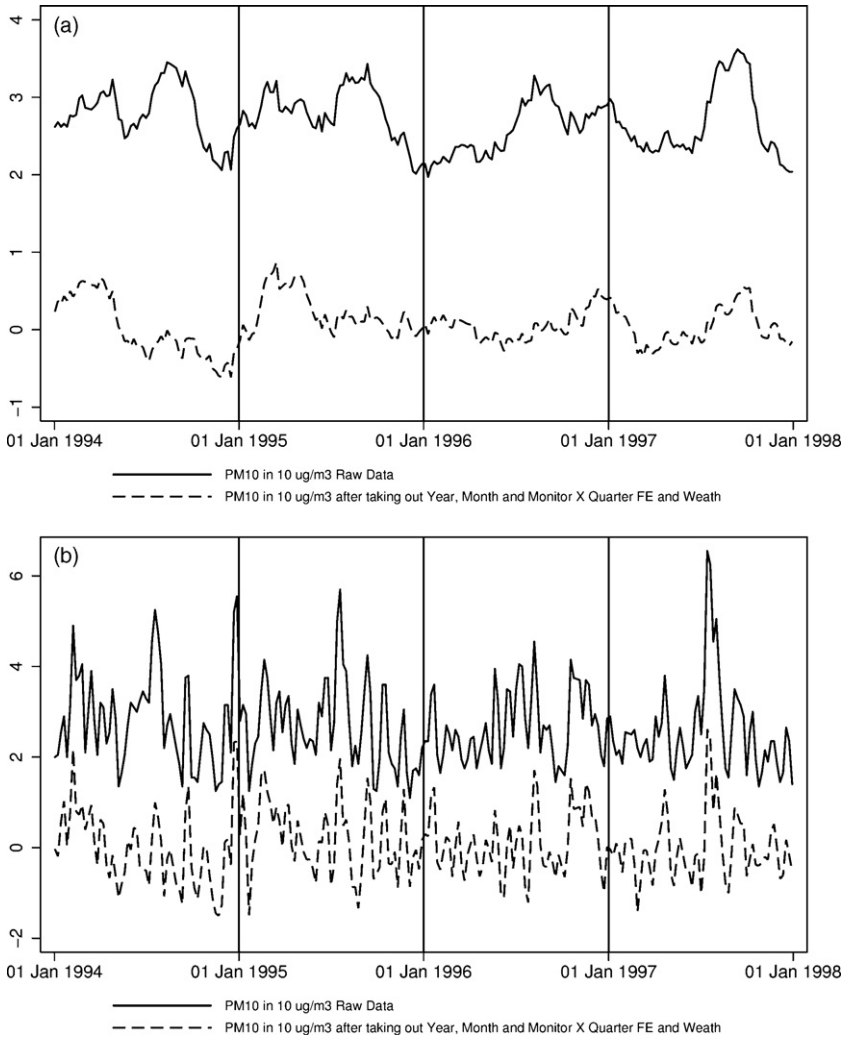

Fig. 4. (a) Air quality at Camden lab monitor, 90 day moving average of PM10. (b) Air quality at Camden lab monitor, 7 day moving average of PM10.

per day. However, as Almond et al. (2005) and Tominey (2007) point out, these estimates are likely to be contaminated by omitted characteristics of the mother that are associated with her smoking behavior.

Including mother fixed effects, which controls for unobserved characteristics of the mother, reduces the estimated effects of smoking considerably, though they remain large: being a smoker is estimated to reduce birth weight by 38.9 g, and each cigarette reduces it a further 2.2 g for a total reduction of about 61 g in infants of women who smoke 10 cigarettes per day. Hence it would take a roughly 3.7 unit change in mean CO levels to have an equivalent impact on birth weight as that from smoking 10 cigarettes per day. Similarly, the effect of smoking 10 cigarettes per day is a bit more than twice as large as the impact of a one unit change in mean CO in terms of the effect on the incidence of low birth weight.

As discussed above, infants of smoking mothers could be either more or less affected than other infants. We investigate this issue in Table 4, which shows estimates for mothers who smoked during both pregnancies. The point estimates in Table 4 are generally much larger than those in Table 2, suggesting the same level of pollution exposure is more harmful to the infants of smokers. Although the effects of CO are no longer statistically significant in the model for birth weight, the point estimate of −39.2 in the model with mother fixed effects is twice as large as the Table 2 coefficient. The coefficient on CO in the models of low birth weight is 0.044 compared to 0.008 in Table 2. For gestation, the Table 4 coefficient on CO is −43 compared to −074 in Table 2. These estimates indicate that the harmful effects from CO are two to six times greater for smoking mothers than for non-smoking mothers, depending on the outcome. Similarly, the impact of ozone is four to six times larger for smoking mothers. Furthermore, we now also find that PM10 in the second and third trimesters has a statistically significant impact on birth weight, while PM10 in the first and second trimesters are both estimated to increase the incidence of low birth weight. PM10 in the second trimester is also estimated to reduce gestation significantly.

Table 5 places the results for smoking mothers in context by showing estimates of the differential effects of CO on other subsets of mothers who may be vulnerable to poor birth outcomes. Since some demographic groups are fairly small, differential effects were estimated using the full sample of births and interacting the vector of pollution measures with the relevant characteristic of the mother. For example, column 1 of Table 5 is based on the same regression as column 3 in Table 2 except that the three pollution measures are also interacted with an indicator for whether the mother was 19 years or younger at the time of birth. Only the estimates on these interactions are shown, as the "main effects" (the estimates that apply to the rest of the sample) are generally compa-

Table 2
Effects of air pollution on health at birth— All mothers < 10 km from a Monito.

	[1] CO	[2] CO	[3] CO	[4] Ozone	[5] Ozone	[6] Ozone	[7] PM10	[8] PM10	[9] PM10
A. Models of birth weight									
3rd trimester pollution	−11.94	−13.81	−16.65	6.31	−3.57	−3.98	−1.91	0.19	−3.66
	[5.521]*	[6.343]*	[7.980]*	[2.753]*	[3.824]	[4.812]	[2.355]	[2.863]	[3.509]
2nd trimester pollution	10.13	−2.01	4.90	0.70	−1.45	−7.98	−4.22	−0.87	−2.17
	[6.510]	[7.325]	[8.492]	[3.166]	[3.846]	[4.518]+	[2.542]+	[3.008]	[3.450]
1st trimester pollution	−1.04	−7.24	−6.38	5.32	3.14	−3.34	−3.31	0.66	−1.69
	[5.447]	[6.503]	[7.785]	[2.914]+	[4.050]	[4.574]	[2.386]	[2.981]	[3.478]
Observations	312,589	312,589	312,589	268,701	268,701	268,701	285,239	285,239	285,239
B. Models of low birth weight (coefficients and standard errors multiplied by 100)									
3rd trimester pollution	0.48	0.71	0.83	−0.35	−0.05	0.18	0.08	0.00	0.15
	[0.245]+	[0.282]*	[0.384]*	[0.137]*	[0.196]	[0.251]	[0.113]	[0.132]	[0.183]
2nd trimester pollution	−0.34	−0.14	−0.36	−0.15	−0.15	−0.11	0.11	0.10	0.07
	[0.310]	[0.346]	[0.453]	[0.162]	[0.193]	[0.252]	[0.124]	[0.150]	[0.186]
1st trimester pollution	−0.04	0.11	0.49	−0.01	0.19	0.43	0.12	0.10	0.34
	[0.247]	[0.304]	[0.401]	[0.141]	[0.188]	[0.234]+	[0.116]	[0.144]	[0.194]+
	313,504	313,504	313,504	269,485	269,485	269,485	286,206	286,206	286,206
C. Models of gestation (coefficients and standard errors multiplied by 100)									
3rd trimester pollution	−4.11	−4.78	−7.41	3.19	−0.96	−0.33	2.11	3.77	2.06
	[2.221]+	[2.603]+	[3.635]*	[1.249]*	[1.769]	[2.255]	[1.023]*	[1.233]**	[1.599]
2nd trimester pollution	3.31	0.06	4.05	0.22	−3.23	−3.28	−2.46	−0.35	−1.12
	[2.624]	[3.130]	[3.955]	[1.480]	[1.793]+	[2.124]	[1.127]*	[1.352]	[1.714]
1st trimester pollution	−0.11	−1.18	−3.95	4.66	2.07	−1.17	−1.70	1.10	−0.07
	[2.273]	[2.678]	[3.582]	[1.319]**	[1.782]	[2.191]	[1.009]+	[1.227]	[1.613]
Observations	305,530	305,530	305,530	262,117	262,117	262,117	276,691	276,691	276,691
Monitor*quarter fixed effects	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes
Mother fixed effects	No	No	Yes	No	No	Yes	No	No	Yes

Notes: Standard errors in brackets, clustered on the census tract level. + indicates statistical significance at the 10% level, * at the 5% level, and ** at the 1% level. All regressions include indicators for maternal age (19–24, 25–34, 35+) education (high school, 13–15 years, 16+), multiple birth, birth order (2, 3, 4+), marital status, male child, maternal race (African American, Hispanic, other race), and maternal smoking as well as the number of cigarettes per day, median family income in the Census tract in 1989, average precipitation and daily minimum and maximum temperature in each trimester before the birth, month dummies, and year dummies. Regressions also include indicators for missing values of the control variables.

Table 3
Effects of smoking on health at birth—all mothers <10 km from a monitor (coefficients from models including CO as pollutant in Table 2).

	[1]	[2]	[3]
A. Models of birth weight			
Mother smokes	−161.8	−161.5	−38.89
	[6.375]**	[6.352]**	[8.265]**
# Cigarettes per day	−5.014	−5.05	−2.243
	[0.482]**	[0.482]**	[0.620]**
# Observations	312,589	312,589	312,589
B. Models of low birth weight (coefficients and standard errors multiplied by 100)			
Mother smokes	4.708	4.671	0.497
	[0.344]**	[0.343]**	[0.496]
# Cigarettes per day	0.196	0.196	0.129
	[0.0265]**	[0.0265]**	[0.0393]**
# Observations	313,504	313,504	313,504
C. Models of gestation (coefficients and standard errors multiplied by 100)			
Mother smokes	−31.59	−31.15	−2.724
	[2.800]**	[2.797]**	[4.118]
# Cigarettes per day	−1.165	−1.171	−0.667
	[0.227]**	[0.228]**	[0.339]*
# Observations	305,530	305,530	305,530
Monitor * quarter fixed effects	No	Yes	Yes
Mother fixed effects	No	No	Yes

Notes: See notes to Table 2. These coefficients are from the models in columns (1)–(3) in Table 2.

rable to those shown in the main specification (column 3, Table 2). The point estimates are substantially larger for very young and very old mothers and for births that had other risk factors.[15] However, there do not seem to be stronger negative effects of pollution on African-American, less educated, or low income mothers. Along with the results for smokers, these estimates suggest that infants at higher risk of poor outcomes for other biological reasons face higher risks from pollution.

Table 4
Effects of air pollution on health at birth—all smoking mothers<10 km from a monitor (mother fixed effects models only).

	[1] CO	[2] Ozone	[3] PM10
A. Models of birth weight			
3rd trimester pollution	−39.22	−19.1	−24.41
	[32.58]	[17.20]	[14.08]+
2nd trimester pollution	10.37	−32.66	−36.42
	[34.15]	[17.82]+	[15.22]*
1st trimester pollution	0.317	−15.29	3.433
	[30.25]	[17.18]	[13.45]
Observations	20,435	20,464	20,041
B. Models of low birth weight (coefficients and standard errors multiplied by 100)			
3rd trimester pollution	4.413	−0.262	0.429
	[2.219]*	[1.144]	[0.950]
2nd trimester pollution	−4.276	1.647	1.773
	[2.311]+	[1.164]	[1.027]+
1st trimester pollution	0.846	1.837	1.636
	[1.982]	[1.081]+	[0.938]+
Observations	20,465	20,501	20,083
C. Models of gestation (coefficients and standard errors multiplied by 100)			
3rd trimester pollution	−42.89	−11.69	−3.209
	[17.92]*	[9.448]	[7.920]
2nd trimester pollution	20.19	−18.5	−14.78
	[18.57]	[9.561]+	[8.102]+
1st trimester pollution	−14.33	−15.15	−8.27
	[17.14]	[9.465]	[7.185]
Observations	19,930	20,118	19,494
Monitor * quarter fixed effects	Yes	Yes	Yes
Mother fixed effects	Yes	Yes	Yes

Notes: See notes to Table 2.

[15] Risk factors are anemia, hypertension (chronic or pregnancy associated), diabetes, heart or lung disease, herpes, hydramnios, previous preterm infant, previous large infant, renal disease, incompetent cervix, rh-sensitivity, uterine bleeding, eclampsia, hemoglobinopathy, or "other complications".

Table 5
Effects of CO on health at birth—mothers from vulnerable groups <10 km from a monitor models with mother fixed effects.

	[1] <age 19	[2] ≥age 35	[3] Risk factors for the preg.	[4] Black	[5] <12 years ed.	[6] Income <30,000
A. Models of birth weight						
3rd trimester pollution	−20.52	−37.81	−24.88	−2.144	−9.186	−10.6
	[12.75]	[11.82]**	[11.15]*	[10.15]	[11.47]	[10.55]
2nd trimester pollution	6.638	20.27	−1.447	12.73	7.131	9.118
	[15.99]	[13.37]	[13.16]	[11.89]	[12.97]	[12.28]
1st trimester pollution	−9.448	0.553	−23.08	−9.429	−12.05	−15.14
	[12.79]	[12.20]	[10.89]*	[9.736]	[11.18]	[10.47]
Observations	312,589	312,589	312,589	312,589	312,589	312,589
B. Models of low birth weight (coefficients and standard errors multiplied by 100)						
3rd trimester pollution	1.075	1.767	1.18	0.322	0.697	0.729
	[0.653]+	[0.640]**	[0.545]*	[0.545]	[0.570]	[0.573]
2nd trimester pollution	−1.06	−0.815	−0.327	−0.581	−1.108	−0.575
	[0.920]	[0.754]	[0.716]	[0.701]	[0.718]	[0.698]
1st trimester pollution	1.107	0.345	1.411	0.423	1.03	0.785
	[0.690]	[0.616]	[0.554]*	[0.567]	[0.609]+	[0.563]
Observations	313,504	313,504	313,504	313,504	313,504	313,504
C. Models of gestation (coefficients and standard errors multiplied by 100)						
3rd trimester pollution	−11.92	−11.83	−10.74	−6.692	−5.438	−5.378
	[5.694]*	[5.479]*	[5.192]*	[4.981]	[5.009]	[4.651]
2nd trimester pollution	6.601	7.081	1.29	1.343	4.245	1.476
	[8.562]	[5.863]	[6.221]	[5.888]	[6.256]	[5.695]
1st trimester pollution	−4.014	−3.129	−11.7	−5.119	−4	−4.384
	[6.703]	[5.129]	[5.103]*	[4.865]	[5.690]	[5.138]
Observations	305,530	305,530	305,530	305,530	305,530	305,530

Notes: The columns show specifications that allow the effect of pollution to vary by characteristics of the mother. The models are estimated as in Table 2, but the pollution measures are interacted with a dummy variable for the characteristic of the mother. For example, in the second column the regression include each trimester CO measures interacted with whether the mother is under age 19, with only the interactions shown. The main effects (not shown) are comparable to the main effects in the corresponding specification in Table 2.

Hence, the effects of pollution appear to be amplified by biological risks but not by non-biological risks. This result also bolsters the case that our identification strategy is working: including the mother fixed effects has taken out the main effect of confounding socioeconomic factors but has not taken out a greater sensitivity to pollution that is linked to biological factors.

Table 6 shows estimates of the effects of pollution on infant mortality from models based on Eq. (4). In these models, we control for birth weight with a series of indicator variables to isolate the effect of pollution after the birth on health. Consistent with the results discussed above, Table 6 suggests that CO matters, rather than exposure to PM10 or ozone. Table 6 suggests that high CO exposures in the first 2 weeks of life increase the risk of death. Since we control for the fact that more deaths occur in the first 2 weeks with our baseline hazard, this estimates reflects the extent to which death within that time is hastened by pollution exposure. We do not, however, find any statistically significant impacts of ozone and PM10 on mortality.

To gauge the magnitude of this estimate, we need to account for the fact that we estimated the impact on the sample of mothers with at least one death, so the base risk of death in this subsample is about 40% (2334 deaths divided by 5848 births). Therefore, we multiply our estimate by the ratio of the overall sample IMR of 6.88 per 1000 births to the subsample IMR of 399 per 1000 births. This calculation suggests 17.6 averted deaths per 100,000 births from a 1 ppm decrease in CO.[16] This estimate is remarkably similar to the 16.5 averted deaths per 100,000 births reported in Currie and Neidell (2005).

As discussed above, we believe that a major contribution of our study is that we can improve the accuracy of our pollution measures because we have the mother's exact addresses. In Table 7 we offer two investigations of this claim. If being closer to a monitor improves measurement, then being farther from a monitor should yield weaker results. Table 7 shows that this is indeed the case: we do not find significant effects on health at birth (or, not shown, on infant mortality) for mothers 10–20 km from a monitor.[17] Similarly, studies often do not have an exact address of the mother but only the zip code of residence, and therefore assign pollution to the zip code centroid using an inverse distance weighted average of monitors near the zip code. In the last three columns of Table 7, we assign pollution to the mother assuming we only know her zip code. In this less precisely merged sample we find generally smaller estimates that are statistically insignificant. Both of these results are consistent with improved measurement from knowing the mother's exact address.

In Table 8, we estimate models that include both CO and ozone. Since the sources of these pollutants are similar and often therefore vary together, it is important to isolate which pollutant drives our results. Although the sample size is somewhat reduced, the estimates for CO are even stronger than those shown in Table 2, as we once again find significant effects of CO on all three infant outcomes. We also find a negative effect of ozone on gestation, though now it is exposure in the last trimester rather than the second trimester which seems to matter.[18]

[16] We do not show separate estimates of the effect of pollution on deaths among infants of smokers because restricting the sample to smokers who had at least one death in the family results in very small sample sizes.

[17] We have also estimated models using mothers who are closer to pollution monitors (within 5 kilometers). Unfortunately, the resulting reduction in sample size increases our standard errors substantially, making it more difficult to draw a clear inference from this exercise.

[18] We also estimated our models including an interaction with CO and an indicator for years after 1995 (midway through our sample) to assess if the effects change over time, but the interaction term was insignificant, suggesting the effects of CO are constant over the period.

Table 6
Effects of air pollution after birth on the probability of infant death all mothers <10 km from a monitor (coefficients and standard errors multiplied by 10,000).

	[1] CO	[2] Ozone	[3] PM10
Mean pollutant weeks 0–2	101.9	8.274	−10.97
	[36.11]**	[18.20]	[25.58]
Mean pollutant weeks 2–4	−21.61	4.478	−6.42
	[15.47]	[9.194]	[10.93]
Mean pollutant weeks 4–6	12.88	9.177	8.122
	[10.77]	[5.543]+	[5.419]
Mean pollutant weeks >6	−8.261	3.266	−0.564
	[5.819]	[3.411]	[1.809]
Birth weight <1500 g	−804.6	278.3	−122.6
	[740.1]	[381.8]	[533.3]
Birth weight 1500–2500 g	−1515.3	−483.6	−908.6
	[737.0]*	[369.5]	[523.0]+
Birth weight 2500–3500 g	−1637.4	−620	−1041.8
	[738.2]*	[371.0]+	[525.1]*
Birth weight ≥3500 g	−1685.6	−664.4	−1066.1
	[739.6]*	[370.9]+	[525.0]*
Week after birth	−1713.7	−1710	−1776.5
	[55.69]**	[57.69]**	[62.64]**
1 (Week after birth ≥1)	1814.8	1643.7	1671.5
	[97.20]**	[111.8]**	[121.8]**
1 (Week after birth ≥2)	−178.1	16.58	21.51
	[83.57]*	[97.69]	[109.6]
1 (Week after birth ≥4)	75.55	41.24	79.84
	[23.81]**	[26.34]	[29.55]**
1 (Week after birth ≥8)	−0.404	8.272	5.087
	[7.681]	[7.814]	[8.314]
1 (Week after birth ≥12)	−1.394	−4.28	−5.07
	[4.156]	[4.829]	[5.089]
1 (Week after birth ≥20)	1.174	2.775	0.917
	[1.379]	[1.493]+	[1.888]
1 (Week after birth ≥32)	1.736	1.293	2.459
	[0.594]**	[0.620]*	[0.786]**
Observations	192,184	163,392	131,837
Number of births	5,848	5,078	4,556
Number of deaths	2,334	2,038	1,870
Number of mothers	2,252	1,962	1,803

Notes: See notes to Table 2. Standard errors are clustered on the census tract level. All models include mother fixed effects.

Table 8
Effects of air pollution on health at birth—all mothers <10 km from a monitor models control for both CO and O3.

	[1] Birth weight	[2] Low Birth weight	[3] Gestation
3rd trimester CO (in ppm)	−20.77	1.056	−9.416
	[8.973]*	[0.429]*	[4.044]*
2nd trimester CO (in ppm)	7.646	−0.784	5.366
	[9.427]	[0.507]	[4.414]
1st trimester CO (in ppm)	−5.765	0.79	−5.044
	[8.443]	[0.445]+	[3.917]
3rd trimester ozone (in 0.01 ppm)	−5.365	0.16	−3.115
	[4.269]	[0.232]	[2.104]
2nd trimester ozone (in 0.01 ppm)	0.271	−0.117	−1.591
	[4.624]	[0.257]	[2.157]
1st trimester ozone (in 0.01 ppm)	−4.384	0.275	−0.849
	[4.172]	[0.241]	[2.032]
Observations	274,358	275,193	267,818

Notes: See Table 2. Coefficients and standard errors are multiplied by 100 in columns 2 and 3. All models include mother fixed effects.

5. Discussion and conclusions

In order to begin to evaluate the costs and benefits of tighter pollution regulation, it is necessary to understand how changes from current, historically low levels of air pollution are likely to affect health. This paper examines the effects of air pollution on infant health using recent data from New Jersey. Our models control for many potential confounders, with our richest model identified using variation in pollution between births among mothers located near particular monitors.

Our strongest and most consistent set of results show that CO has negative effects on infant health both before and after birth. Since most CO emissions come from transportation sources, these findings are germane to the current contentious debate over proposals to further tighten automobile emissions standards. For example, the state of California's most recent proposal to increase emis-

Table 7
Effects of air pollution on health at birth—alternative ways to assign pollution.

	Mothers >10 km and <20 km from a monitor			Assigning pollution using zip code		
	[1] CO	[2] Ozone	[3] PM10	[4] CO	[5] Ozone	[6] PM10
A. Models of birth weight						
3rd trimester pollution	−1.11	−5.353	−8.613	−14.16	−2.097	−6.207
	[9.537]	[4.467]	[4.846]+	[9.536]	[5.184]	[4.546]
2nd trimester pollution	−11.25	2.696	−3.964	7.449	−7.957	−4.087
	[9.937]	[4.624]	[5.651]	[9.811]	[4.814]+	[3.941]
1st trimester pollution	−17.47	−2.464	−7.223	−0.551	1.6	−3.082
	[10.02]+	[4.410]	[5.079]	[8.695]	[4.662]	[3.681]
Observations	248,230	270,668	137,123	312,589	268,701	285,239
B. Models of low birth weight (coefficients and standard errors multiplied by 100)						
3rd trimester pollution	0.43	0.303	0.317	0.85	0.167	0.298
	[0.474]	[0.207]	[0.246]	[0.463]+	[0.273]	[0.236]
2nd trimester pollution	0.152	−0.156	−0.0303	−0.447	−0.0535	0.149
	[0.492]	[0.215]	[0.242]	[0.506]	[0.254]	[0.208]
1st trimester pollution	0.406	0.146	0.346	0.246	0.23	0.388
	[0.478]	[0.203]	[0.241]	[0.463]	[0.228]	[0.210]+
Observations	249,163	271,605	137,748	313,504	269,485	286,206
C. Models of gestation (coefficients and standard errors multiplied by 100)						
3rd trimester pollution	−0.618	0.0659	−2.434	0.71	1.028	1.413
	[4.359]	[1.993]	[2.195]	[4.192]	[2.387]	[2.034]
2nd trimester pollution	0.991	−0.142	0.0585	4.939	−3.561	−1.347
	[4.385]	[2.059]	[2.395]	[4.464]	[2.242]	[1.942]
1st trimester pollution	−1.241	0.762	−1.737	3.263	−0.121	0.842
	[4.109]	[1.849]	[2.041]	[3.892]	[2.195]	[1.769]
Observations	243,028	263,952	133,723	305,530	262,117	276,691

Notes: See notes to Table 2. All models include mother fixed effects. The models in columns [4] to [6] assign pollution to the child assuming we only knew the mother's zip code of residence and computing the inverse distance weighted average of monitor values from the zip code centroid.

sions standards has been blocked by the Environmental Protection Agency. The Agency first argued that it had no authority to regulate the greenhouse gases in auto exhaust. When that argument was dismissed by the Supreme Court in April 2007, the agency then denied California's request for the waiver necessary to implement its law, claiming that uniform federal standards were superior to the piecemeal approach offered by the state. The state is currently suing the federal government over the issue. Should the state prevail, at least 16 other states are set to implement California's regulations (Maynard, 2007; Barringer, 2008).

It is noteworthy that we find negative effects of exposure to CO even at the low levels of ambient CO currently observed. Some areas in our study saw a reduction in mean CO levels from 4 ppm to 1 ppm over our sample period. Our estimates of the effects of CO on birth weight and gestation suggest that this reduction had an effect roughly equivalent to getting a women smoking 10 cigarettes a day to quit. We also find that infants of smokers are at much greater risk of negative effects from CO exposure. We also find some evidence of significant effects of PM10 and ozone on health at birth, particularly among smokers, though these estimates are less robust than our CO estimates. We further find that a one unit decrease in mean CO levels in the first 2 weeks of life saves roughly 18 lives per 100,000 births, which represents a reduction in the probability of infant death of about 2.5%.

To value the impact of recent declines in CO throughout the U.S., we perform the following illustrative calculations.[19] To value the improvements in birth weight, we compute the percentage change in birth weight from a unit change in pollution by dividing the estimated impact of third-trimester CO on birth weight (−16.65) by the mean birth weight in our sibling sample (3236). We multiply this by the estimated elasticity between birth weight and earnings of 0.1 from Black et al. (2007) to obtain the percentage change in earnings. We then multiply this by the average earnings of all full time workers per state in 2003[20] and the total number of births per state in 2003 to get the change in earnings per birth cohort per state from a 1 ppm change in CO. We then multiply this by the change in annual average 8-h CO concentrations from 1989 to 2003 per state to obtain the increase in annual earnings for the 2003 birth cohort. Finally, we compute the present discounted value of the annual earnings increase assuming a 6% discount rate and 30 years of labor force participation, which gives us an estimated increase in nationwide earnings of $720 million for the 2003 birth cohort due to the fact that CO had fallen from 1989 levels. This is clearly a lower bound, since the assumed discount rate of 6% is relatively high and we ignore the fact that mean earnings for this cohort will certainly grow in the future. Furthermore the decline in actual exposure was likely larger than is indicated by the mean decline over the monitors, since at least in New Jersey, people tend to live in the more heavily polluted areas that experienced the largest declines.

In order to value the improvements in infant mortality, we multiply our estimate of 17.6 lives saved per 100,000 births for a 1 ppm change in CO by the number of births per state and the decreases in CO levels per state to obtain the nationwide number of deaths avoided. This gives us a total of 449 deaths averted in 2003 by the reduction in CO from 1989 levels. We compute the benefits from these avoided deaths using a value of statistical life of $4.8 million as used by the EPA, which yields an estimated $2.2 billion in annual savings.[21]

While we recognize the strong assumptions behind these calculations, the magnitude of these benefits suggests potentially substantial benefits from the improvements in CO over time. Moreover, there are several reasons why our estimates may understate the health impact from pollution exposure. Unlike small-scale epidemiological studies that use personal air quality monitors strapped to persons, we use a crude proxy for individual exposures. Our noisier measures of exposure may lead us to falsely accept a null hypothesis. And since the literature does not give much guidance about the type of exposures that are most likely to be harmful (in terms of length of exposure, when it occurred during pregnancy, or intensity of exposure) it is possible that more precise measures taken at key points in the pregnancy would uncover larger effects. Furthermore, our study is based on the population of live births. It is possible that pollution causes fetal losses or it impairs fertility. If high levels of pollution cause vulnerable fetuses to be lost, or cause women who might have had low birth weight babies not to become pregnant, then mean levels of birth weight and gestation will be increased. For all these reasons, we regard these estimates as lower bounds on the benefits of pollution control to infants. As such, they may still provide a useful benchmark for assessing the benefits of further reductions in air pollution in terms of infant health.

[19] For these calculations we assume a homogeneous relationship between pollution and birth weight or infant mortality. While it is not possible to properly assess this, we do note that the marginal impact of CO on infant mortality we estimate here is virtually identical to the marginal impact of 17 deaths per 100,000 births found in Currie and Neidell (2005).

[20] Available from the Bureau of Labor Statistics at http://www.bls.gov/cew/.

[21] Full details of these calculations are available from the authors' upon request.

Appendix A. Effects of air pollution on health at birth - displaying coefficients on all covariates.

	[1] CO	[2] CO	[3] CO	[4] Ozone	[5] Ozone	[6] Ozone	[7] PM10	[8] PM10	[9] PM10
Models of birth weight									
3rd trimester pollution	−11.94	−13.81	−16.65	6.312	−3.566	−3.978	−1.906	0.19	−3.657
	[5.521]*	[6.343]*	[7.980]*	[2.753]*	[3.824]	[4.812]	[2.355]	[2.863]	[3.509]
2nd trimester pollution	10.13	−2.009	4.904	0.695	−1.453	−7.975	−4.219	−0.865	−2.174
	[6.510]	[7.325]	[8.492]	[3.166]	[3.846]	[4.518]+	[2.542]+	[3.008]	[3.450]
1st trimester pollution	−1.039	−7.24	−6.379	5.321	3.139	−3.34	−3.31	0.662	−1.691
	[5.447]	[6.503]	[7.785]	[2.914]+	[4.050]	[4.574]	[2.386]	[2.981]	[3.478]
Mother age 19–24	40.51	40.92	31.89	40.63	41.34	30.69	44.56	45.19	35.16
	[6.195]**	[6.210]**	[6.840]**	[6.445]**	[6.480]**	[7.959]**	[5.989]**	[6.002]**	[7.329]**
Mother age 25–34	61.3	62.14	32.54	50.51	51.57	29.4	61.17	62.08	33.73
	[6.975]**	[7.001]**	[8.655]**	[7.120]**	[7.158]**	[9.810]**	[6.735]**	[6.746]**	[9.267]**
Mother age 35 or higher	62.36	63.5	32.94	49.92	51.2	35.63	61.62	63	36.98
	[7.702]**	[7.708]**	[11.22]**	[7.933]**	[7.984]**	[13.09]**	[7.528]**	[7.554]**	[12.36]**
High School	27.47	27.39	−1.738	23.99	23.58	4.496	25.37	25.43	−1.139
	[3.642]**	[3.651]**	[5.436]	[4.113]**	[4.143]**	[5.819]	[3.795]**	[3.796]**	[5.517]
13–15 years education	52.29	52.25	7.928	50.31	49.35	8.641	49.56	49.33	8.576
	[4.108]**	[4.099]**	[7.200]	[4.808]**	[4.841]**	[7.771]	[4.301]**	[4.284]**	[7.466]
16 or more years of education	57.5	57.32	−5.929	56.64	56.11	6.044	54.54	55.06	−6.013
	[4.523]**	[4.523]**	[9.059]	[5.290]**	[5.291]**	[10.54]	[4.892]**	[4.882]**	[9.418]
Multiple birth	−1029.8	−1029.9	−1009.7	−1029.2	−1028.6	−1004.7	−1031.6	−1030.9	−1011.3
	[6.913]**	[6.923]**	[14.48]**	[7.770]**	[7.778]**	[14.67]**	[7.636]**	[7.636]**	[14.57]**
Birth order 2	87.06	87.64	57.06	100.4	99.92	57.53	91.73	92.3	56.36
	[4.300]**	[4.308]**	[5.558]**	[4.926]**	[4.947]**	[6.429]**	[5.252]**	[5.258]**	[6.776]**
Birth order 3	99.97	100.6	32.66	112.2	111.4	21.36	103.2	103.4	20.71
	[5.954]**	[5.968]**	[7.960]**	[6.589]**	[6.629]**	[9.236]*	[6.803]**	[6.786]**	[9.331]*
Birth order 4 or higher	71.27	72.04	−11	85.23	83.79	−24.14	69.01	68.95	−19.5
	[7.842]**	[7.867]**	[10.01]	[8.016]**	[8.088]**	[10.79]*	[8.239]**	[8.247]**	[10.40]+
Mother married	86.22	85.88	31.67	90.94	90.73	40.1	87.27	86.99	36.69
	[3.349]**	[3.341]**	[5.503]**	[3.682]**	[3.704]**	[5.999]**	[3.547]**	[3.519]**	[5.769]**
Mother is smoking	−161.8	−161.5	−38.89	−156	−156.7	−41.42	−160.7	−161.4	−43.47
	[6.375]**	[6.352]**	[8.265]**	[6.385]**	[6.399]**	[8.630]**	[6.348]**	[6.349]**	[8.328]**
Number of cigarettes per day	−5.014	−5.05	−2.243	−5.845	−5.88	−3.03	−5.566	−5.592	−2.979
	[0.482]**	[0.482]**	[0.620]**	[0.504]**	[0.504]**	[0.614]**	[0.503]**	[0.502]**	[0.606]**
Male	114	113.8	120.1	114.9	114.8	122.5	115	114.8	120.8
	[2.166]**	[2.159]**	[2.503]**	[2.336]**	[2.331]**	[2.841]**	[2.296]**	[2.293]**	[2.684]**
Med fam income 1989 in $10,000	3.463	1.543	3.994	1.018	2.576	7.333	2.738	5.668	3.844
	[1.675]*	[1.844]	[2.639]	[1.644]	[1.727]	[3.003]*	[1.794]	[1.834]**	[2.883]
Fraction of people poor in 1989	−197.6	−199.6	−1.40	−173.7	−164.9	9.063	−195.5	−151.4	−1.10
	[21.25]**	[22.49]**	[29.51]	[20.50]**	[20.84]**	[30.99]	[22.86]**	[23.78]**	[30.32]
Precipitation third trimester	0.239	−0.0704	0.02	−0.056	−0.554	−0.564	0.26	0.00816	0.0491
	[0.315]	[0.330]	[0.376]	[0.335]	[0.347]	[0.429]	[0.337]	[0.352]	[0.413]
Precipitation second trimester	−0.301	−0.368	0.26	−0.0863	0.0536	−0.134	−0.328	−0.124	0.501
	[0.333]	[0.338]	[0.392]	[0.364]	[0.368]	[0.440]	[0.338]	[0.345]	[0.430]
Precipitation first trimester	0.0421	−0.015	−0.208	−0.358	−0.254	−0.428	−0.0606	−0.0329	−0.511
	[0.308]	[0.314]	[0.387]	[0.348]	[0.353]	[0.423]	[0.347]	[0.360]	[0.434]
Mean daily min temp third trimester	−0.414	0.151	−0.118	−0.852	−0.555	−0.677	−0.332	0.181	−0.101
	[0.284]	[0.292]	[0.360]	[0.315]**	[0.322]+	[0.399]+	[0.301]	[0.308]	[0.394]
Mean daily min temp second trimester	−0.19	0.226	0.0736	0.049	0.264	0.0783	−0.014	0.147	−0.183
	[0.287]	[0.310]	[0.369]	[0.312]	[0.335]	[0.400]	[0.303]	[0.315]	[0.372]
Mean daily min temp first trimester	−0.653	−0.245	−0.269	−0.718	−0.421	−0.204	0.0284	0.186	−0.0115
	[0.271]*	[0.279]	[0.343]	[0.315]*	[0.326]	[0.392]	[0.293]	[0.315]	[0.394]
Mean daily max temp third trimester	1.448	−0.147	−0.105	1.057	−0.0419	0.423	1.778	−0.27	−0.25
	[0.476]**	[0.558]	[0.647]	[0.559]+	[0.608]	[0.750]	[0.517]**	[0.577]	[0.669]
Mean daily max temp second trimester	0.855	−0.132	−0.373	0.64	−0.237	0.585	1.00	0.199	−0.0112
	[0.444]+	[0.459]	[0.561]	[0.517]	[0.574]	[0.708]	[0.482]*	[0.486]	[0.564]
Mean daily max temp first trimester	2.172	0.74	0.393	1.361	−0.242	−0.365	1.799	0.437	−0.377
	[0.424]**	[0.521]	[0.643]	[0.483]**	[0.572]	[0.725]	[0.459]**	[0.546]	[0.702]
Mother age missing	−197.9	−196.5	21.44	−728.2	−749.1	−10.31	354.3	346.8	–
	[377.5]	[371.1]	[13.54]	[15.74]**	[17.51]**	[13.16]	[14.29]**	[15.87]**	
Education variable missing	−35.39	−39.11	−51.4	−39.01	−43.79	−57.71	−31.79	−36.58	−57.57
	[7.283]**	[7.175]**	[9.412]**	[7.273]**	[7.298]**	[9.087]**	[7.291]**	[7.238]**	[8.802]**
Multiple birth missing	−262.2	−263	−174.1	−144.6	−143	3.059	−181.8	−179	−90.88
	[55.45]**	[55.14]**	[91.12]+	[58.52]*	[58.40]*	[86.42]	[51.78]**	[51.36]**	[89.62]
Birth order missing	36.62	40.88	15.89	106.1	103.8	37.01	63.41	64.4	−6.089
	[38.14]	[38.22]	[42.23]	[37.28]**	[37.36]**	[43.80]	[38.67]	[38.81]+	[43.08]
Mother married missing	−240.8	−239.7	−175	−151.3	−148.6	−97.11	−293	−290	−171.6
	[85.85]**	[85.62]**	[92.86]+	[76.33]*	[76.04]+	[83.20]	[77.53]**	[77.09]**	[92.98]+
Male missing	−713.5	−712.6	−175.8	−1681.8	−1676.6	−1324.5	−940.2	−926.4	−457.8
	[419.1]+	[415.6]+	[433.1]	[506.3]**	[502.7]**	[671.3]*	[420.8]*	[416.7]*	[507.4]
Mother is smoking missing	−102.6	−107.4	−44.53	−113.5	−115.5	−46.31	−108	−115.8	−49.11
	[8.471]**	[8.501]**	[9.788]**	[8.562]**	[8.555]**	[10.08]**	[8.419]**	[8.428]**	[9.503]**
Mother African American	−198.2	−193.6		−211.8	−204.6		−204.5	−197.9	
	[4.028]**	[4.189]**		[4.176]**	[4.177]**		[4.167]**	[4.272]**	
Mother Hispanic	−43.51	−42.93		−61.36	−56.8		−51.91	−47.36	
	[4.027]**	[3.953]**		[4.333]**	[4.393]**		[4.154]**	[4.034]**	

Appendix A (*Continued*)

	[1] CO	[2] CO	[3] CO	[4] Ozone	[5] Ozone	[6] Ozone	[7] PM10	[8] PM10	[9] PM10
Other race or race missing	−230.3	−228.1		−232.4	−226.4		−234.9	−228.7	
	[6.147]**	[6.337]**		[7.343]**	[7.465]**		[7.061]**	[7.310]**	
Constant	3174.2	3438.1	3319.5	2947.5	3191.9	3363.6	2626	2825.8	3280.2
	[380.4]**	[372.3]**	[85.77]**	[67.47]**	[75.56]**	[107.7]**	[60.59]**	[72.16]**	[106.1]**
Observations	312589	312589	312589	268701	268701	268701	285239	285239	285239
Monitor* quarter fixed effects	no	yes	yes	no	yes	yes	no	yes	yes
Mother fixed effects	no	no	yes	no	no	yes	no	no	yes

Notes: The table corresponds to the same regressions as Table 2 Panel A, but displays all covariates (except for year, month and monitor dummies).

References

Allison, Paul, 1982. Discrete-time methods for the analysis of event histories. Sociological Methodology XII, 61–98.

Almond, Douglas, Kenneth, Chay, David Lee, 2005. The cost of low birth weight. Quarterly Journal of Economics 121, 1031.

Basu, R., Woodruff, T.J., Parker, J.D., Saulnier, L., Schoendorf, K.C., 2004. Comparing exposure metrics in the relationship between Pm2 5 and birth weight in California. Journal of Exposure Analysis and Environmental Epidemiology 14 (5), 391–396.

Barringer, Felicity, 2008. California sues EPA over denial of waiver. The New York Times, January 3.

Bell, M., Ebisu, K., Belanger, K., 2007. Is risk of low birth weight affected by maternal exposure to air pollution? Epidemiology 18 (5), S128–S228.

Black, Sandra, Paul Devereux, Kjell Salvanes, 2007. From the cradle to the labor market? The effect of birth weight on adult outcomes. Quarterly Journal of Economics 122, 409–439.

Brauer, M., Lencar, C., Tamburic, L., Koehoorn, M., Demers, P., Karr, C., 2008. A cohort study of traffic-related air pollution impacts on birth outcomes. Environmental Health Perspectives 116 (5), 680–686.

Chen, L., Yang, W., Jennison, B.L., Goodrich, A., Omaye, S.T., 2002. Air pollution and birth weight in northern Nevada 1991–1999. Inhalation Toxicology 14 (2), 141–157.

Cnattingius, Sven, 1997. Maternal age modifies the effect of maternal smoking on intrauterine growth retardation but not on late fetal death and placental aburption. American Journal of Epidemiology 145 (4), 319.

Chay, K., Greenstone, M., 2003a. the impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession. Quarterly Journal of Economics CXVIII.

Chay, K., Greenstone, M., 2003b. Air Quality, Infant Mortality, and the Clean Air Act of 1970, NBER working paper 10053.

Currie, Janet, 2008. Healthy, wealthy, and wise? Socioeconomic status, poor health in childhood, and human capital development. Journal of Economic Literature.

Currie, Janet, Neidell, Matthew, 2005. Air pollution and infant health: what can we learn from California's recent experience? Quarterly Journal of Economics 120 (3).

Dugandzic, Rose, Dodds, Linda, Stieb, David, Smith-Doiron, Marc, 2006. The association between low level exposures to ambient air pollution and term low birth weight: a retrospective cohort study. Environmental Health 5, 3.

Environmental Protection Agency, Automobiles and carbon monoxide, EPA Office of Mobile Sources Fact Sheet OMS-3, January 1993, http://www.epa.gov/oms/consumer/03-co.pdf.

Environmental Protection Agency, 2003. Criteria pollutants, February 20. http://www.epa.gov/oar/oaqps/greenbk/o3co.html.

Friedman, Michael, Kenneth Powell, Lori Hutwagner, LeRoy Graham, W., Gerald Teague, 2001. Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. Journal of the American Medical Association CCLXXXV,897–905.

Huel, G., Godin, J., Frery, N., Girard, F., Moreau, T., Nessmann, C., Blot, P., 1993. Aryl hydrocarbon hydroxylase activity in human placenta and threatened preterm delivery. Journal of Exposure Analysis and Environmental Epidemiology 3 (Suppl. 1), 187–189.

Huynh, M., Woodruff, T.J., Parker, J.D., Schoendorf, K.C., 2006. Relationships between air pollution and preterm birth in California. Paediatric and Perinatal Epidemiology 20 (6), 454–461.

Lee, S.J., Hajat, S., Steer, P.J., Filippi, V., 2008. A time-series analysis of any short-term effects of meteorological and air pollution factors on preterm births in London, UK. Environmental Research 106 (2), 185–194.

Lipfert, F.W., Zhang, J., Wyzga, R.E., 2000. Infant mortality and air pollution: a comprehensive analysis of US data for 1990. Journal of the Air & Waste Management Association 50 (8), 1350–1366.

Liu, Shiliang, Daniel, K., Yuanli, S., Yue, C., Richard Burnett, 2003. Association between gaseous ambient air pollutants and adverse pregnancy outcomes in Vancouver, Canada. Environmental Health Perspectives CXI, 1773–1778.

Liu, S.L., Krewski, D., Shi, Y., Chen, Y., Burnett, R.T., 2007. Association between maternal exposure to ambient air pollutants during pregnancy and fetal growth restriction. Journal of Exposure Science and Environmental Epidemiology 17 (5), 426–432.

Logan, W.P.D., Glasg, M.D., 1953. Mortality in London fog incident. Lancet I 1952, 336–338.

Maisonet, Mildred, Timothy, B, Adolfo, C, Jouni, J, 2001. Relation between ambient air pollution and low birth weight in the northeastern United States. Environmental Health Perspectives CIX, 351–356.

Maynard, Micheline, 2007. EPA denies California's emissions waiver. The New York Times, December 19.

Parker, J.D., Mendola, P., Woodruff, T.J., 2008. Preterm birth after the Utah Valley steel mill closure a natural experiment. Epidemiology 19 (6), 820–823.

Parker, J.D., Woodruff, T.J., 2008. Influences of study design and location on the relationship between particulate matter air pollution and birthweight. Paediatric and Perinatal Epidemiology 22 (3), 214–227.

Parker, J.D., Woodruff, T.J., Basu, R., Schoendorf, K.C., 2005. Air pollution and birth weight among term infants in California. Pediatrics 115 (1), 121–128.

Pope, C. Arden, 1989. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. American Journal of Public Health LXXIX, 623–628.

Pope, C. Arden, Schwartz, Joel, Ransom, Michael, 1992. Daily mortality and PM10 pollution in Utah Valley. Archives of Environmental Health XLVII, 211–216.

Ransom, Michael, Pope, C. Arden, 1992. Elementary school absences and PM10 pollution in Utah Valley. Environmental Research LVIII, 204–219.

Ritz, B., Wilhelm, M., Hoggatt, K.J., Ghosh, J.K.C., 2007. Ambient Air Pollution and Preterm Birth in the Environment and Pregnancy Outcomes Study at the University of California, 166. American Journal of Epidemiology, Los Angeles, 1045-52.

Ritz, B., Wilhelm, M., Zhao, Y.X., 2006. Air Pollution and infant death in southern California 1989–2000. Pediatrics 118 (2), 493–502.

Ritz, Beate, Fei Yu, 1999. The effects of ambient carbon monoxide on low birth weight among children born in southern California Between 1989 and 1993. Environmental Health Perspectives CVII, 17–25.

Ritz, Beate, Fei Yu, Guadalupe Chapa, Scott Fruin, 2000. Effect of air pollution on preterm birth among children born in southern california between 1989 and 1993. Epidemiology XI, 502–511.

Rogers, J.F., Dunlop, A.L., 2006. Air pollution and very low birth weight infants: a target population? Pediatrics 118 (1), 156–164.

Rogers, J.F., Thompson, S.J., Addy, C.L., McKeown, R.E., Cowen, D.J., Decoufle, P., 2000. Association of very low birth weight with exposures to environmental sulfur dioxide and total suspended particulates. American Journal of Epidemiology 151 (6), 602–613.

Sagiv, S.K., Mendola, P., Loomis, D., Herring, A.H., Neas, L.M., Savitz, D.A., Poole, C., 2005. A time-series analysis of air pollution and preterm birth in Pennsylvania 1997–2001. Environmental Health Perspectives 113 (5), 602–606.

Salam, M.T., Millstein, J., Li, Y.F., Lurmann, F.W., Margolis, H.G., Gilliland, F.D., 2005. Birth outcomes and prenatal exposure to ozone, carbon monoxide, and particulate matter: results from the Children's Health Study. Environmental Health Perspectives 113 (11), 1638–1644.

Samet, Jonathan, Scott Zeger, Julia Kelsall, Jing Xu, Laurence Kalstein, 1997. Does weather confound or modify the association of particulate air pollution with mortality? Environmental Research Section A LXXVII, 9–19.

Seaton, Anthony, et al., 1995. Particulate air pollution and acute health effects. Lancet CCCLIV, 176–178.

Spengler, John, Samet, Jonathan, McCarthy, J.F., 2000. Indoor Air quality Handbook. McGraw Hill Book Co, New York, NY.

Tominey, Emma, 2007. Maternal smoking during pregnancy and child birth weight. London School of Economics Center for Economic Performance Working Paper DP0828, October.

Xu, X., Wang, L., 1998. Synergistic effects of air pollution and personal smoking on adult pulmonary function. Archives of Environmental Health 53, 4–53.

Wilhelm, M., Ritz, B., 2005. Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, 113, 9, Environmental Health Perspectives, California USA, 1212–1221.

Wilson, William, Mage, David, Grant, Lester, 2000. Estimating separately personal exposure to ambient and nonambient particulate matter for epidemiology and risk assessment: why and how? Journal of the Air Waste Management Association L, 1167–1183.

Woodruff, T.J., Darrow, L.A., Parker, J.D., 2008. Air pollution and postneonatal infant mortality in the United States 1999–2002. Environmental Health Perspectives 116 (1), 110–115.

Woodruff, T.J., Grillo, J., Schoendorf, K.C., 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. Environmental Health Perspectives CV, 608–612.

World Health Organisation (WHO), 2000. Carbon monoxide. WHO Air Quality Guidelines. 2nd ed. WHO Regional Office for Europe, Copenhagen.