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AIR POLLUTION AND INFANT HEALTH: LESSONS FROM NEW JERSEY

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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. In addition to large sample size, our work offers three important innovations: First, because we know the exact addresses of mothers, we select those mothers closest to air monitors to ensure a more accurate measure of air quality. Second, since we follow mothers over time, we control for unobserved characteristics of mothers using maternal fixed effects. Third, we examine interactions of air pollution with smoking and other predictors of poor infant health outcomes. We find consistently negative effects of exposure to pollution, especially carbon monoxide, both during and after birth. The effects are considerably larger for smokers than for nonsmokers as well as for older mothers. Since automobiles are the main source of carbon monoxide emissions, our results have important implications for regulation of automobile emissions.

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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 (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 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, 2003) families with higher incomes or preferences for cleaner air are likely to sort into locations with better air quality. Our data permits us to follow mothers over time, so we can include pollution monitor and maternal fixed effects to capture all time-invariant characteristics of the mother and neighborhood. 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 might exacerbate the effect of air pollution by increasing inflammatory responses and airway reactivity (Wang and Xu, 1998). Alternatively, since cigarette smoke contains high levels of pollutants, including as carbon monoxide, 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 air pollution has a significant effect on fetal health even at the relatively low levels of pollution experienced 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 carbon monoxide (CO) during the last trimester of pregnancy would increase the risk of low birth weight by 8 percent. Furthermore, a one unit change in mean CO during the first two weeks after birth would increase the risk of infant mortality by one percent relative to baseline levels. We also find that the effects of air pollution on infant health at birth are two to six times larger for smokers and for mothers over age 35 relative to the whole population. 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 I provides necessary background about the ways in which pollution may affect infant health and the previous literature. Section II describes our data, while methods are described in Section III. Section IV presents our results, and Section V details our conclusions.

I. BACKGROUND

We begin with a discussion of the ways in which the three criterion air pollutants we consider can affect infant health. Carbon Monoxide (CO) is an odorless, colorless gas which is poisonous at high levels. CO

bonds with hemoglobin more easily than oxygen, so that it reduces the body's ability to deliver oxygen to organs and tissues. Because infants are small, and many have respiratory problems to begin with, CO may be particularly harmful to them. Nationwide, 77 percent of CO comes from transportation sources, while as much as 90 percent of CO in cities comes from motor vehicle exhaust (Environmental Protection Agency, January 1993, 2003b). Among smokers, cigarettes are a major source of CO, and smokers have higher baseline levels of CO in the blood than non-smokers (Environmental Protection Agency, 2000).

Particulate matter can take many forms, including ash and dust, and motor vehicle exhaust is a major source. It is thought that the most damage comes from the smallest particles since they are inhaled deep into the lungs [Environmental Protection Agency, 2003b]. The mechanisms through which particles harm health are controversial. A leading theory is that they cause an inflammatory response which weakens the immune system [Seaton, et al. 1995].

Ozone (the major component of smog) 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. Ozone 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 not generally found in homes because it quickly reacts with household surfaces (http://www.hc-sc.gc.ca/hecs-sesc/air_quality/faq.htm).

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. We also know little about the extent that infants are protected from the negative effects of pollution while they are in the womb. Pollution exposure could affect the health of the mother by, for example, weakening her immune system, which could have negative effects on the fetus. Pollution could directly affect an infant by weakening the immune system and making them more susceptible to illness or death from a wide range of causes.[1] But

[1] Alternatively, since motor vehicle exhaust is a major contributor of CO and PM10, these pollutants may

the available research gives little guidance about what levels of pollution might be necessary to induce negative effects or about when fetuses or infants are most vulnerable.

Many 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. 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 2,500 grams), while Ritz et al. (2000) report that CO exposure in the six weeks before birth is correlated with gestation in some regions of southern California but not in others. Alderman et al. (1987) found that CO in the last trimester had no effect on low birth weight once maternal education and race were controlled. Ritz et al. (2000) report that PM10 exposure 6 weeks before birth increases preterm birth, while Mainsonet et al. (2001) find that PM10 has no effect on low birth weight.

Results of studies of the effects of pollution on infant mortality are also mixed. 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, Zhang, and Wyzga (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. Families with higher incomes or greater preferences for cleaner air may be more likely to sort into neighborhoods with better air quality.

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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 which may include 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].

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.

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 lead 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, and only particulates were measured during the time period they examine, so that it was not possible for them to examine the effects of other pollutants.

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]

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[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, Schwartz, and Ransom, 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, Samet and McCarthy, 2000; Wilson, Mage, and Grant, 2000] suggests that much of what is outdoors comes indoors. Furthermore, although the cross-sectional correlation between ambient air quality and personal

III. METHODS

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 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.

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

$$(1) \qquad O_{ijmt} = \sum_{s=1}^{3} \left(P_{mt}^{s} \beta^{s} + w_{mt}^{s} \gamma^{s} \right) + x_{ijmt} \delta + Y_{t} + \varepsilon_{ijmt}$$

where O is an infant health outcome and 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 pollution from the monitor closest to the mother's residence in each of the the first, second, and third trimesters of her pregnancy, denoted by s. We construct these measures by taking the average pollution measure over the trimester, so β^{s} reflects the effect from a change in mean pollution levels for trimester s.[4] This measure captures high ambient levels sustained over a period of time. The w_{mt} represents daily precipitation, daily minimum and daily maximum temperature measures averaged over each trimester of the pregnancy. We control for weather because it may have independent effects on infant health outcomes (Samet et al. 1997).

The vector x_{ijmt} includes mother and child specific characteristics taken from the birth certificate.

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exposure is low (between .2 and .6 in most studies of PM 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 have also estimated models using the maximum daily value of pollution over the same intervals, but found that

These characteristics include dummy variables for the mother's age (19-24, 25-34, 35+) and education (12, 13-15, or 16+ years), an indicator for whether it is a multiple birth, controls for birth order (2^{nd}, 3^{rd}, 4^{th} or higher), indicators for whether the mother is married, whether the child is male, whether the mother is African-American, Hispanic, and other or unknown race, an indicator for whether the mother smokes, and the number of cigarettes if she smokes. These are all significant determinants of birth outcomes that have been included in many other studies. 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 Census block group as a proxy. We include dummy variables for missing maternal education, marital status, race, and smoking status, so as to be able to keep observations with missing independent variables. 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 reductions in infant mortality.

As previously mentioned, a limitation of model (1) is that pollution exposure is likely to be correlated with omitted characteristics of families that are themselves 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 to inland areas), we estimate models of the form:

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

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 to capture average neighborhood amenities to the extent they are similar.

Model (2) may still suffer from omitted variables bias. In particular, unobserved characteristics

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it was not statistically significant in any of our models.

of mothers (such as her regard for her own health) may be important, and may also be correlated with her choice of neighborhoods. Hence, we estimate:

$$(3) \qquad O_{ijmt} = \sum_{s=1}^{3}\left(P_{mt}^{s}\beta^{s} + w_{mt}^{s}\gamma^{s}\right) + x_{ijmt}\delta + Y_{t} + \varphi_{mt} * Q_{t} + \varsigma_{j} + \varepsilon_{ijmt}$$

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 are identified by variation in pollution at a particular monitor that a women is exposed to between pregnancies. Much of this variation is driven by changes in pollution levels over time and within the year. Air quality improvements over time are largely due to air quality regulations, and variation within the year (after controlling for seasonal effects and weather) are largely due to unpredictable variations in human activity.

It is important for identification that variation in infants' pollution exposure be uncorrelated with other characteristics of the infants or 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 .939 and .947, respectively, for CO are .301 and .271, respectively, and for PM are .410 and .407, respectively. This suggests that mothers do not appear to be systematically moving to cleaner or dirtier areas between births.

In order to examine infant mortality conditional on health at birth, we modify the models 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.

We then estimate a model in which the probability of death D_{ijmt} is specified as:

$$(4) \qquad D_{ijmt} = \alpha(t) + \sum_{s=1}^{4}\left(\Delta^s P_{mt}\beta^s\right) + w^s_{mt}\gamma^s + x_{ijmt}\delta + O_{ijmt}\pi + Y_t + \varphi_{mt} * Q_t + \varsigma_j + \varepsilon_{ijmt}$$

where $\alpha(t)$ is a measure of duration dependence and is specified as a linear spline function in the weeks since the infant's birth, with breaks after 1, 2, 4, 8, 12, 20, and 32 weeks. These break points were chosen 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 Δ^s indicating times since birth. Δ^1 equals one if time since birth is between 0 and 2 weeks, Δ^2 (Δ^3) between 2 and 4 (4 and 6) weeks and Δ^4 for over 6 weeks. Thus the effect of pollution as measured by β^s 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_{imt}) to the list of independent variables. To the extent that birth weight is a sufficient statistic for health at birth, π from equation (4) will capture the 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 focus on mothers who lost at least one child. These families may have other characteristics (besides pollution exposure) that lead to a higher risk of infant death. 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). These characteristics will be captured by the mother fixed effects specification. It would be more concerning if families with deaths were exposed to systematically different levels of pollution, but this does not seem to be the case. Mean ozone, CO, and PM10 measures in the trimester before birth are virtually identical in families with deaths and those without.

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 marginal infant. If the marginal infant who dies because of an increase in pollution is similar to one who is at risk of death because of the additional pollution but survives, then our results will tell us about the effects of variations in pollution in the observed range.

II. DATA

The location of each of 57 monitors, and what each one measures is shown in Figure 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 samples and models, though we will also show one specification that includes both CO and Ozone, the two pollutants that have the largest effects individually. Figure 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.

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 Agency (EPA).[5] 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 health at birth models) or for each week after birth (for the infant mortality hazard model). For the pollutants of interest, the daily measures we use are the 8-hour maximums of CO and O3 and the 24-hour average of PM10.[6] County level weather data come from the Surface Summary of the Day (TD3200) from the National Climatic Data Center.[7]

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 three columns show means for the full sample, the sample within 10 kilometers of an ozone monitor, and the sample of births to smoking mothers within 10 kilometers of an ozone monitor. Because different monitors measure different pollutants, the subsamples used in the

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

[6] The 8-hour maximum corresponds to taking the maximum 8-period moving average within a 24 hour period. We chose these measures because pollution standards are based on them. Since PM10 is not measured every day, the weekly mean for PM10 may be noisier than those for other pollutants. The measures we use are highly correlated with measures of short-term spikes in pollutants. For example, the correlation between the maximum 1 hour reading for CO and the maximum 8 hour average for CO ranges from .91 to .95, depending on the month of the year. For ozone, the comparable figures are .89 to .97.

[7] 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.

regression models are slightly different.[8] Of the 1.5 million births in New Jersey over our sample period, 42% were within 10 kilometers of an ozone monitor, with roughly 10% of these births to mothers who smoked. Columns 4 and 5 restrict the sample further to children with a sibling within the sample. These are the infants who identify the effects of pollution in the models with maternal fixed effects. Almost 20% of the total births are in the sibling sample and within 10 kilometers of a monitor. Finally, column 5 shows that when we look at the subset of mothers who had more than one child in the sample, lived within 10 kilometers of a monitor and were smokers (at both births), the sample becomes much smaller, but is still sizable at 21,116 births.

A comparison of columns 1, 2, and 3 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 1,000 births overall, 7.7 in the sample closer to monitors, and 9.9 among the smokers. Comparing column 2 to column 4 or column 3 to column 5 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.

Panel B gives means of the pollution measures for the subsets of the sample within 10 kilometers of a monitor. A comparison of columns 2 and 3 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 live in similar neighborhoods in terms of air quality to mothers with a single birth.[9]

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 a level close to 1 ppm by 2005. Figures 2 through 4

[8] Sample sizes also vary slightly for different outcomes because of missing values of the outcomes.
[9] 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 hour 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 PM10, the 24 hour average must not exceed 150 $\mu g/m^3$ more than once per year on average over three years (see http://www.epa.gov/air/criteria.html). For the period of our sample,

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.[10] The "a" series plot 3 month moving averages (corresponding to the measures of pollution we use in models of health at birth), while the "b" series plot 7 day moving averages (corresponding to the measures of pollution we use in the models of infant mortality). These plots show that adjusting for these factors accounts for seasonal and annual trends, though there is still considerable variation left that can identify the effects of pollution.

The third panel of Table 1 shows means of the control variables available in the Vital Statistics data. Mothers within 10 kilometers of a monitor are almost a year younger on average than the sample mean. It is striking that mothers within 10 kilometers of a monitor are also much more likely to be African American or Hispanic and have a half 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 kilometers of a monitor. In contrast, mothers with more than one birth in the sample look quite similar to mothers who are observed to have had only one birth.

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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.

[10] 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.

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

IV. RESULTS

Estimates of the effects of pollution on all mothers within 10 kilometers of a monitor are shown in Table 2. The first column shows estimates of equations (1), (2), and (3) for each pollutant. The mother fixed effects model, equation (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 (4) of Table 1). 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. 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. However, once we control for monitors and monitor*quarter (as in columns (2), (5), and (8)) the estimates suggest that CO in the last trimester of the pregnancy reduce birth weight, and CO also increases low birth weight. Now the only wrong-signed coefficient suggests that PM10 in the first trimester of pregnancy increases gestation. Finally, when we further control for mother fixed effects in columns (3), (6), and (9), the estimates for CO become slightly larger. Ozone in the second trimester now has a statistically significant negative effect on birth weight and gestation, and none of the estimates for PM10 are statistically significant. This pattern of results across specifications suggests the importance of controlling for both maternal and neighborhood fixed effects for accounting for confounding factors. It also suggests that in New Jersey, conditional on other observable characteristics of mothers, mothers in more polluted (more urban areas) have unobserved characteristics that make them more likely to have healthy infants.

The estimates indicate consistently negative effects of pollution on birth outcomes. The estimated effects are most robust and consistent for CO, where it is statistically significant for all three

outcomes and increases in magnitude as omitted variables are more carefully controlled. 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 a standard deviation is .79) would reduce average birth weight by 18.07 grams (from a base of 3,237 grams) — 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 .0083 (from a base of .106) – an eight percent increase in the incidence of low birth weight. The fact that the effect is greater 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. Finally, a one unit change in mean CO is estimated to reduce gestation by .08 weeks (from a base of 38.55 weeks) – a reduction in mean gestation of .2 percent.

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 165 grams in models that include monitor fixed effects, and each additional cigarette smoked reduces birth weight by 4.9 grams, for a total reduction of approximately 214 grams at the mean of 10 cigarettes per day. However, as Almond, Chay and Lee (2003) 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: In these models, being a smoker is estimated to reduce birth weight by 42.6 grams, and each cigarette reduces it a further 2.2 grams for a total reduction of about 65 grams in infants of women who smoke 10 cigarettes per day. Hence it would take a roughly 3.5 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 roughly two and a half times 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 a broader array of pollutants are harmful to the infants of smokers. Although the effects of CO on birth weight are no longer statistically significant in the model for birth weight, the point estimate of -39.1 in the model with mother fixed effects is twice as large as the Table 2 coefficient. The Table 4 coefficient on CO in the models of low birth weight is .04 compared to .008 in Table 2. For gestation, the Table 4 coefficient on CO is -.43 compared to -.07 in Table 2. These estimates indicate that the harmful effects of a one unit increase in 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 comparable 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.[11] 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 6 shows estimates of the effects of pollution on infant mortality from models based on equation (4). In these models, we control for birth weight in an effort to isolate the effect of pollution after the birth on health. Table 6 suggests that high CO exposures in the first two weeks of life increase the risk of death. Note that we control for the fact that more deaths occur in the first two weeks with our baseline hazard, so this estimates reflects the extent to which death within that time is hastened by pollution exposure. The coefficient on CO implies that a one unit increase in CO in the previous week would increase the risk of death by about 1 percentage point. As discussed above, we have chosen our sample to include only children who died and their siblings, so the base risk of death in this subsample is about 40% (2285 deaths divided by 5735 births). Hence, our estimate implies that a one unit increase in pollution in the previous week increases the risk of death by about 2.5%. We do not find any statistically significant impacts of ozone and PM10 on mortality. 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.

Tables 7 and 8 offer two specification checks. As discussed above, we believe that a major contribution of our study is that since we have mother's exact addresses, we can measure pollution exposure relatively accurately. If this is indeed a contribution, then we should see that models using mothers who are further away from monitors yield weaker results. Table 7 shows that this is indeed the case: If we focus on mothers who are 10 to 20 kilometers from a monitor, we do not find significant

[11] 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".

effects on health at birth (or, not shown, on infant mortality).[12]

In Table 8, we estimate models that include both CO and ozone. Although the sample size is somewhat reduced, the estimates for CO are even stronger than those shown in Table 2. 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.

V. 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 emissions standards has been blocked by the Environmental Protection Agency. The Agency first argued that it had no authority to regulate the green house 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 piece-meal approach offered by the state. The state is currently suing the federal government over the issue. Should the state

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

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 equivalent to getting a women smoking 10 cigarettes a day to quit. We also find some evidence of significant effects of PM10 and ozone on health at birth, particularly among smokers, and we find that infants of smokers are at much greater risk of negative effects from pollution exposure. We further find that a one unit increase in mean CO levels in the first two weeks of life increases the probability of infant death by about 2.5 percent. Over our sample period, average levels of CO in New Jersey declined 1.4 ppm from 2.3 to .9 ppm. Our estimates imply that this decline led to about 28 fewer deaths per year by 2003.[13]

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, when they occurred in the pregnancy, or intensity) 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. If high levels of pollution cause vulnerable fetuses to be lost, 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.

[13] This is calculated using the mean infant mortality rate of 6.88 per 1,000 over the sample period and the number of births in 2003 (about 120,000).

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Figure 1: Location of Air Monitors in New Jersey

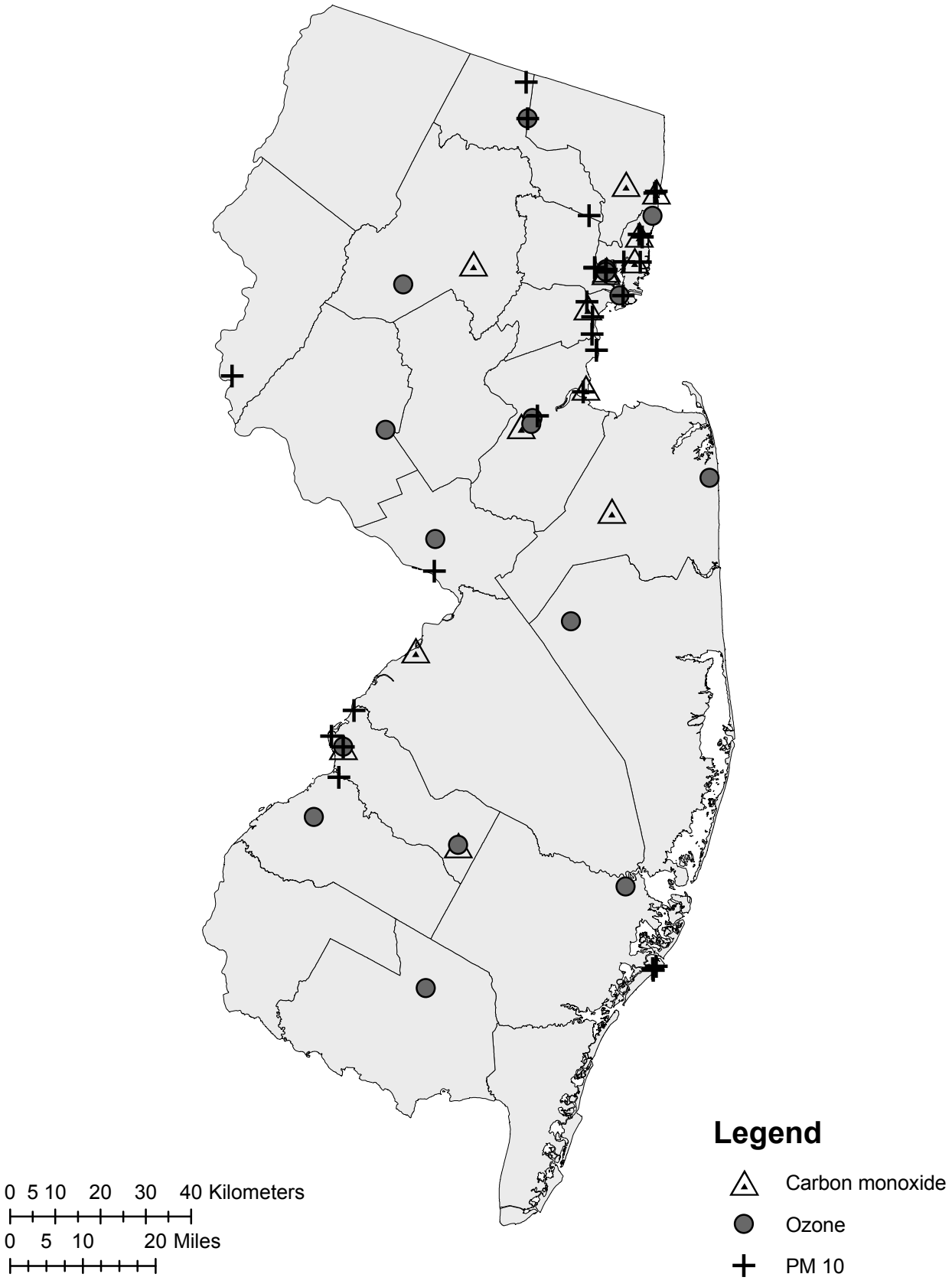

Figure 2a: Air Quality at Camden Lab Monitor, 90 Day Moving Average of CO

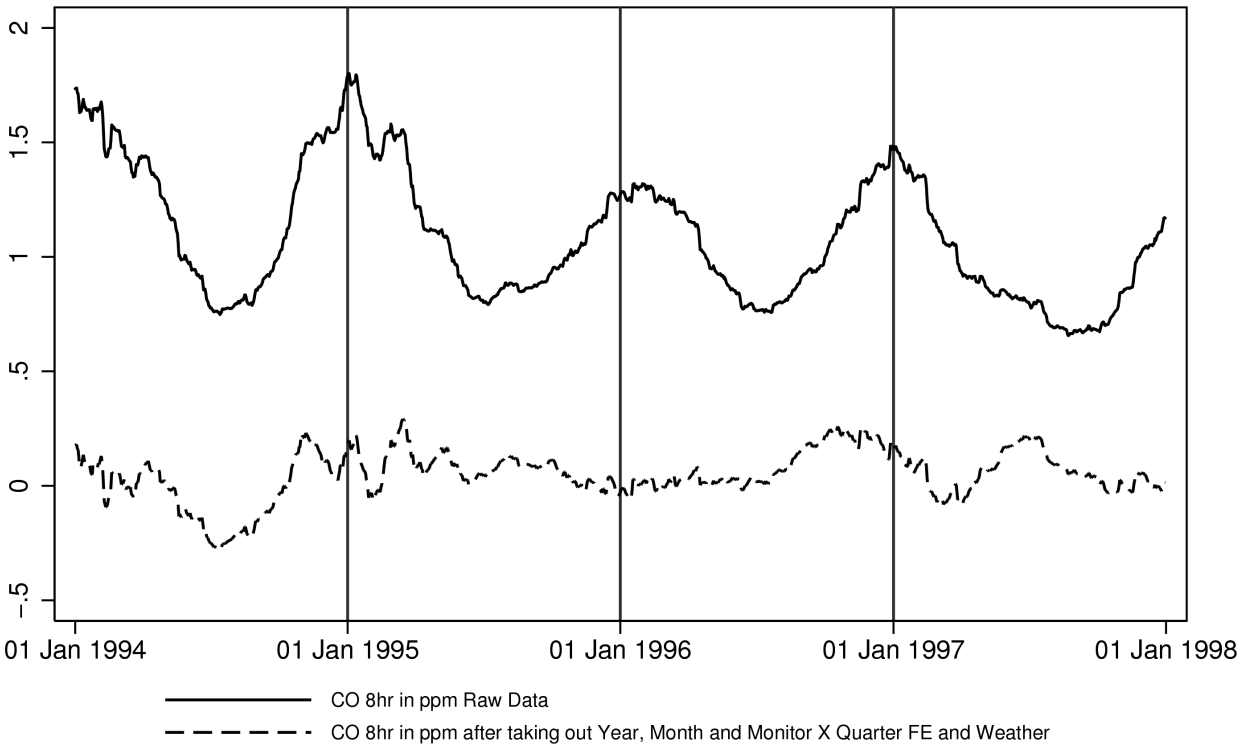

Figure 2b: Air Quality at Camden Lab Monitor, 7 Day Moving Average of CO

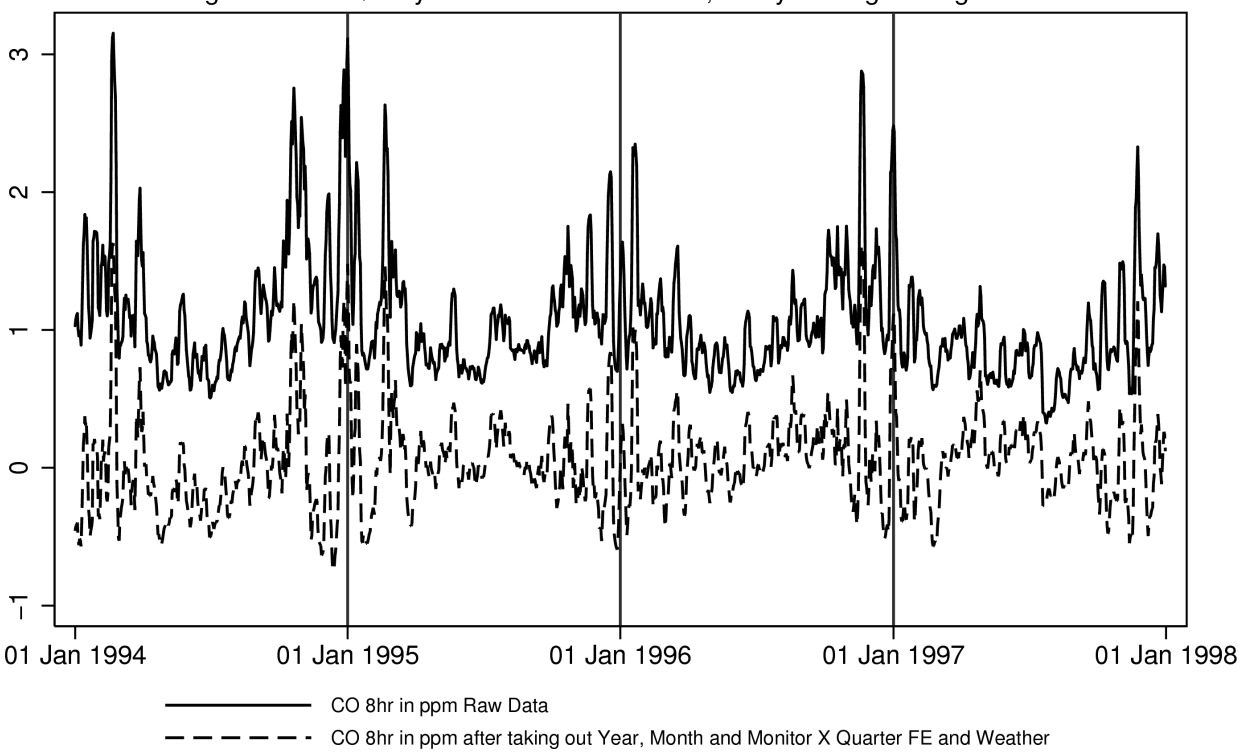

Figure 3a: Air Quality at Camden Lab Monitor, 90 Day Moving Average of OZ

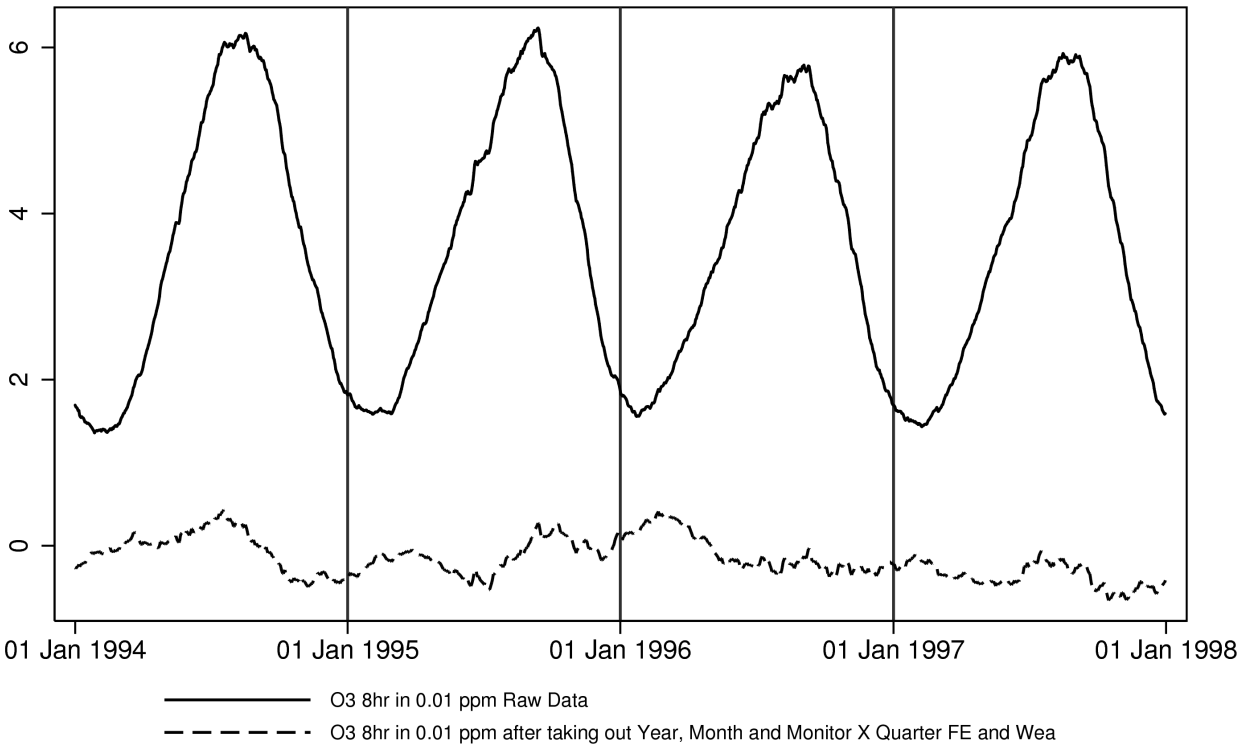

Figure 3b: Air Quality at Camden Lab Monitor, 7 Day Moving Average of OZ

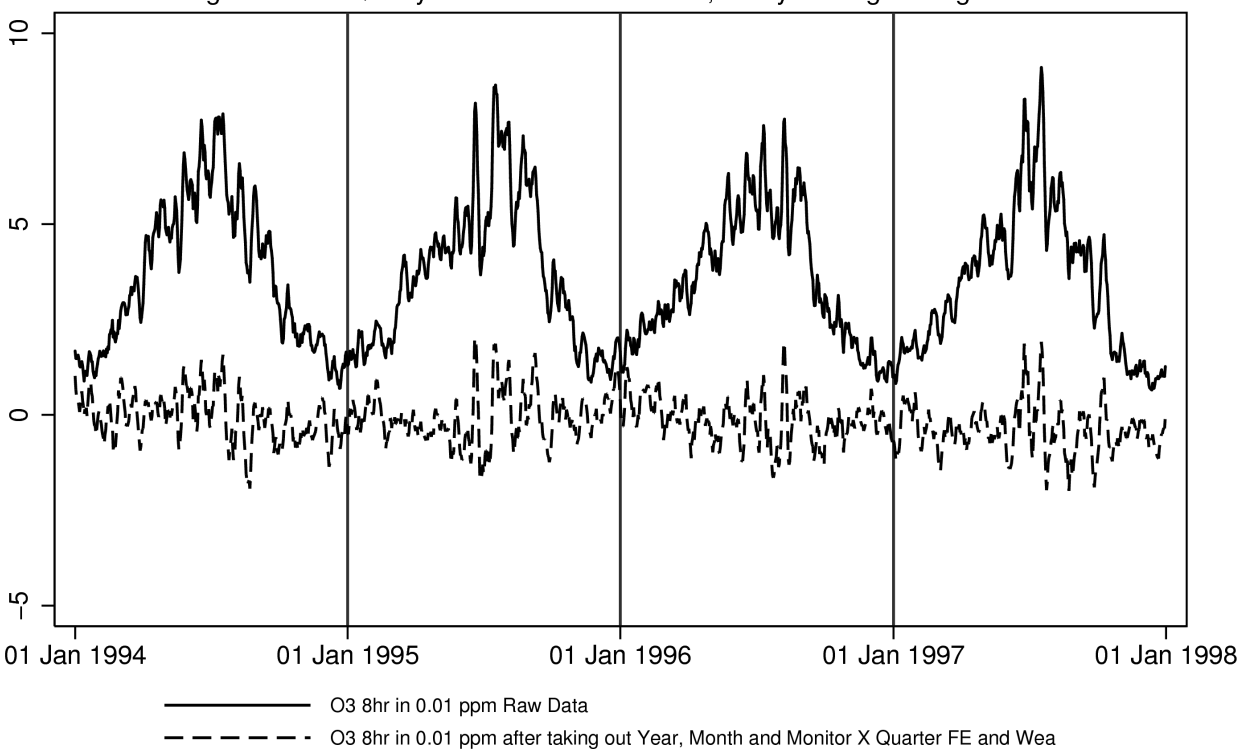

Figure 4a: Air Quality at Camden Lab Monitor, 90 Day Moving Average of PM10

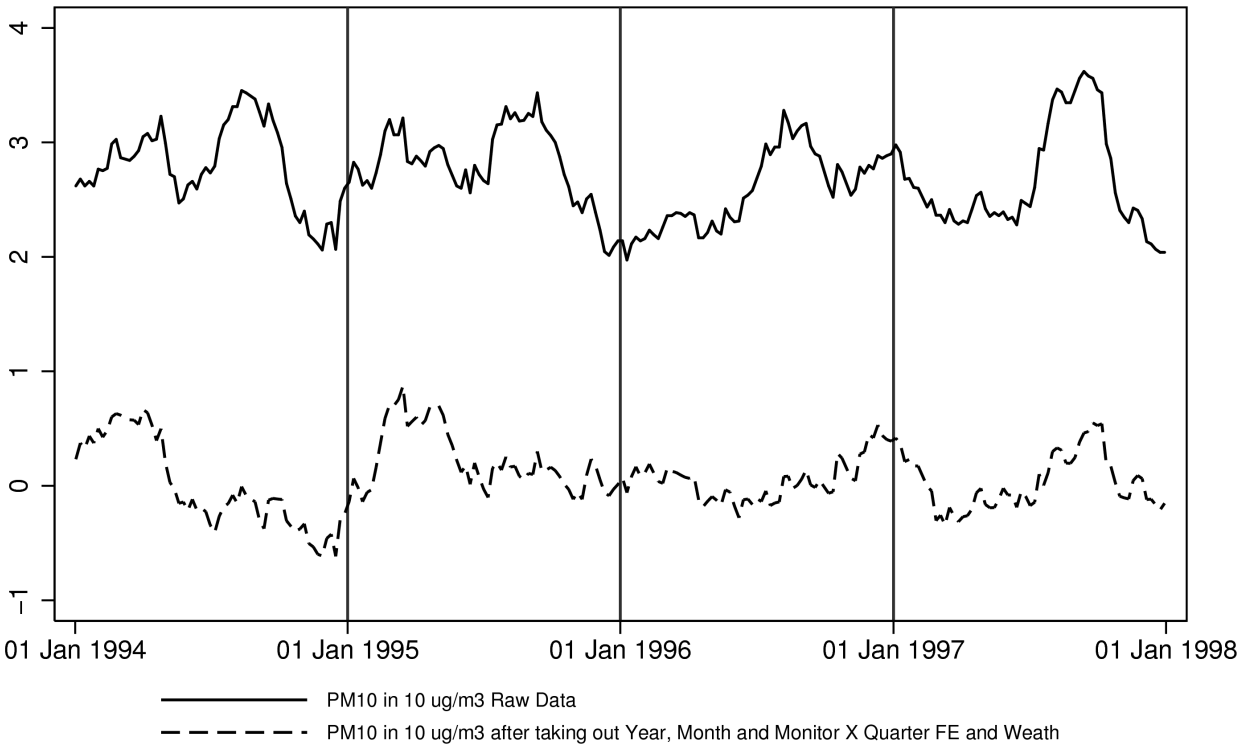

Figure 4b: Air Quality at Camden Lab Monitor, 7 Day Moving Average of PM10

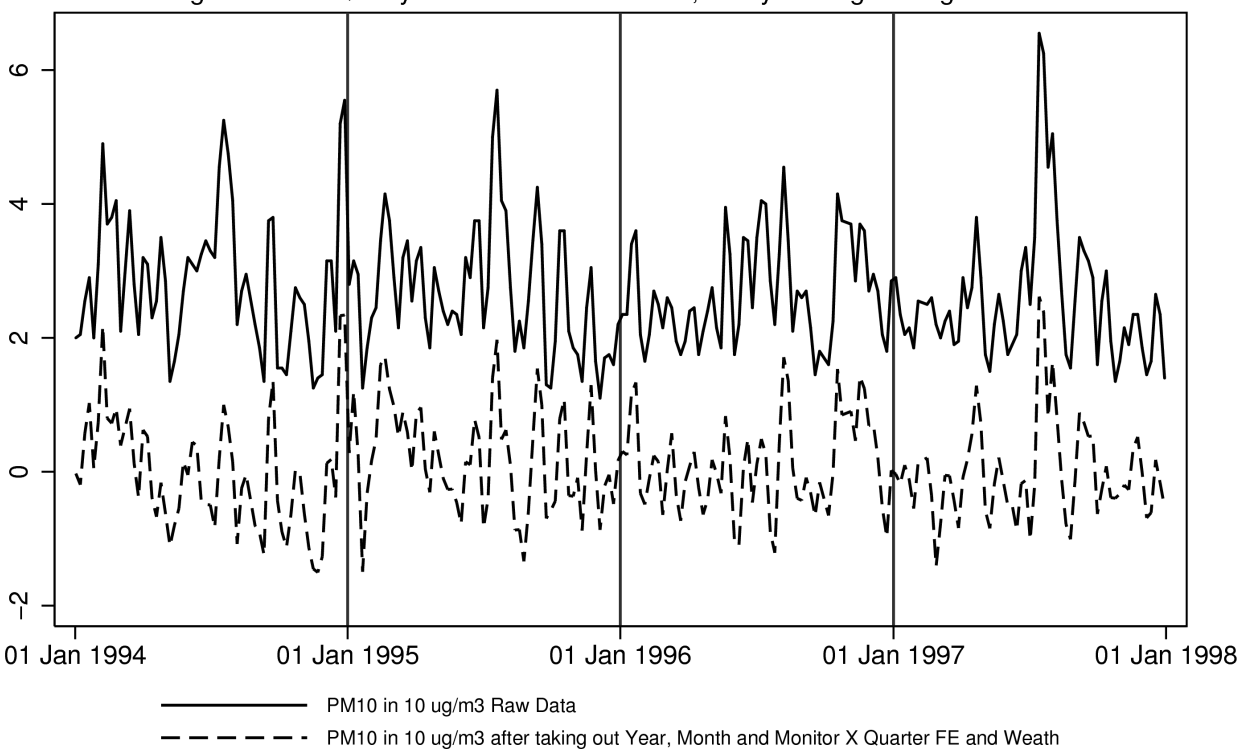

Table 1: Sample Means

	1	2	3	4	5
	All	<10km monitor	<10km monitor & smoking	like (2) but >=1 sibling	like (3) but >=1 sibling
Number of observations	1511180	629953	62068	283882	21116
Panel A: Outcomes					
Birth weight in Grams	3319.7	3267.37	3054.71	3236.51	2937.58
	[615.58]	[650.54]	[656.04]	[660.54]	[682.10]
Infant death	0.0069	0.0077	0.0099	0.0086	0.0128
Gestation	38.83	38.71	38.28	38.55	37.84
	[2.30]	[2.47]	[2.89]	[2.64]	[3.21]
Low birth weight	0.076	0.088	0.157	0.106	0.210
Panel B: Pollution Measures Last Trimester Before Birth					
Ozone (8 hour moving average in .01 ppm)		3.60	3.61	3.60	3.57
		[1.49]	[1.52]	[1.50]	[1.52]
CO (8 hour moving average in ppm)		1.64	1.55	1.60	1.51
		[0.79]	[0.77]	[0.76]	[0.73]
PM10 (24 hour moving average in 10 ug/m3)		2.99	2.99	2.97	3.01
		[0.74]	[0.74]	[0.74]	[0.75]
Ozone (8 hour moving average in .01 ppm)		3.60	3.60	3.62	3.55
		[1.79]	[1.82]	[1.80]	[1.82]
CO (8 hour moving average in ppm)		1.64	1.55	1.60	1.51
		[0.88]	[0.86]	[0.85]	[0.82]
PM10 (24 hour moving average in 10 ug/m3)		2.98	2.97	2.95	2.99
		[1.50]	[1.49]	[1.48]	[1.50]
Panel C: Control Variables					
Mother Age in Years	29.23	28.25	27.44	27.75	26.92
	[6.00]	[6.16]	[5.99]	[6.00]	[5.65]
Mother African American	0.19	0.30	0.41	0.35	0.54
Mother Hispanic	0.18	0.23	0.14	0.20	0.10
Mother Years of Education	13.28	12.79	11.77	12.74	11.46
	[2.64]	[2.68]	[1.95]	[2.57]	[1.84]
Multiple Birth	0.032	0.029	0.026	0.060	0.069
Mother married	0.72	0.61	0.36	0.59	0.29
Birth parity	1.98	2.00	2.46	2.44	3.33
	[1.15]	[1.19]	[1.61]	[1.29]	[1.86]
Child Male	0.51	0.51	0.51	0.51	0.51
Mother smoking	0.09	0.10	1.00	0.12	1.00
Number of cigarettes per day	1.01	1.03	10.06	1.16	10.35
	[3.90]	[3.91]	[7.62]	[4.10]	[7.57]
Median family income census tract 1989 ($10,000)	4.66	4.05	3.53	3.97	3.25
	[1.77]	[1.59]	[1.38]	[1.62]	[1.31]
Fraction poor in census tract 1989	0.09	0.13	0.17	0.14	0.20
	[0.10]	[0.13]	[0.14]	[0.13]	[0.14]
Mean precipitation in previous 90 days	13.02	13.05	13.11	12.98	13.03
	[4.21]	[4.15]	[4.16]	[4.08]	[4.07]
Mean of daily max temperature previous 90 days	63.69	64.09	64.42	64.10	64.67
	[14.65]	[14.74]	[14.70]	[14.74]	[14.74]
Mean of daily min temperature previous 90 days	21.33	22.04	22.25	21.87	22.43
	[15.19]	[15.16]	[15.11]	[15.18]	[15.12]

Notes: Standard deviations in brackets. Column [5] contains births of smoking mothers that have a least one sibling where the mother was also smoking during the pregnancy.

Table 2: Effects of Air Pollution on Health at Birth - All Mothers < 10 km from a Monitor

	1	2	3	4	5	6	7	8	9
		CO			Ozone			PM10	
A. Models of Birth Weight									
3rd trimester pollution	-12.73	-14.98	-18.07	7.003	-2.667	-3.701	-1.364	0.149	-2.979
	[5.393]*	[6.505]*	[6.690]*	[2.828]*	[3.938]	[3.966]	[2.499]	[2.960]	[3.016]
2nd trimester pollution	10.90	-0.990	4.912	0.685	-2.417	-8.455	-3.799	-0.986	-2.090
	[6.453]	[7.236]	[7.396]	[3.329]	[4.039]	[4.138]*	[2.487]	[3.095]	[3.131]
1st trimester pollution	-0.701	-5.431	-5.664	4.827	2.013	-3.757	-4.249	-0.821	-2.846
	[5.180]	[6.273]	[6.429]	[2.879]	[3.833]	[3.933]	[2.439]	[2.919]	[2.936]
Observations	301577	301577	301577	257502	257502	257502	272933	272933	272933
B. Models of Low Birth Weight (Coefficients and standard errors multiplied by 100)									
3rd trimester pollution	0.512	0.710	0.832	-0.396	-0.112	0.121	0.0616	-0.00717	0.0715
	[0.253]*	[0.307]*	[0.359]*	[0.137]*	[0.192]	[0.217]	[0.120]	[0.144]	[0.163]
2nd trimester pollution	-0.385	-0.181	-0.374	-0.116	-0.0998	-0.0737	0.0874	0.104	0.0785
	[0.303]	[0.341]	[0.397]	[0.162]	[0.198]	[0.226]	[0.120]	[0.149]	[0.169]
1st trimester pollution	-0.0306	0.0853	0.519	-0.0291	0.206	0.389	0.164	0.150	0.390
	[0.243]	[0.298]	[0.345]	[0.138]	[0.186]	[0.215]	[0.116]	[0.139]	[0.159]*
	302012	302012	302012	257920	257920	257920	273420	273420	273420
C. Models of Gestation (Coefficients and standard errors multiplied by 100)									
3rd trimester pollution	-4.377	-5.409	-7.737	3.159	-1.224	-0.531	2.044	3.431	1.956
	[2.292]	[2.776]	[3.012]*	[1.224]*	[1.726]	[1.814]	[1.066]	[1.268]*	[1.375]
2nd trimester pollution	3.188	-0.237	3.990	0.427	-3.279	-3.713	-2.534	-0.808	-1.523
	[2.737]	[3.085]	[3.331]	[1.454]	[1.760]	[1.893]*	[1.069]*	[1.344]	[1.428]
1st trimester pollution	0.270	-0.721	-3.778	4.237	0.884	-1.440	-1.944	0.596	-0.521
	[2.182]	[2.669]	[2.894]	[1.239]*	[1.649]	[1.801]	[1.051]	[1.268]	[1.339]
Observations	297079	297079	297079	253664	253664	253664	267701	267701	267701
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: 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, average daily minimum and maximum temperature in each 3 month period before the birth, month dummies, and year dummies. Regressions also include indicators for missing values. Standard errors in brackets, clustered on the mother level except for mother fixed effects specification. An asterisk indicates that the coefficient is significant at the 95% level of confidence.

Table 3: Effects of Smoking on Health at Birth - All Smoking 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	-165.4	-165.0	-42.57
	[6.515]*	[6.513]*	[6.645]*
# Cigarettes per day	-4.907	-4.939	-2.159
	[0.492]*	[0.492]*	[0.481]*
# Observations	301577	301577	301577
B. Models of Low Birth Weight (Coefficients and standard errors multiplied by 100)			
Mother smokes	4.949	4.916	0.714
	[0.357]*	[0.357]*	[0.357]*
# Cigarettes per day	0.191	0.190	0.126
	[0.0282]*	[0.0282]*	[0.0259]*
# Observations	302012	302012	302012
C. Models of Gestation (Coefficients and standard errors multiplied by 100)			
Mother smokes	-32.25	-31.78	-2.842
	[2.911]*	[2.908]*	[2.998]
# Cigarettes per day	-1.144	-1.150	-0.604
	[0.224]*	[0.224]*	[0.218]*
# Observations	297079	297079	297079
Monitor * Quarter Fixed Effects	no	yes	yes
Mother Fixed Effects	no	no	yes

Notes: These coefficients are from the models in columns (1)-(3) in Table 2
See Table 2 for further notes

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	-39.10	-19.29	-24.41
	[27.94]	[14.34]	[11.71]*
2nd trimester pollution	10.43	-32.43	-36.43
	[31.29]	[15.29]*	[12.13]*
1st trimester pollution	0.237	-15.41	3.437
	[27.38]	[14.67]	[11.67]
Observations	20439	20467	20044
B. Models of Low Birth Weight (Coefficients and standard errors multiplied by 100)			
3rd trimester pollution	4.401	-0.255	0.429
	[1.959]*	[1.005]	[0.825]
2nd trimester pollution	-4.282	1.641	1.775
	[2.192]	[1.072]	[0.855]*
1st trimester pollution	0.854	1.838	1.635
	[1.918]	[1.028]	[0.822]*
Observations	20469	20504	20086
C. Models of Gestation (Coefficients and standard errors multiplied by 100)			
3rd trimester pollution	-42.77	-11.83	-3.211
	[15.26]*	[7.748]	[6.404]
2nd trimester pollution	20.25	-18.38	-14.75
	[17.09]	[8.248]*	[6.624]*
1st trimester pollution	-14.40	-15.21	-8.280
	[14.91]	[7.932]	[6.377]
Observations	19934	20121	19497
Monitor * Quarter Fixed Effects	yes	yes	yes
Mother Fixed Effects	yes	yes	yes

Notes: See Table 2.

Table 5: Effects of CO on Health at Birth - Mothers from Vulnerable Groups
< 10 km From a Monitor - Models with Mother Fixed Effects

	1	2	3	4	5	6
	<age 19	>= age 35	Risk factors for the preg.	Black	<12 yrs ed.	Income <30,000
A. Models of Birth Weight						
Mean CO 1-3 months	-24.85	-42.84	-25.03	-4.951	-13.07	-12.55
before birth	[10.37]*	[10.31]*	[8.876]*	[8.686]	[9.264]	[8.809]
Mean CO 4-6 months	7.028	20.72	-0.616	12.63	10.29	9.368
before birth	[12.86]	[12.08]	[10.57]	[10.11]	[11.07]	[10.49]
Mean CO 7-9 months	-6.292	2.675	-24.30	-7.15	-11.27	-13.64
before birth	[10.06]	[9.837]	[8.506]*	[8.375]	[8.901]	[8.450]
Observations	301577	301577	301577	301577	301577	301577
B. Models of Low Birth Weight (Coefficients and standard errors multiplied by 100)						
Mean CO 1-3 months	1.298	1.797	1.097	0.394	0.818	0.694
before birth	[0.557]*	[0.554]*	[0.477]*	[0.467]	[0.498]	[0.473]
Mean CO 4-6 months	-1.239	-0.724	-0.456	-0.671	-1.239	-0.572
before birth	[0.691]	[0.649]	[0.568]	[0.543]	[0.595]*	[0.563]
	1.076	0.371	1.626	0.421	0.985	0.761
before birth	[0.541]*	[0.529]	[0.457]*	[0.450]	[0.478]*	[0.454]
Observations	302012	302012	302012	302012	302012	302012
C. Models of Gestation (Coefficients and standard errors multiplied by 100)						
Mean CO 1-3 months	-12.93	-12.27	-10.43	-6.896	-7.309	-6.265
before birth	[4.664]*	[4.638]*	[3.989]*	[3.907]	[4.163]	[3.959]
Mean CO 4-6 months	7.143	5.778	0.872	0.907	4.999	1.499
before birth	[5.778]	[5.433]	[4.746]	[4.547]	[4.975]	[4.708]
Mean CO 7-9 months	-3.585	-2.279	-11.70	-4.648	-3.485	-3.497
before birth	[4.520]	[4.427]	[3.821]*	[3.767]	[3.998]	[3.798]
Observations	297079	297079	297079	297079	297079	297079

Notes: The columns show specifications that allow the effect of pollution vary by characteristics of the mother. The models are estimated as in Table 2, but the pollution measures are interacted with a dummy for the characteristic of the mother. For example in the second column the regression included three CO measures (for each trimester) for mothers under the age of 19 and 3 CO measures for mothers 20 or older. The first set would be set to zeroes for mothers older than 20, the second set for mothers 19 or younger. Only the set of interactions with the demographic group denoted in the column title are reported. The other set of interactions is not shown, but generally very similar to the main effects in the first column.

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 100)

	1 CO	2 Ozone	3 PM10
Mean pollutant if 0 <= week <2	107.0	8.407	-11.13
	[37.60]*	[18.61]	[25.19]
Mean pollutant if 2 <= week <4	-22.29	4.154	-7.886
	[15.79]	[9.075]	[10.72]
Mean pollutant if 4 <= week <6	14.65	9.014	8.583
	[10.89]	[5.464]	[5.908]
Mean pollutant if 6 <= week	-5.993	2.752	-0.244
	[5.877]	[3.426]	[1.845]
Week after birth	-1720.0	-1713.1	-1775.6
	[54.89]*	[58.24]*	[63.82]*
Week after birth-1 * Indicator	1838.9	1654.5	1673.7
for week >= 1	[100.4]*	[110.8]*	[121.5]*
Week after birth-2 * Indicator	-199.8	5.859	13.92
for week >= 2	[86.22]*	[97.58]	[108.5]
Week after birth-4 * Indicator	78.48	44.53	83.77
	[24.09]*	[26.96]	[30.36]*
Week after birth-8 * Indicator	1.073	8.407	6.223
for week >= 8	[7.483]	[8.050]	[8.991]
Week after birth-12 *	-2.018	-4.844	-5.716
Indicator for week >= 12	[4.375]	[4.852]	[5.508]
Week after birth-20 *	1.146	2.931	0.810
Indicator for week >= 20	[1.552]	[1.696]	[2.074]
Week after birth-32 *	1.883	1.274	2.689
Indicator for week >= 32	[0.625]*	[0.672]	[0.827]*
Observations	188690	161758	130251
Number of Births	5735	5017	4490
Number of Deaths	2285	2010	1840
Number of Mothers	2207	1938	1778

Notes: See Table 2. Models also control for infant's birth weight. Standard errors are clustered on mother level in all models. All models include mother fixed effects.

Table 7: Effects of Air Pollution on Health at Birth - All Mothers > 10 km and < 20 km from a Monitor

	1 CO	2 Ozone	3 PM10
A. Models of Birth Weight			
3rd trimester pollution	0.847	-5.760	-9.575
	[7.958]	[3.757]	[4.307]*
2nd trimester pollution	-15.90	1.114	-3.166
	[8.543]	[3.852]	[4.465]
1st trimester pollution	-18.93	-2.414	-7.470
	[7.823]*	[3.662]	[4.052]
Observations	221973	242833	120347
B. Models of Low Birth Weight (Coefficients and standard errors multiplied by 100)			
3rd trimester pollution	0.587	0.367	0.345
	[0.400]	[0.190]	[0.219]
2nd trimester pollution	0.320	-0.102	0.0222
	[0.430]	[0.194]	[0.227]
1st trimester pollution	0.418	0.160	0.435
	[0.393]	[0.185]	[0.206]*
Observations	222387	243269	120588
C. Models of Gestation (Coefficients and standard errors multiplied by 100)			
3rd trimester pollution	-1.811	-0.480	-1.740
	[3.266]	[1.546]	[1.750]
2nd trimester pollution	-0.853	-0.462	-0.353
	[3.509]	[1.587]	[1.814]
1st trimester pollution	-1.888	1.120	-2.672
	[3.212]	[1.509]	[1.647]
Observations	219806	239393	118971

Notes: See Table 2. 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)	-22.58	1.028	-9.995
	[7.397]*	[0.401]*	[3.363]*
2nd trimester CO (in ppm)	8.456	-0.820	5.492
	[8.110]	[0.439]	[3.687]
1st trimester CO (in ppm)	-5.771	0.864	-4.926
	[7.024]	[0.380]*	[3.193]
3rd trimester ozone (in .01 ppm)	-6.028	0.125	-3.616
	[3.693]	[0.200]	[1.678]*
2nd trimester ozone (in .01 ppm)	0.441	-0.105	-1.837
	[4.131]	[0.224]	[1.877]
1st trimester ozone (in .01 ppm)	-5.149	0.291	-0.974
	[3.776]	[0.204]	[1.716]
Observations	264814	265214	260560

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