Air Pollution and Infant Health:

What Can We Learn From California's Recent Experience?

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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 over the existing literature. First, most previous studies examine populations subject to greater levels of pollution, either because they lived further in the past or in some more heavily polluted area. In contrast, the experience of California in the 1990s is clearly relevant to the current policy debate over the regulation of pollution. Second, many studies examine a few routinely monitored pollutants in isolation, generally because of data limitations. We examine four "criterion" pollutants in a common framework Third, we develop an identification strategy based on within zip code variation in pollution levels that controls for potentially important unobserved characteristics of high pollution areas. Fourth, we use rich individual-level data to investigate effects on infant mortality, fetal deaths, low birth weight and prematurity in a common framework. We find that both Carbon Monoxide and particulates (PM10) exposures are associated with increased risk of death. We find that the reduction in Carbon monoxide (CO) that occurred over the 1990s in California resulted in a reduction of 535 deaths, while reductions in PM10 saved a further 509 lives. However, we find little consistent evidence of pollution effects on fetal deaths, low birth weight or short gestation.

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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). Higher standards for O3 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 U.S. Environmental Protection Agency (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 death 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]

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[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, most previous studies examine populations subject to greater levels of pollution, either because they lived further in the past (Chay and Greenstone, 2001 a,b) or in some more heavily polluted place (Xu, Ding, and Wang, 1995; Wang, Ding, Ryan, and Xu, 1997, Bobak, 2000, Dejmek et al. 1999, Bobak and Leon, 1999). 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 few routinely monitored pollutants in isolation, generally because of data limitations. We examine four "criterion" pollutants that are commonly monitored in the U.S.: Ozone (O3), carbon monoxide (CO), particulate matter (PM10), and nitrogen dioxide (NO2). Thus our results will enable us to say something about which pollutants appear to be the most harmful to infants.

Third, 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 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 level fixed effects, which will capture any unobserved characteristics of zip codes that are unchanged over time.

Fourth, we exploit rich individual-level data to estimate hazard models, where the hazard is defined either over weeks or months, and the baseline hazard is specified as a flexible non-parametric spline. This specification allows us to control separately for the effects of pollution exposure before and after the birth. Fifth, we examine effects on infant mortality, fetal death, low birth weight and prematurity in a common framework.

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. Our estimates suggest that the reductions in CO and PM10 that occurred over the 1990s saved more than 1,000 infant lives. However, we find little consistent evidence that pollution in the prenatal period affects birth weight, the probability of short gestation, or the risk of fetal death, at least at the levels of pollution that we observe.

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, and poisonous gas that reduces the delivery of oxygen to organs and tissues. Nitrogen Dioxide is a brown, reactive gas that irritates the lungs and may lower resistance to respiratory infections. Particulate matter can take many forms, including ash and dust. It is thought that the most damage comes from the smallest particles, since they are inhaled deep into the lungs (U.S. EPA, 2003b). Motor vehicles are a major source of PM10, NO2, and especially of CO--as much as 90% of CO in cities comes from motor vehicle exhaust (EPA, January 1993).[3]

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[3] Sulphur Dioxide and lead are the other two criterion pollutants. We do not examine them because levels are now so low in California that many monitors have been removed from service.

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 high in the atmosphere through reactions between nitrogen oxides (such as NO2) and volatile organic compounds (which are found in auto emissions, among other sources).

Compliance with standards for NO2 and PM10 is assessed by looking at annual means. Compliance with standards for O3 and CO is assessed by examining whether the level of pollution exceeded the standard over an eight-hour period during the year. These different approaches to standards suggest that the effects of NO2 and 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 or about the extent that infants are protected from the negative effects of pollution while they are in the womb. Infant mortality is defined as mortality in the first year of life, although the majority of infant deaths occur in the first month of life, often from some form of respiratory failure. These facts suggest that air pollution could be implicated in infant deaths. Air pollution could also affect fetal health: Some pollutants are known to cross the placenta, or to disrupt the flow of blood to the fetus and may therefore affect the fetus directly. Others may impair the health of the mother (e.g. by weakening her immune system) and hence affect the fetus indirectly, or cause premature labor (which has been linked to maternal infection).

Only some of these potential mechanisms have been examined. For example, it has long been known that CO can disturb the functioning of the placenta, that it crosses the placenta, and that it tends to concentrate in the fetus at higher levels than in the mother (Longo, 1977) ; it has also been shown in studies using rats that CO can have a negative effect on brain development (Garvey and Longo, 1978). However, the placenta may still offer some protection against episodic exposure.

Other studies have examined the negative effects of chemicals that are associated with high levels of CO and PM10; since motor vehicle exhaust is a major contributor of these two monitored pollutants, 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).

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. More recent studies have focused on the link between poor infant outcomes and high levels of pollution. For example, Xu, Ding, and Wang (1995) and Wang, Ding, Ryan, and Xu (1997) examine Chinese women delivering in Beijing in 1988. They found that there was a positive relationship between exposure to SO2 and Total Suspended Particles (TSPs) (the only two pollutants measured in Beijing at the

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[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.

time) and two infant health outcomes: preterm birth and low birth weight.[5] Bobak (2000), Dejmek et al. (1999) and Bobak and Leon (1999) examine Czech women and report that higher TSPs are associated with increases in low birth weight, preterm birth, and infant mortality due to respiratory causes (conditional on birth weight and gestation). The effects were highest in the post neonatal period, and only TSPs were statistically significant when the researchers also controlled for SO2 and nitrogen oxides.

Studies in the U.S. have also found a link between air pollution and infant health. For example, a study conducted in the early 1970s in Los Angeles (Williams, Spence, and Tideman, 1977) reported lower mean birth weights in areas with high pollution among women who were non-smokers. Woodruff et al. (1997) report that cities with higher levels of air pollution also tend to have higher infant mortality rates, even conditional on differences in socioeconomic status between cities. This study has been very influential, and has been used as a causal estimate of the effects of pollution in order to calculate harms (c.f. Kaiser et al. (2001)). On the other hand, Lipfert, Zhang, and Wyzga (2000) use linked U.S. birth and infant death records for 1990 and find little consistant relationship between annual county-level measures of ambient air quality and infant deaths.

Two recent studies by Ritz and her collaborators have examined the effects of air pollution in Southern California between 1989 and 1993 (Ritz et al. 2000; Ritz and Yu, 1999). In models that examine the same four criterion pollutants as this study, they demonstrate a relationship between high levels of CO and an increased risk of preterm

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[5] Note that PM10 refers to particles of a particular size, while many of the studies reviewed in this section discuss Total Suspended Particles or TSPs. In general one would expect TSP and PM10 to move together because PM10 is a component of TSP.

birth. They also find a relationship between CO, PM10, and low birth weight among full-term infants.

One drawback 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. Suppose for example, that areas with high levels of air pollution also tended to have high levels of water pollution. Then one might falsely conclude that air pollution was to blame for infant deaths, with potentially negative consequences for remediation efforts.

Two studies by Chay and Greenstone deal with this problem 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. On average, TSPs fell from 95 to 60 micrograms per cubic meter of air between 1970 and 1984. However, they show that both the Clean Air Act and the recession induced sharper reductions in TSPs in some areas than in others, and they use this exogenous variation in levels of pollution 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 but had little effect on the rate of low birth weight (i.e. birth weight less than 2500 grams).[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 that pollution has already been reduced

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

below that threshold. Secondly, given the available data, Chay and Greenstone were not able to directly compare the effects of prenatal and post-natal pollution exposure in order to determine whether pollution works mainly by harming fetuses or by harming vulnerable infants, or both.[7] Finally, the Chay and Greenstone studies cannot speak to whether other pollutants affect infant health, since only TSPs were measured during the time period that they study.

In the current paper, we propose an alternative identification strategy based on exploiting within-zip code variation in pollution levels. As we show below, even after controlling for seasonal effects and weather, there is a great deal of within-zip code variation in pollution levels. The zip code fixed effects control for many factors (such as poverty) which are both strongly geographically concentrated, and associated with poorer prospects for infants. Using this strategy allows us to identify the effects of pollution in more recent data, to compare the effects of several criterion pollutants, and to distinguish between the effects of prenatal and post-natal pollution exposure.

A final issue is that this paper (like the others discussed above) examines the effect of outdoor air quality measured using a fixed monitor. Much recent research focuses on the link between outdoor (ambient) air quality and total personal exposures to pollution measured using meters attached to persons. The latter will be 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.

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[7] They examine the effects of pollution on deaths in the first month of life (neonatal mortality), and show that most of the effect on infant mortality can be accounted for by a reduction in these deaths. However, since most infant deaths occur in the first month of life, any factor that significantly reduced infant deaths, would be likely to reduce neonatal deaths.

The research on the relationship between indoor and outdoor air quality has established several results. First, much of what is outdoors comes indoors–one study calculated that 46% of the fine particles a person who spent most of his or her time indoors in an air-conditioned home would be exposed would come from outdoor sources, while 84% of the particles someone who spent a lot of time indoors in a house with the windows open would be exposed would come from outdoors (Wilson, Mage, and Grant, 2000). Ozone is an interesting exception because it reacts with household surfaces and thus is not typically found in high concentrations within houses (http://www.hc-sc.gc.ca/hecs-sesc/air_quality/faq.htm). 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 between ambient air quality and personal exposure is higher. This is because there is a great deal of idiosyncratic variation across individuals in exposures to indoor air pollution, but for a given individual, indoor air quality may be relatively constant. So for a given individual, much of the variation in air quality comes from variation in ambient pollution levels (Wilson, Mage, and Grant, 2000).

Finally, indoor and outdoor air pollutants come from different sources and may have different health effects, so it is of interest to study the effects of ambient pollutants. Moreover, the effects of ambient air quality are of policy interest because the Environmental Protection Agency is mandated to monitor outdoor air and has no regulatory authority for indoor air quality.

III Data

Detailed data on atmospheric pollution comes from the 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.[8]

Following Neidell (2002), 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 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, of the births included in our sample, 76% were within 10 miles of a monitor, and we obtain very similar results if we limit our analysis to this subsample.

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[8] 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). Neidell (2002) shows that the levels of pollution calculated using all monitors, and the levels calculated using only continuously operated monitors are very highly correlated.

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 and for NO2 (.92 and .90, respectively). Correlations for PM10 and CO are somewhat lower, but still high (.77 and .78) suggesting that our measure is reasonably accurate.

Descriptive statistics for the pollution variables are shown in the first panel of Table 1, which also describes the units.[9] Table 1 shows that there is considerable variation in these measures, both between and within zip codes over our sample period. For example, the within zip code standard deviation for CO is .777 compared to the between zip code standard deviation of .677.

The pollutants we examine exhibit different seasonal patterns, as shown in Figure 1.[10] Ambient levels CO, PM10 and NO2 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, PM10, and NO2 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 will show below, the negative correlation of ozone with other pollutants can yield wrong-signed effects in single-pollutant models.

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[9] 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.

[10] Sulphur Dioxide and lead are the other two criterion pollutants. We do not examine them because levels are now so low in California that many monitors have been removed from service.

Our models include monthly fixed effects to control for seasonal effects, which removes some of the variation in pollution, but Figure 2 shows that a great deal of within zip code variation remains. Figure 2 plots residual levels of pollution after the zip code dummies, month and year dummies, weather indicators and all of the other variables included in our models have been controlled for. (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.)

Data on birth weight, gestational age, and infant 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 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. Most infants who are low birth weight are also premature (defined as gestation less than 37 weeks), so we also look at these outcomes. Note, that there is no birth cohort file for 1998, so this year is excluded from our analysis.

Low birth weight and/or premature infants are at high risk both of an infant death and of fetal death. The distinction between these two concepts is that a child must be born alive in order to be registered as an infant death. 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 for our examination of birth weight, gestation, and fetal death. Examination of the effects of pollution on this sample will give us estimates of the effects of pollution on birth outcomes that are not biased by fetal selection that occurs after 26 weeks. While pollution might also cause fetal deaths before 26 weeks, the data does not support an analysis of this issue.

Descriptive statistics for these variables are also shown in Table 1. The infant mortality rate here is the number of infants less than one year old alive in any particular quarter who die, hence to compare to published figures one would multiply this number by four. The estimates indicate that over the sample period, about 6.56 children per 1,000 died in their first year. About nine percent of pregnancies lasting at least 26 weeks have gestation less than 37 weeks, while about 5 percent of pregnancies result in a low birth weight delivery. Finally, the rate of fetal death is similar to the infant mortality rate.

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, 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 (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, since this information is not included on California's birth certificate. Still, this will only pose problems for the analysis if that part of maternal smoking that is not captured by other included variables is systematically correlated with the within-zip code variation in levels of air pollution.

The third panel of Table 1 shows trends in pollution levels over the sample period. All four pollutants show considerable declines. Some of this improvement is perhaps due to new federal "Tier 1" automobile tailpipe pollution standards passed in 1990 which became effective in 1994-1996.

The final panel of Table 1 shows that although the infant mortality rate fell sharply over a relatively short time, trends in low birth weight and gestation were much flatter. This table suggests then, that declines in mortality were largely due to events occurring after the birth, rather than to improvements in prenatal health.

Table 2 shows mean 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 four 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 more than twice as high 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. 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, Table 2 shows that more polluted areas tend to have more mothers who are black and unmarried, and have fewer mothers who are college educated. On the other hand, more polluted areas have higher fractions of Hispanic mothers, which would cause them to have better birth outcomes, given that Hispanic women tend to bear healthier infants other things being equal. In what follows, we will control for these important observable differences between locations, as well as for unobservable zip code-level characteristics by including zip code-level fixed effects.

IV. Methods

We begin by estimating models of the effects of post-natal pollution exposure on the probability of infant death, conditional on prenatal pollution exposure. Specifically, we estimate a discrete-time hazard model where the unit of time is the week. Our model allows for time-varying covariates, non-parametric duration dependence, and zip code level fixed effects. Allison (1982) shows that estimates from models of this type converge to those obtained from continuous time models, as discussed further in the appendix.

The hazard rate (P_{izt}) is specified as:

$$P_{izt} = {''}(t) + w_{iz}\,(+ h_z H + x_{zt}\$ + N_z + m_t, \tag{1}$$

where P_{izt} is the probability of death. (Note that we have also estimated models using $f(P_{izt})$ as the dependent variable, where f is the logit transformation as discussed further below). In (1), ${''}(t)$ 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 and use of government insurance; the h_z are time-invariant measures of the infants health at the time of the birth, including indicators for low birth weight and short gestation; the x_{zt} are time-varying covariates, including pollution and weather; N_z is a zip code specific fixed effect; and m_t is a vector of month dummies. In this model, the main coefficient of interest is $\$$, the effect of post-natal pollution exposure on the probability of death. Any effect of prenatal exposures is assumed to be captured via the effects on birth weight and gestation, which are controlled.

We will estimate a second model, explicitly examining the effects of pollution before and after birth:

$$P_{izt} = \text{''}(t) + w_{iz}(+ p_z 0 + x_{zt}\$ + N_z + m_t, \qquad (2)$$

In this model, birth weight and gestation are omitted, and a vector p_z measures prenatal pollution exposure (which cannot vary after the child is born). In this model, we can compare the estimated effects of prenatal and postnatal exposures, 0 and $\$$.

In order to implement this estimation strategy, we treat an individual who lived for n weeks as if they contributed n observations to the sample. The dependent variable (Y_{it}) 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. Y_{it} is then regressed on the covariates specified in (1) by ordinary least squares.

Because this procedure yields a very large number of observations, with relatively few deaths, we employ case-control sampling to reduce the number of observations. First, we keep all individuals who died (the cases). 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 who were in the same zip code. 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 period, we randomly chose five times as many non-deaths as deaths. We lose some observations due to missing covariates which means that the probability of death in the estimation sample is .1589 rather than .1666 (the total number of deaths is 25,256). 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)).[11]

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 is referred to as "harvesting" (Schwartz (2001)). If harvesting 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. Although we also

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[11] 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 adds a constant to the log odds ratio in the Appendix.

estimate models with lagged pollution levels, it is not feasible to estimate models with very long lag structures, and so models estimated using weekly measures may miss the longer-term effects of pollution exposure.

On the other hand, a problem with models using longer time units such as months is that the measure of pollution is imprecisely assigned. For example, if we use the month as the time unit, children who die on the first day of their second month of life are incorrectly assigned average pollution levels for all of the days in the month. Thus, using longer time periods involves more measurement error, which could bias coefficients downwards, especially if it is the acute effects of exposure that matter. Still, it is important to note that PM10, in particular, is only measured once per week, and is quite variable, so that readings over a few weeks might actually give a more accurate picture of the amount of pollution a child was exposed to.

In order to deal with these problems, we compare estimates from models using weeks to estimates from models using months as the time unit. As we show below, the monthly models yield very similar estimates of the effects of CO, suggesting that the estimated effects in the weekly models are not driven by harvesting. On the other hand, the effects of PM10 become larger when months are used as the time unit, suggesting that there may in fact be more measurement error in the weekly than in the monthly measure of PM10.

Note that 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 precipitation in the vector x_{zt}. These controls are specified to be in the same time units as the pollutants—for example, if both pollution in the weeks after birth and pollution in the last trimester are included in the model, then variables measuring the weather during these periods are also included. To the extent that weather affects pollution without having an independent effect on infant health, including the weather variables will reduce the amount of legitimate variation in our pollution measures, and attenuate the estimated effects. Thus, inclusion of these variables will yield a conservative estimate of the effect of pollution.

Figure 3 compares actual infant mortality rates to those that would be predicted by model (1), if we excluded the pollution measures from the model. Figure 3 shows that there is still a fair amount of unexplained variation in infant mortality rates, which could be driven by unexpected changes in pollution levels.

We go on to directly examine the effects of prenatal pollution exposure on low birth weight, prematurity, and fetal death in a 20 percent random sample of pregnancies that lasted at least 26 weeks (regardless of whether or not the pregnancy ended in a live birth). These models have the form:

$$P_{iz} = w_{iz} (+ p_z O + N_z + m_t, \tag{3}$$

where now P_{iz} is defined as the probability of low birth weight or short gestation, and the other variables are defined as above.

V. Results

a) Effects on Infant Mortality

Table 3 shows estimates of model (1). For convenience, the coefficients and standard errors on the pollutants and on the weather variables are multiplied by 1000. For comparison with previous work, we first estimate cross sectional models for each pollutant separately. These “single pollutant” models without zip code fixed effects shown in columns (1) through (4), indicate that exposure to CO after birth increases the probability of infant death. On the other hand, O3 after birth has a wrong-signed coefficient.

Column (5) shows that if we include all four pollutants only CO has a significant positive effect on mortality after birth. Since CO, PM10, and NO2 are highly correlated, and it is possible that these estimates suffer from multi-collinearity, we also present a multi-pollutant model excluding one pollutant, NO2, in column (6). This model is consistent with the column (5) model.

Columns (7) through (12) of Table 3 show the same models estimated using zip code fixed effects. The estimated effects of pollution after birth become much stronger compared to the cross-sectional models. Now CO, PM10, and NO2 exposures after birth are all estimated to increase the risk of death (and O3 is again estimated to have a wrong-signed effect) in the single-pollutant models shown in columns (7) through (10). However, the multi-pollutant models again suggest that only CO exposure after birth significantly increases infant mortality. This result is robust to whether we include all four pollutants or only three, as a comparison of columns (11) and (12) shows.

The other covariates shown in Table 3 have largely the expected signs. For example, the probability of death is much higher among low birth weight and premature infants, and is higher in the first week than subsequently. Children with black mothers have a lower probability of death conditional on being low birth weight, as do children of Hispanic and foreign-born mothers. Other factors that increase the risk of death are having a teen mother, having a high school dropout mother, being of high birth order, and having government insurance (rather than private insurance; very few births to mothers in

California are not covered by insurance). Including the fixed effects has relatively little effect on the estimated effects of these individual-level covariates. In the rest of this discussion, we omit these covariates from the tables.

Table 4 presents an alternative specification of model (1) which includes zipcode*year fixed effects. The results are very similar to those discussed above. However, this is not our preferred specification, given that there are a substantial number of zipcode*year cells without deaths

Table 5 begins to examine the question of whether prenatal exposures matter, by estimating models of form (2). Panel 1 adds average pollution measured over the last trimester of each child's pregnancy, while panel two adds both first and last trimester pollution variables. To conserve space, we focus only on the models with zip-code fixed effects, and drop the model with all four pollutants. We found that NO2 was never significant in the multi-pollutant models, but that collinearity with CO and PM10 became an issue when multiple measures of the same pollutants were added to the model.

Panel 1 of Table 5 shows that the estimated effects of pollution after birth are very similar to those shown in Tables 3 and 4. None of the measures of pollution in the last trimester are significant at the 95 percent level of confidence. But PM10 in the last trimester is signficant at the 90 percent level, and the coefficient is large, suggesting that perhaps PM10 exposure in the last trimester has a negative effect on infant health. Panel 2 shows that including pollution in the first trimester as well has little effect on the other coefficients, and that pollution in the first trimester does not appear to have a significant effect on infant mortality.

Table 6 shows a model similar to that estimate in Panel 1 of Table 5, except that the hazard is estimated over months, rather than weeks of life. As discussed above, if the effects we observe were driven by “harvesting”, then we might expect the size of the estimated effects to fall when we move to a longer time horizon. However, rather than decreasing the size of the estimated effects, this change in time interval tends to increase the effect of PM10 after birth, while the effect of CO is unchanged. The greatly increased size and significance of the coefficient on PM10 may indicate that exposures may be measured more accurately over a longer time horizon (since PM10 is only measured in one 24 hour period per week) or that PM10 has cumulative effects. Some evidence in favor of the latter possibility is that PM10 in the last trimester still appears to have a positive, if imprecisely estimated effect in these models.

In addition to the alternative specifications shown in Tables 4, 5, and 6, we have estimated models using pollution measured from monitors within a 10-mile (rather than a 20 mile radius); models using a logit rather than a linear probability model, and models that exclude deaths in the first week of life. The rationale for this last specification check, was that infants at risk of death might be more likely than others to spend their first week in an environment such as an incubator in which they would not be exposed to ambient air quality. These models all produced similar results to those in Tables 5 and 6, as shown in Appendix Table 1.[12] Finally, we estimated models that interacted the effects of pollution with race, in order to see whether there were any systematic differences in the effects of pollution. None of the interaction terms were significant, as shown in Appendix Table 1.

To summarize, CO and PM10 appear to have the most significant effects on infant

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[12] Using closer monitors results in the loss of data (since fewer zip codes are within 10 miles of a monitor). However, we found that the correlations between pollution measures obtained using a 20 mile radius, and those obtained using a 10 mile radius were very high (.96, .95, .96, and .97 for O3, CO, PM10, and NO2, respectively) so perhaps it is not surprising that the two measures produce similar results.

mortality. The estimated effect of CO is remarkably robust to many changes in specification. The Table 6 coefficient of approximately .0033 (recall that all coefficients and standard errors on the pollutants are multiplied by 1,000) implies that the 1.021 unit decline in CO that occurred over our sample period was associated with approximately 535 fewer deaths, or about 11 fewer deaths per 100,000 live births.

The coefficient on PM10 is more sensitive to specification, but the Table 6 coefficient of approximately .020 implies that the .159 unit decline in PM10 over our sample period resulted in 509 fewer deaths, which implies a reduction of 10.9 deaths per 100,000, per unit of PM10.

To compare this estimate to those of Chay and Greenstone, it is necessary to recall that we divided the PM10 measure by 100, so there was actually a 15.9 micro grams per meter cubed per hour decline in PM10 over our sample, or a reduction of .7 deaths per 100,000 per standard unit. Of course, Chay and Greenstone estimate single pollutant models, so a better comparison might involve the coefficient of .033 from column 2 of Table 6. This coefficient implies a reduction of 1.2 deaths per 100,000 per standard unit of PM10 reduction, which is smaller than the effects estimated by Chay and Greenstone. The smaller estimate may reflect a non-linear effect of particulates on infant health, the fact that TSPs are a broader measure than PM10 (while roughly half of TSPs are less than 10 microns in diameter, smaller particles are thought to have the worst effects), perhaps a California-specific effect given that Chay and Greenstone use national data. It is also possible that the estimated effect is larger in more aggregated data, a possibility we investigate below.

B) Effects on Low Birth Weight, Prematurity, and Fetal Death

As Table 1 showed, infant death is a rare outcome, and it is possible that prenatal pollution exposure could have effects on infant health even if it did not result in death. Also, the sample of children born alive is a selected one, so it is of interest to examine the effects of pollution on a fuller sample of pregnancies, including both those born alive and those born dead. Hence, in Table 7 we examine these questions directly by estimating model (3). Columns (1) through (5) estimate cross-sectional models, while Columns (6) through (10) include zip code fixed effects.

The estimates shown in the single-pollutant cross-sectional models are consistent with those of the prior literature, in that they suggest that all of the criterion pollutants reduce birth weight and/or gestation. For example, CO, PM10, and NO2 are all estimated to contribute to low birth weight, although in the multi-pollutant model, only PM10 remains statistically significant. Similarly, multi-pollutant models of prematurity suggest that CO, PM10 and O3 all increase prematurity. Fetal deaths have not been previously examined, but we find little evidence that they are related to pollution, even in the cross section.

Unlike the infant mortality results presented above, these estimates are not robust to the inclusion of zip code level fixed effects. In the fixed effects models, none of the pollutants have a significant effect on the probability of low birth weight, prematurity, or fetal death. We have also estimated these models using only the sample of live births with very similar results, and have experimented with including pollution in the first trimester as well as pollution in the last trimester. Neither measure of pollution exposure was statistically significant in the fixed effects models.

c) Estimated Effects in Aggregate Data

Several previous studies have used aggregate rather than individual-level data and it is of interest to compare our results with theirs. Hence, we have aggregated our data to the zip code-quarter level and estimated models similar to (1) and (2). Note that in the infant mortality regressions, we now control only for pollution in the quarter of birth, and cannot distinguish between exposure before and after birth. These models are shown in Table 8. 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 is significant in the multi-pollutant models. The point estimate of .468 in column (2) indicates that the decline in PM10 from 48.8 to 32.9 micrograms per cubic meter of air that occurred between 1989 and 1997 led to about 1.9 infant fewer deaths per 100,000, per unit decline in PM10, which suggests that the estimated effects of PM10 may be larger in more aggregated data. The rest of the Table shows that once again, we find little effect of pollution on the incidence of low birth weight, prematurity, or fetal death.

VI. Discussion and Conclusions

Environmental policy continues to be extremely 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).[13] Similarly, there is a great deal of controversy over the Bush

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[13] 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

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. 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 indeed 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 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 variation in pollution, so that we are able to control for unobservable fixed characteristics of zip codes as well as for a detailed group of observable time-varying characteristics. Controlling for area fixed effects causes us to overturn some of the findings in the (largely cross-sectional) epidemiological literature concerning prenatal pollution exposures. For example, we find little effect of prenatal pollution exposure on the probability of low birth weight or prematurity once zip code fixed effects are included in the models.

In "single pollutant" models that include fixed effects, we find that CO, PM10, and NO2 all increase infant mortality. But in multi-pollutant models, we find that CO and PM10 have the strongest effects. Our estimates imply that reductions in CO and PM10 over the time interval we study saved over 1,000 infant lives. These findings areis clearly relevant to policy debates over automobile emissions and the Clear Skies Initiative, for example.

A complete evaluation of the costs and benefits of improvements in air quality is far beyond the scope of this paper (see for example, 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). Note however that there are several reasons why the health benefit that we measure here would not be capitalized into housing prices. 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 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 $1.7 billion.[14] If we use the EPA(1999) value of $4.8 million, the benefit would grow to $5 billion. These estimates ignore other benefits of pollution reduction, such as improvements in health which are not at the life/death margin, and so are lower-bound estimates of the benefit. But they may still be a useful benchmark for assessing the benefits of further reductions in air pollution in terms of infant health.

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[14] Chay and Greenstone (2001a) use this $1.6 million value. However, Viscusi (1993) suggested that the value of a life was between $3.5 and $8.5 million, and U.S. EPA (1999) valued infant lives lost due to lead at $4.8 million, the same value that they used for adult lives.

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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:

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:

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:

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

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. For example, one could set d_1 to 1 and d_2 to ~.16 as we have done. 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

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

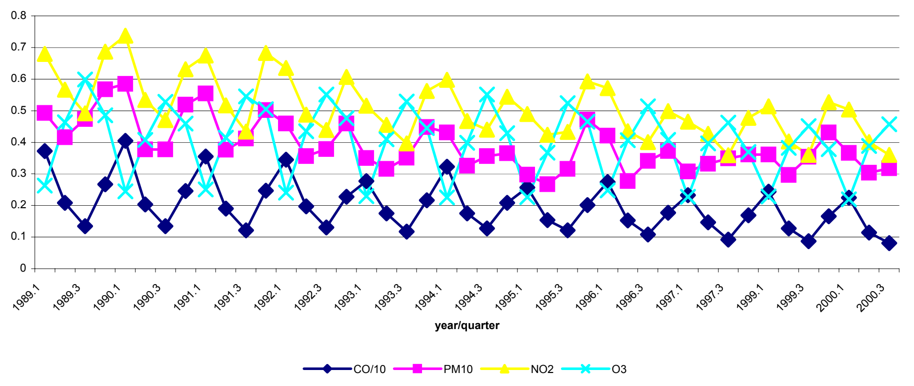
or the log odds

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

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."

yearq	CO	CO/10	PM10	NO2	O3
1989.1	3.717823	0.371782	0.492629	0.679481	0.262495
1989.2	2.086403	0.20864	0.415296	0.566852	0.463648
1989.3	1.341853	0.134185	0.473456	0.491766	0.599021
1989.4	2.666479	0.266648	0.566975	0.686221	0.48542
1990.1	4.044464	0.404446	0.584118	0.736906	0.244813
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
1994.1	3.224715	0.322472	0.430792	0.597581	0.225304
1994.2	1.750595	0.17506	0.326117	0.467667	0.398644
1994.3	1.273965	0.127397	0.356087	0.439707	0.551016
1994.4	2.081572	0.208157	0.365098	0.544296	0.42919
1995.1	2.565088	0.256509	0.297169	0.489783	0.226227
1995.2	1.541892	0.154189	0.267019	0.423343	0.367046
1995.3	1.213551	0.121355	0.315238	0.433065	0.523474
1995.4	2.013943	0.201394	0.470906	0.592657	0.465395
1996.1	2.736386	0.273639	0.420965	0.571536	0.247932
1996.2	1.52767	0.152767	0.277481	0.434587	0.406193
1996.3	1.08558	0.108558	0.340922	0.40013	0.51374
1996.4	1.765868	0.176587	0.372432	0.498498	0.407162
1997.1	2.323437	0.232344	0.307716	0.464997	0.227501
1997.2	1.464453	0.146445	0.331418	0.426641	0.396679
1997.3	0.920483	0.092048	0.349255	0.358628	0.461875
1997.4	1.684802	0.16848	0.360902	0.477551	0.369038
1999.1	2.434757	0.243476	0.361447	0.513488	0.228255
1999.2	1.278792	0.127879	0.296067	0.402989	0.382664
1999.3	0.867694	0.086769	0.354267	0.360508	0.451446
1999.4	1.660605	0.166061	0.430618	0.527017	0.378613
2000.1	2.245327	0.224533	0.366382	0.503481	0.219788
2000.2	1.141004	0.1141	0.302934	0.400211	0.387977
2000.3	0.811396	0.08114	0.318022	0.359895	0.456868

Figure 1. Seasonal Variation in Pollution



yearq	O3	CO	PM10	NO2
1989.1	-0.009296	0.013918	-0.045405	-0.017571
1989.2	7.24E-05	-0.038522	-0.005488	0.004173
1989.3	0.008549	-0.062345	0.014059	-0.002565
1992.4	0.012015	0.004099	-0.004159	0.007394
1993.1	0.018907	-0.183172	-0.005793	0.00095
1993.2	-0.011578	0.024584	0.002575	0.007758
1993.3	-0.00909	0.117439	0.000624	0.011719
1993.4	-0.001006	0.070171	0.009589	0.005688
1994.1	-0.009823	-0.143302	0.019257	-0.013753
1994.2	-0.005086	0.00418	0.025665	0.00804
1994.3	0.002446	0.044381	0.016084	0.012385
1994.4	0.010264	-0.02595	-0.045859	-0.014467
1995.1	0.013893	-0.268214	-0.017153	-0.028117
1995.2	-0.01313	0.100662	-0.011621	9.43E-05
1995.3	-0.015428	0.172124	-0.009733	0.015785
1995.4	0.014931	-0.03586	0.052291	0.00814
1996.1	0.028037	-0.220652	0.01434	0.01247
1996.2	0.001509	0.019338	-0.01141	0.002539
1996.3	-0.008622	0.160834	0.00969	0.011888
1996.4	-0.008752	0.016988	-0.016065	-0.015056
1997.1	0.037011	-0.454555	-0.075502	-0.05023
1997.2	0.022185	0.131973	0.05075	0.034551
1997.3	-0.02718	0.224852	0.034402	0.024678
1997.4	-0.012767	0.085622	-0.020478	0.002512
1999.1	0.050429	-0.249589	-0.039596	-0.014034
1999.2	0.039667	0.137548	0.004252	0.02403
1999.3	-0.024944	0.239401	0.018543	0.013015
1999.4	-0.026061	-0.089309	0.008084	-0.014161
2000.1	0.033328	-0.232035	-0.007834	-0.001617
2000.2	0.007905	-0.003724	0.013569	-0.001772
2000.3	-0.027106	0.292187	-0.010166	0.013731

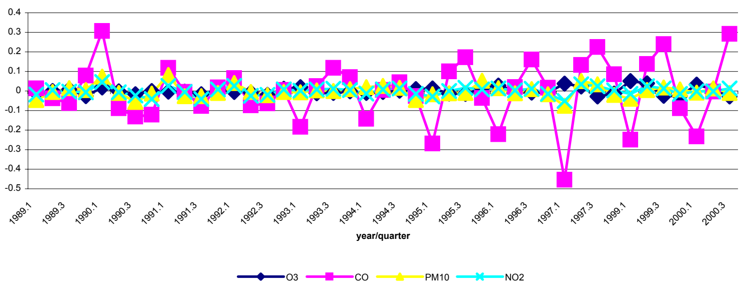
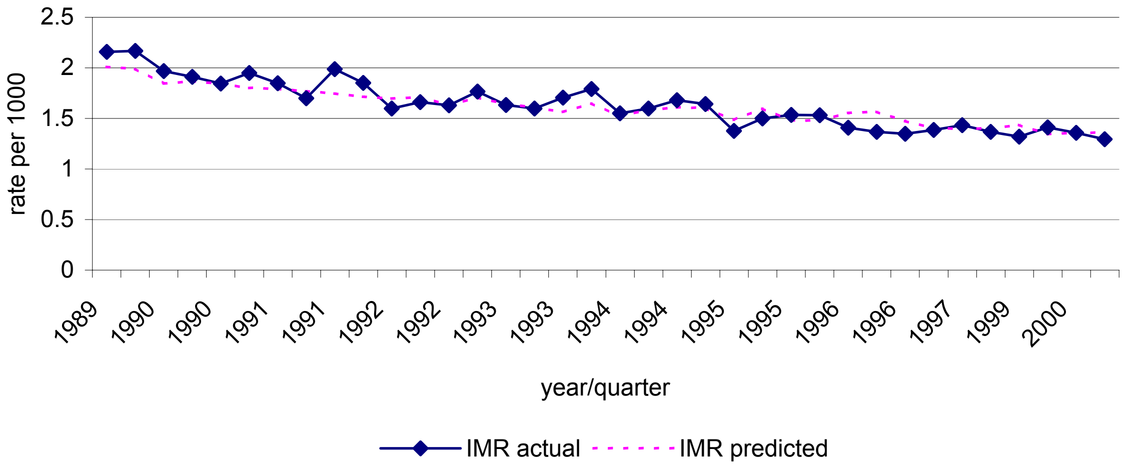


Figure 2. Residual Variation in Pollution

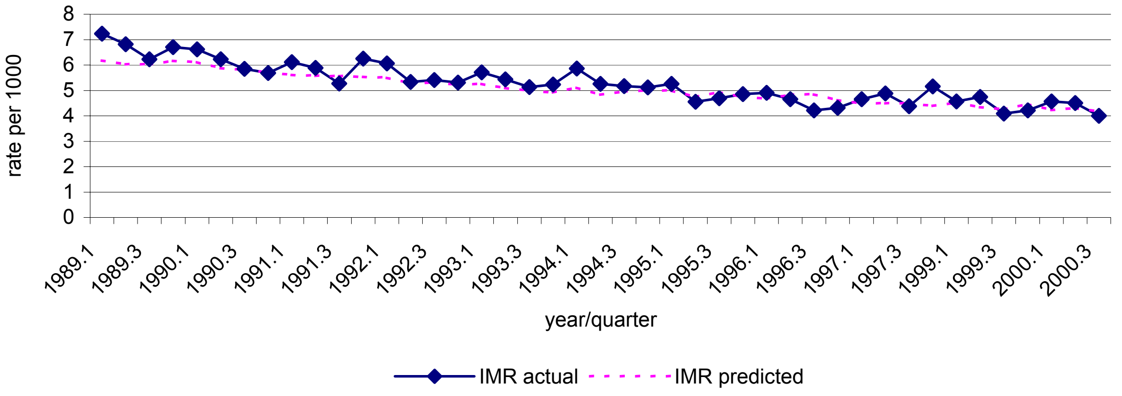
quarterly annual

Figure 5. Plot of Actual and Predicted IMR (annual)



1993.1	5.707997	5.2565	1993.4	1.705443	1.5625
1993.2	5.439817	5.0933	1994.1	1.791934	1.6487

Figure 3. Plot of Actual and Predicted IMR (quarterly)



1997.2	4.881277	4.4944	2000.1	1.409817	1.3452
1997.3	4.37183	4.4873	2000.2	1.358679	1.3599
1997.4	5.16095	4.3882	2000.3	1.294	1.3534
1999.1	4.561981	4.5132			
1999.2	4.739056	4.3459			
1999.3	4.090824	4.2474			
1999.4	4.213202	4.4611			
2000.1	4.561512	4.2299			
2000.2	4.502883	4.2973			
2000.3	4.006234	4.17			

Table 1: Levels and Trends in Pollution and Infant Health

Variable	Mean	Std. Dev.	Between zip std. Dev.	Within zip std. Dev.
Panel 1				
CO 8-hr	1.975	1.101	0.677	0.777
PM10 24-hr	0.391	0.142	0.108	0.092
NO2 1-hr	0.509	0.184	0.154	0.095
O3 8-hr	0.404	0.159	0.099	0.118
Panel 2				
infant mortality rate	1.64	1.95	4.07	11.28
gestation<37 rate	92.60	30.91	25.72	48.64
rate of low birthweight	48.80	21.59	14.94	39.08
fetal death rate	5.86	6.58	3.38	13.64
*per 1000				

Panel 3

year	CO	PM10	NO2	O3
1989	2.409	0.488	0.603	0.460
1990	2.435	0.462	0.590	0.414
1991	2.252	0.460	0.574	0.433
1992	2.243	0.413	0.542	0.427
1993	1.940	0.366	0.481	0.409
1994	2.071	0.369	0.511	0.403
1995	1.822	0.337	0.484	0.398
1996	1.767	0.353	0.475	0.396
1997	1.585	0.338	0.431	0.366
1998	1.544	0.361	0.449	0.363
1999	1.388	0.329	0.420	0.357

Panel 4 year	IMR annual	Gestation < 37 weeks	Low Birth Weight	Fetal Deaths
1989	2.16	95.67	51.11	6.49
1990	1.97	93.31	48.73	6.27
1991	1.87	92.39	47.91	6.04
1992	1.69	91.73	48.76	5.92
1993	1.67	92.73	48.98	5.78
1994	1.66	92.22	49.94	5.75
1995	1.51	92.15	48.86	5.86
1996	1.41	92.23	48.80	5.88
1997	1.40	92.08	48.56	5.40
1999	1.32	92.56	47.04	5.36
2000	1.35	91.28	47.72	5.60

Note: O3 and NO2 are measured as the hourly pollution level in parts per million. CO is measured in parts per million over an eight hour period, while PM10 is measured as micro grams per meter cubed per hour, and is only measured over one 24 hour period each week. To make units comparable, we multiply the measures of 03 and NO2 by multiplying by 10, and divide the measure of PM10 by 100. To construct quarterly data, we aggregate these same data.

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	1.157	1.883	2.786
PM10 24-hr	0.251	0.390	0.536
NO2 1-hr	0.310	0.495	0.689
O3 8-hr	0.337	0.401	0.471
infant mortality rate	1.512	1.788	1.965
gestation<37 rate	78.385	89.626	92.388
low BW rate	43.281	47.243	48.673
fetal death rate	5.129	5.743	5.862
% male	0.488	0.488	0.489
% black	0.064	0.071	0.078
% hispanic	0.256	0.393	0.440
% asian	0.134	0.108	0.099
% other race	0.015	0.008	0.006
% married	0.742	0.700	0.669
% foreign mom	0.333	0.416	0.455
% racial diff b/w parents	0.185	0.172	0.154
% HS grads	0.334	0.339	0.344
% AD degree	0.153	0.140	0.134
% college grads	0.297	0.236	0.196
% educ. diff b/w parents	0.373	0.375	0.371
% age 19 to 25	0.267	0.303	0.325
% age 26 to 30	0.276	0.282	0.288
% age 31 to 35	0.258	0.230	0.214
% age >= 36	0.144	0.121	0.103
% first born	0.431	0.413	0.408
% second born	0.323	0.310	0.304
% third born	0.148	0.159	0.162
% gov't insurance	0.338	0.408	0.416
% prenatal care in 1st trimester	0.826	0.807	0.769

Notes: Zip-code years were ranked by first standardizing all of the pollution measures using a Z-score, and then taking the average of the four measures.

Table 3: Effect of Pollution on Infant Mortality, A Comparison of Cross Sectional and Fixed Effects Models

	1	2	3	4	5	6	7	8	9	10	11	12
	CS	CS	CS	CS	CS	CS	FE	FE	FE	FE	FE	FE
CO after birth	1.918				3.3189	1.7085	3.9189				4.6131	3.4462
	[0.5727]**				[1.1550]**	[0.7260]*	[0.7256]**				[1.2780]**	[0.8862]**
PM10 after birth		-0.4978			-1.6207	-3.8889		10.3787			2.6435	0.7353
		[3.5712]			[4.6144]	[4.4119]		[4.3296]*			[5.3598]	[5.0995]
NO2 after birth			3.6507		-11.861				15.624		-10.1063	
			[3.2436]		[6.5079]				[4.5493]**		[8.0208]	
Ozone after birth				-20.7595	-11.3175	-14.7446				-21.848	-7.215	-9.8735
				[5.0884]**	[6.0301]	[5.7505]*				[6.3423]**	[6.9632]	[6.8601]
Maximum temp. after birth	-0.7267	-0.801	-0.806	-0.5148	-0.4986	-0.5217	-0.5601	-0.7531	-0.7363	-0.4631	-0.4419	-0.4552
	[0.0710]**	[0.0682]**	[0.0677]**	[0.0967]**	[0.0976]**	[0.0967]**	[0.0778]**	[0.0739]**	[0.0736]**	[0.1061]**	[0.1070]**	[0.1062]**
Precipitation after birth	-0.0303	-0.0514	-0.0457	-0.0289	-0.0226	-0.0256	0.0019	-0.0228	-0.0229	-0.0279	0.0075	0.0067
	[0.0597]	[0.0599]	[0.0596]	[0.0597]	[0.0603]	[0.0603]	[0.0656]	[0.0656]	[0.0651]	[0.0658]	[0.0665]	[0.0664]
1-2 weeks old	-0.3485	-0.3484	-0.3485	-0.3485	-0.3485	-0.3486	-0.348	-0.348	-0.348	-0.3481	-0.348	-0.348
	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**	[0.0068]**
3-4 weeks old	-0.0384	-0.0383	-0.0383	-0.0383	-0.0383	-0.0383	-0.0387	-0.0387	-0.0387	-0.0387	-0.0387	-0.0387
	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**
5-8 weeks old	-0.0045	-0.0045	-0.0045	-0.0045	-0.0045	-0.0045	-0.0043	-0.0043	-0.0043	-0.0043	-0.0043	-0.0043
	[0.0017]**	[0.0017]**	[0.0017]**	[0.0017]**	[0.0017]**	[0.0017]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**
9-12 weeks old	-0.0058	-0.0058	-0.0058	-0.0058	-0.0058	-0.0058	-0.0059	-0.0059	-0.0059	-0.0059	-0.0059	-0.0059
	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**
13-20 weeks old	-0.007	-0.007	-0.007	-0.0071	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007
	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**	[0.0005]**
21-32 weeks old	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032	-0.0032
	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**	[0.0002]**
> 32 weeks old	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008	-0.0008
	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**	[0.0001]**
Birthweight	-0.1298	-0.1298	-0.1298	-0.1298	-0.1298	-0.1298	-0.1303	-0.1303	-0.1303	-0.1303	-0.1303	-0.1303
	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0015]**	[0.0015]**	[0.0015]**	[0.0015]**	[0.0015]**	[0.0015]**
Gestation	-0.386	-0.3863	-0.3861	-0.3868	-0.3865	-0.3865	-0.3857	-0.3858	-0.3858	-0.3861	-0.3858	-0.3858
	[0.0264]**	[0.0264]**	[0.0264]**	[0.0264]**	[0.0264]**	[0.0264]**	[0.0281]**	[0.0281]**	[0.0281]**	[0.0281]**	[0.0281]**	[0.0281]**
male	-0.03	-0.03	-0.03	-0.03	-0.03	-0.03	-0.0301	-0.0301	-0.0301	-0.0301	-0.0301	-0.0301
	[0.0013]**	[0.0013]**	[0.0013]**	[0.0013]**	[0.0013]**	[0.0013]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**	[0.0014]**
black	-0.0154	-0.0146	-0.0148	-0.0155	-0.0158	-0.0159	-0.0005	-0.0004	-0.0005	-0.0005	-0.0005	-0.0005
	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0027]**	[0.0027]**	[0.0031]	[0.0031]	[0.0031]	[0.0031]	[0.0031]	[0.0031]
hispanic	-0.0211	-0.0205	-0.0207	-0.0206	-0.0207	-0.021	-0.0171	-0.0171	-0.0171	-0.0171	-0.0171	-0.0171
	[0.0020]**	[0.0020]**	[0.0020]**	[0.0020]**	[0.0020]**	[0.0020]**	[0.0022]**	[0.0022]**	[0.0022]**	[0.0022]**	[0.0022]**	[0.0022]**
asian	-0.013	-0.013	-0.013	-0.0134	-0.0132	-0.0132	-0.0098	-0.0098	-0.0098	-0.0097	-0.0098	-0.0098
	[0.0027]**	[0.0027]**	[0.0027]**	[0.0028]**	[0.0028]**	[0.0028]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**

Table 3: Effect of Pollution on Infant Mortality (continued)

other race	-0.005	-0.0052	-0.0051	-0.0054	-0.0052	-0.0052	-0.0031	-0.003	-0.0031	-0.0031	-0.003	-0.0031
	[0.0076]	[0.0076]	[0.0076]	[0.0076]	[0.0076]	[0.0076]	[0.0077]	[0.0078]	[0.0077]	[0.0078]	[0.0078]	[0.0077]
married mother	0.0008	0.0007	0.0008	0.0008	0.0007	0.0008	-0.0003	-0.0001	-0.0002	-0.0001	-0.0003	-0.0003
	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]	[0.0030]
foreign born mother	-0.0214	-0.021	-0.0211	-0.0213	-0.0214	-0.0216	-0.0207	-0.0207	-0.0207	-0.0207	-0.0207	-0.0207
	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**
parents diff race	0.0107	0.0106	0.0106	0.0106	0.0106	0.0107	0.0106	0.0106	0.0106	0.0106	0.0106	0.0106
	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**
HS grad mother	-0.0085	-0.0086	-0.0086	-0.0085	-0.0085	-0.0085	-0.0097	-0.0097	-0.0097	-0.0097	-0.0097	-0.0097
	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**
AD degree	-0.0113	-0.0114	-0.0114	-0.0113	-0.0113	-0.0113	-0.0135	-0.0135	-0.0135	-0.0135	-0.0135	-0.0135
	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**
college grad	-0.0146	-0.0147	-0.0147	-0.0147	-0.0147	-0.0147	-0.0184	-0.0183	-0.0184	-0.0184	-0.0184	-0.0184
	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**
educ diff parents	0.0003	0.0003	0.0003	0.0004	0.0004	0.0004	0.0004	0.0004	0.0004	0.0004	0.0004	0.0004
	[0.0014]	[0.0014]	[0.0014]	[0.0014]	[0.0014]	[0.0014]	[0.0015]	[0.0015]	[0.0015]	[0.0015]	[0.0015]	[0.0015]
19-25 mother	-0.009	-0.009	-0.009	-0.0091	-0.0091	-0.0091	-0.0097	-0.0097	-0.0097	-0.0097	-0.0097	-0.0097
	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**
26-30 mother	-0.0175	-0.0174	-0.0174	-0.0175	-0.0175	-0.0176	-0.0187	-0.0187	-0.0187	-0.0187	-0.0187	-0.0187
	[0.0031]**	[0.0031]**	[0.0031]**	[0.0031]**	[0.0031]**	[0.0031]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**
31-35 mother	-0.0191	-0.019	-0.019	-0.0191	-0.0192	-0.0192	-0.0209	-0.0209	-0.0209	-0.0209	-0.0209	-0.0209
	[0.0034]**	[0.0034]**	[0.0034]**	[0.0034]**	[0.0034]**	[0.0034]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**
mother >=36	-0.0144	-0.0143	-0.0144	-0.0146	-0.0146	-0.0146	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016
	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0038]**	[0.0040]**	[0.0040]**	[0.0040]**	[0.0040]**	[0.0040]**	[0.0040]**
first born	-0.0446	-0.0446	-0.0446	-0.0448	-0.0448	-0.0448	-0.0469	-0.0469	-0.0469	-0.0469	-0.0469	-0.0469
	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**
second born	-0.0216	-0.0216	-0.0216	-0.0217	-0.0217	-0.0218	-0.0233	-0.0234	-0.0234	-0.0234	-0.0233	-0.0233
	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**
third born	-0.0141	-0.014	-0.0141	-0.0141	-0.0141	-0.0142	-0.0152	-0.0152	-0.0152	-0.0152	-0.0152	-0.0152
	[0.0025]**	[0.0025]**	[0.0025]**	[0.0025]**	[0.0025]**	[0.0025]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**	[0.0024]**
gov't insurance	0.0089	0.0088	0.0089	0.0089	0.0088	0.0089	0.0114	0.0114	0.0114	0.0113	0.0113	0.0114
for birth	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**	[0.0016]**
prenatal care 1st	-0.0114	-0.0114	-0.0114	-0.0115	-0.0115	-0.0115	-0.0116	-0.0116	-0.0116	-0.0117	-0.0117	-0.0116
trimester	[0.0017]**	[0.0017]**	[0.0017]**	[0.0017]**	[0.0017]**	[0.0017]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**
Observations	158869	158869	158869	158869	158869	158869	158869	158869	158869	158869	158869	158869
R-squared	0.49	0.49	0.49	0.49	0.49	0.49	0.49	0.49	0.49	0.49	0.49	0.49
Number of fixed effects							899	899	899	899	899	899

Notes: Standard errors in brackets. * indicates significance at the 95% level, ** at the 99% level. All regressions also included year, month, and quarter of conception dummies. Coefficients and standard errors on polllutants and weather measures have been multiplied by 1000. The high values for r-square are due to the case-control sampling in which we over-sample deaths relative to non-deaths. Using a 10% randomly drawn sample, we obtain r-sqaures of approximately 0.01.

Table 4: Models Controlling for Zip-code*Year Fixed Effects

	1	**2**	**3**	**4**	**5**	**6**
CO after birth	3.8487				4.7181	3.431
	[0.7581]**				[1.3850]**	[0.9301]**
PM10 after birth		9.6454			1.9096	-0.2317
		[4.5014]*			[5.6010]	[5.3293]
NO2 after birth			15.1777		-11.1188	
			[4.7350]**		[8.6796]	
Ozone after birth				-22.9564	-7.3207	-10.2386
				[6.6839]**	[7.3595]	[7.2341]
Observations	158869	158869	158869	158869	158869	158869
R-squared	0.52	0.52	0.52	0.52	0.52	0.52
# fixed effects	8637	8637	8637	8637	8637	8637

Notes: These regression as the same as table 3 except with zip code-year fixed effect

Table 5: Effect of Post-Natal and Pre-Natal Pollution Exposure on Infant Mortality

1. Include Pollution in Last Trimester

	1	2	3	4	5
	FE	FE	FE	FE	FE
CO after birth	4.119				3.601
	[0.7918]**				[0.9561]**
PM10 after birth		10.665			0.517
		[4.6654]*			[5.4397]
NO2 after birth			14.934		
			[5.0290]**		
Ozone after birth				-24.694	-11.552
				[6.7615]**	[7.3656]
CO last trimester	-1.350				-2.014
	[1.5985]				[1.6876]
PM10 last trimester		19.540			21.613
		[10.0486]			[11.0454]
NO2 last trimester			9.107		
			[13.0755]		
Ozone last trimester				18.967	5.937
				[10.3169]	[11.3861]
Observations	158929	158929	158929	158929	158929
R-squared	0.420	0.420	0.420	0.420	0.420
# of fixed effects	899	899	899	899	899

2. Include Pollution in First and Last Trimester

	1	2	3	4	5
	FE	FE	FE	FE	FE
CO after birth	3.9485				3.2839
	[0.7768]**				[0.9389]**
PM10 after birth		9.5524			0.1738
		[4.6583]*			[5.4191]
NO2 after birth			14.2468		
			[4.9114]**		
Ozone after birth				-25.6816	-12.5183
				[6.7561]**	[7.3749]
CO last trimester	-3.3422				-3.9475
	[2.6009]				[2.5939]
CO first trimester	-2.7659				-1.1186
	[3.0718]				[2.8268]
PM10 last trimester		16.3407			23.37
		[11.1737]			[11.6001]*
PM10 first trimester		-19.0058			-20.4998
		[13.0834]			[13.3492]
NO2 last trimester			0.6965		
			[14.5616]		
NO2 first trimester			-20.3831		
			[19.4712]		
Ozone last trimester				24.9255	7.2852
				[12.9689]	[13.1859]
Ozone first trimester				9.498	16.9699
				[15.1943]	[15.5203]
Observations	158588	157679	158780	158854	157419
R-squared	0.43	0.43	0.43	0.43	0.43
# of fixed effects	899	899	899	899	899

Notes: Standard errors in brackets.   * indicates significance at the 95% level, ** at the 99% level. All regress
conception dummies. Coefficients and standard errors on polllutants and weather measures have been multi
due to the case-control sampling in which we over-sample deaths relative to non-deaths. Using a 10% rando
approximately 0.01.

sions also included year, month, and quarter of
iplied by 1000. The high values for r-square are
omly drawn sample, we obtain r-sqaures of

Table 6: Monthly Hazard for Mortality

1. Including Third Trimester Pollution

	1	**2**	**3**	**4**	**5**
CO after birth	5.1779				3.2887
	[1.1942]**				[1.4109]*
PM10 after birth		33.0728			19.6756
		[7.4821]**			[7.9810]*
NO2 after birth			35.4074		
			[8.6040]**		
Ozone after birth				-24.6961	-10.1899
				[9.2452]**	[10.4414]
CO last trimester	-1.0197				-1.3305
	[1.7431]				[1.8916]
PM10 last trimester		21.1155			19.823
		[11.1062]			[13.0487]
NO2 last trimester			12.8164		
			[14.2782]		
Ozone last trimester				23.664	11.1876
				[11.5478]*	[12.8772]
# Observations	159480	159480	159480	159480	159480
R-squared	0.33	0.33	0.33	0.33	0.33
# fixed effects	897	897	897	897	897

Notes: These regressions include zip-code fixed effects and are of the same form as those in Table 4.

Table 7: Prenatal Pollution and the Probability of Poor Birth Outcomes

	1	2	3	4	5	6	7	8	9	10
	CS	CS	CS	CS	CS	FE	FE	FE	FE	FE
1. Low Birth Weight										
CO last trimester	0.7719**				0.441	0.131				0.723
	[0.2788]				[0.3356]	[1.4123]				[1.0917]
PM10 last trimester		7.7422**			5.8935*		-4.190			-7.289
		[1.9505]			[2.4867]		[7.3253]			[6.7991]
NO2 last trimester			4.0163**					-2.610		
			[1.5074]					[12.2516]		
Ozone last trimester				3.106	1.477				1.581	4.974
				[2.3531]	[2.8103]				[7.6838]	[7.5827]
Observations	921661	921661	921661	921661	921661	921661	921661	921661	921661	921661
R-squared	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
# fixed effects						915	915	915	915	915
2. Gestation Less than 37 Weeks										
CO last trimester	1.7937**				0.9619*	0.634				1.041
	[0.3839]				[0.4587]	[3.6809]				[2.7288]
PM10 last trimester		25.9510**			18.9103**		4.933			-0.090
		[2.6580]			[3.4181]		[18.6968]			[17.1281]
NO2 last trimester			15.6315**					11.078		
			[2.0562]					[31.2045]		
Ozone last trimester				20.1126**	13.9816**				7.577	10.255
				[3.1811]	[3.8177]				[19.7108]	[19.2820]
Observations	922158	922158	922158	922158	922158	922158	922158	922158	922158	922158
R-squared	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
# fixed effects						936	936	936	936	936
3. Fetal Deaths >= 26 weeks										
CO last trimester	-0.1589				-0.2001	0.0152				0.0323
	[0.0890]				[0.1045]	[0.1857]				[0.1755]
PM10 last trimester		-0.2533			0.5037		-0.5371			-0.5713
		[0.6064]			[0.7695]		[1.0691]			[1.1235]
NO2 last trimester			-1.2388**					0.0793		
			[0.4745]					[1.5436]		
Ozone last trimester				0.2267	-0.3976				-0.5653	-0.3625
				[0.7164]	[0.8594]				[1.1979]	[1.2576]
Observations	874697	874697	874697	874697	874697	874697	874697	874697	874697	874697
R-squared	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003
# fixed effects						731	731	731	731	731

Table 8: Estimates Using Data Aggregated to Quarterly Level

1. Infant Mortality	**1**	**2**	**3**	**4**	6
CO, quarter of death	0.047				0.03
	[0.0307]				[0.0326]
PM10, quarter of death		0.4684***			0.4002**
		[0.1668]			[0.1765]
NO2, quarter of death			0.5191*		
			[0.2840]		
Ozone, quarter of death				0.187	0.043
				[0.1737]	[0.1835]
# Observations	30238	30238	30238	30238	30238
R-squared	0.09	0.09	0.09	0.09	0.09

2. Low Birthweight					
CO, quarter of birth	0.367				0.5808*
	[0.3225]				[0.3118]
PM10, quarter of birth		-1.349			-3.323
		[1.7078]			[2.0458]
NO2, quarter of birth			0.355		
			[2.4388]		
Ozone, quarter of birth				0.867	2.659
				[1.7738]	[2.0602]
# Observations	35219	35219	35219	35219	35219
R-squared	0.26	0.26	0.26	0.26	0.26

3. Gestation < 37 weeks					
CO, quarter of birth	0.356				0.33
	[0.5285]				[0.6010]
PM10, quarter of birth		1.262			0.661
		[3.3474]			[3.8362]
NO2, quarter of birth			2.174		
			[3.7345]		
Ozone, quarter of birth				0.093	0.15
				[2.9789]	[2.9373]
# Observations	35219	35219	35219	35219	35219
R-squared	0.34	0.34	0.34	0.34	0.34

4. Fetal Deaths >= 26 weeks					
CO, quarter of birth	0.037				0.055
	[0.1163]				[0.1199]
PM10, quarter of birth		0.163			-0.131
		[0.5114]			[0.6301]
NO2, quarter of birth			0.951		
			[0.8119]		
Ozone, quarter of birth				0.430	0.53
				[0.5941]	[0.6979]
# Observations	35219	35219	35219	35219	35219
R-squared	0.06	0.06	0.06	0.06	0.06

Notes: Specifications are similar to those in Tables 3 and 7, with all data aggregated to quarterly level.
All models have zipcode fixed effects.

Appendix Table 1: Alternative Specifications for Infant Mortality Models

1. Pollutants Measured Using Monitors Within 10 Miles vs. 20 Miles

	20 mile measure w 20 mile sample	20 mile measure w 10 mile sample	10 mile measure w 10 mile sample	20 mile measure w 20 mile sample	20 mile measure w 10 mile sample	10 mile measure w 10 mile sample
CO after birth	5.266	4.973	4.316	3.601	2.743	3.076
	[1.3630]**	[1.5072]**	[1.2571]**	[0.9561]**	[1.0424]**	[0.9220]**
PM10 after birth	3.401	5.613	2.913	0.517	1.766	0.395
	[5.6996]	[6.3747]	[5.6998]	[5.4397]	[6.1192]	[5.4519]
NO2 after birth	-14.570	-19.612	-12.276			
	[8.6593]	[9.4708]*	[8.3522]			
Ozone after birth	-7.755	-15.197	-11.871	-11.552	-20.378	-14.358
	[7.4582]	[8.6239]	[7.7448]	[7.3656]	[8.4716]*	[7.7010]
CO last trimester	-2.766	-2.456	-2.221	-2.014	-1.733	-1.784
	[2.1238]	[2.3143]	[1.8657]	[1.6876]	[1.8157]	[1.5331]
PM10 last trimester	19.833	20.406	21.769	21.613	22.226	23.056
	[11.0754]	[13.1675]	[11.5441]	[11.0454]	[12.9646]	[11.4474]*
NO2 last trimester	10.168	9.860	7.066			
	[17.7793]	[20.3613]	[17.1261]			
Ozone last trimester	3.308	16.789	16.310	5.937	19.300	17.975
	[12.5586]	[14.6099]	[12.8070]	[11.3861]	[13.1317]	[11.7055]
Observations	158929	120962	120962	158929	120962	120962
R-squared	0.42	0.42	0.42	0.42	0.42	0.42
# fixed effects	899	621	621	899	621	621

2. Logit Model Similar to Specification in Table 4

CO after birth	50.860				47.240
	[8.9849]**				[11.4122]**
PM10 after birth		103.718			-19.701
		[53.7617]			[63.4381]
NO2 after birth			180.910		
			[60.2830]**		
Ozone after birth				-306.233	-132.082
				[83.8269]**	[94.5778]
CO last trimester	-9.070				-18.761
	[18.6361]				[20.1527]
PM10 last trimester		232.989			264.145
		[119.5512]			[137.0946]
NO2 last trimester			143.487		
			[155.6154]		
Ozone last trimester				204.656	35.662
				[131.5708]	[148.8100]
Observations	158927	158927	158927	158927	158927
# fixed effects	897	897	897	897	897

Appendix Table 1, continued.

3. Logit Model Similar to Specification in Table 6

CO after birth	50.860				47.240
	[8.9849]**				[11.4122]**
PM10 after birth		103.718			-19.701
		[53.7617]			[63.4381]
NO2 after birth			180.910		
			[60.2830]**		
Ozone after birth				-306.233	-132.082
				[83.8269]**	[94.5778]
CO last trimester	-9.070				-18.761
	[18.6361]				[20.1527]
PM10 last trimester		232.989			264.145
		[119.5512]			[137.0946]
NO2 last trimester			143.487		
			[155.6154]		
Ozone last trimester				204.656	35.662
				[131.5708]	[148.8100]
Observations	158927	158927	158927	158927	158927
# fixed effects	897	897	897	897	897

4. Infant Mortality Excluding Deaths in First Week of Life, Weekly Hazard

CO after birth	4.3769				3.7812
	[0.7812]**				[0.9517]**
PM10 after birth		11.3822			0.6591
		[4.6628]*			[5.4557]
NO2 after birth			16.1484		
			[5.1088]**		
Ozone after birth				-26.7073	-12.9168
				[6.9562]**	[7.6142]
CO last trimester	-1.2543				-1.6508
	[1.6655]				[1.7648]
PM10 last trimester		16.2893			16.2639
		[9.8987]			[11.1528]
NO2 last trimester			11.8711		
			[12.9085]		
Ozone last trimester				17.9686	8.1968
				[10.3082]	[11.5367]
Observations	142582	142582	142582	142582	142582
R-squared	0.08	0.08	0.08	0.08	0.08
# fixed effects	839	839	839	839	839

5. Pollution Interacted with Mother's Race

CO after birth	4.1544				3.472
	[0.7741]**				[0.9541]**
CO after birth*black	-1.4195				0.7681
	[1.8096]				[2.4487]
PM10 after birth		10.9927			1.3272
		[4.4790]*			[5.2510]
PM10 after birth*black		-8.4547			-11.6848
		[13.9528]			[16.7986]
NO2 after birth			15.8887		
			[4.8139]**		

NO2 after birth*black			-3.8368		
			[13.1553]		
Ozone after birth				-23.4153	-11.2746
				[6.4571]**	[7.1040]
Ozone after birth*black				5.5143	13.3575
				[13.6929]	[17.4665]
CO last trimester	-1.5985				-1.5882
	[1.4553]				[1.5524]
CO last trimester*black	0.886				-3.2808
	[1.9738]				[3.0436]
PM10 last trimester		16.5068			15.2174
		[9.2339]			[10.5179]
PM10 last trimester*black		3.3767			25.1158
		[18.8065]			[25.0480]
NO2 last trimester			10.373		
			[11.5544]		
NO2 last trimester*black			-6.0114		
			[16.2662]		
Ozone last trimester				22.1206	12.4128
				[9.6517]*	[10.6712]
Ozone last trimester*black				-18.9987	-37.047
				[15.1330]	[21.8365]
Observations	158869	158869	158869	158869	158869
R-squared	0.49	0.49	0.49	0.49	0.49

Notes: These models are of the same form as those shown in Tables 5 and 6.