Air Pollution and Infant Health:

What Can We Learn From California's Recent Experience?

Janet Currie, NBER and Dept. Of Economics UCLA, 405 Hilgard Ave.

Los Angeles CA, 90095-1477, currie@ucla.edu

Matthew Neidell, Columbia University

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Abstract: We examine the impact of air pollution on infant death in California over the 1990s. Our work offers several innovations: First, many previous studies examine populations subject to far greater levels of pollution. In contrast, the experience of California in the 1990s is clearly relevant to current debates over the regulation of pollution. Second, many studies examine a single pollutant in isolation, generally because of data limitations. We examine three "criteria" pollutants in a common framework Third, we use rich individual-level data to investigate effects of pollution on infant mortality and low birth weight in a common framework, and we consider the potential impact of pollution on fetal deaths. Fourth, we develop an identification strategy based on within zip code-month variation in pollution levels to control for potentially important unobserved characteristics of high pollution areas as well as seasonal factors. We find a significant effect of CO on infant mortality and conclude that reductions in carbon monoxide over the 1990s saved approximately 1,000 infant lives in California.

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Air quality regulations are costly to both producers and consumers and the optimal level of pollution abatement is hotly contested. For example, in October 2002, the Bush administration joined Daimler Chrysler and General Motors in a lawsuit against Californian regulations that would have mandated that one in ten cars sold in California be "low emission" or "zero-emission" vehicles, beginning in 2003 (Doggett, 2002; New York Times, October 14, 2002). New standards for ozone and particulates were proposed by the Environmental Protection Agency (EPA) in 1997 but were held up in the courts until a Supreme Court decision in 2001 (Stafford, 2001).

Pollution abatement is often justified as something that will promote health yet there is still much to be learned about the specific health effects. The EPA did not include infant mortality in the primary quantitative benefit analysis of the 1990 Clean Air Act Amendments in 1999 (U.S. EPA 1999) because the weight of the scientific evidence linking infant health to air pollution was viewed as insufficient.[1]

This paper addresses this issue by examining the impact of air pollution on infant health in California over the 1990s. Infants are of interest for two reasons. First, policy makers and the public are highly motivated to protect these most vulnerable members of society. Second, in the case of infant death, the link between cause and effect is immediate, whereas for adults, diseases today may reflect pollution exposure that occurred many years ago.[2]

[1] As of May 12, 2003, the EPA's Scientific Advisory Board was debating whether to include an analysis of infant health effects in its 2003 report to Congress on the benefits of the Clean Air Act. However, they had determined that "[these] estimates are not meant to be additive to the primary estimates of mortality" (U.S. EPA, 2003, page 6-13).

[2] California's experience is also of special interest, since under the Clean Air Act of 1970, it is the only state allowed to set automobile emission standards at a level higher than the federal standard. Other states may adopt California's standards, but may not draft their own.

Our work offers several innovations over the existing literature. First, many previous studies examine populations subject to greater levels of pollution because they lived further in the past or in some more heavily polluted place. In contrast, the experience of California in the 1990s is clearly relevant to the contemporary debate over pollution levels in the United States.

Second, many studies examine a single pollutant in isolation, generally because of data limitations. We examine three "criteria" pollutants that are commonly monitored in the U.S.: Ozone (O3), carbon monoxide (CO), and particulate matter (PM10). Thus our results may shed light on the important question of which pollutants are most harmful to infants.[3]

Third, we exploit rich individual-level data to estimate linear models that approximate hazard models, where the hazard is defined over weeks of life and the baseline hazard is specified as a flexible non-parametric spline. A fourth innovation is that we consider possible effects of pollution on fetal death, rather than focusing only on live births.

Fifth, while epidemiological studies have documented correlations between pollution and poor infant outcomes, it is possible that these correlations reflect some omitted characteristics (such as differences in socio-economic status or pollution of ground water) that are correlated with both air pollution and infant health outcomes. We

3 An earlier version of this paper also examined Nitrogen Dioxide (NO2). NO2 is an important precurser of particulate matter and is highly correlated with both CO and PM10 as it comes from many of the same sources. We found little evidence that NO2 had an independent effect on infant death and so we have excluded it here. We do not examine the two other criteria pollutants, SO2 and lead because levels are now so low that many monitors have been removed from service.

will control for this possibility both by including a rich set of covariates, such as whether the birth was covered by public health insurance, and by estimating models with zip code-month level fixed effects, which will capture any unobserved characteristics of zip codes that are unchanged over time as well as seasonal effects. Hence, only unusual changes in pollution within zip codes and months are used to identify the effects.

Our estimates confirm that air pollution has a significant effect on infant mortality even at the relatively low levels of pollution experienced in recent years. In particular the reductions in CO that occurred over the 1990s saved approximately 1,000 infant lives in California. We find little evidence that pollution affects fetal death or the probability of low birth weight among infants with gestation of 26 weeks or more.

The rest of the paper is laid out as follows: Section II provides necessary background information about the previous literature and the ways in which pollution may affect infant health. Section III describes our data while methods are described in Section IV. Section V offers results, and Section VI ends with a discussion and conclusions.

II. Background

Carbon Monoxide 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 in any case, CO may be particularly harmful to them. As much as 90% of CO in cities comes from motor vehicle exhaust (EPA, January 1993).

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 (U.S. EPA, 2003b). The mechanism through which particles harm health are controversial. The leading theory is that they cause an inflammatory response which weakens the immune system (Seaton, et al. 1995). We focus on PM10, particles less than 10 microns in diameter, although many older studies use measures of Total Suspended Particles or TSPs. In general one would expect TSP and PM10 to move together because PM10 is a component of TSP, but some of the larger particles included in TSP may be less damaging than the particles found in PM10.

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 O3 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).

Compliance with standards for PM10 is assessed by looking at annual means as well as 24-hour means while compliance with standards for O3 and CO is assessed by examining whether the level of pollution exceeded the standard over any eight-hour period during the year. These different approaches to standards suggest that the effects of PM10 may be expected to be cumulative while the effects of CO and O3 are expected to be more acute.

A link between air pollution and infant health has long been suspected although the exact biological mechanisms through which it occurs are not known. We also know little about what levels of these pollutants are sufficient to affect infant mortality (death in the first year of life) or 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. In infants, a weakened immune system could make them more susceptible to death from a wide range of causes.

Since motor vehicle exhaust is a major contributor of CO and PM10, these pollutants may themselves be markers for other components of exhaust such as polycyclic aromatic hydrocarbons (PAHs), acetonitrile, benzene, butadiene, and cyanide. Many of these compounds have been shown to have effects on developing fetuses in animal studies which may include retarded growth.[4] Studies in humans have shown elevated levels of an enzyme induced by PAHs in women about to have preterm deliveries (Huel et al., 1993). However, this research gives very little guidance about what levels of pollution might be necessary to induce negative effects or about when fetuses are most vulnerable.

Many studies have demonstrated links between very severe pollution episodes and increased mortality of infants and others. For example, Logan and Glasg (1953) found dramatic increases in cardiopulmonary mortality during a killer fog that occurred in London, England in 1952. Chart 1 summarizes some of the more recent studies,

[4] The web site http://www.epa.gov/ttn/atw/hapindex.html provides a list of the chemicals present in vehicle exhaust, and evidence regarding their health effects.

dividing them into two groups. The first group focus on the link between poor infant outcomes and high levels of pollution. Most of these studies report negative associations between pollution and outcomes. The second part of the chart focuses on U.S. studies, many of which also report a link between air pollution and infant health, although some do not. For example, Lipfert, Zhang, and Wyzga (2000) find that while they can replicate previous findings of a negative effect of PM10 on infant health, the result is not robust to changes in specification.

An important limitation of all of these studies is that it is possible that the observed relationships could reflect an unobserved factor that was correlated with both air pollution and child outcomes. This is likely to be a greater problem in studies such as Lui et al. (2003) that do not control for factors like maternal education, but it may be a problem even in studies that include such controls. Suppose for example, that areas with high levels of air pollution also tended to have high levels of water pollution. If water pollution causes infant deaths but is unobserved, then one might falsely conclude that air pollution was to blame for infant deaths, with potentially negative consequences for remediation efforts. Similarly, as we will show below, zip codes with high pollution have many other characteristics that may have a direct effect on infant outcomes, such as high rates of teen parenthood and low average levels of education. Many previous studies have not controlled adequately for these characteristics.

Two studies by Chay and Greenstone deal with the problem of omitted confounders by focusing on "natural experiments" provided by the implementation of the Clean Air Act of 1970 and geographic variation in pollution levels induced by the

recession of the early 1980s.[5] Chay and Greenstone show that on average TSPs fell from 95 to 60 micrograms per cubic meter of air between 1970 and 1984. However, both the Clean Air Act and the recession induced sharper reductions in TSPs 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 TSPs associated with the Clean Air Act (recession) led to between five and eight (four and seven) fewer infant deaths per 100,000 live births but had little effect on the incidence of low birth weight.[6]

Although these studies provide compelling evidence of the link between pollution and infant health, it is not clear that reductions from the much lower levels of ambient pollution today would have the same effect. For example, it might be the case that only pollution above some threshold is harmful, and pollution has already been reduced below that threshold. Moreover, the Chay and Greenstone studies cannot speak to the question of whether other pollutants affect infant health because only TSPs were measured during the time period that they study.

In this paper, we propose an alternative identification strategy using individual-level data and exploiting within-zip code-month variation in pollution levels. We create measures of pollution at the zip code-week level and control for individual differences

5 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.

6 Although Almond, Chay, and Lee (2002) argue that birth weight does not have a causal effect on infant mortality, low birth weight is still widely acknowledged to be the leading indicator of poor health at birth.

between mothers that may be associated with variation in birth outcomes. As we show below, even after controlling for seasonal differences at the zip code-month level, there is sufficient variation in pollution levels to identify an effect. Using this strategy allows us to identify the effects of pollution in more recent data and to compare the effects of several criteria pollutants. The zip code-month fixed effects and the individual data allow us to control for many factors (such as poverty) which are both strongly geographically concentrated and associated with poorer prospects for infants. They also control for the possible seasonal differences in mortality within zip codes, although as we show below there is little evidence of a seasonal pattern in mortality in California.

A final 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.

The research on the relationship between indoor and outdoor air quality has established several results (see Spengler, Samet and McCarthy (2000) for a survey of the literature on indoor air pollution). First, much of what is outdoors comes indoors–estimates of the fraction of indoor fine particles that originated outdoors range from 46% to 84% depending on whether the house was air-conditioned and whether windows were left open (Wilson, Mage, and Grant, 2000). The rate at which outdoor air circulates through a house depends on the season and the weather, variables we will control for in our analysis.

Second, although the cross-sectional correlation between ambient air quality and personal 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 (Wilson, Mage, and Grant, 2000)

III Data

Detailed data on atmospheric pollution comes from the California Environmental Protection Agency's air monitoring stations. These monitors record ambient levels of "criteria pollutants", which are those air pollutants considered most responsible for urban air pollution. Monitors tend to be located in the most densely populated areas of the state, and also in those that are most polluted. The location of monitors may also change over time. Hence, in this analysis, we use only those monitors that existed continuously throughout the period, although using all monitors does not change our results.[7]

Following Neidell (2004), we use the monitor data to construct a measure of pollution for each zip code in the state as follows: First, we calculate the centroid of each zip code. We then measure the distance between the EPA monitor and the center of the zip code. Finally, we calculate a weighted average pollution level using all monitors within a 20-mile radius of the zip code's center, using the inverse of the distance to the

[7] The data is the California Ambient Air Quality Data from the California Air Resources Board, a department of the California Environmental Protection Agency (available at http://www.arb.ca.gov/aqd/aqdcd/aqdcd.htm).

monitor as the weight. We use this method to construct a pollution measure for each zip code and time period. Using this method, we are able to assign a pollution level to zip codes covering about 70 percent of the births in the state. Zip codes that we were not able to assign pollution levels to are overwhelmingly rural. While not every urban zip code has a monitor, 76% of the births included in our sample were within 10 miles of a monitor and we obtain very similar results if we limit our analysis to this subsample.

In order to assess the accuracy of our measure, we compare the actual level of pollution at each monitor location with the level of pollution that we would assign using our method (i.e. using the distance weighted average of data from all other monitors less than 20 miles away), if the monitor in question was not there. The correlations between the actual and predicted levels of pollution are remarkably high for O3 (.92). Correlations for PM10 and CO are somewhat lower, but still high (.77 and .78) suggesting that our measure is reasonably accurate. Note that as long as there is no systematic pattern to these errors, measurement error will tend to bias our estimates of the effects of pollution towards zero.

Descriptive statistics for the pollution variables are shown in the first panel of Table 1, which also describes the units.[8] Table 1 shows that there is considerable variation in these measures both between zip code-months and within zip code-months over our sample period. For example, the within zip code-month standard deviation for

[8] These measures 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.

CO is .447 compared to the between zip code standard deviation of 1.018 (which can be compared to the mean of 1.998 units.)

The pollutants we examine display strong seasonal patterns as shown in Figure 1. The vertical lines show the first quarter of each year. In California, ambient levels of CO and PM10 tend to increase in cold weather when they are trapped by damp cold air. PM10 also spikes in cold weather because it is produced by combustion sources used for heating. In general, levels of CO and PM10 are highly correlated which may make it difficult to disentangle their effects. On the other hand, ozone forms at a higher rate in heat and sunlight. Thus ozone emissions spike during the summer. As we show below, the negative correlation of ozone with other pollutants can yield wrong-signed effects in single-pollutant models.

Our models include zip code-month fixed effects in order to control for seasonal effects which could be different in northern and southern California, for example. These effects also remove some of the variation in pollution, but Figure 2 shows that a great deal of residual variation remains. Figure 2 plots residual levels of pollution after the zip code-month dummies, year dummies, weather indicators and all of the other variables included in our base models (described further below) have been controlled for. Residuals are normalized by mean pollution levels so that they are expressed in percentage terms. It is very important to establish that there is significant within zip code-month variation since mean differences in the level of pollutants between zip code-months are not used to identify the effects of pollution in the zip code-month fixed effects models.

Since weather is a key determinant of pollution levels but could also have independent effects on infant health, we include controls for maximum temperatures and average precipitation in our models. The weather data come from the Surface Summary of the Day (TD3200) from the National Climatic Data Center available at http://www4.ncdc.noaa.gov/cgi-win/wwcgi.dll?wwAW~MP#MR. Weather stations are not particularly well matched to pollution monitors. We use county-level average weather data in our models. Although these measures are somewhat crude, they should capture the effects of, for example, unusual heat waves or rainy spells that are not captured by our zip code-month fixed effects. To the extent that weather affects pollution without having an independent effect on infant health, including the weather variables will reduce the amount of variation in our pollution measures and make it more difficult to detect its effects (Samet et al., 1997).

Data on birth weight, infant deaths and fetal deaths come from the California Birth Cohort files for 1989 to 2000. These data are abstracted from birth, death, and fetal death certificates. Birth weight is the single most widely used summary measure of infant health, and low birth weight (defined as birth weight less than 2500 grams) is a marker for higher rates of infant mortality and other negative outcomes. Note that there is no birth cohort file for 1998, so this year is excluded from our analysis.

The distinction between fetal and infant death is that a child must be born alive in order to be registered as an infant death. In California, a live birth is defined as "the complete expulsion or extraction from its mother of a product of conception...which, after such separation, breathes or shows any other evidence of life...", while a fetal death

is a "death prior to complete expulsion or extraction from its mother of a product of conception" (California Code of Regulations, Title 17, sections 915 and 916).

Hence, a premature delivery that ended in a child dying before birth would be classified not as an infant death but as a fetal death. If pollution has an effect on fetal deaths, then examining only the population of live births may yield biased estimates of its true effects. For example, if pollution causes a fetus that would have been born alive, but low birth weight to be stillborn, then it could even appear that pollution increased birth weight.

Since fetal death certificates give birth weight and gestation, we combined live births and fetal deaths in order to create a sample of pregnancies lasting at least 26 weeks.[9] Examination of the effects of pollution on this sample will give us estimates of the effects of pollution that are not biased by fetal selection that occurs after 26 weeks. While pollution might also cause fetal deaths before 26 weeks, fetal deaths before 26 weeks are not accurately reported. We also confine our analysis of infant outcomes to infants with at least 26 weeks gestation so that we can define pollution exposure in the first, second, and third trimesters of the pregnancy.

Since we do not examine the effects of pollution on gestation or on infants with less than 26 weeks gestation our results leave open the possibility that pollution could lead to premature termination of pregnancies and/or high rates of infant death in this population. Hence, our estimates will under-state the total effect of pollution on infant

9 This yields a sample of 4,593,001 live births. There were 127,189 live births with gestation less than 26 weeks.

health if it causes fetal losses before 26 weeks, or an increased probability of death in surviving infants with very short gestations.

Figure 3 shows seasonal variation in infant mortality, fetal death, and birth weight over time. In contrast to Figure 1, there is no strong seasonal pattern in these outcomes. A comparison of Figure 1 and Figure 3 shows that while both pollution and infant mortality have been trending downwards, the rate of low birth weight has remained flat. Hence, the idea that reductions in pollution may have reduced infant mortality without having much impact on birth weight is consistent with the aggregate data.

Descriptive statistics for these outcome variables are also shown in Table 1. Over the sample period 3.91 children per 1,000 with gestation of 26 weeks or more died in their first year. The incidence of fetal death was slightly lower while 48.35 children per 1,000 were low birth weight.

In addition to the infant health measures, Birth Cohort File variables relevant for our analysis include the date of birth, mother's age, race and ethnicity, education, marital status, and the 5-digit zip code of maternal residence, as well as information about use of prenatal care and whether the birth was covered by public health insurance. The rapid increase in the fraction of births covered by Medicaid is a potential confounding factor when examining birth outcomes because there is evidence that Medicaid coverage changed the way that at risk infants were treated (c.f. Currie and Gruber, 1996), so it is fortunate that we can control for Medicaid coverage of the birth directly. Unfortunately, it is not possible to control for maternal smoking because this information is not included on California's birth certificate. To the extent that smoking is correlated with other variables included in our model, bias due to this omission will be reduced.

The third panel of Table 1 shows trends in pollution levels over the sample period. All the pollutants show considerable declines. Like Figure 3, the fourth panel of Table 1 shows that although the infant mortality rate fell sharply over a relatively short time, trends in low birth weight were much flatter. This part of the table suggests then that declines in mortality were largely due to events occurring after the birth, rather than to improvements in prenatal health. Finally, the last panel of Table 1 lists the federal standards for the pollutants we examine. A comparison of the first and last panels of the tables suggests that on an average day, pollution levels in California are well under the thresholds for these standards. However the fact that Los Angeles is consistently out of compliance for both ozone and CO indicates that there is substantial variability in pollution levels around these means.

Table 2 shows mean annual outcomes and pollution levels as well as means of various control variables by zip code pollution level. In order to rank zip code-years by pollution level, we first standardized all of the pollution measures using a "z-score" and then took the average of the three measures. While this is a rough way to rank areas, Table 2 indicates that it is informative--there are sharp differences in ambient pollution levels between the most polluted and the least polluted areas of the state. For example, the CO measure is almost three times higher in the most polluted areas compared to the least polluted ones.

These gradients correspond to gradients in birth outcomes: The most polluted areas have uniformly worse outcomes than the least polluted ones. As the third part of the table shows, this association could be due to the fact that pollution levels are highly correlated with socioeconomic characteristics that are themselves predictive of poorer

birth outcomes. For example, 73 percent of mothers are married in the least polluted areas compared to 63 percent in the most polluted areas; 25 percent are high school dropouts in the cleaner areas compared to 41 percent in the dirtiest; and the comparable figures for use of government insurance are 38 percent and 50 percent. These are very large differences in the average characteristics of mothers and failure to adequately control for them could generate spurious relationships between pollution and birth outcomes. In our models, we will control for these important observable differences between locations as well as for unobservable zip code-level characteristics and seasonal differences by including zip code-month level fixed effects.

IV. Methods

Evidently, air pollution affects 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. However, one might wonder whether effects observed after birth actually reflect the lingering effects of exposures before birth. In order to control for this possibility we estimate models that include the infant's birth weight and gestation. These variables can be regarded as summary statistics for the infant's health at birth and hence will help to capture any effects of pollution before the birth. We also include controls for average pollution levels during the infants first trimester, second trimester, and third trimester of gestation, though as we show below this has no effect on the estimated effect of post-natal pollution exposure.

The probability of death P_{izt} is specified as:

$$P_{izt} = \alpha(t) + w_{iz}\gamma + h_{iz}\varsigma + x_{zt1}\beta_1 + x_{zt2}\beta_2 + \phi_{zt} + Y_t, \quad (1)$$

where *i* indexes the individual, *z* indexes the zip code, *t* indexes the time period and $\alpha(t)$ is a measure of duration dependence and is specified as a linear spline in the weeks since the child's birth, with breaks after 1, 2, 4, 8, 12, 20, and 32 weeks. These break points reflect the fact that death is much more common in the first weeks than thereafter. The w_{iz} are time-invariant covariates measured at the individual level, such as the mother's demographic and background characteristics; the h_{iz} are time-invariant measures of the infant's health and pollution exposure at the time of the birth including indicators for low birth weight and short gestation and for pollution exposure in the 1st, 2nd, and 3rd trimesters; the x_{zt1} are time-varying measures of pollution exposure after the birth, the x_{zt2} are weather indicators; ϕ_{zt} is a vector of zip code-month specific fixed effects; and Y_t is a vector of year dummies that allows for state-wide trends in these outcomes. We consider several variations on (1) including estimation of models that include both zip code-month and small area-year fixed effects which are described further below. The main coefficient of interest is β_1, the effect of post-natal pollution exposure on the probability of death.

This model can be thought of as a flexible, discrete-time, hazard model that allows for time-varying covariates, non-parametric duration dependence, and zip code-month level fixed effects. The model imposes little "structure" on the pattern of coefficients, allowing the data to "speak for itself", a consideration that is particularly important given the lack of guidance in the literature regarding mechanisms and functional form. Allison (1982) shows that estimates from models of this type converge

to those obtained from continuous time models, as discussed further in the appendix. (Note that we have also estimated models using $f(P_{izt})$ as the dependent variable, where f is the logit transformation–the results were very similar).

In order to implement this estimation strategy, we treat an individual 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 is repeated for every period, while the time-varying covariates are updated each period. P_{izt} is then regressed on the covariates specified in (1) by ordinary least squares.

This procedure yields a very large number of observations. Most infants survive all 52 weeks of their first year, yielding a sample of 250 million weekly observations. Hence, we employ case-control sampling to reduce the number of observations. First, we keep all individuals who died (the cases) in the week that they died. Then, in order to select controls, we choose randomly among all the observations on children who lived for at least as many periods as the index child and take the control child's observation for that week. That is, if a child died in week 3, the controls would be chosen from observations on all children who lived at least 3 weeks regardless of whether they later died. For each week, we randomly chose fifteen times as many non-deaths as deaths. This method greatly reduces computational burden while yielding unbiased estimates of the effects of pollution on the probability of death (Mantel (1973), Prentice and Breslow (1978), Lubin and Gail (1984)).[10]

[10] In contrast, suppose we took all children who died, and selected a control group by sampling all children who survived their first year. At any point in time during the year, we would have a sample that excluded infants who were at risk of death, but survived only to die later. We reproduce Mantel's discussion of why retaining individuals on the basis of their outcomes only

As discussed above, we chose a week as the unit of time in our base specification. A potential problem with choosing such a small interval is that children who die from exposure to high amounts of pollution in week t might have died at t+1 in any case. This problem of mortality displacement is sometimes referred to as "harvesting" (Schwartz (2001)). If mortality displacement is an important phenomenon, then estimates based on weekly pollution measures will tend to overstate the loss of life caused by pollution. For example, the actual loss of life might be only one week rather than average life expectancy at birth. Moreover, models estimated using weekly pollution focus on the short-term effects of pollution exposure and the cumulative impacts of post-natal exposure might also be important.

Estimating models using longer time units, such as months, involves more measurement error because the measure of pollution is imprecisely assigned. For example, if we use the month as the time unit, children who die in their first week of life are incorrectly assigned average pollution levels for all of the days in the month. Moreover, if it really was a sharp spike in a pollutant that caused death, these spikes would tend to be averaged out in more aggregate data. An alternative is to estimate models that include cumulative pollution measures, in order to see whether it is truly short-term effects of pollution exposure that matter.

In addition to the specification checks discussed above we show that our results are robust to several other changes to model (1). First, we drop observations from the

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adds a constant to the log odds ratio in the Appendix. Since we begin with the entire universe of births and can choose the sample to analyze, we have followed the case control literature that specifies the correct way to choose an analysis sample rather than the economics literature on "choice-based sampling" which suggests estimation methods to deal with samples that have been chosen non-randomly (c.f. Manski and Lerman, 1977; Imbens, 1992).

first week of life since many infants who die in the first week may never leave the hospital and thus may never be exposed to outdoor air. We also estimate models that include fixed effects for the interaction between minor civil division and year in addition to the zip code-month effects. A minor civil division is a small cluster of two or three contiguous zip codes. Examples in Los Angeles county include Santa Monica and Pasadena. It did not prove practical to include both zip code-month and zip code-year fixed effects in our models for computational reasons. The inclusion of MCD-year effects controls for characteristics of local areas that might change over time, such as access to medical facilities.[11]

Finally, we estimate models that include leads of the weekly pollution measures. Pollution that has not yet occurred should have no impact on mortality once contemporaneous pollution levels are controlled, and we show that leads are indeed statistically insignificant.

Our results show a very robust effect of post-natal CO exposure on infant mortality. In contrast, we find no significant effect of prenatal exposures. In order to investigate the effect of prenatal exposures further, we go on to estimate models of the effects of prenatal exposure on the probability of fetal death and on the probability of low birth weight in a 10 percent random sample of all pregnancies that lasted at least 26 weeks. These models have the form:

$$P_{iz} = w_{iz}\gamma + p_{z1}\eta_1 + p_{z2}\eta_2 + \phi_{zt} + \mathrm{Y_t}, \qquad (2)$$

11 In California, an MCD corresponds to a township, which is a subdivision of a county. In many cases, the MCD may be the same as a Census "Place", which is an incorporated or unincorporated population center. We have also estimated our models using place-year fixed effects and obtained very similar estimates.

where P_{iz} is the relevant probability; the w_{iz} are time-invariant covariates measured at the individual level, such as the mother's demographic and background characteristics; the vector p_{z1} measures prenatal pollution exposure in each trimester; p_{z2} is a vector of weather variables; ϕ_{zt} is a zip code-month specific fixed effect; and Y_t is a vector of year dummies that allows for state-wide trends in these outcomes. In this model the main coefficient of interest is η_1, the effect of prenatal pollution exposure on the probability of a negative outcome.

V. Results

a) Effects on Infant Mortality

Table 3 shows estimates of model (1). For comparison with previous work we first estimate cross sectional models for each pollutant separately. These cross-sectional models include indicator variables for each month and year but do not include any controls for zip codes. The "single pollutant" models without zip code-month fixed effects are shown in columns (1) through (3). They indicate that post-natal exposure to CO increases infant mortality while, as discussed above, O3 has a perverse negative effect. This negative effect disappears however, when all three pollutants are included in a single cross-sectional model, as shown in column (4). Columns (5) through (8) show estimates of similar models including zip code-month effects. It is remarkable that the the addition of these controls has little effect on the estimated effect of CO, and in fact increases it slightly.

Of the measures of prenatal pollution exposure included in model (8), only the

coefficient for 2nd trimester exposure to PM10 is significant and it is wrong signed. However, while it is not quite significant, the coefficient on 3rd trimester exposure to PM10 is of similar magnitude and positive. This pattern of coefficients is what one would expect if there were collinearity in the measures of prenatal pollution. Therefore, in order to guage the overall effect of prenatal pollution we test to see whether we can reject the null hypothesis that the sum of the coefficients on the prenatal pollution measures are equal to zero. The F-tests shown at the bottom of Table 3 indicate that we cannot, suggesting that prenatal pollution exposure has little effect. We have investigated the sensitivity of these results by including controls only for pollution in the first trimester, the last three months of pregnancy, or the last month of pregnancy, with similar insignificant results.

Because of the case-control sampling, the coefficients in Table 3 are difficult to interpret. The bottom of Table 3 presents calculations of the magnitude of the effects in terms of the implied number of deaths per 100,000 births (of gestation 26 weeks or more) associated with a one unit increase in the pollutant in question. Given the 4,593,001 such births in areas where pollution could be assigned over our sample period, the estimate of 18.125 in the last column suggests that the 1.1 unit decline in CO that took place saved 991 infant lives. (Note that we do not consider possible lives saved in areas without pollution monitors. If these areas did not have monitors because they had little pollution and/or were sparely populated, then reductions in pollution could be expected to have relatively little effect).

Table 3 also shows the estimated effects of the other covariates included in our models. Birth weight and gestation are significant predictors of mortality, consistent

with other research. Maximum temperatures are estimated to reduce mortality in the cross-sectional models, but have little effect in the models that include zip-month fixed effects, suggesting that the inclusion of these variables does help to control for the effects of weather. The coefficients on $\alpha(t)$ indicate that most infants who die, do so in the first two weeks, which is consistent with past research. Males, Hispanics, children of foreign-born mothers, and children whose mothers commenced prenatal care in the first trimester are all less likely to die, while children of high school dropouts, teen mothers, people on government insurance, and babies of high parity are more likely to die, consistent with our expectations.

Table 4 shows that the effect of post-natal CO exposure is robust to many changes in specification. The first column shows that dropping infants who died in the first week (who may not have been exposed to much outdoor air) does not change the qualitative result. (Although the coefficient rises, the implied number of deaths remains similar at 1,148 because the mortality rate among infants falls after the first week).

Column (2) shows that the key coefficient is not affected by dropping the prenatal pollution measures entirely. We have also tried dropping birth weight and gestation with similar results. Column (3) shows estimates without the weather variables, which again are similar to those reported in Table 3.

Columns (4) and (5) show the effect of restricting our sample to people within 10 miles of a zip code monitor. In column (4), the pollution measure is calculated using data from zip codes that lie within 20 miles of the zip code centroid, while in column (5), only monitors within 10 miles are used to create the pollution measure. Again, these changes make little difference to the estimated coefficients.

Column (6) shows estimates using the month rather than the week as the unit of observation. This is the only specification in which the effect of CO is not statistically signficant. Nevertheless, the coefficient estimate is remarkably similar to that shown in Table 3 and the increase in the standard error is consistent with the idea that there is more measurement error in pollution measured at the monthly level. In monthly models with zip code rather than zip code-month fixed effects the coefficient on CO is quite similar to those reported here and statistically significant.

Column (7) shows estimates including MCD-year effects as well as zip-month effects. Including these effects causes the estimated coefficient on CO to rise slightly. The coefficient of 3.623 implies that the reduction in CO over the 90s saved 1,243 infant lives. This specification check suggests that the results in Table 3 yields conservative estimates of the number of lives saved.

Table 5 offers two additional specification checks. Columns (1) and (2) show models that include leads of pollution. An infant who dies in week t should not be affected by pollution in week t+1 and in fact, Table 5 shows that leads are not statistically significant and that their inclusion has little impact on the estimated effect of CO. This finding is remarkable given the strong seasonal correlations in CO and suggests that we really are capturing the effect of acute exposure in a given week.

Column (3) takes a somewhat different approach by including a measure of average cumulative exposure in addition to the weekly pollution measure. Only the weekly pollution measure is significant, and again, the coefficient on CO is robust. It is worth noting however that for PM10 the cumulative effect of PM10 is greater than the weekly effect, although it is not statistically significant in the multi-pollutant models. In

single-pollutant models that only include PM10 (not shown), the coefficient on the cumulative measure reaches significance at the 90 percent level of confidence. We also found that in monthly hazard models with separate zip code and month fixed effects rather than zip code-month fixed effects, PM10 had a significant effect. All this suggests that it is possible that PM10 has a cumulative post-natal effect that is difficult to capture in models that rely on weekly variation in PM10 within zip code-months.

b) Effects on Birth Weight and Fetal Death

The results so far show that post-natal exposure to CO has a remarkably robust effect on infant mortality. In contrast, the F-statistics indicate that the effects of prenatal pollution exposure sum to zero, suggesting that the estimated effect of post-natal exposure is not driven by prenatal pollution exposure.

However, prenatal pollution exposures might still have harmful effects on birth weight and fetal death. In order to investigate these effects, we present estimates of model (2) for birth weight in Table 6. In Table 6 the only coefficients that are individually statistically significant are those on 2nd trimester CO exposure, and they are wrong-signed. However, the F-tests shown at the bottom of the page indicate that we cannot reject the null hypothesis that the coefficients on prenatal CO exposure (and other prenatal pollution exposures) sum to zero.

Table 7 presents similar estimates for fetal deaths. We find no evidence of any effect of prenatal pollution exposure, though as discussed above, we cannot reliably examine the effects on fetal deaths before 26 weeks.

c) Estimated Effects in More Aggregate Data

Several previous studies have used aggregate rather than individual-level data and it is of interest to see what happens if we move to more aggregate data. Most previous studies use data aggregated to the county-year level. We aggregate up to the zip code-quarter level and estimate models similar to (1) and (2) which include zip code fixed effects. Previous estimates using more aggregated data have not attempted to sort out the effects of post-natal and pre-natal exposures, presumably because of the difficulty of precisely assigning such exposures in aggregate data. We do not do so here either. The sample size for the infant mortality regressions is slightly smaller than for the birth outcome regressions, because for 1989, the rate can only be calculated for the last quarter of the year.

The first panel of Table 8 shows that in the aggregate-level data, only PM10 has a statistically significant effect in the multi-pollutant models--there is no statistically significant effect of CO on infant mortality. This observation suggests that estimates based on aggregate data will significantly under-estimate the effects of CO perhaps because it is acute exposure to CO that matters. The point estimate of .0034 on PM10 in column (4) indicates that there was a decline of .34 deaths per 100,000 per unit of PM10 reduction per quarter, which is smaller than the Chay and Greenstone estimate of the effects of TSPs. The smaller estimate may reflect a non-linear effect of particulates on infant health, the fact that TSPs are a broader measure than PM10, or a California-specific effect given that Chay and Greenstone use national data.

Panel 2 indicates that the coefficient on CO is significant in the multi-pollutant model for low birth weight and very large. However, the fact that CO is not significant

in the single-pollutant model suggests that the finding in the multi-pollutant model may be due to collinearity between the pollution measures in this more aggregate data. Panel 3 shows that, once again, we find little effect of pollution on the probability of fetal death.

VI. Discussion and Conclusions

Environmental policy continues to be contentious. For example, the EPA has responded to the threat posed by increased diesel emissions by proposing new rules that would require refiners to phase in cleaner diesel fuel between 2006 and 2010, but the American Petroleum Institute and the National Petro-Chemical and Refiners Association have filed suit in an effort to block implementation of these standards (Stafford, 2001).[12] Similarly, there is controversy over the Bush administration's recent "Clear Skies" initiative, which would eliminate the requirement that older power plants upgrade their pollution controls when they upgrade or modernize their equipment and replace them with "cap and trade" provisions. Critics contend that the plan would not regulate CO production, provides weaker caps than alternative legislation introduced in the Senate, and will not necessarily reduce pollution in the most polluted areas, an important consideration if the effects of pollution are non-linear (Environmental Defense, 2003).

In order to begin to evaluate the costs and benefits of such policies, it is necessary to understand how changes from current, historically low levels of air pollution are likely

[12] Due to increased driving, trucks burning diesel emitted more nitrogen oxides and particles in 1997, than they did in 1970 when the Clean Air Act was passed

to affect health and which pollutants have the greatest health effects. This paper examines the effects of air pollution on infant health, using recent data from California. Our models are identified using within zip code-month variation in pollution so that we are able to control for unobservable fixed characteristics of zip codes, seasonal effects, and a detailed group of observable time-varying characteristics.

Our most interesting and novel finding is that high levels of post-natal exposure to CO have a significant effect on infant mortality. We believe that this effect has been overlooked because it is acute and hard to detect in data that has been aggregated even to the monthly level. This finding is remarkably robust to many changes in specification and suggests that decreases in CO levels over the 1990s saved about 1,000 infant lives in California. These findings are clearly relevant to policy debates over automobile emissions and the Clear Skies Initiative, for example.

We test carefully to see if the estimated effect of post-natal CO exposure on infant mortality actually reflects the lingering impact of prenatal exposures but we find little evidence in favor of this hypothesis. Prenatal pollution exposures are not jointly significant and prenatal pollution exposure has no impact on birth weight or on fetal death in models estimated using individual-level data for gestations that lasted at least 26 weeks.

PM10, which has been the focus on most research to date on the effects of pollution on health, is seldom statistically significant in our multi-pollutant models. One interpretation of this result is that previous studies have wrongly attributed the effects of CO to PM10. But there is some suggestive evidence that it is the cumulative effects of PM10 that matter and our hazard models may be better suited to detecting acute rather

than cumulative effects. Given that CO and PM10 are highly correlated and come from many of the same sources, policies aimed at reducing one are likely to have significant effects on ambient levels of both.

A complete evaluation of the costs and benefits of improvements in air quality is far beyond the scope of this paper.[13] It is likely that the costs of reducing pollution may be greater at low levels of pollution than at higher levels. But there are also several reasons why conventional measures of the benefits of pollution abatement (such as the effects of pollution levels on housing prices) might understate them. First, the effects of pollution on infant health are not well known—that is a starting point for this research. Second, CO is a colorless, odorless gas and people may not be willing to pay for reductions in pollution that they do not observe. Third, to the extent that parents place a lower value on infant health relative to other goods than infants would, the value of their health will not be fully captured by the parents' willingness to pay for pollution reduction.

What is the value then, of improvements in infant health due to reductions in pollution? If, following Chay and Greenstone (2001a), we value a life at a very conservative $1.6 million, then the estimated reduction in infant deaths due to reduced air pollution in California over the 1990s would be valued at about $1.6 billion.[14] If we use the EPA(1999) value of $4.8 million, the benefit would grow to $4.8 billion. These

13 See Greenstone (2002) who calculates the cost of the 1970 and 1977 Clean Air Act Amendments or Sieg et al. (2000) who examine willingness to pay for air quality improvements in the context of a general equilibrium model of housing prices.

14 Viscusi (1993) suggests that the value of a life was between $3.5 and $8.5 million, and U.S. EPA (1999) values infant lives lost due to lead at $4.8 million, the same value that they used for adult lives.

estimates are based on a conservative estimation strategy which nets out pollution changes that are correlated with seasons or weather. These estimates also ignore the value of improvements in health in infants who are not at the life-death margin. Hence, we regard these estimates as lower bounds on the benefits to infants. But they may still provide a useful benchmark for assessing the benefits of further reductions in air pollution in terms of infant health.

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Appendix:

1. Description of the survival model

The description of this model follows Allison (1982). Define a discrete-time hazard rate:

$$P_{it} = \Pr[T_i = t \mid T_i > t, x_{it}]$$

where P_{it} is the probability of death for individual i in period t, T is the time of occurrence, and x are covariates that affect death.

We can now specify the likelihood function:

$$L = \prod_{i=1}^{n} [\Pr(T_i = t)]^{\partial_i} [\Pr(T_i > t)]^{1-\partial_i}$$

where δ_i is a dummy variable equal to 1 if the observation is uncensored and 0 otherwise. This is analogous to the continuous time model in that each individual contributes to the likelihood function the hazard rate if uncensored and the survivor function if censored.

Using conditional probabilities, we can restate the hazard and survivor function as:

$$\Pr(T_i = t) = P_{it} \prod_{j=1}^{t-1} (1 - P_{ij})$$

$$\Pr(T_i > t) = \prod_{j=1}^{t} (1 - P_{ij})$$

After substituting these into the likelihood function, taking logs, and rearranging terms, we are left with:

$$\log L = \sum_{i=1}^{n} \sum_{j=1}^{ti} y_{it} \log\left(\frac{P_{it}}{1 - P_{it}}\right) + \sum_{i=1}^{n} \sum_{j=1}^{ti} \log\left(1 - P_{it}\right)$$

where y_{it} = 1 if person *i* dies in period *t*, and 0 otherwise. This now amounts to the analysis of binary data, and, after specifying the hazard as a function of the covariates, can be estimated by logit model. Alternatively, we can specify the hazard as a linear probability model and estimate it by least squares.

2. A Note on Case Control Sampling

Mantel (1973, pages 481-482) provides a simple explanation of case-control sampling. In his analysis, a random proportion d_1 of cases, and a random proportion d_2 of controls are chosen. It is key that people be chosen from both groups randomly. Intuitively, there is little to be gained by arbitrarily increasing the size of the control group, if the size of the treatment group is fixed. However, it still seems that selecting the individuals to be retained on the basis of their outcome will introduce a bias. Mantel shows however, that only the intercept of the log odds ratio is changed. Specifically,

"The possible outcomes for individual I with vector X_i are:

1) he can develop disease and be in the sample, with probability $d_1P(Y_i=1|X_i)$;

2) he can develop disease and not be in the sample, with probability $(1-d_1)P(Y_i=1|X_i)$;

3) he can remain disease free and be in the sample, with probability $d_2P(Y_i=0|X_i)$;

4) he can remain disease free and not be in the sample, with probability $(1-d_2)P(Y_i=0|X_i)$.

We now make use of the fact that for any truncated multinomial...the probability P', for a particular observable outcome is its unconditional probability divided by the total of probabilities for observable outcomes. Thus we may write

$$P'(Y_i=1|X_i) = d_1P(Y_i=1|X_i)/[d_1P(Y_i=1|X_i) + d_2P(Y_i=0|X_i)] \quad (1)$$

in consequence of which

$$P'(Y_i=1|X_i)/P'(Y_i=0|X_i) = d_1P(Y_i=1|X_i)/d_2P(Y_i=0|X_i) \quad (2)$$

or the log odds

$$\log\{P'(Y_i=1|X_i)/P'(Y_i=0|X_i) = \log(d_1/d_2) + \log\{P(Y_i=1|X_i)/P(Y_i=0|X_i). \quad (3)$$

What this implies is that the conditional log odds for being a case has the same dependence on X_i as the unconditional log odds; only the intercept is changed."

Figure 1. Seasonal Variation in Pollution

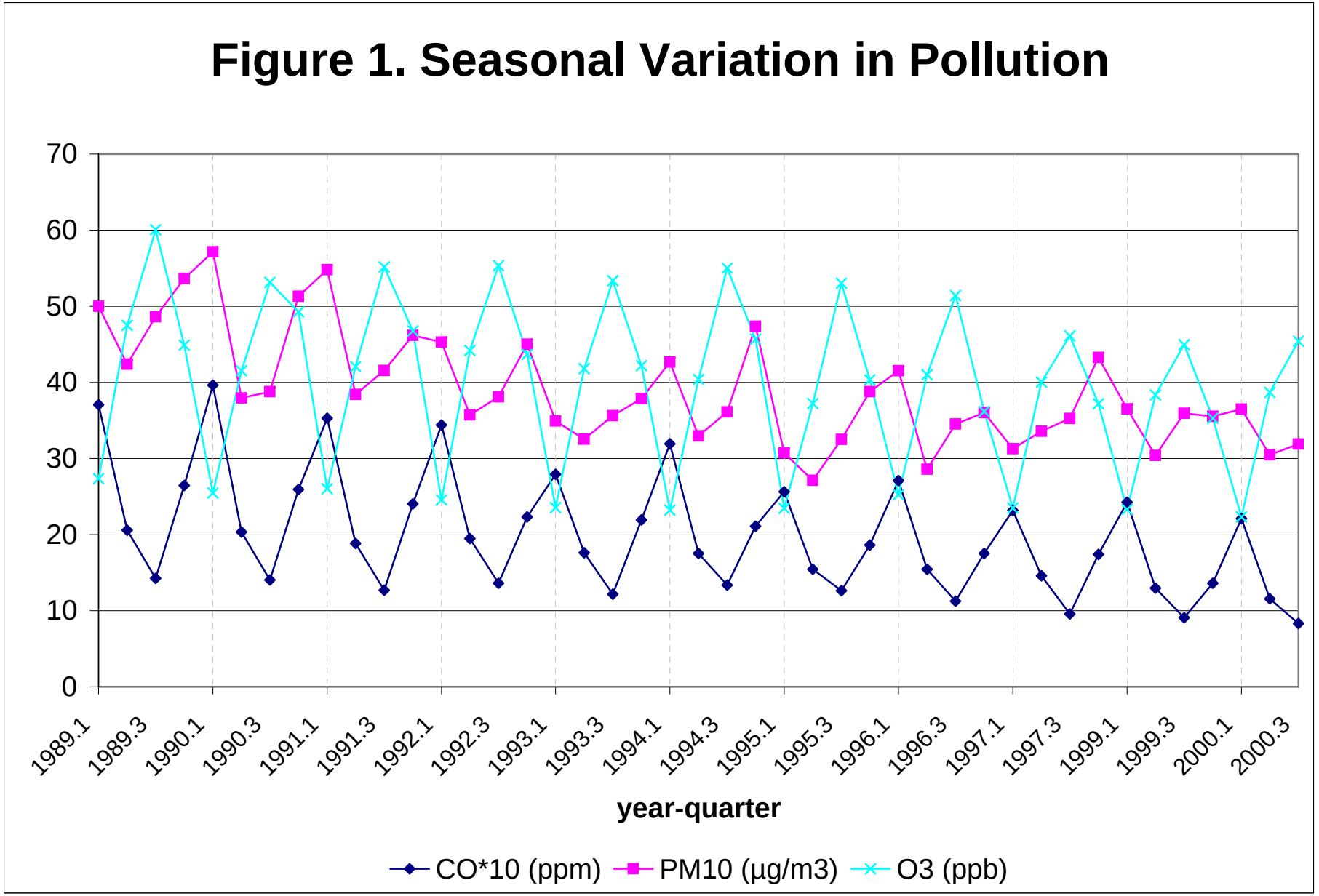

Figure 2. Percent Residual Variation in Pollution

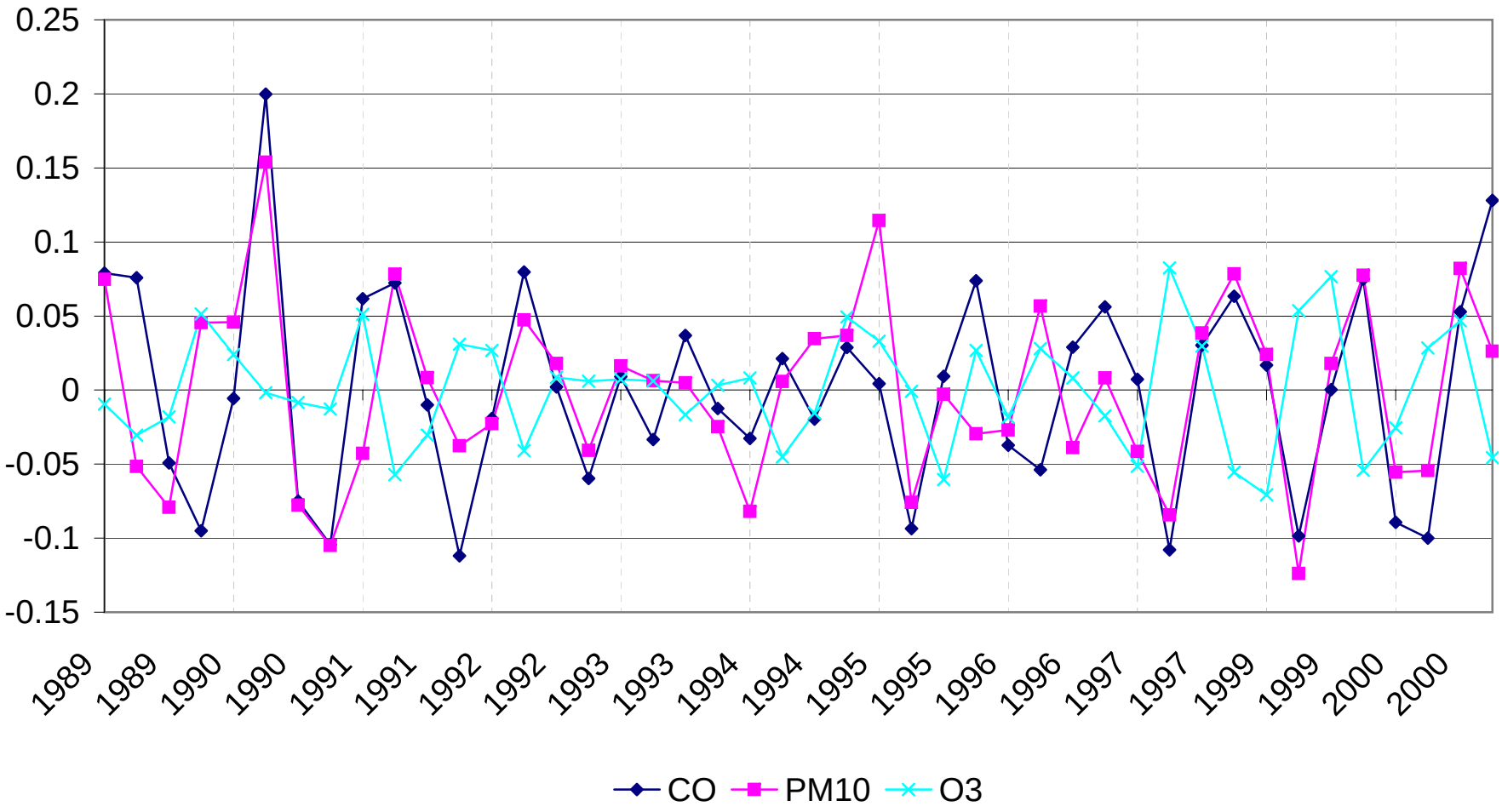

Figure 3. Seasonal Variation in Infant Health Outcomes

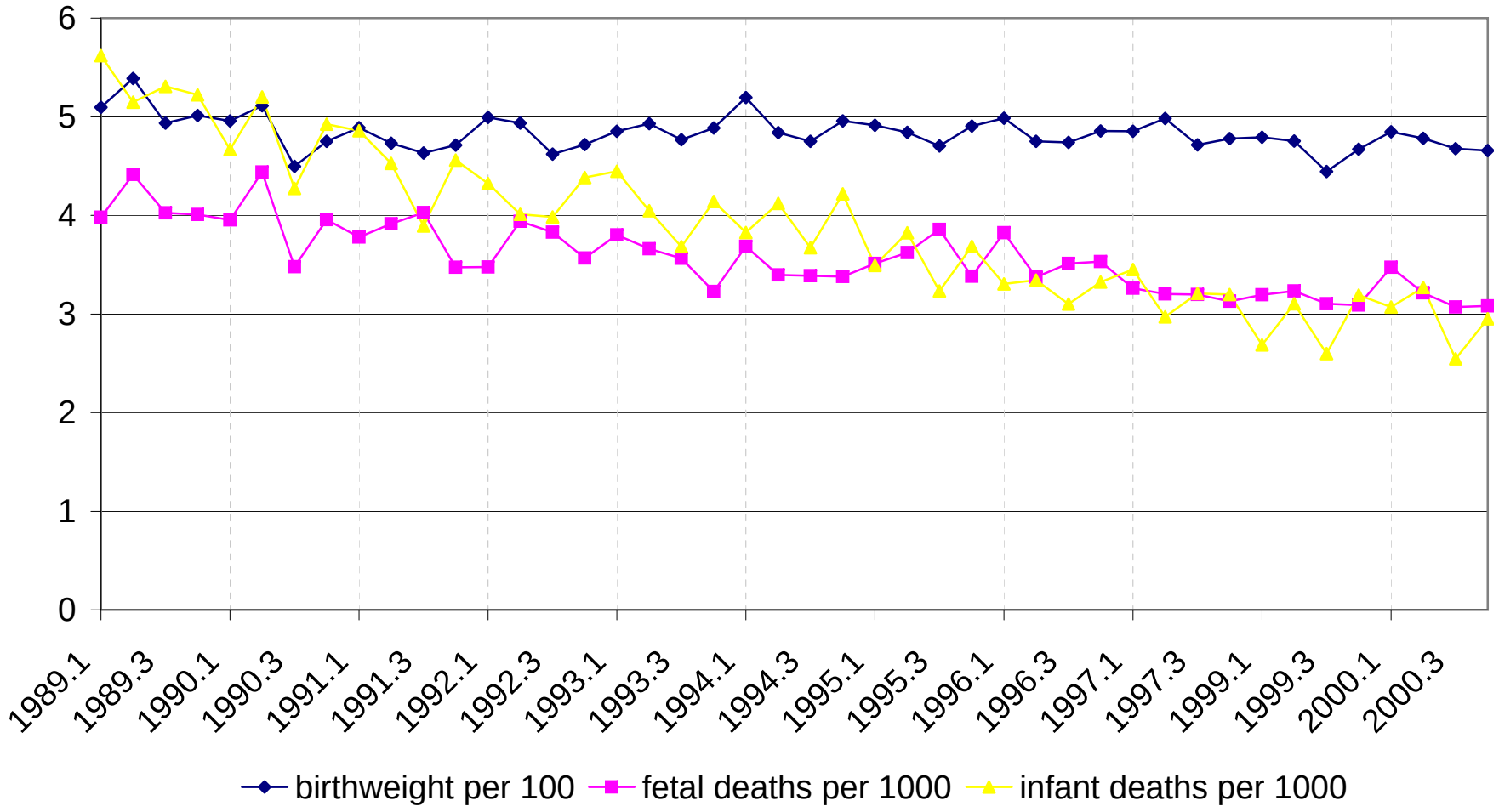

Chart 1: Selected Epidemiological Studies of Effects of Pollution on Infant Health

A: Studies Outside the U.S. and Canada

Study	Location	Years	Outcomes	Pollutants	Effects
Bobak (2000)	Czech Republic	1991	low birth weight preterm birth, growth retard.	SO2, TSP, NOX	Mean exposure during pregnancy related to increased risk of LBW and preterm birth. Effects greatest for exposure in first trimester.
Bobak and Leon (1999)	Czech Republic	1989-91	infant mortality	SO2, TSP, NOX	Mean lifetime exposure to TSPs increased mortality due to respiratory causes when all pollutants entered in model.
Dejmek et al. (1999)	Northern Bohemia, Europe	1994-96	Growth retardation	PM10, PM2.5	Exposure in 1st month of pregnancy related to interuterine growth retardation.
Loomis et al. (1999)	Mexico City	1993-95	infant mortality	PM10	PM10 associated with higher risk of mortality within 3 to 5 days.
Luiz et al (1998)	Sao Paulo, Brazil	1991,92, 95	fetal death	index of CO, NO2 SO2, O3, PM10	Index associated with increased risk of fetal death within 5 days.
Wang, Ding, Ryan, and Xu (1997)	Beijing, China	1988-91	low birth weight	SO2 & TSP	Exposure in last trimester increases risk of low birth weight.
Xu, Ding, and Wang (1995)	Beijing, China	1988	preterm birth	SO2 & TSP	7-day lagged moving average of each pollutant associated with increased risk of preterm birth.

B: Studies of the U.S. and Canada

Alderman et al. (1987)	Colorado	1975-83	low birth weight	CO	No association CO in last trimester in LBW once maternal education and race were controlled.
Lipfert, Zhang, and Wyzga (2	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 birth weight preterm birth, growth retard.	CO, NO2, SO2, O3	SO2 in 1st month increases LBW. SO2 and CO in last month increases preterm birth. Growth retard-ation associated with CO, NO2, SO2 in 1st month.
Mainsonet et al. (2001)	Northeastern U.S.	1994-96	low birth weight	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 birth weight	CO, NO2, O3, & PM10	CO exposure in last trimester increased incidence of low birth weight.
Williams, Spence, & Tideman (1977)	Los Angeles	early 1970s	low birth weight	TSP	Lower mean birth weight in areas with high pollution among women who were non-smokers.
Woodruff et al. (1997)	86 U.S. MSAs	1989-91	infant mortality	PM10	Infants with high exposure more likely to die in postneonatal period.

Table 1: Levels and Trends in Pollution and Infant Health

Variable	Mean	Std. Dev.	Between Zip-Month Std. Dev.	Within Zip Month Std. Dev.
Panel 1				
CO 8-hr ppm	1.998	1.169	1.018	0.447
PM10 24-hr ug/m3	39.448	14.755	12.899	7.869
O3 8-hr ppb	40.456	17.107	15.832	5.509
Panel 2				
IMR per 1000	3.91	6.24	1.35	6.23
low birth weight per 1,000	48.35	21.45	3.45	21.40
fetal deaths per 1,000	3.58	5.97	0.84	5.96
Panel 3				
year	**CO**	**PM10**	**O3**	
1989	2.458	49.651	46.139	
1990	2.472	46.575	41.664	
1991	2.288	46.377	43.516	
1992	2.279	41.285	42.830	
1993	1.974	37.040	41.089	
1994	2.111	37.384	40.351	
1995	1.857	34.256	40.037	
1996	1.798	35.790	39.681	
1997	1.608	34.052	36.630	
1999	1.580	36.510	36.109	
2000	1.376	33.572	35.657	
Panel 4		**Low Birth**	**Fetal**	**Sample #**
year	**IMR**	**Weight**	**Deaths**	**of births**
1989	5.33	51.02	4.10	388,097
1990	4.76	48.23	3.95	444,021
1991	4.46	47.41	3.79	454,902
1992	4.18	48.15	3.70	445,760
1993	4.08	48.59	3.55	449,374
1994	3.96	49.33	3.46	441,080
1995	3.56	48.42	3.59	419,948
1996	3.27	48.32	3.56	407,923
1997	3.21	48.31	3.20	386,137
1999	2.90	46.64	3.15	372,232
2000	2.96	47.39	3.21	383,527

National Ambient Air Quality Standards

O3	85 ppb	8-hr
	125 ppb	1-hr
CO	9.5 ppm	8-hr
	35.5 ppm	1-hr
PM10	155 ug/m3	24-hr

Table 2: Pollution Levels for Bottom, Middle, and Top Third of Zipcode-Years Ranked by Mean Pollution Levels

Variable	**bottom 1/3**	**middle 1/3**	**top 1/3**
CO 8-hr	1.176	1.907	2.912
PM10 24-hr	25.647	38.558	54.139
O3 8-hr	34.837	39.828	46.705
IMR	3.583	3.728	4.406
low BW per 1,000	47.094	48.448	49.506
fetal death per 1,000	3.370	3.528	3.840
% male	0.487	0.489	0.488
% black	0.083	0.081	0.083
% hispanic	0.317	0.513	0.550
% asian	0.161	0.105	0.089
% other race	0.012	0.006	0.005
% married	0.725	0.653	0.629
% foreign mom	0.394	0.503	0.524
% racial diff b/w parents	0.189	0.156	0.139
% HS dropout	0.254	0.370	0.408
% HS grads	0.359	0.347	0.348
% AD degree	0.148	0.121	0.114
% college grads	0.239	0.161	0.130
% teen mothers	0.064	0.077	0.081
% age 19 to 25	0.304	0.346	0.366
% age 26 to 30	0.281	0.279	0.281
% age 31 to 35	0.233	0.202	0.187
% age >= 36	0.117	0.097	0.084
% first born	0.419	0.392	0.391
% second born	0.318	0.306	0.299
% third born	0.154	0.168	0.170
% gov't insurance	0.384	0.490	0.495
% prenatal care in 1st trimester	0.816	0.788	0.742

Table 3: Effects of Pollution on Infant Mortality

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	CS	CS	CS	CS	FE	FE	FE	FE
CO	2.458			2.466	2.631			2.89
	[0.488]**			[0.651]**	[0.977]**			[1.040]**
PM10		0.053		-0.026		0.002		-0.036
		[0.031]		[0.037]		[0.039]		[0.042]
O3			-0.141	-0.038			-0.077	-0.046
			[0.043]**	[0.050]			[0.065]	[0.067]
CO 1st trim.	0.513			-0.29	1.708			1.095
	[0.763]			[0.913]	[1.444]			[1.539]
CO 2nd trim.	-2.493			-1.137	-2.065			-0.565
	[0.889]**			[1.029]	[1.022]*			[1.157]
CO 3rd trim.	0.015			-0.969	1.418			0.323
	[0.807]			[0.938]	[1.553]			[1.607]
PM10 1st trim.		0.03		0.06		-0.009		-0.002
		[0.055]		[0.067]		[0.078]		[0.088]
PM10 2nd trim.		-0.253		-0.205		-0.256		-0.245
		[0.060]**		[0.071]**		[0.067]**		[0.081]**
PM10 3rd trim.		0.228		0.214		0.198		0.172
		[0.057]**		[0.072]**		[0.081]*		[0.088]
O3 1st trim.			-0.067	-0.07			-0.062	-0.009
			[0.060]	[0.075]			[0.091]	[0.101]
O3 2nd trim.			-0.031	0.002			0.012	0.07
			[0.070]	[0.080]			[0.073]	[0.085]
O3 3rd trim.			0.187	0.056			0.171	0.085
			[0.054]**	[0.064]			[0.085]*	[0.090]
Birthweight	-0.006	-0.006	-0.006	-0.006	-0.006	-0.006	-0.006	-0.006
	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**
Gestation	-0.081	-0.081	-0.081	-0.081	-0.081	-0.081	-0.081	-0.081
	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
Maximum temp.	-0.312	-0.442	-0.262	-0.269	-0.276	-0.169	-0.114	-0.225
after birth	[0.057]**	[0.052]**	[0.073]**	[0.073]**	[0.122]*	[0.118]	[0.127]	[0.134]
Precipitation	0.026	0.005	0.003	0.021	0.013	-0.016	-0.012	0.012
after birth	[0.042]	[0.042]	[0.041]	[0.042]	[0.049]	[0.048]	[0.048]	[0.049]
Max Temp. 1st trim.	0.218	0.338	0.355	0.404	0.131	0.266	0.294	0.359
	[0.123]	[0.130]**	[0.144]*	[0.149]**	[0.165]	[0.168]	[0.186]	[0.189]
Max Temp. 2nd trim.	0.236	0.197	0.236	0.165	0.155	0.113	0.142	0.069
	[0.127]	[0.133]	[0.147]	[0.154]	[0.157]	[0.162]	[0.174]	[0.182]
Max Temp. 3rd trim.	-0.348	-0.313	-0.352	-0.336	-1.357	-1.340	-1.305	-1.361
	[0.142]*	[0.145]*	[0.146]*	[0.148]*	[1.481]	[1.481]	[1.481]	[1.480]
Precip. 1st trim.	0.003	0.081	0.041	0.076	0.047	0.086	0.065	0.092
	[0.118]	[0.120]	[0.118]	[0.122]	[0.129]	[0.128]	[0.129]	[0.130]
Precip. 2nd trim.	-0.107	-0.173	-0.072	-0.184	-0.105	-0.166	-0.062	-0.201
	[0.124]	[0.123]	[0.121]	[0.127]	[0.140]	[0.142]	[0.142]	[0.144]
Precip. 3rd trim.	0.099	0.109	0.092	0.111	-0.202	-0.202	-0.175	-0.211
	[0.055]	[0.056]*	[0.055]	[0.063]	[1.003]	[1.004]	[1.005]	[1.004]
1-2 weeks old	-0.353	-0.353	-0.353	-0.353	-0.353	-0.353	-0.353	-0.353
	[0.007]**	[0.007]**	[0.007]**	[0.007]**	[0.006]**	[0.006]**	[0.006]**	[0.006]**
3-4 weeks old	-0.025	-0.025	-0.025	-0.025	-0.025	-0.025	-0.025	-0.025
	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**
5-8 weeks old	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001
	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]
9-12 weeks old	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004

	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
13-20 weeks old	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004	-0.004
	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**
21-32 weeks old	-0.002	-0.002	-0.002	-0.002	-0.002	-0.002	-0.002	-0.002
	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**
> 32 weeks old	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**
Male	-0.018	-0.018	-0.018	-0.018	-0.017	-0.017	-0.017	-0.017
	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
Black	0.003	0.004	0.003	0.003	-0.001	-0.001	0.000	-0.001
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
Hispanic	-0.011	-0.011	-0.011	-0.011	-0.012	-0.012	-0.012	-0.012
	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
Asian	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
Other race	-0.001	-0.001	-0.001	-0.001	-0.002	-0.002	-0.002	-0.002
	[0.006]	[0.006]	[0.006]	[0.006]	[0.006]	[0.006]	[0.006]	[0.006]
Married mother	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
Foreign born mother	-0.013	-0.013	-0.013	-0.013	-0.013	-0.013	-0.013	-0.013
	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
Parents diff. race	0.008	0.008	0.008	0.008	0.008	0.008	0.008	0.008
	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
HS grad mother	-0.008	-0.008	-0.008	-0.008	-0.008	-0.008	-0.008	-0.008
	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
AD degree mother	-0.010	-0.010	-0.010	-0.010	-0.009	-0.009	-0.009	-0.009
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
College grad.	-0.016	-0.016	-0.016	-0.016	-0.014	-0.014	-0.014	-0.014
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
Parents educ. Differs	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001
	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]
19-25 mother	-0.008	-0.008	-0.008	-0.008	-0.007	-0.007	-0.007	-0.007
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
26-30 mother	-0.012	-0.012	-0.012	-0.012	-0.011	-0.011	-0.011	-0.011
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
31-35 mother	-0.015	-0.015	-0.015	-0.015	-0.014	-0.014	-0.014	-0.014
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**
Mother >=36	-0.012	-0.012	-0.012	-0.012	-0.010	-0.010	-0.010	-0.010
	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**	[0.003]**
1st born	-0.031	-0.031	-0.031	-0.031	-0.030	-0.030	-0.030	-0.030
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
2nd born	-0.018	-0.018	-0.018	-0.018	-0.016	-0.016	-0.016	-0.016
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
3rd born	-0.011	-0.011	-0.011	-0.011	-0.010	-0.010	-0.010	-0.010
	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**	[0.002]**
Gov't insurance	0.008	0.008	0.008	0.008	0.007	0.007	0.007	0.007
for birth	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
Prenatal care 1st	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007
trimester	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
Observations	206352	206352	206352	206352	206352	206352	206352	206352
R-squared	0.27	0.27	0.27	0.27	0.29	0.29	0.29	0.29

Notes: Robust standard errors in parentheses. A ** indicates significance at the 99% level of confidence.
A * indicates significance at the 95% level of confidence.

Table 3: Continued.

F tests of the hypothesis that prenatal pollution coefficients sum to zero								
CO	4.77			4.31	0.12			0.07
	[0.029]			[0.038]	[0.732]			[0.787]
PM10		0.01		0.80		0.20		0.22
		[0.929]		[0.372]		[0.657]		[0.638]
O3			1.36	0.02			0.56	0.79
			[0.244]	[0.897]			[0.453]	[0.374]

Notes: The first value is the F-statistic, the value in brackets is the p-value.

Magnitude of Effects on Infant Mortality								
CO	2.458			2.466	2.631			2.89
	15.416			**15.466**	**16.501**			**18.125**
PM10		0.053		-0.026		0.002		-0.036
		0.332		**-0.163**		**0.013**		**-0.226**
O3			-0.141	-0.038			-0.077	-0.046
			-0.884	**-0.238**			**-0.483**	**-0.288**

Note: The first value is the coefficient estimate, the second (in bold) is the number of lives saved per unit reduction pollutant, which is the coefficient*100*(the overall IMR/sample IMR)) where overall IMR = 17,939/4,576,562.

Table 4: Alternative Specifications of the Infant Mortality Models

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Drop 1st week	w/o prenatal pollution	w/o weather	Monitor within 10 m	Monitor within 10 m	Monthly hazard	MCD-year FE
CO	4.648	2.986	2.169	2.879	3.491	2.854	3.623
	[1.504]**	[1.017]**	[0.952]*	[1.045]**	[0.920]**	[1.719]	[1.070]**
PM10	-0.038	-0.035	-0.038	-0.044	-0.061	0.128	-0.075
	[0.058]	[0.042]	[0.042]	[0.042]	[0.039]	[0.089]	[0.044]
O3	-0.141	-0.041	-0.099	-0.041	0.001	-0.128	-0.054
	[0.095]	[0.065]	[0.061]	[0.069]	[0.065]	[0.118]	[0.072]
CO 1st trim	1.981		1.07	1.228	1.382	0.7	4.281
	[2.130]		[1.539]	[1.553]	[1.302]	[1.638]	[2.034]*
CO 2nd trim	-0.577		-0.514	-0.475	-0.778	-1.958	0.049
	[1.560]		[1.134]	[1.173]	[0.934]	[1.290]	[1.272]
CO 3rd trim	1.67		-0.057	0.635	0.747	-0.605	3.252
	[2.254]		[1.613]	[1.627]	[1.371]	[1.618]	[2.142]
PM10 1st trim	-0.069		-0.043	0.004	-0.037	-0.051	-0.158
	[0.123]		[0.085]	[0.089]	[0.079]	[0.094]	[0.105]
PM10 2nd trim	-0.293		-0.203	-0.256	-0.222	-0.07	-0.354
	[0.112]**		[0.080]*	[0.083]**	[0.075]**	[0.097]	[0.096]**
PM10 3rd trim	0.175		0.175	0.16	0.142	0.112	0.032
	[0.119]		[0.087]*	[0.090]	[0.082]	[0.090]	[0.107]
O3 1st trim	0.053		0.075	-0.006	-0.004	-0.018	0.034
	[0.143]		[0.094]	[0.102]	[0.086]	[0.116]	[0.128]
O3 2nd trim	0.176		0.065	0.073	0.065	-0.016	0.109
	[0.119]		[0.077]	[0.087]	[0.077]	[0.090]	[0.096]
O3 3rd trim	0.096		0.063	0.094	0.069	0.08	0.154
	[0.122]		[0.087]	[0.091]	[0.079]	[0.114]	[0.118]
Birthweight	-0.006	-0.006	-0.006	-0.006	-0.006	-0.006	-0.006
	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.000]**	[0.0003]**
Gestation	-0.08	-0.081	-0.081	-0.082	-0.081	-0.089	-0.081
	[0.002]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**	[0.001]**
Observations	131488	206352	206352	201990	201990	205214	206352
R-squared	0.16	0.29	0.29	0.29	0.29	0.23	0.296

Notes: Robust standard errors in parentheses. A ** indicates significance at the 99% level of confidence. A * indicates significance at the 95% level of confidence. The difference between columns (4) and (5) is that in column (4) pollution is still measured using monitors up to 20 miles away, while in column (5) only monitors within 10 miles are used to assign pollution.

Table 5: Additional Specification Checks for Infant Mortality

	(1)	(2)	(3)
	1 lead	2 leads	Cumulative
CO	2.907	2.969	2.607
	[1.192]*	[1.235]*	[1.021]*
CO lead or	-0.171	-0.247	1.048
cumulative	[1.142]	[1.247]	[2.146]
CO 2nd lead		0.013	
		[1.157]	
PM10	-0.049	-0.052	-0.063
	[0.043]	[0.044]	[0.040]
PM10 lead or	0.025	0.024	0.175
cumulative	[0.045]	[0.049]	[0.120]
PM10 2nd lead		0.016	
		[0.047]	
O3	-0.026	-0.032	-0.014
	[0.076]	[0.078]	[0.068]
O3 lead or	-0.047	-0.062	-0.106
cumulative	[0.076]	[0.086]	[0.125]
O3 2nd lead		0.014	
		[0.078]	
CO 1st trim	1.249	1.402	1.1
	[1.551]	[1.576]	[1.629]
CO 2nd trim	-0.562	-0.604	-0.128
	[1.160]	[1.184]	[1.239]
CO 3rd trim	0.427	0.518	0.307
	[1.609]	[1.641]	[1.613]
PM10 1st trim	-0.005	-0.026	-0.008
	[0.088]	[0.090]	[0.089]
PM10 2nd trim	-0.246	-0.236	-0.236
	[0.081]**	[0.083]**	[0.081]**
PM10 3rd trim	0.182	0.182	0.147
	[0.088]*	[0.089]*	[0.088]
O3 1st trim	0.005	0.016	-0.004
	[0.102]	[0.105]	[0.108]
O3 2nd trim	0.078	0.077	0.075
	[0.085]	[0.087]	[0.088]
O3 3rd trim	0.089	0.103	0.083
	[0.090]	[0.092]	[0.091]
Birthweight	-0.006	-0.006	-0.006
	[0.000]**	[0.000]**	[0.000]**
Gestation	-0.081	-0.082	-0.081
	[0.001]**	[0.001]**	[0.001]**
Observations	205981	201898	206352
R-squared	0.29	0.29	0.29

Notes: Robust standard errors in parentheses. A ** indicates significance at the 99% level of confidence. A * indicates significance at the 95% level of confidence.

Table 6: Prenatal Pollution and Probability of Low Birthweight

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	CS	CS	CS	CS	FE	FE	FE	FE
CO 1st trim	0.564			0.147	-2.048			-2.029
	[0.468]			[0.585]	[3.059]			[2.906]
CO 2nd trim	-1.692**			-2.225**	-5.556			-6.427*
	[0.531]			[0.638]	[3.230]			[3.150]
CO 3rd trim	0.461			0.652	-0.83			-0.318
	[0.504]			[0.604]	[2.432]			[2.392]
pm10 1st trim		0.018		0.021		-0.05		-0.038
		[0.032]		[0.041]		[0.120]		[0.117]
pm10 2nd trim		-0.093*		0.008		-0.229		-0.09
		[0.039]		[0.046]		[0.134]		[0.124]
pm10 3rd trim		0.07		0.025		0.03		0.001
		[0.039]		[0.049]		[0.118]		[0.115]
o3 1st trim			-0.036	-0.037			-0.007	0.072
			[0.037]	[0.047]			[0.187]	[0.181]
o3 2nd trim			-0.079	-0.135**			-0.168	-0.272
			[0.042]	[0.050]			[0.194]	[0.194]
o3 3rd trim			0.055	0.052			0.025	-0.005
			[0.033]	[0.041]			[0.197]	[0.198]
Observations	459837	454974	461027	453756	459837	454974	461027	453756
R-squared	0.01	0.01	0.01	0.01	0.03	0.03	0.03	0.03

Notes: Robust standard errors in parentheses. A ** indicates significance at the 99% level of confidence.
A * indicates significance at the 95% level of confidence.

F tests of the hypothesis that prenatal pollution coefficients sum to zero

CO	1.56			4.89	2.71			3.30
	[0.211]			[0.027]	[0.100]			[0.069]
PM10		0.01		1.49		2.33		0.67
		[0.913]		[0.223]		[0.127]		[0.413]
O3			2.05	5.26			0.40	0.77
			[0.152]	[0.022]			[0.530]	[0.381]

Notes: The first value is the F-statistic, the value in brackets is the p-value.

Table 7: Prenatal Pollution and Fetal Deaths

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	CS	CS	CS	CS	FE	FE	FE	FE
CO 1st trim	-0.159			-0.266	-0.329			-0.399
	[0.134]			[0.166]	[0.470]			[0.509]
CO 2nd trim	-0.138			-0.058	-0.061			-0.074
	[0.155]			[0.186]	[0.493]			[0.542]
CO 3rd trim	-0.019			-0.106	0.356			0.471
	[0.157]			[0.189]	[0.491]			[0.553]
pm10 1st trim		0.001		0.01		0.007		0.017
		[0.009]		[0.012]		[0.021]		[0.022]
pm10 2nd trim		-0.012		-0.011		-0.012		-0.014
		[0.011]		[0.014]		[0.023]		[0.024]
pm10 3rd trim		0.015		0.017		0.011		-0.001
		[0.012]		[0.016]		[0.023]		[0.025]
o3 1st trim			-0.001	-0.011			0.015	-0.001
			[0.011]	[0.014]			[0.034]	[0.037]
o3 2nd trim			0.002	0.004			-0.01	-0.008
			[0.013]	[0.015]			[0.037]	[0.040]
o3 3rd trim			0.002	-0.006			-0.013	-0.002
			[0.010]	[0.013]			[0.039]	[0.042]
Observations	461628	456748	462822	455526	461628	456748	462822	455526
R-squared	0	0	0	0	0.02	0.02	0.02	0.02

Notes: Robust standard errors in parentheses. A ** indicates significance at the 99% level of confidence.
A * indicates significance at the 95% level of confidence.

F tests of the hypothesis that prenatal pollution coefficients sum to zero

CO	3.80			4.71	0.00			0.00
	[0.051]			[0.030]	[0.970]			[0.998]
PM10		0.08		1.27		0.03		0.00
		[0.774]		[0.260]		[0.854]		[0.949]
O3			0.05	0.72			0.03	0.06
			[0.832]	[0.396]			[0.857]	[0.803]

Notes: The first value is the F-statistic, the value in brackets is the p-value.

Table 8: Estimates Using Data Aggregated to Quarterly Level

	(1)	(2)	(3)	(4)
1. Infant Mortality				
CO, quarter of death	0.043			0.030
	[0.0362]			[0.0379]
PM10, quarter of death		0.0044***		0.0034**
		[0.0017]		[0.0017]
Ozone, quarter of death			0.002	0.001
			[0.0017]	[0.0017]
# Observations	29452	29452	29452	29452
R-squared	0.09	0.09	0.09	0.09
2. Low Birthweight				
CO, quarter of birth	0.354			0.5274*
	[0.3252]			[0.3150]
PM10, quarter of birth		-0.010		-0.027
		[0.0168]		[0.0205]
Ozone, quarter of birth			0.009	0.024
			[0.0191]	[0.0224]
# Observations	34269	34269	34269	34269
R-squared	0.25	0.25	0.25	0.25
3. Fetal Deaths				
CO, quarter of birth	-0.015			0.007
	[0.1174]			[0.1197]
PM10, quarter of birth		0.000		-0.002
		[0.0052]		[0.0062]
Ozone, quarter of birth			0.004	0.005
			[0.0058]	[0.0068]
# Observations	34269	34269	34269	34269
R-squared	0.06	0.06	0.06	0.06

Note: The dependent variable in all cases is events per 1,000, per quarter. To get rates per 100,000, multiply by 100. Robust standard errors in brackets. A * and ** indicate significance at the 5 and 1% levels,respectively. Controls are similar to those shown in Tables 3 except that they are aggregated to the quarterly level.