

**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 estimate hazard models that investigate whether infant deaths are more affected by pollution exposure before or after the birth.

Our results suggest that both carbon monoxide (CO) and particulates (PM10) exposures are associated with increased risk of death. We find that the 42 percent reduction in CO that occurred over the 1990s in California resulted in a reduction of 627 deaths (in approximately 3.9 million live births), while reductions in PM10 saved a further 432 lives. This reduction in deaths was accomplished primarily through reductions in pollution exposure after birth, though we find some evidence that NO2 exposure may have increased the probability of 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). The Administration argues that California’s regulation usurped federal authority to regulate fuel efficiency, and has obtained a two-year injunction preventing implementation of the regulations. 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 effects of pollution on health. 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.¹

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,

¹ 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

2000, Dejmek et al. 1999, Bobak and Leon, 1999). While these studies demonstrate a link between high levels of pollution and infant mortality, it is not clear that their results can be extrapolated to the contemporary debate over pollution levels in the United States if pollution has non-linear effects on health. In contrast, the experience of California in the 1990s is clearly relevant to this debate.

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 pollution of ground water) that are correlated with both air pollution and infant health outcomes. We 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 semi-parametric survival models to control separately for the effects of pollution exposure before and after the birth. We also define the hazard either over weeks or months in order to determine the degree of "harvesting," or mortality displacement, that might occur from higher frequency measurements.

Our estimates confirm that air pollution has a significant effect on infant

standard. Other states may adopt California’s standards, but may not draft their own.

mortality, even at the relatively low levels of pollution experienced in recent years. In “single pollutant” models that include zip code fixed effects, CO, PM10, and NO2 all increase infant mortality, while in “multiple pollutant” models, CO and PM10 have the most significant effects. Our estimates suggest that the reductions in CO and PM10 that occurred over the 1990s saved 627 and 432 infant lives, respectively. Although it is known that at least some of these pollutants can cross the placenta, we find that it is primarily exposure to pollution after birth that contributes to infant deaths. We also find little consistent evidence that pollution in the prenatal period affects birth weight, though NO2 exposure is estimated to increase the probability of short gestation

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, and it is thought that the most damage comes from the smallest particles, since they are inhaled deep into the lungs (U.S. EPA, 2003). 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. However, we 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.

A link between air pollution and infant health has long been suspected, although the exact biological mechanisms through which it occurs are not known. Infant mortality, although defined as mortality in the first year of life, mostly occurs 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). 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, such as retarded growth.² 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 time) and two infant health outcomes: preterm birth and low birth weight.³ 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,

² 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.

³ 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.

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.

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 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 recent 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 but 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).⁴

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 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.⁵ 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 (both over time and across seasons) 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

⁴ 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.

⁵ 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

several criterion pollutants, and to distinguish between the effects of prenatal and post-natal pollution exposure.

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. Figure 1 shows ozone monitors by county in 1999, while Figure 2 shows ozone monitors by zip code within Los Angeles county. Monitors tend to be located in the most densely populated areas of the state (indicated in gray on the figure), 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.⁶

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

significantly reduced infant deaths, would be likely to reduce neonatal deaths.

⁶ 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.

able to assign pollution levels to are overwhelmingly rural, as indicated in Figures 1 and 2.

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 inverse-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. 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 in micro grams per meter cubed over one 24 hour period every six days. In order to make units roughly comparable, we scale measures of O3 and NO2 by multiplying by 10, and divide the measure of PM10 by 100. When we turn to analyses of aggregate data, we use these same measures averaged up to the quarterly level. Taking the average of our measures of NO2 and PM10 over the four quarters of the year would yield an annual mean similar to that used to monitor compliance with federal pollution standards. 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 (U.S. Environmental Protection Agency, 2002).⁷ Table 1 shows that there is considerable

⁷ These measures are highly correlated with measures of short-term spikes in pollutants. For

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 .75 compared to the between zip code standard deviation of .70.

The pollutants we examine come from different sources and exhibit somewhat different seasonal patterns, as shown in Figure 3.⁸ 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). Ambient levels of these pollutants tend to increase in cold weather when they are trapped by damp cold air. PM10 also tends to increase 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 is formed higher in the atmosphere through reactions between nitrogen oxides (such as NO2) and volatile organic compounds (which are found in auto emissions, among other sources), and 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. Our models include season fixed effects, which although remove some of the variation in these measures, as we will show below, a great deal of within zip code variation remains.

Data on birth weight, gestational age, and infant deaths come from the California Birth Cohort files for 1989 to 1997. These data are abstracted from birth, death, and fetal

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.

⁸ Sulphur Dioxide and lead are the other two criterion pollutants. We do not examine them

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), or very premature (gestation less than 32 weeks), so we also look at these outcomes.

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 of low birth weight to instead 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 and gestation. 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. Moreover, fetal deaths in the first months of pregnancy are generally thought to reflect chromosomal damage, while deaths after 26 weeks are often due to complications of labor and delivery and hence are more comparable to deaths that occur after birth.

Descriptive statistics for these variables are also shown in Table 1. About nine

because levels are now so low in California that many monitors have been removed from service.

percent of pregnancies lasting at least 26 weeks have gestation less than 37 weeks, while only 1.2 percent have gestation less than 32 weeks. About 5 percent of pregnancies result in a low birth weight delivery.

In addition to the infant health measures, 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. It is noteworthy that these reductions in pollution occurred against a backdrop of increases in total miles driven, and the increased popularity of vehicles which were not subject to the same standards as other passenger cars, such as sport utility vehicles.

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-quarters by pollution level, we first standardized all of the pollution measures using a “z-score” and then took the average of the four measures. Table 2 indicates that 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 in part 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, semi-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:

$$P_{izt} = \alpha(t) + w_{iz}\gamma + p_z\eta + x_{zt}\beta + \varphi_z, \quad (1)$$

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), $\alpha(t)$ 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.

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 p_z are time-invariant covariates defined at the zip code level, such as pre-natal pollution exposure (which cannot vary after the child is born); the x_{zt} are time-varying covariates, including pollution and weather; and φ_z is a zip code specific fixed effect.

The main coefficients of interest are η , the effect of pre-natal exposure, and β , the effect of post-natal exposure, on the probability of death.

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, yielding a probability of death in the estimation sample of .1543 rather than .1666 (the total number of deaths is 22,513). 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)).⁹

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. Moreover, models estimated using weekly pollution focus on the short-term effects of pollution exposure. Although we also estimate models with lagged pollution

⁹ 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

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

sample that excluded infants who were at risk of death, but survived only to die later.

pollution in the last trimester are included in the model, then variables measuring the weather during these periods are also included.

Our estimates suggest that pollution after the birth has a much greater impact on infant deaths than pollution prior to birth. In order to further probe these results, we go on to directly examine the effects of prenatal pollution exposure on birth weight and gestation 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:

$$P_{iz} = w_{iz}\gamma + p_z\eta + \varphi_z, \quad (2)$$

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

These models are identified by exploiting within-zip code variation in pollution levels, infant mortality, and birth outcomes. Figure 3 suggests that some of this variation is seasonal, as one might expect given the relationships between temperature, precipitation levels, and air pollution in California. However, Figure 4 indicates that even after we control for the zip code dummies, the season dummies, the weather, and the other variables in the vectors w_{iz} and p_z , there is still a good deal of residual variation in these pollution levels. Figure 5 indicates that there is little seasonal pattern in infant mortality or low birth weight, though short gestation does seem to decline slightly in the winter months. However, Figure 6 shows that conditional on all of the other covariates included in our models except pollution, there is little seasonal pattern in infant health outcomes. Thus, our results are not likely to be driven by a spurious correlation between seasonal patterns in pollution and seasonal patterns in infant mortality or birth outcomes.

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. The “single pollutant” models without zip code fixed effects shown in columns (1) through (4) indicate that exposure to CO, PM10, and NO2 after birth increases the probability of infant death. On the other hand, O3 after birth has a wrong-signed coefficient, though exposure to ozone in the last trimester is estimated to have a significant positive effect on mortality. Of the other three pollutants, only prenatal exposure to CO is statistically significant, and it is wrong-signed.

Column (5) shows the results of estimating a multi-pollutant model, still without zip code fixed effects. In this specification, only CO has a significant positive effect on mortality after birth, while NO2 exposure in the last trimester also has a significant positive effect. However, CO in the last trimester has a perverse negative effect on mortality. 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 suggests that only CO has a significant effect on mortality after birth, while O3 exposure in the last trimester also increases mortality.

Columns (7) through (12) of Table 3 show the same models estimated using zip code fixed effects. The estimated effects of pollution after birth are remarkably similar to those of the cross-sectional models. However, including fixed effects eliminates the

impact of prenatal exposures. While 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), the multi-pollutant models 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 in the first week than subsequently. Children with black mothers have a higher probability of death, while children of Hispanic and foreign-born mothers have a lower probability of death. 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.

Tables 4 and 5 present several alternative specifications of the model (1). In the first panel, we include pollution in the first trimester rather than pollution in the last trimester in order to probe whether pollution measured at the outset of the pregnancy has a greater effect. The results are similar to those in Table 3, in that only CO exposure after birth appears to have a significant effect on mortality in the multi-pollutant models. The second panel of Table 4 presents a model similar to that in Table 3, except that it also controls for birth weight and gestation. By controlling for birth weight and gestation, we

ask whether exposure to pollution after birth has an effect conditional on health at birth. The effects of birth weight and gestation are highly significant (note that the coefficients and standard errors on these variables are multiplied by 1,000), but the estimated effects of pollution are very similar to those presented in Table 3.

Table 5 explores the timing of pollution exposure. In the first panel, we add two lags of pollution. These estimates address the issue of whether it is very short-term acute exposure or a somewhat more cumulative exposure to pollution that matters. The single-pollutant models indicate that it is the first lags of CO, PM10, and NO2 that have the largest effects. These lagged effects may reflect the assignment problem discussed above (i.e. for a child who dies on the first day of the second week of life, pollution during the first week is more relevant than pollution during the second week). On the other hand, the second lags are never statistically significant and tend to be much smaller than the contemporaneous effect and first lag, suggesting that cumulative effects (at least over a few weeks) are not important. The multi-pollutant model with lags demands a lot of the data, so perhaps it is not surprising that little is statistically significant. It is however, interesting to note that in this specification, the first lag of PM10 is significant. Also, F-tests on the two groups of coefficients indicate that both the CO and PM10 coefficients are significant at the 90 percent level of confidence.

The second panel of Table 5 adds a “lead” of the pollution data. While the first lag may matter because of a measurement problem as described above, pollution measured after the child dies should certainly not have any effect on the probability of death. It is encouraging then, that we find no significant effect of leads in our data.

Table 6 shows a model similar to panel 2 of Table 4, except that the hazard is

estimated over months of life rather than weeks. Rather than decreasing the estimated effects, this change in time interval tends to increase the estimated effects of CO, PM10, and NO2 in the single-pollutant models. In the multi-pollutant model shown in column 5, nothing is statistically significant, indicating some difficulty in distinguishing the separate effects of these pollutants. However, if we drop NO2 from the model, CO and PM10 are significant at the 95 and 90 percent levels of confidence, respectively, as shown in Table 6. The fact that the coefficient estimates become larger as the time unit is increased suggests that the results are not driven by harvesting. In particular, the greatly increased size and significance of the coefficient on PM10 both in the weekly models with lags and in the monthly models suggests that exposures may actually be measured better over a longer time interval, given that the PM10 measure is only recorded once per week.

In addition to the alternative specifications shown in Tables 4 and 5, we have also estimated logit models, and models using pollution measured from monitors within a 10-mile (rather than a 20 mile) radius. These models all produced similar results to those in Table 3, as shown in Appendix Table 1.¹⁰

To summarize, we find very little effect of prenatal exposure to pollution in models with zip code fixed effects, while the cross-sectional estimates of the effects of post-natal exposures are robust to the addition of fixed effects. Our results indicate that of the pollutants we examine, CO and PM10 appear to have the most significant effects on infant mortality. The estimated effect of CO is remarkably robust to many changes in

¹⁰ 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

specification. The coefficient of approximately .004 (recall that all coefficients and standard errors on the pollutants are multiplied by 1,000) implies that the 1.074 unit decline in CO that occurred over our sample period was associated with approximately 627 fewer deaths, or about 16 fewer deaths per 100,000 live births.

The coefficient on PM10 is more sensitive to specification, but in the weekly models with lags (shown in column 6 of Table 5), the sum of the lags is .0116 which can be compared to the coefficient of .0129 on PM10 in the monthly hazard model (shown in column 6 of Table 6). The coefficient of .0129 suggests that the 23 unit decline in PM10 over our sample period resulted in 432 fewer deaths, which implies a reduction of .5 deaths per 100,000 per unit of PM10.

This estimate is small relative to the effects estimated by Chay and Greenstone. However, single-pollutant models which only include PM10 may be more directly comparable to their work. In these models, the estimated effect of PM10 is somewhat greater. For example, in column 3 of Table 5, the sum of the lags is .0236, and in column 3 of Table 6, the coefficient is .0289. Hence, in these single-pollutant models, our estimates imply a reduction of approximately 1.1 deaths per 100,000, per unit of PM10, which is closer to the range of between 4 and 8 deaths found by Chay and Greenstone. The smaller effect may reflect a non-linear effect of particulates on infant health, the fact that TSPs are a broader measure than PM10, or perhaps a California-specific effect given that Chay and Greenstone use national data.

b) Effects on Birth weight and Gestation

As discussed above, our results suggest that pollution exposure prior to birth has

produce similar results.

little effect on the risk of infant death. However, 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 (2). Columns (1) through (6) estimate cross-sectional models, while Columns (7) through (12) 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 short gestation suggest that PM10 has an important effect, although in the model for gestation less than 37 weeks O3 is also important.

Unlike the results presented above, most of these estimates are not robust to the inclusion of zip code level fixed effects. In the models with fixed effects, none of the pollutants have a significant effect on the probability of low birth weight. However, NO2 is estimated to have a large and significant effect on the probability of short gestation, in both single and multiple pollutant models, and for both gestation less than 32 weeks and gestation less than 37 weeks. For example, the point estimate of .017 in the model for gestation less than 37 weeks implies that the decrease of 26 units in NO2 that occurred over our sample period decreased the probability of short gestation by 4.5

percent.¹¹

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 first panel of Table 8 shows that in the aggregate-level data, CO, PM10, and NO2 are all significant in single-pollutant models of infant mortality with zip code fixed effects. However, only PM10 is significant in the multi-pollutant models, and then only when we drop NO2. The point estimate of .478 in column (3) indicates that the decline in PM10 from 56.6 to 33.7 micrograms per cubic meter of air that occurred between 1989 and 1997 led to approximately 427 fewer infant deaths, or about .5 deaths per 100,000, per unit decline in PM10. This estimate is remarkably similar to what we obtained in the individual-level data using either weekly hazards with lags, or monthly hazards.

The coefficient on CO in the single-pollutant model shown in column 2 implies a much smaller effect of CO—here the one unit decrease over the period is found to have saved only 195 lives. Hence, it appears that the effects of CO are much attenuated in the aggregate data, perhaps because of the increased measurement error involved in moving to a longer time period.

¹¹ We have also estimated these models using only the sample of live births, and the results were quite similar to those presented in Table 7.

The rest of the Table shows that once again, we find little effect of pollution on the incidence of low birth weight in the aggregate data, and we also find little effect on the probability of short gestation.

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).¹² Similarly, there is a great deal of 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 substitute "cap and trade" provisions that would allow companies above the caps for sulphur dioxide, nitrogen oxides, and mercury to buy credits from other firms with pollution levels below the caps. 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

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 understand how changes from the 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 pollution on birth weight once zip code fixed effects are controlled for, and our results suggest that only NO₂ affects gestation.

We also find little effect of O₃, despite the fact that this is the most closely monitored pollutant under the Clean Air Act, and the fact that many urban areas of California are out of compliance with federal standards for O₃ (for example, the Los Angeles Basin is classified as “extreme” in its degree of noncompliance).

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 a total of 1059 infant lives. Although CO is known to cross the placenta, we found that only pollution exposure after birth significantly increased mortality. The finding that CO has an important impact on infant health is clearly relevant to policy debates over automobile emissions and the Clear Skies Initiative.

Our estimates indicate then, that reductions in pollution to levels well below

federal standards continue to have positive effects on infant health, and that CO, NO₂, and PM₁₀ may all have effects on infant health, though given the high degree of correlation between these pollutants, it is difficult to determine their exact magnitudes. Moreover, given that automobile exhaust is a major source of all three pollutants, determining which pollutant is most harmful may be less important than the finding that even currently low levels of pollution have harmful effects.

If we value a life at a very conservative \$1.6 million, then the estimated reduction of 1059 infant deaths due to reduced air pollution would be valued at \$1.7 billion.¹³ This estimate ignores other benefits of pollution reduction, such as improvements in health which are not at the life/death margin, and so is certainly a lower-bound estimate of the benefit. But it might be a useful benchmark for assessing the costs and benefits of further reductions in air pollution.

¹³ Chay and Greenstone (2001a) use this value. However, Viscusi (1993) suggested that the value of a life was between \$3.5 and \$8.5 million.

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Appendix: 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:

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

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:

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

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.

Figure 1: Ozone Monitors in California

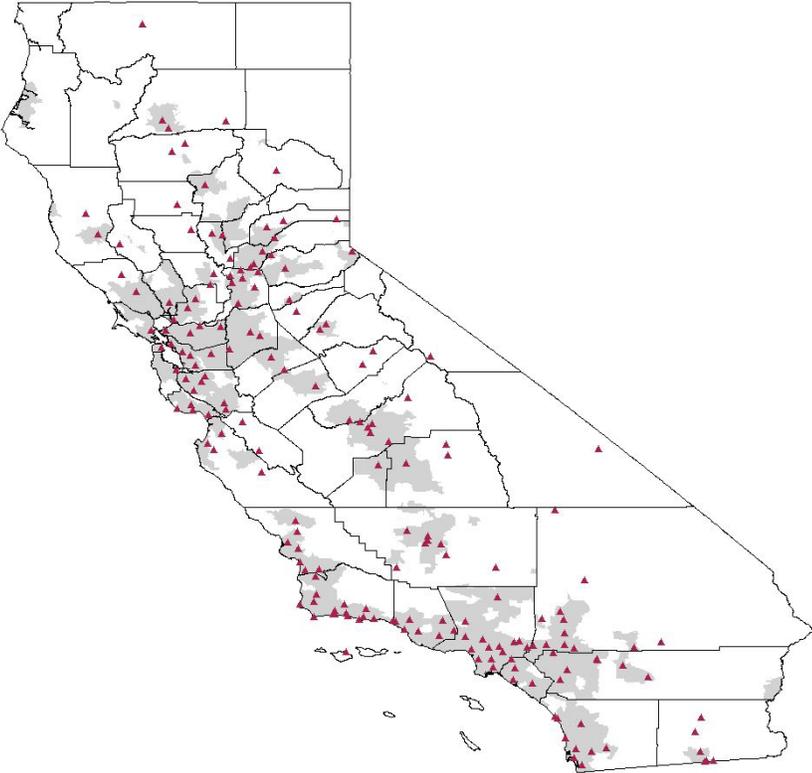


Figure 2: Ozone Monitors in Los Angeles County

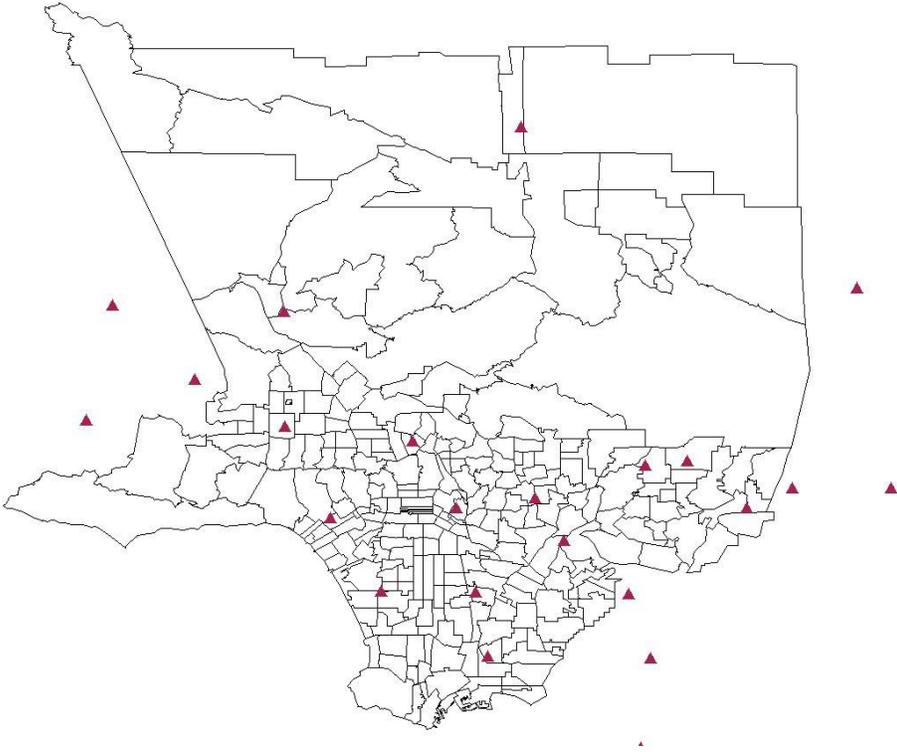


Figure 3. Seasonal Patterns in Pollution

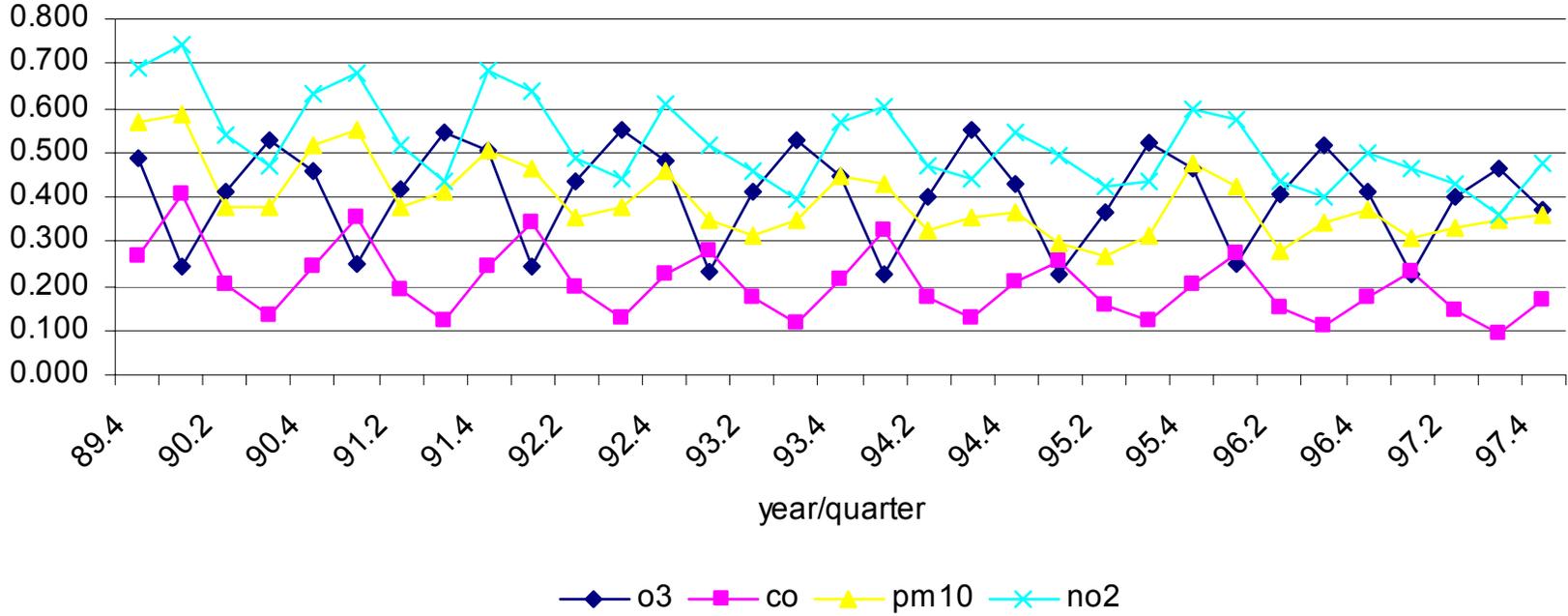


Figure 4. Residual Variation in Pollution

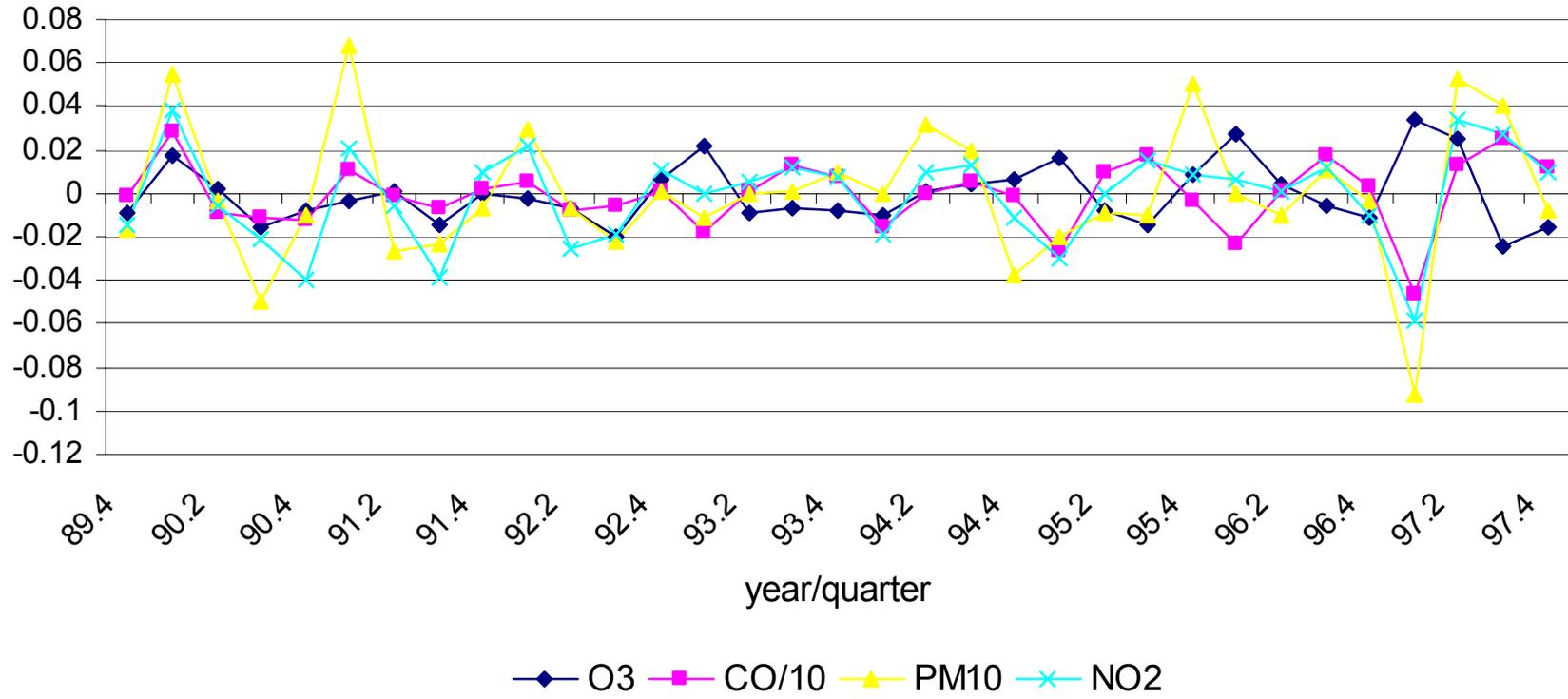


Figure 5: Seasonal Patterns in Infant Health

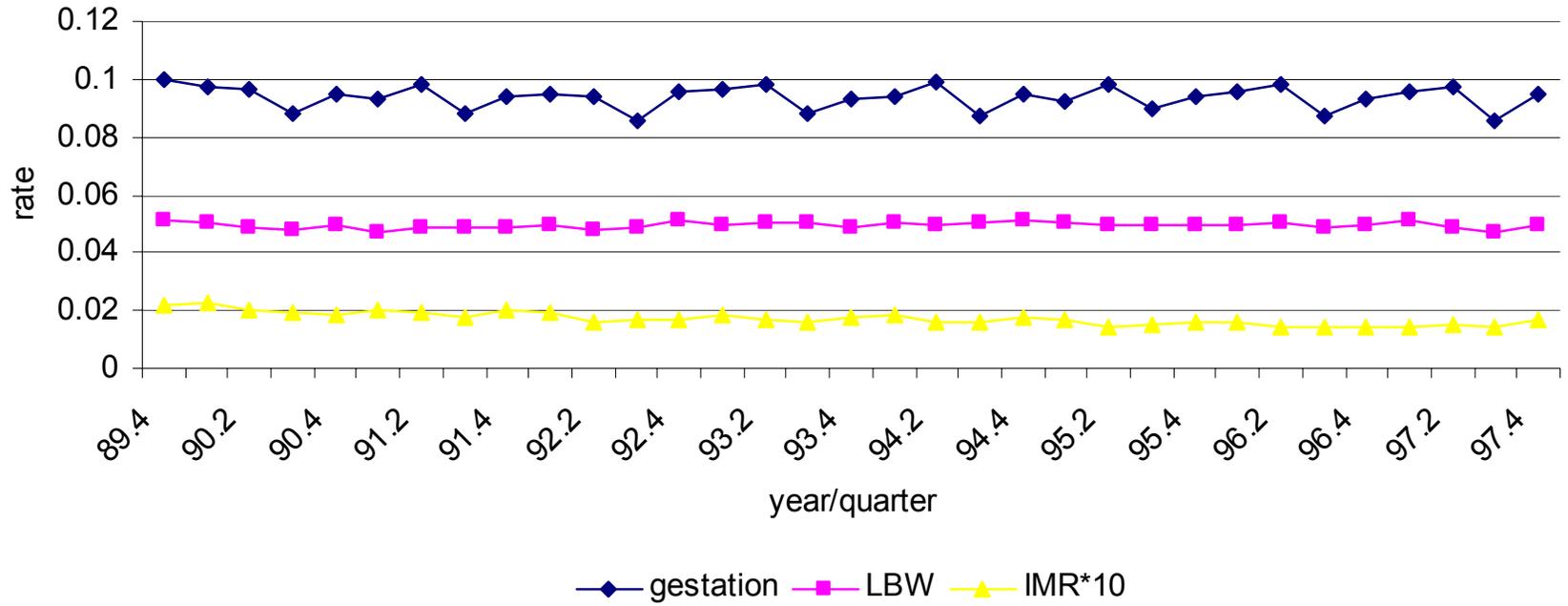


Figure 6. Predicted Outcomes from Covariates Excluding Pollution

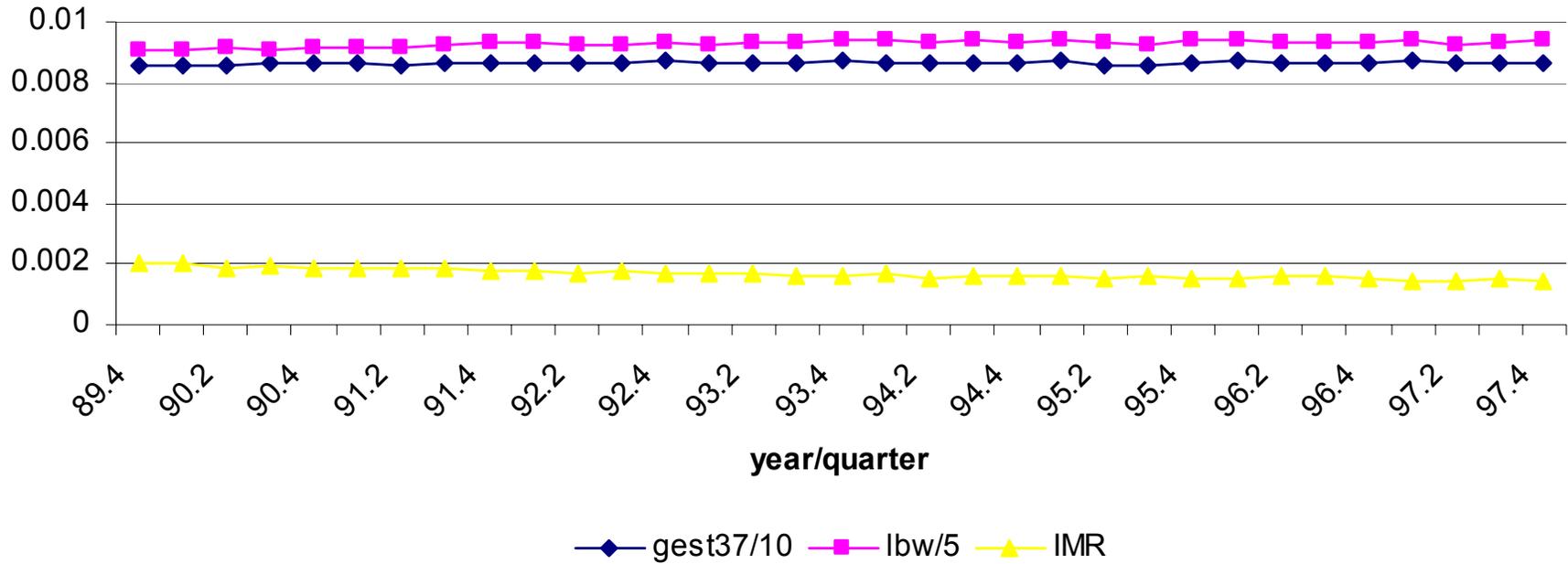


Table 1: Levels and Trends in Pollution and Infant Health

Variable	Mean	Std. Dev.	Between zip std. Dev.	Within zip std. Dev.
<u>Panel 1</u>				
CO 8-hr	2.0374	1.0956	0.7020	0.7479
PM10	0.3930	0.1438	0.1088	0.0948
NO2	0.5171	0.1857	0.1562	0.0936
O3	0.4093	0.1590	0.1028	0.1151
<u>Panel 2</u>				
infant mortality rate	0.0017	0.0020	0.0045	0.0117
gestation<37 rate	0.0925	0.0306	0.0282	0.0480
gestation<32 rate	0.0120	0.0098	0.0076	0.0187
rate of low birthweight	0.0488	0.0213	0.0188	0.0386
<u>Panel 3</u>				
year	CO	PM10	NO2	O3
89	2.654	0.566	0.688	0.488
90	2.430	0.462	0.593	0.414
91	2.242	0.459	0.576	0.434
92	2.236	0.413	0.544	0.428
93	1.936	0.365	0.483	0.409
94	2.068	0.368	0.514	0.404
95	1.823	0.338	0.487	0.398
96	1.769	0.353	0.477	0.396
97	1.580	0.337	0.431	0.368
<u>Panel 4</u>				
year	IMR	Low Birth Weight	Gestation < 37 weeks	Gestation < 32 weeks
89	0.0022	0.0507	0.0983	0.0132
90	0.0020	0.0487	0.0932	0.0123
91	0.0019	0.0479	0.0923	0.0121
92	0.0017	0.0487	0.0917	0.0118
93	0.0017	0.0489	0.0926	0.0120
94	0.0017	0.0499	0.0922	0.0121
95	0.0016	0.0488	0.0921	0.0120
96	0.0014	0.0488	0.0922	0.0117
97	0.0015	0.0485	0.0920	0.0115

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	1.2020	1.9690	2.8340
PM10	0.2534	0.3895	0.5423
NO2 1-hr	0.3136	0.5040	0.6990
O3 8-hr	0.3374	0.4123	0.4751
gestation<37 rate	0.0784	0.0886	0.0922
gestation<32 rate	0.0101	0.0113	0.0117
low BW rate	0.0433	0.0473	0.0487
infant mortality rate*	0.0016	0.0019	0.0020
% male	0.487	0.487	0.490
% black	0.064	0.073	0.076
% hispanic	0.255	0.388	0.447
% asian	0.129	0.106	0.100
% other race	0.014	0.007	0.006
% married	0.739	0.698	0.669
% foreign mom	0.323	0.413	0.461
% racial diff b/w parents	0.181	0.166	0.152
% HS grads	0.340	0.338	0.341
% AD degree	0.154	0.141	0.133
% college grads	0.284	0.232	0.193
% educ. diff b/w parents	0.375	0.374	0.369
% age 19 to 25	0.271	0.305	0.326
% age 26 to 30	0.278	0.284	0.287
% age 31 to 35	0.256	0.230	0.214
% age >= 36	0.138	0.117	0.102
% first born	0.434	0.420	0.410
% second born	0.323	0.309	0.306
% third born	0.147	0.157	0.162
% gov't insurance	0.356	0.416	0.428
% prenatal care in 1st trimester	0.821	0.798	0.768

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

	1	2	3	4	5	6	7	8	9	10	11	12
	CS	CS	CS	CS	CS	CS	FE	FE	FE	FE	FE	FE
asian	0.011	0.011	0.011	0.011	0.011	0.011	0.013	0.013	0.013	0.013	0.013	0.013
	[0.0031]**	[0.0031]**	[0.0031]**	[0.0032]**	[0.0031]**	[0.0031]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**
other race	0.002	0.002	0.002	0.002	0.002	0.002	0.006	0.006	0.006	0.006	0.006	0.006
	[0.0090]	[0.0090]	[0.0090]	[0.0090]	[0.0090]	[0.0090]	[0.0086]	[0.0086]	[0.0086]	[0.0086]	[0.0086]	[0.0086]
married mother	-0.007	-0.007	-0.007	-0.007	-0.007	-0.007	-0.008	-0.008	-0.008	-0.008	-0.008	-0.008
	[0.0048]	[0.0048]	[0.0048]	[0.0048]	[0.0048]	[0.0048]	[0.0049]	[0.0049]	[0.0049]	[0.0049]	[0.0049]	[0.0049]
foreign born mother	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026	-0.026
	[0.0023]**	[0.0023]**	[0.0023]**	[0.0023]**	[0.0023]**	[0.0023]**	[0.0021]**	[0.0021]**	[0.0021]**	[0.0021]**	[0.0021]**	[0.0021]**
parents diff race	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.015	0.015
	[0.0023]**	[0.0023]**	[0.0023]**	[0.0023]**	[0.0023]**	[0.0023]**	[0.0021]**	[0.0021]**	[0.0021]**	[0.0021]**	[0.0021]**	[0.0021]**
HS grad mother	-0.011	-0.011	-0.011	-0.011	-0.011	-0.011	-0.013	-0.012	-0.012	-0.012	-0.013	-0.013
	[0.0022]**	[0.0022]**	[0.0022]**	[0.0022]**	[0.0022]**	[0.0022]**	[0.0020]**	[0.0020]**	[0.0020]**	[0.0020]**	[0.0020]**	[0.0020]**
AD degree	-0.019	-0.019	-0.019	-0.019	-0.019	-0.019	-0.021	-0.021	-0.021	-0.021	-0.021	-0.021
	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**
college grad	-0.025	-0.025	-0.025	-0.025	-0.025	-0.025	-0.029	-0.029	-0.029	-0.029	-0.029	-0.029
	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0031]**	[0.0031]**	[0.0031]**	[0.0031]**	[0.0031]**	[0.0031]**
educ diff parents	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002
	[0.0015]	[0.0015]	[0.0015]	[0.0015]	[0.0015]	[0.0015]	[0.0016]	[0.0016]	[0.0016]	[0.0016]	[0.0016]	[0.0016]
19-25 mother	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.017	-0.017	-0.017	-0.017	-0.017	-0.017
	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0032]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**
26-30 mother	-0.027	-0.027	-0.027	-0.027	-0.028	-0.027	-0.029	-0.029	-0.029	-0.029	-0.029	-0.029
	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0033]**	[0.0033]**	[0.0033]**	[0.0033]**	[0.0033]**	[0.0033]**
31-35 mother	-0.023	-0.023	-0.023	-0.023	-0.023	-0.023	-0.025	-0.025	-0.025	-0.025	-0.025	-0.025
	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0035]**	[0.0036]**	[0.0036]**	[0.0036]**	[0.0036]**	[0.0036]**	[0.0036]**
mother >=36	-0.014	-0.014	-0.014	-0.014	-0.014	-0.014	-0.015	-0.015	-0.015	-0.015	-0.015	-0.015
	[0.0043]**	[0.0043]**	[0.0043]**	[0.0043]**	[0.0043]**	[0.0043]**	[0.0041]**	[0.0041]**	[0.0041]**	[0.0041]**	[0.0041]**	[0.0041]**
first born	-0.030	-0.030	-0.030	-0.030	-0.030	-0.030	-0.032	-0.032	-0.032	-0.032	-0.032	-0.032
	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**	[0.0026]**
second born	-0.020	-0.020	-0.020	-0.020	-0.020	-0.020	-0.021	-0.021	-0.021	-0.021	-0.021	-0.021
	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0029]**	[0.0025]**	[0.0025]**	[0.0025]**	[0.0025]**	[0.0025]**	[0.0025]**
third born	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.017	-0.017	-0.017	-0.017	-0.017	-0.017
	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0030]**	[0.0027]**	[0.0027]**	[0.0027]**	[0.0027]**	[0.0027]**	[0.0027]**
gov't insurance for birth	0.015	0.015	0.015	0.015	0.015	0.015	0.018	0.018	0.018	0.018	0.018	0.018
	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**
prenatal care 1st trimester	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016	-0.016
	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0019]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**	[0.0018]**
Constant	0.955	0.960	0.958	0.966	0.967	0.967	0.944	0.952	0.948	0.956	0.944	0.946
	[0.0131]**	[0.0128]**	[0.0128]**	[0.0145]**	[0.0148]**	[0.0149]**	[0.0160]**	[0.0151]**	[0.0158]**	[0.0154]**	[0.0176]**	[0.0172]**
Observations	145869	145869	145869	145869	145869	145869	145869	145869	145869	145869	145869	145869
R-squared	0.410	0.410	0.410	0.410	0.410	0.410	0.410	0.410	0.410	0.410	0.410	0.410
Number of zip							884	884	884	884	884	884

Notes: Standard errors in brackets. * indicates significance at the 95% level, ** at the 99% level. All regressions also included year and quarter dummies. Coefficients and standard errors on pollutants 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-squares of approximately 0.01.

Table 4: Pollution and Probability of Infant Death - Alternative Specifications

	1	2	3	4	5	6
1. Include Pollution in First Trimester vs. Last Trimester						
CO after birth	2.677 [0.7003]**				3.866 [1.3337]**	2.682 [0.9033]**
PM10 after birth		8.100 [4.3123]			2.215 [5.3820]	0.247 [5.1493]
NO2 after birth			11.128 [4.6274]*		-10.141 [8.4642]	
Ozone after birth				-8.726 [6.5862]	4.173 [7.8491]	1.066 [7.4851]
CO first trimester	0.121 [1.1930]				1.070 [1.9123]	0.192 [1.4423]
PM10 first trimester		-5.284 [8.5463]			-2.253 [10.8159]	-4.002 [10.3663]
NO2 first trimester			-6.156 [9.7509]		-10.864 [15.4198]	
Ozone first trimester				-9.394 [9.9558]	-3.939 [12.2374]	-6.700 [11.7966]
# Observations	145499	144657	145684	145747	144361	144409
R-squared	0.42	0.42	0.42	0.42	0.42	0.42
# fixed effects	883	878	883	883	878	878
2. Add Birthweight and Gestation as Controls for Prenatal Exposure						
CO after birth	3.499 [0.6596]**				3.4904 [1.2499]**	2.9913 [0.8487]**
PM10 after birth		11.7969 [4.0333]**			4.0314 [5.0337]	3.2726 [4.8134]
NO2 after birth			16.7115 [4.3618]**		-4.2801 [7.9465]	
Ozone after birth				-16.6198 [6.2082]**	-5.0307 [7.3794]	-6.24 [7.0427]
CO last trimester	-1.5951 [1.0505]				-1.0363 [1.8053]	-1.2162 [1.3364]
PM10 last trimester		-2.101 [8.0653]			0.87 [10.2183]	0.4497 [9.7670]
NO2 last trimester			-8.3673 [8.4236]		-2.1277 [13.9701]	
Ozone last trimester				14.5674 [9.4760]	10.5688 [11.2821]	10.1328 [10.9272]
Birthweight	-0.1279 [0.0011]**	-0.1279 [0.0011]**	-0.1279 [0.0011]**	-0.1279 [0.0011]**	-0.1279 [0.0011]**	-0.1279 [0.0011]**
Gestation	-0.3693 [0.0207]**	-0.3688 [0.0207]**	-0.3696 [0.0207]**	-0.369 [0.0207]**	-0.3694 [0.0207]**	-0.3693 [0.0207]**
# Observations	145813	145813	145813	145813	145813	145813
R-squared	0.48	0.48	0.48	0.48	0.48	0.48
# fixed effects	883	883	883	883	883	883

Notes: All regressions include zip-code fixed effects and are of the same form as those in Table 3. See Table 3 notes. Coefficients and standard errors on birthweight and gestation have also been multiplied by 1,000.

Table 5: Mortality and the Timing of Pollution Exposure

	1	2	3	4	5	6
1. Include Lags of Weekly Pollution Levels						
CO after birth	1.4387 [1.3230]				3.1683 [2.1209]	1.6442 [1.4698]
CO after birth, 1 lag	2.9223 [1.7130]				-0.1592 [2.5665]	1.5386 [1.8285]
CO after birth, 2 lags	0.0893 [1.4096]				0.6288 [2.1192]	0.1947 [1.5058]
PM10 after birth		4.7778 [4.8283]			-0.2593 [5.7039]	-1.4405 [5.5347]
PM10 after birth, 1 lag		17.1978 [5.3340]**			12.0075 [6.0918]*	13.1097 [5.9385]*
PM10 after birth, 2 lags		1.6563 [4.8872]			0.5173 [5.7003]	-0.0456 [5.5312]
NO2 after birth			4.1626 [6.6115]		-10.6272 [11.2143]	
NO2 after birth, 1 lag			20.7079 [7.8700]**		12.1302 [12.7497]	
NO2 after birth, 2 lags			0.4311 [6.9714]		-4.0191 [11.1747]	
Ozone after birth				-11.8697 [9.7138]	1.0691 [10.7928]	-2.071 [10.2125]
Ozone after birth, 1 lag				3.0282 [11.1285]	-2.4453 [12.6645]	2.0357 [11.8658]
Ozone after birth, 2 lags				-11.5678 [9.4739]	-5.3388 [10.8644]	-7.401 [10.1847]
CO last trimester	-1.3014 [1.0672]				-0.5143 [1.8355]	-0.7898 [1.3598]
PM10 last trimester		1.0266 [8.2003]			2.4044 [10.4323]	1.7651 [9.9660]
NO2 last trimester			-6.412 [8.5429]		-3.1635 [14.2084]	
Ozone last trimester				19.1707 [9.5922]*	15.8848 [11.4177]	15.2771 [11.0559]
2. Include Leads of Weekly Pollution Levels						
CO after birth	3.7007 [1.1515]**				2.4596 [2.0149]	3.3086 [1.3163]*
CO after birth, one lead	0.1448 [1.1531]				2.2426 [2.0189]	0.3195 [1.3005]
PM10 after birth		11.1786 [4.5797]*			3.9685 [5.6194]	4.0081 [5.4585]
PM10 after birth, one lead		3.013 [4.6358]			-0.5884 [5.6480]	-2.5949 [5.4668]
NO2 after birth			18.4681 [5.6024]**		4.1081 [10.7384]	
NO2 after birth, one lead			-0.7187 [5.5971]		-13.9391 [10.7374]	
Ozone after birth				-8.4663 [8.9752]	-9.4781 [10.6604]	-7.1099 [9.7807]
Ozone after birth, one lead				-10.5294 [8.3081]	8.631 [10.1298]	3.7755 [9.1986]
CO last trimester	-1.2218 [1.0655]				-0.348 [1.8314]	-0.8284 [1.3559]
PM10 last trimester		1.1927 [8.1841]			3.2733 [10.3869]	2.1415 [9.9316]
NO2 last trimester			-6.5675 [8.5304]		-5.555 [14.1604]	
Ozone last trimester				17.3442 [9.5733]	14.7535 [11.3922]	13.59 [11.0278]

Notes: These regressions include zip-code fixed effects and are of the same form as those in Table 3 except that they also include birthweight and gestation. See Table 4 notes.

Table 6: Monthly Hazard for Mortality

	1	2	3	4	5	6
CO after birth	4.9582 [0.7956]**				2.6643 [1.4903]	3.8915 [1.0972]**
PM10 after birth		28.8948 [5.9520]**			9.6306 [7.9202]	12.8686 [7.4110]
NO2 after birth			34.1386 [5.7579]**		12.4437 [10.3270]	
Ozone after birth				-19.3366 [7.6454]*	-5.9762 [9.2088]	-2.9919 [8.8822]
CO last trimester	-1.9206 [1.0798]				-1.7348 [1.8582]	-1.4829 [1.3749]
PM10 last trimester		0.544 [8.3316]			3.1762 [10.6706]	3.7604 [10.1216]
NO2 last trimester			-6.25 [8.5537]		3.0175 [14.3933]	
Ozone last trimester				22.5104 [9.7457]*	15.2813 [11.5796]	15.966 [11.2191]
Birthweight	-0.1561 [0.0011]**	-0.1561 [0.0011]**	-0.1561 [0.0011]**	-0.1561 [0.0011]**	-0.1561 [0.0011]**	-0.156 [0.0011]**
Gestation	-0.5516 [0.0210]**	-0.5505 [0.0210]**	-0.5512 [0.0210]**	-0.5517 [0.0210]**	-0.5517 [0.0210]**	-0.5518 [0.0210]**
# Observations	145907	145907	145907	145907	145907	145907
R-squared	0.46	0.46	0.46	0.46	0.46	0.46
# fixed effects	880	880	880	880	880	880

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

	1	2	3	4	5	6	7	8	9	10	11	12
	CS	CS	CS	CS	CS	CS	FE	FE	FE	FE	FE	FE
1. Low Birth Weight												
CO last trimester	0.6751*				0.048	0.303	0.191				0.326	0.467
	[0.3154]				[0.5781]	[0.3747]	[0.4052]				[0.6638]	[0.5025]
PM10 last trimester		8.6131**			6.4623*	6.9268*		0.406			-2.221	-1.878
		[2.4186]			[3.1950]	[3.0313]		[3.0181]			[3.7779]	[3.6260]
NO2 last trimester			5.1721**		1.853				2.308		1.639	
			[1.8304]		[3.4541]				[3.1977]		[5.0686]	
Ozone last trimester				4.914	1.758	2.316				2.721	4.062	4.430
				[2.5804]	[3.2173]	[3.1273]				[3.3908]	[4.0765]	[3.9145]
Observations	785755	785755	785755	785755	785755	785755	785755	785755	785755	785755	785755	785755
R-squared	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Number of zip							892	892	892	892	892	892
2. Gestation Less than 32 Weeks												
CO last trimester	0.243				-0.327	-0.046	0.4257*				-0.8902**	0.393
	[0.1805]				[0.2959]	[0.1923]	[0.2060]				[0.3377]	[0.2556]
PM10 last trimester		4.8678**			4.3698**	4.8824**		3.5993*			-1.140	1.990
		[1.2287]			[1.5992]	[1.5081]		[1.5372]			[1.9250]	[1.8483]
NO2 last trimester			2.5579**		2.044				9.3596**		14.9868**	
			[0.9561]		[1.6765]				[1.6254]		[2.5775]	
Ozone last trimester				2.7846*	-0.142	0.472				1.455	-1.322	2.039
				[1.3919]	[1.7647]	[1.7107]				[1.7251]	[2.0730]	[1.9908]
Observations	776726	776726	776726	776726	776726	776726	776726	776726	776726	776726	776726	776726
R-squared	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003	0.003
Number of zip							816	816	816	816	816	816
3. Gestation Less than 37 Weeks												
CO last trimester	1.4691**				-0.1291	0.6266	0.6827				-0.3055	1.1107
	[0.4994]				[0.9253]	[0.5884]	[0.5427]				[0.8889]	[0.6729]
PM10 last trimester		23.8196**			16.8066**	18.1822**		6.3668			-2.813	0.6467
		[3.2001]			[4.3646]	[4.1893]		[4.0410]			[5.0579]	[4.8548]
NO2 last trimester			13.2961**		5.4915				13.5548**		16.5485*	
			[2.7097]		[5.3091]				[4.2820]		[6.7872]	
Ozone last trimester				19.2177**	10.4232*	12.0769*				9.1959*	8.324	12.0388*
				[4.2034]	[5.1605]	[4.8598]				[4.5408]	[5.4592]	[5.2423]
Observations	786300	786300	786300	786300	786300	786300	786300	786300	786300	786300	786300	786300
R-squared	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
Number of zip							913	913	913	913	913	913

Table 8: Estimates Using Data Aggregated to Quarterly Level

	1	2	3	4	5	6
1. Infant Mortality						
CO, quarter of death	0.0532* [0.0291]				0.004 [0.0373]	0.033 [0.0318]
PM10, quarter of death		0.4776*** [0.1787]			0.347 [0.2162]	0.4223** [0.2103]
NO2, quarter of death			0.6346*** [0.2283]		0.476 [0.3149]	
Ozone, quarter of death				0.130 [0.1846]	-0.152 [0.2300]	-0.010 [0.2099]
# Observations	31204	31204	31204	31204	31204	31204
R-squared	0.09	0.09	0.09	0.09	0.09	0.09
2. Low Birthweight						
CO, quarter of birth	0.403 [0.2874]				0.8184** [0.3705]	0.6979** [0.3160]
PM10, quarter of birth		-1.884 [1.7670]			-4.1147* [2.1336]	-4.4113** [2.0798]
NO2, quarter of birth			0.459 [2.2756]		-1.936 [3.1070]	
Ozone, quarter of birth				0.932 [1.8472]	3.9288* [2.2763]	3.379 [2.0980]
# Observations	27686	27686	27686	27686	27686	27686
R-squared	0.27	0.27	0.27	0.27	0.27	0.27
3. Gestation < 37 weeks						
CO, quarter of birth	0.067 [0.3890]				0.036 [0.5015]	0.071 [0.4278]
PM10, quarter of birth		1.715 [2.3917]			0.778 [2.8882]	0.864 [2.8153]
NO2, quarter of birth			1.965 [3.0800]		0.561 [4.2058]	
Ozone, quarter of birth				2.239 [2.5002]	1.802 [3.0814]	1.961 [2.8400]
# Observations	27686	27686	27686	27686	27686	27686
R-squared	0.35	0.35	0.35	0.35	0.35	0.35
4. Gestation < 32 weeks						
CO, quarter of birth	0.124 [0.1421]				0.216 [0.1832]	0.143 [0.1563]
PM10, quarter of birth		-0.298 [0.8739]			-0.305 [1.0552]	-0.484 [1.0286]
NO2, quarter of birth			-0.455 [1.1253]		-1.172 [1.5366]	
Ozone, quarter of birth				-0.543 [0.9135]	0.123 [1.1258]	-0.210 [1.0376]
# Observations	27686	27686	27686	27686	27686	27686
R-squared	0.15	0.15	0.15	0.15	0.15	0.15

Notes: Specifications are similar to those in Tables 3 and 6, with all data aggregated to quarterly level. All models have zipcode fixed effects.

Appendix Table 1: Alternative Specifications for Infant Mortality Models

1. Logit Model Similar to Specification in Table 3

CO after birth	48.034				53.978	43.033
	[9.9640]**				[18.5771]**	[12.9146]**
PM10 after birth		149.504			52.758	32.329
		[59.8123]*			[74.8756]	[71.8823]
NO2 after birth			223.621		-107.100	
			[67.1547]**		[120.3778]	
Ozone after birth				-237.129	-58.485	-85.619
				[94.9856]*	[113.3059]	[108.8719]
CO last trimester	-13.783				-10.926	-12.159
	[16.3752]				[27.5402]	[20.3310]
PM10 last trimester		31.016			61.722	51.389
		[122.5290]			[154.8436]	[148.4846]
NO2 last trimester			-38.865		-28.850	
			[129.8099]		[212.5154]	
Ozone last trimester				167.289	107.799	103.579
				[142.7516]	[170.4416]	[165.7376]
Observations	145810	145810	145810	145810	145810	145810
# zipcode fixed effects	880	880	880	880	880	880

2. 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	3.892	4.089	3.517	2.664	2.941	2.620
	[1.0972]**	[1.2148]**	[1.0426]**	[1.4903]	[1.6550]	[1.3425]
PM10 after birth	12.869	14.073	16.323	9.631	10.936	13.281
	[7.4110]	[8.5393]	[7.8050]*	[7.9202]	[9.0876]	[8.3034]
NO2 after birth				12.444	11.868	10.678
				[10.3270]	[11.6217]	[10.0144]
Ozone after birth	-2.992	-3.925	-7.015	-5.976	-6.719	-8.872
	[8.8822]	[10.3646]	[9.1654]	[9.2088]	[10.7221]	[9.3345]
CO last trimester	-1.483	-0.666	-0.373	-1.735	-0.577	-0.142
	[1.3749]	[1.5283]	[1.2933]	[1.8582]	[2.0801]	[1.6458]
PM10 last trimester	3.760	2.423	-0.513	3.176	2.768	0.315
	[10.1216]	[11.6982]	[10.5921]	[10.6706]	[12.2956]	[11.0714]
NO2 last trimester				3.018	-0.936	-2.905
				[14.3933]	[16.4431]	[13.6605]
Ozone last trimester	15.966	24.270	21.463	15.281	24.396	21.932
	[11.2191]	[13.1700]	[11.4642]	[11.5796]	[13.5609]	[11.6761]
Observations	145907	112577	112577	145907	112577	112577
# zipcode fixed effects	880	607	607	880	607	607
R-squared	0.46	0.46	0.46	0.46	0.46	0.46

Notes: Models with zipcode*week interactions are based on a smaller sample because zipcode-weeks with no deaths are excluded. All of these models include birthweight and gestation and so are most comparable to panel 2 of Table 4.