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Air Pollution and Infant Health:

What Can We Learn From California

Abstract

We examine the impact of air pollution on infant death in California over the 1990s. Our work offers several innovations: First, many previous studies examine populations subject to far greater levels of pollution. In contrast, the experience of California in the 1990s is clearly relevant to current debates over the regulation of pollution. Second, many studies examine a few routinely monitored pollutants in isolation, generally because of data limitations. We examine four "criteria" 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 of pollution on infant mortality, fetal deaths, low birth weight and prematurity in a common framework. We find that the reductions in carbon monoxide (CO) and particulates (PM10) over the 1990s in California saved over 1,000 infant 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). New standards for ozone and particulates were proposed by the Environmental Protection Agency (EPA) in 1997, but were held up in the courts until a Supreme Court decision in 2001 (Stafford, 2001).

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

This paper addresses this issue by examining the impact of air pollution on infant 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

Our work offers several innovations over the existing literature. First, many previous studies examine populations subject to greater levels of pollution, either because they lived

[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

of the effects on low birth weight and prematurity take the possibility of fetal selection into account.

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 in California. However, in contrast to much of the epidemiological literature, 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. Finally, we show that the estimated per unit effects of pollution are similar for blacks and whites and for children of more and less educated people, and that between-zip code differences in pollution levels account for relatively little of observed between-group differences in infant mortality rates.

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

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). Carbon Monoxide is an odorless, colorless gas which is poisonous at high levels. CO bonds with hemoglobin more easily than oxygen, so that it reduces the body's ability to deliver oxygen to organs and tissues. Because infants are small, and many have respiratory problems in any case, CO may be particularly harmful to them. Nitrogen Dioxide is a brown, reactive gas that irritates the lungs

and may lower resistance to respiratory infections. It is also an important precursor to particulate matter in California.

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). The mechanism through which particles harm health are controversial, however the leading theory is that they cause an inflammatory response which weakens the immune system (Seaton, et al. 1995). In infants, a weakened immune system could make them more susceptible to death from a wide range of causes. PM10 exposure could also affect the health of the mother, for example, by weakening her immune system, and hence affect the fetus.

Ozone (the major component of smog) is a highly reactive compound that damages tissue, reduces lung function, and sensitizes the lungs to other irritants. For example, exposure to O3 during exercise reduces lung functioning in adults, and causes symptoms such as chest pain, coughing, and pulmonary congestion. Ozone is formed through reactions between nitrogen oxides (such as NO2) and volatile organic compounds (which are found in auto emissions, among other sources) in heat and sunlight. Interestingly, ozone is not generally found in homes because it is highly reactive, and quickly reacts with household surfaces (http://www.hc-sc.gc.ca/hecs-sesc/air_quality/faq.htm).

Compliance with standards for NO2 and PM10 is assessed by looking at annual means (there is a 24-hour standard for PM10 as well). Compliance with standards for O3 and CO is assessed by examining whether the level of pollution exceeded the standard over any eight-hour period during the year. These different approaches to standards suggest that the effects of NO2 and PM10 may be expected to be cumulative while the effects of CO and O3 are expected to be

more acute. We do not examine the two other criteria pollutants, SO2 and lead, because levels are now so low that many monitors have been removed from service.

A link between air pollution and infant health has long been suspected, although the exact biological mechanisms through which it occurs are not known. We also know little about what levels of these pollutants are sufficient to affect infant mortality (death in the first year of life) or about the extent that infants are protected from the negative effects of pollution while they are in the womb.

Only some 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, the pl;(ined the negativu 423rom)4.2423 317.2796 Tm(a)Tj12 0 1p.(d Longo, 19787aiwn7

Many studies have demonstrated links between very severe pollution episodes and increased mortality of infants and others. For example, Logan and Glasg (1953) found dramatic increases in cardiopulmonary mortality during a killer fog that occurred in London England in 1952. Chart 1 summarizes some of the more recent studies, dividing them into two groups. The first group focuses on the link between poor infant outcomes and pollution in areas with high levels of pollution; most report negative associations between pollution and infant outcomes.4

The second part of the chart focuses on U.S. studies, many of which also report a link between air pollution and infant health, although some do not. For example, Lipfert, Zhang, and Wyzga (2000) find that while they can replicate previous findings of a negative effect of PM10 on infant health, the result is not robust to changes in specification.

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

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

Two studies by Chay and Greenstone deal with the problem of omitted confounders by focusing on “natural experiments” provided by the implementation of the Clean Air Act of 1970, and geographic variation in pollution levels induced by the recession of the early 1980s. These studies are similar in spirit to a sequence of papers by C. Arden Pope, who investigated the health effects of the temporary closing of a Utah steel mill (Pope, 1989; Ransom and Pope, 1992; Pope, Schwartz, and Ransom, 1992) and to Friedman et al. (2001) who examine the effect of changes in traffic patterns in Atlanta due to the 1996 Olympic games. However, these studies did not look specifically at infants.

Chay and Greenstone show that 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 live births but had little effect on the incidence of low birth weight.5

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 oday would oday would o7 Tw B. com

In the current paper, we propose an alternative identification strategy using individual-level data and exploiting within-zip code variation in pollution levels. This strategy enables us to create finer measures of pollution and to control for individual differences between mothers that may be associated with birth outcomes. 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 criteria 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. Actual personal exposures are affected by ambient air quality, indoor air quality, and the time the individual spends indoors and outdoors. One might expect, for example, that infants spend little time outdoors, so that outdoor air quality might not be relevant.

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

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

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

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. Moreover, NO2 is a major precursor of PM10 in California, which suggests that these two pollutants may be particularly highly correlated. We will examine the sensitivity to our estimates to excluding NO2 below. 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.

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. Residuals are normalized by mean pollution levels so that they are expressed in percentage terms. (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.) It is very important to establish that there is significant within zip code variation, since mean differences in the level of pollutants between zip codes are not used to identify the effects of pollution in the zip code fixed effects models.

Data on birth weight, gestational age,(yDay)]ythaTiwanTs7wre emi the Surface S09 Tc -0.0005 Tw 20.5

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 this outcome. Note, that there is no birth cohort file for 1998, so this year is excluded from our analysis.

The distinction between fetal and infant death is that a child must be born alive in order to be registered as an infant death. In California, a live birth is defined as "the complete expulsion or extraction from its mother of a product of conception...which, after such separation, breathes or shows any other evidence of life...", while a fetal death is a "death prior to complete expulsion or extraction from its mother of a product of conception" (California Code of Regulations, Title 17, sections 915 and 916).

Hence, a premature delivery that ended in a child dying before birth would be classified

less than one year old; hence to compare to published figures one would multiply this numb[redacted] four. The estimates indicate that over the sample period, about 6.56 children per 1,000 died i[redacted] their first year. Table 1 shows that 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 of maternal residence, as well as information about use of prenatal care a[redacted] whether the birth was covered by public health insurance. The rapid increase in the fraction of[redacted] births covered by Medicaid is a potential confounding factor when examining birth ou[redacted] because there is evidence that Medicaid coverage changed the way that at risk infants were treated (c.f. Currie and Gruber, 1996), so it is fortunate that we can control for M[redacted] 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.

The third panel of Table 1 shows trends in pollution levels over the sample period. All four pollutants show considerable declines. The fourth panel of Table 1 shows that althoug[redacted] infant mortality rate fell sharply over a relatively short time, trends in low birth weight and gestation were much flatter. This part of the table suggests then, that declines in mortality were largely due to events occurring after the birth, rather than to improvements in pr[redacted] Finally, the last 12cntr6yificate2i.roeow cTm(rndard so ie2i.rp(four polte or when exam)Tj12 8 0 7 120 12 72 17

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 them

reflect the fact that death is much more common in the first weeks than thereafter. The w_{iz} are defined as above; the h_{iz} 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_{zt1} are time-varying measures of pollution exposure, the x_{zt2} are weather indicators; and the φ_z, Γ_t, and Y_t are defined as in (1). In this model, any effect of prenatal exposures is assumed to be captured via the effects on birth weight and gestation, which are controlled. The main coefficient of interest is β_1, the effect of post-natal pollution exposure on the probability of death.

This model can be thought of as a flexible, discrete-time, hazard model that allows for time-varying covariates, non-parametric dur92 Tmt

individuals who died (the cases) in the week that they died. Then, in order to select controls, we choose randomly among all the observations on children who lived for at least as many periods as the index child, and who were in the same zip code, and we take the control child's observation for that week. That is, if a child died in week 3, the controls would be chosen from observations on all children who lived at least 3 weeks regardless of whether they later died. For each zip code-week, we randomly chose five times as many non-deaths as deaths (we show below that results using 15 times as many non-deaths are very similar). This method greatly reduces computational burden while yielding unbiased estim

time unit, children who die in their first week of life are incorrectly assigned average pollution levels for all of the days in the month.

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 every six days, 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 will compare estimates from models using weeks to estimates from models using months as the time unit, and we also try augmenting our weekly model by including average cumulative weekly exposures.

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 average precipitation in the vector x_{zt2}. 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 (Samet et al., 1997). However, as shown below, we find that the exclusion of the weather variables has little effect on our estimates.

Manski and Lerman, 1977; Imbens, 1992).

V. Results

a) Effects of Prenatal Exposures

Table 3 shows estimates of model (1), where the dependent variable is the probability of short gestation. 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. The "single pollutant" models without zip code fixed effects shown in columns (1) through (4), indicate that exposure to PM10, NO2, and Ozone in the month before birth all increase the probability of short gestation. Column (5) shows that if we include all four pollutants, only PM10 and Ozone have significant effects. The estimated effects are slightly larger if NO2 is excluded from the model, as shown in column (6).

However, as discussed above, the pollution measures may be capturing other characteristics of zip codes. Columns (7) through (12) of Table 3 show the same models estimated using zip code fixed effects. The estimated effects of pollution on the probability of short gestation disappear, suggesting that it is very important to control for omitted variables.

Table 3 also displays the other covariates included in our models. These variables have largely the expected signs and are not much affected by the inclusion of the fixed effects. For example, infants born to black, unmarried, less educated mothers are more likely to suffer from short gestation than other infants, as are infants of high parity.

The first two panels of Table 4 present estimates from similar models of low birth weight, and the probability of fetal death. Only the estimated effects of pollution are shown; the coefficients on the other covariates are supressed in order to save space. Even in the cross section, we find little consistent evidence that pollution affects these outcomes once the

b) Effects of Pollution on Infant Mortality

Table 6 shows estimates of model (2). A comparison of the cross-sectional and fixed effects estimates shows that the estimates are quite robust to the inclusion of zip code fixed effects, in contrast to the models of prenatal exposures discussed above. The single-pollutant models suggest that CO, PM10, and NO2 exposures all increase the probability of death, while ozone has a counter-intuitive negative effect. However, when all four pollutants (or when CO, PM10, and ozone are included) only CO has a significant effect.

The figures in bold are the implied number of deaths associated with a one unit increase in the pollutant in question, per 100,000 births. Given the 4,720,190 births in areas where pollution could be assigned over our sample period, the estimate of 13.864 in the last column of Table 6 suggests that the one unit decline in CO that took place over the sample period saved 654 infant lives. (Note that we do not consider possible lives saved in areas without pollution monitors. If these areas did not have monitors because they had little pollution and/or were sparely populated, then reductions in pollution could be expected to have relatively little effect).

Table 7 shows that the estimated effect of CO is extremely robust to many changes in specification. Except for columns 5 and 6, all of the models included zip code fixed effects. The coefficients on the other covariates are excluded from the table in order to conserve space.

Columns 1 and 2 of Table 7 use a sample in which 15 times as many non-deaths as deaths were chosen (rather than 5 tim

it is not our preferred specification, because some zip code-year cells have no deaths. Nevertheless, it is reassuring to find that the inclusion of these additional fixed effects has little effect on the estimates.

Columns 7 and 8 show estimates from models that exclude deaths (and controls) from the first week of life. Our rationale for this specification check is that infants who are very sick may never leave the hospital, and the quality of the air they are exposed to may be strictly controlled (if they are in an incubator, for example). Once again, this change has relatively little effect on our estimates.

Columns 9 and 10 show estimates from models that use data from monitors within a 10 mile radius of each zip code centroid. The sample size is smaller, given that those who live

Table 8 shows estimates from a model intended to get at possible cumulative effects of pollution in an alternative way. In addition to the weekly measures of pollution exposure, the models shown in Table 8 also include the average weekly exposure over the child's life. This modification has little effect on the estimated coefficient on CO. However, in column 5, the cumulative effect of NO2 is large and statistically significant, while column 6 shows that the cumulative effect of PM10 is significant when NO2 is omitted. Thus, the last two rows of Table 7 and Table 8 suggest that PM10 (and possibly its precursor NO2) have cumulative effects on infant health which increase the probability of mortality. Still, the estimated effect of PM10 in Table 8 is considerably smaller than in Table 7, suggesting that exposure in the last few weeks may matter more than cumulative exposure over the infant's entire lifetime.

We have also investigated possible non-linearities in the effects of pollution on infant mortality, as well as the possibility that pollution has different effects on different groups by estimating models similar to those shown in Table 5. None of the interaction terms in these models were statistically significant. Hence, we find little evidence of a non-linear effect of pollution on infant mortality (at least over the range of pollution measured in our data), and conclude that the same exposure to pollution will have the same effect on mortality risk regardless of race or maternal education.

The estimates in Table 2 suggested that there were small differences in the average pollution levels experienced by children of mothers with and without a high school education. We calculate that the mean difference of .229 units in CO exposure could be responsible for an extra 51.82 deaths in the less educated group relative to the more educated group over the sample period, while the 3.077 unit difference in PM10 exposure could have accounted for 27.82

extra deaths in the less educated group. Still, this is a relatively small fraction of the overall difference (4,885) in the number of deaths between the two groups.

To summarize, 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 specification, and implies that reduction in CO over our sample period saved approximately 654 lives in California. The coefficient on PM10 is more sensitive to specification, but the models using monthly measures suggest that the decline in PM10 saved 415 lives over the same period.

The estimated effect of PM10 is smaller than the Chay and Greenstone estimates of the effects of TSPs. The single pollutant model for PM10 using the monthly measure (which is not shown) implies that each one unit reduction led to a decline of 1.17 deaths per 100,000. However, it must be kept in mind that the estimates are not directly comparable, given 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), and that Chay and Greenstone use more aggregate data. The effect of PM10 in aggregate data is investigated further in the next section.

c) Estimated Effects in More 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). All of the models in Table 9 control for zip code fixed effects. Note that in the infant mortality regressions, we now control only for pollution in the quarter of birth. These models are shown in Table 9. 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. In order to compare the

effect sizes to those indicated in bold in Tables 6 and 7, it is necessary to multiply coefficients and standard errors by 100 to give the effect per 100,000 live births.

The first panel of Table 9 shows that in the aggregate-level data, only PM10 has a statistically significant effect in the multi-pollutant models--there is no statistically significant effect of CO. This observation suggests that estimates based on aggregate data will significantly under-estimate the effects of CO, perhaps because acute exposures matter. On the other hand, the point estimate of .004 on PM10 in column (6) indicates that there was a decline of .4 deaths per 100,000 per unit of PM10 reduction per quarter, or a reduction of 1.6 deaths per 100,000 annually, which is larger than the estimate of .554 per 100,000 implied by the comparable Table 7 estimate. Hence, it does appear that the estimated effects of PM10 are larger in more aggregate data.

The rest of the Table shows that once again, we find little consistent effect of pollution on the incidence of prematurity or fetal death in the fixed effects models. Panel 2 indicates however, that the coefficient on CO is significant in the multi-pollutant model for low birth weight, and very large. However, the fact that CO is not significant in the single-pollutant model suggests that the finding in the multi-pollutant model may be due to collinearity between the pollution measures.

VI. Discussion and Ctqu 2ŋ1easures.

block implementation of these standards (Stafford, 2001).9 Similarly, there is controversy over the Bush administration's recent "Clear Skies" initiative, which would eliminate the requirement that older power plants upgrade their pollution controls when they upgrade or modernize their equipment and replace them with "cap and trade" provisions. Critics contend that the plan would not regulate CO production, provides weaker caps than alternative legislation introduced in the Senate, and will not necessarily reduce pollution in the most polluted areas, an important consideration if the effects of pollution are non-linear (Environmental Defense, 2003).

In order to begin to evaluate the costs and benefits of such policies, it is necessary to understand how changes from current, historically low levels of air pollution are likely 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 detailed characteristics of individuals including zip code of residence causes us to overturn some of the findings in the cross-sectional epidemiological literature concerning prenatal pollution exposures. For example, we find little average effect of prenatal pollution exposure on the probability of low birth weight, short gestation, or fetal death once zip code fixed effects are included in individual-level models, although there is some evidence that people in high pollution areas, and children whose mothers have less than a high school education, may be more subject to fetal death induced by pollution.

[9] Due to increased driving, trucks burning diesel emitted m

In "single pollutant" models that include fixed effects, we find that CO, PM10, and NO2 all increase infant mortality. Our results for CO also hold in "multi-pollutant" models and are extremely robust to many changes in specification. The estimated effects of PM10 and NO2 (an important precursor) are more sensitive to specification, and show some signs of collinearity. However, our preferred estimates imply that reductions in CO and PM10 over the time interval we study saved over 1,000 infant lives in California alone. These findings are 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 might 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.10 If we use the EPA(1999) value of $4.8 million, the benefit would grow to $5.1 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 benefits to infants. But they may still provide a useful benchmark for assessing the benefits of further reductions in air pollution in terms of infant health.

[10] 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:

$$P_{it} = \Pr[T_i = t \mid T_i > t, x_{it}] \qquad \text{(A1)}$$

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

We can now specify the likelihood function:

$$L = \prod_{i=1}^{n} [\Pr(T_i = t)]^{\partial_i} [\Pr(T_i > t)]^{1-\partial_i} \qquad \text{(A2)}$$

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:

$$\Pr(\quad) \quad {}_{1}^{1}(1 \quad {}_{ij})$$

$$\Pr(\quad)\ {}_{1}(1 \quad {}_{j}$$

"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

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

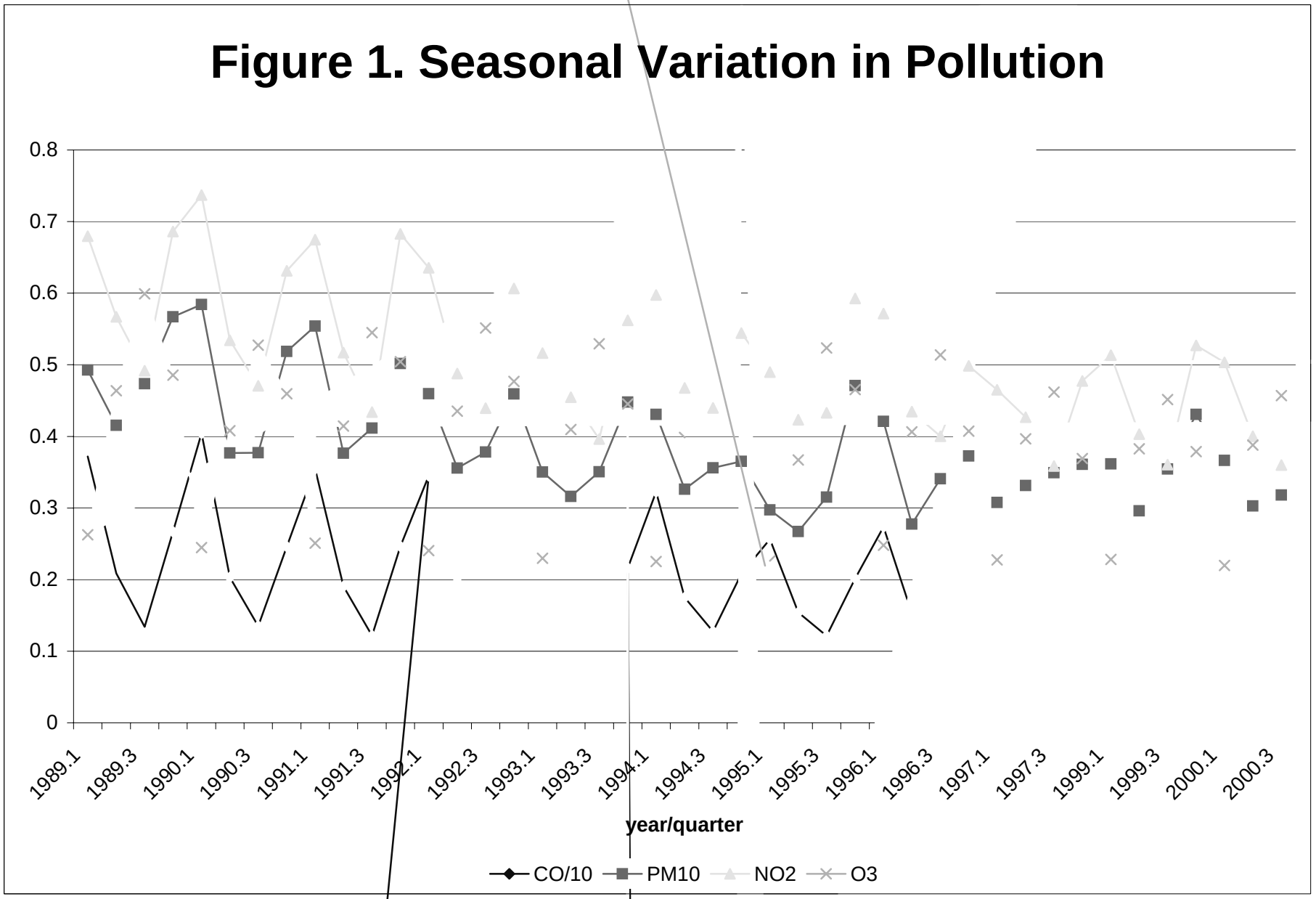
A: Studies of High Pollution Areas

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

B: Studies of the U.S. and Canada

Alderman et al. (1987)	Colorado	1975-83	low birth weight	CO	No association between CO in last trimester and LBW once maternal education and race were controlled.

Figure 1. Seasonal Variation in Pollution



Fig

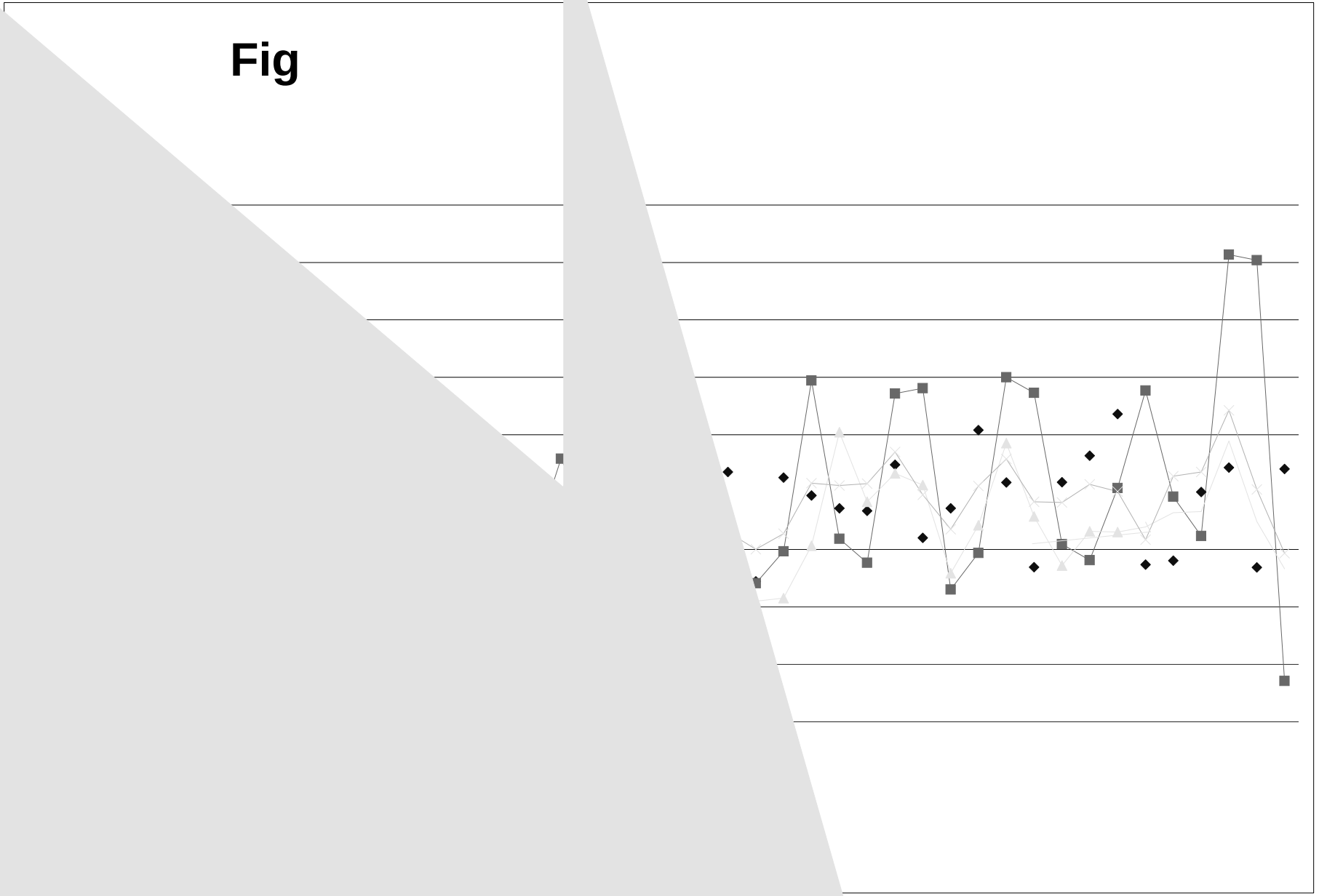


Table 1: Levels and Trends in Pollution and Infant Health

Variable	Mean	Std. Dev.	Between zip std. Dev.	Within zip std. Dev.
Panel 1				
CO 8-hr ppm	1.975	1.101	0.677	0.777
PM10 24-hr ug/m3	39.125	14.165	10.833	9.244
NO2 1-hr ppb	50.919	18.370	15.356	9.457
O3 8-hr ppb	40.424	15.929	9.937	11.802
Panel 2				
Quarterly IMR per 1000	1.64	1.95	4.07	11.28
gestation<37 per 1,000	92.60	30.91	25.72	48.64
low birth weight per 1,000	48.80	21.59	14.94	39.08
fetal deaths per 1,000	5.86	6.58	3.38	13.64

Panel 3 year	CO	PM10	NO2	O3
1989	2.409	48.817	60.340	45.993
1990	2.435	46.174	58.986	41.400
1991	2.252	45.965	57.426	43.326
1992	2.243	41.339	54.208	42.709
1993	1.940	36.645	48.058	40.879
1994	2.071	36.923	51.123	40.309
1995	1.822	33.728	48.383	39.799
1996	1.767	35.253	47.488	39.581
1997	1.585	33.774	43.083	36.593
1999	1.544	36.098	44.936	36.259
2000	1.388	32.891	42.001	35.685

Panel 4 year	Quarterly IMR	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

National Ambient Air Quality Standards

CO	9.5 ppm	8-hr
	35.5 ppm	1-hr
PM10	155 ug/m3	24-hr
NO2	54 ppb	annual
O3	85 ppb	8-hr
	125 ppb	1-hr

Note: What we refer to as the quarterly IMR is the number of infants < 12 months old who died in a quarter.

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	25.136	39.036	53.580
NO2 1-hr	30.972	49.538	68.865
O3 8-hr	33.698	40.095	47.136
quarterly IMR	1.512	1.788	1.965
gestation<37 per 1,000	78.385	89.626	92.388
low BW per 1,000	43.281	47.243	48.673
fetal death per 1,000	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 dropout	0.216	0.285	0.326
% HS grads	0.334	0.339	0.344
% AD degree	0.153	0.140	0.134
% college grads	0.297	0.236	0.196
% teen mothers	0.055	0.064	0.070
% 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

Average Zipcode Pollution Levels by Individual Race and Education:

	O3	NO2	CO	PM
All	40.274	51.156	1.968	39.090
Blacks	38.135	51.916	2.124	38.878

Table 3: The Effect of Pollution on the Probability of Short Gestation
Pollution Measure=Average During Last Month of Pregnancy

	1 CS	2 CS	3 CS	4 CS	5 CS	6 CS	7 FE	8 FE	9 FE	10 FE	11 FE	12 FE
CO	0.867				0.005	0.293	0.248				0.211	0.387
	[0.502]				[0.870]	[0.587]	[3.022]				[1.097]	[2.387]
PM10		0.211**			0.162**	0.167**		0.087			0.061	0.065
		[0.033]			[0.042]	[0.041]		[0.137]			[0.086]	[0.108]
NO2			0.094**		0.022				0.054		0.021	
			[0.028]		[0.051]				[0.268]		[0.284]	
Ozone				0.167**	0.110*	0.117**				0.087	0.078	0.084
				[0.039]	[0.048]	[0.045]				[0.154]	[0.186]	[0.135]
Max temp. last	-0.078	-0.200*	-0.141	-0.336**	-0.360**	-0.354**	-0.415	-0.441	-0.435	-0.534	-0.536	-0.531
30 days	[0.078]	[0.082]	[0.082]	[0.098]	[0.101]	[0.100]	[0.434]	[0.485]	[0.516]	[0.494]	[0.510]	[0.496]
Avg. Precip.	-0.167*	-0.083	-0.153*	-0.195*	-0.118	-0.118	-0.193	-0.155	-0.182	-0.204	-0.172	-0.173
last 30 days	[0.077]	[0.077]	[0.077]	[0.076]	[0.079]	[0.079]	[0.219]	[0.213]	[0.228]	[0.210]	[0.241]	[0.229]
Infant Characteristics												
Male	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**	-0.012**
	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]
first birth	-0.018**	-0.018**	-0.018**	-0.017**	-0.018**	-0.018**	-0.016**	-0.016**	-0.016**	-0.016**	-0.016**	-0.016**
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
second birth	-0.022**	-0.022**	-0.022**	-0.022**	-0.022**	-0.022**	-0.021**	-0.021**	-0.021**	-0.020**	-0.021**	-0.021**
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
third birth	-0.017**	-0.017**	-0.017**	-0.016**	-0.017**	-0.017**	-0.016**	-0.016**	-0.016**	-0.015**	-0.016**	-0.016**
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
Mother Characteristics												
Black	0.056**	0.056**	0.055**	0.056**	0.056**	0.056**	0.052**	0.052**	0.052**	0.052**	0.052**	0.052**
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
Hispanic	0.010**	0.009**	0.009**	0.010**	0.009**	0.009**	0.008**	0.007**	0.008**	0.008**	0.007**	0.007**
	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]
Asian	0.022**	0.022**	0.022**	0.022**	0.022**	0.022**	0.023**	0.022**	0.023**	0.023**	0.022**	0.022**
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
Other Race	0.01	0.01	0.01	0.010*	0.009	0.009	0.009	0.009	0.009	0.01	0.008	0.008
	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]	[0.005]
Married	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**	-0.013**
	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]	[0.002]
Foreign Born	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**	-0.009**
	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]
Parents of	0.008**	0.008**	0.008**	0.008**	0.008**	0.008**	0.009**	0.009**	0.009**	0.009**	0.009**	0.009**
Different Race	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]	[0.001]

Table 3: The Effect of Pollution on the Probability of Short Gestation (continued)
Pollution Measure=Average During Last Month of Pregnancy

	1	2	3	4	5	6	7	8	9	10	11	12

Table 5: Differential Effects of Pollution on Birth Outcomes, Zipcode Fixed Effects Models

	1	2	3	4	5	6
Interaction Variable:	**High Poll.**	**High Poll.**	**< HS**	**< HS**	**Black**	**Black**
1. Dependent Variable=Low Birth Weight						
CO	4.139	-0.708	1.534	0.695	1.365	0.535

Table 8: Effects of Pollution on Infant Mortality, Including Cumulative Average Weekly Exposure Zipcode Fixed Effects Models

	1	2	3	4	5	6

Table 9: Estimates Using Data Aggregated to Quarterly Level