AIR POLLUTION AND INFANT HEALTH: WHAT CAN WE LEARN FROM CALIFORNIA'S RECENT EXPERIENCE?*

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We examine the impact of air pollution on infant death in California over the 1990s. Our work offers several innovations: first, most previous studies examine populations subject to far greater levels of pollution. Second, many studies examine a single pollutant in isolation. We examine three "criteria" pollutants in a common framework. Third, we use rich individual-level data and pollution measured at the weekly level. Our most novel finding is a significant effect of CO on infant mortality: we find that reductions in carbon monoxide over the 1990s saved approximately 1000 infant lives in California.

Air quality regulations are costly to both producers and consumers, and the optimal level of pollution abatement is hotly contested. 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 [Environmental Protection Agency 1999] because the weight of the scientific evidence linking infant health to air pollution was viewed as insufficient.[1]

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

Our work offers several innovations over the existing literature. First, many previous studies examine populations subject to

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

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greater levels of pollution because they lived further in the past or in some more heavily polluted place. In contrast, the experience of California in the 1990s is clearly relevant to the contemporary debate over pollution levels in the United States. Second, many studies examine a single pollutant in isolation (usually particulate matter), generally because of data limitations. We examine three "criteria" pollutants that are commonly monitored in the United States: Ozone (O3), carbon monoxide (CO), and particulate matter (PM10).[2] Third, we exploit weekly pollution data and rich individual-level data to estimate linear models that approximate hazard models, where the risk of death is defined over weeks of life and we control for the length of life as a flexible nonparametric spline.

Our framework allows us to control for a wide array of potential confounders in an effort to identify causal effects. In addition to controls for both postnatal and prenatal pollution exposure, we control for weather, the age of the child, observable characteristics of the mother and child (such as race, maternal age, child gender), and for a variety of unobservable characteristics that can be captured by month, year, zip code fixed effects (or combinations of these fixed effects). In our richest specification, the effects of pollution are identified using only variation within cells defined at the zip code, month, and year level. Of these controls, we find that those for the age of the child appear to be most important, which reflects the fact that the probability of death in the first weeks is much higher than the probability of death later in the first year.

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, and suggest that previous studies may have overlooked a potentially important role for CO. In particular, we estimate that the reductions in CO that occurred over the 1990s saved approximately 1000 infant lives in California. This finding is robust to many sensitivity analyses, and does not appear to be due to mortality displacement, or "harvesting." In keeping with some of the previous research in

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

this area, we also find an effect of PM10 in some specifications, but this finding is not as robust as the finding for CO.

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

I. Background

We begin with a discussion of the ways in which the three criteria air pollutants can affect infant health. Carbon monoxide is an odorless, colorless gas which is poisonous at high levels. CO bonds with hemoglobin more easily than oxygen, so that it reduces the body's ability to deliver oxygen to organs and tissues. Because infants are small, and many have respiratory problems in any case, CO may be particularly harmful to them. As much as 90 percent of CO in cities comes from motor vehicle exhaust [Environmental Protection Agency January 1993].

Particulate matter can take many forms, including ash and dust, and motor vehicle exhaust is a major source. It is thought that the most damage comes from the smallest particles since they are inhaled deep into the lungs [Environmental Protection Agency 2003b]. The mechanisms through which particles harm health are controversial. The leading theory is that they cause an inflammatory response which weakens the immune system [Seaton et al. 1995]. We focus on PM10, particles less than 10 microns in diameter, although many older studies use measures of Total Suspended Particles (TSPs). In general, one would expect TSP and PM10 to move together because PM10 is a component of TSP, but some of the larger particles included in TSP may be less damaging than the particles found in PM10.

Ozone (the major component of smog) is a highly reactive compound that damages tissue, reduces lung function, and sensitizes the lungs to other irritants. For example, exposure to O3 during exercise reduces lung functioning in adults and causes symptoms such as chest pain, coughing, and pulmonary congestion. Ozone is formed through reactions between nitrogen oxides and volatile organic compounds (which are found in auto emissions, among other sources) in heat and sunlight. Ozone is not generally found in homes because it quickly reacts with

household surfaces (http://www.hc-sc.gc.ca/hecs-sesc/air_quality/faq.htm).

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

A link between air pollution and infant health has long been suspected, although the exact biological mechanisms through which it occurs are not known. We also know little about what levels of these pollutants are sufficient to affect infant mortality (death in the first year of life) or about the extent that infants are protected from the negative effects of pollution while they are in the womb. Pollution exposure could affect the health of the mother by, for example, weakening her immune system, which could have negative effects on the fetus. In infants, a weakened immune system could make them more susceptible to death from a wide range of causes. Alternatively, since motor vehicle exhaust is a major contributor of CO and PM10, these pollutants may themselves be markers for other components of exhaust which injure infants.[3] However, the available research gives very little guidance about what levels of pollution might be necessary to induce negative effects or about when fetuses or infants are most vulnerable.

Many studies have demonstrated links between very severe pollution episodes and increased mortality of infants and others. One of the most famous focused on a "killer fog" in London, England, and found dramatic increases in cardiopulmonary mortality [Logan and Glasg 1953]. Many subsequent studies have found negative associations between pollution and positive infant outcomes.[4] An important limitation of all of these studies is that it is possible that the observed relationships could reflect an

3. Components such as polycyclic aromatic hydrocarbons (PAHs), acetonitrile, benzene, butadiene, and cyanide (see http://www.epa.gov/ttn/atw/hapindex.html) have been shown to have effects on developing fetuses in animal studies which may include retarded growth. Studies in humans have shown elevated levels of an enzyme induced by PAHs in women about to have preterm deliveries [Huel et al, 1993].

4. These studies are summarized in a previous version of this paper. A few of the most recent pertaining to North America include Ritz et al. [2000], Ritz and Yu [1999], Mainsonet et al. [2001], and Liu et al. [2003].

unobserved factor that was correlated with both air pollution and child outcomes. This is likely to be a greater problem in studies that do not control for factors like maternal education, but it may be a problem even in studies that include such controls. Suppose, for example, that areas with high levels of air pollution also tended to have high levels of water pollution. If water pollution causes infant deaths but is unobserved, then one might falsely conclude that air pollution was to blame for infant deaths, with potentially negative consequences for remediation efforts.

Two studies by Chay and Greenstone [2003a, 2000b] deal with the problem of omitted confounders by focusing on "natural experiments" provided by the implementation of the Clean Air Act of 1970 and the recession of the early 1980s.[5] Chay and Greenstone show that on average TSPs fell from 95 to 60 micrograms per cubic meter of air between 1970 and 1984. However, both the Clean Air Act and the recession induced sharper reductions in TSPs in some counties than in others, and they use this exogenous variation in levels of pollution at the county-year level to identify its effects. They estimate that a one-unit decline in TSPs associated with the Clean Air Act (recession) led to between five and eight (four and seven) fewer infant deaths per 100,000 live births.

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

In this paper we use individual-level data and weekly zip code-level pollution measures and control for many potential confounders in an effort to identify causal effects. Using this strategy

5. These studies are similar in spirit to a sequence of papers by C. Arden Pope, who investigated the health effects of the temporary closing of a Utah steel mill [Pope, 1989; Ransom and Pope 1992; Pope, Schwartz, and Ransom 1992] and to Friedman et al. [2001] who examine the effect of changes in traffic patterns in Atlanta due to the 1996 Olympic games. However, these studies did not look specifically at infants.

allows us to identify the effects of pollution in more recent data and to compare the effects of several criteria pollutants.[6]

II. Data

Detailed data on atmospheric pollution come from the California Environmental Protection Agency's air monitoring stations.[7] Following Neidell [2004], we use the monitor data to construct a weekly measure of pollution for each zip code in the state. To do this, we compute a weekly level of pollution for each monitor taking the mean of the daily values. For the three pollutants of interest, the daily measures we use are the eight-hour maximum CO and O3, and the 24-hour average PM10.[8] To assign these weekly values of pollution from the monitors to zip codes, we proceed 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 using the geographic coordinates of the monitor. Finally, we calculate a weighted average of the weekly pollution level using all monitors within a twenty-mile radius of the zip code's center, using the inverse of the distance to the monitor as the weight. This method enables us to construct a pollution measure for each zip code and week.

6. A final issue is that this paper (like the others discussed above) examines the effect of outdoor air quality measured using monitors in fixed locations. Actual personal exposures are affected by ambient air quality, indoor air quality, and the time the individual spends indoors and outdoors. One might expect, for example, that infants spend little time outdoors so that outdoor air quality might not be relevant. Research on the relationship between indoor and outdoor air quality [Spengler, Samet, and McCarthy 2000; Wilson, Mage, and Grant 2000] suggests that much of what is outdoors comes indoors. Second, although the cross-sectional correlation between ambient air quality and personal exposure is low (between .2 and .6 in most studies of PM, e.g.), the time-series correlation is higher. This is because for a given individual indoor sources of air pollution may be relatively constant and uncorrelated with outdoor air quality. So for a given individual much of the variation in air quality comes from variation in ambient pollution levels.

7. Monitors tend to be located in the most densely populated areas of the state, and also in those that are most polluted. The location of monitors may also change over time. Hence, in this analysis, we use only those monitors that existed continuously throughout the period, although using all monitors does not change our results.

8. The data are the California Ambient Air Quality Data from the California Air Resources Board, a department of the California Environmental Protection Agency (available at http://www.arb.ca.gov/aqd/aqdcd/aqdcd.htm). The 8-hour maximum corresponds to taking the maximum 8-period moving average within a 24-hour period. Since PM10 is only measured once every six days, the weekly mean includes at most two monitor readings of PM10. These measures are highly correlated with measures of short-term spikes in pollutants. For example, the correlation between the maximum one-hour reading for CO and the maximum eight-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.

Using this method, we are able to assign a pollution level to zip codes covering about 70 percent of the births in the state. Zip codes that we were not able to assign pollution levels to are overwhelmingly rural. While not every urban zip code has a monitor, 76 percent of the births included in our sample were within ten miles of a monitor, and we obtain very similar results if we limit our analysis to this subsample, as shown below.

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

Descriptive statistics for the pollution variables are shown in the first and second panels of Table I. Panel 2 indicates that there has been considerable decline in pollution levels over our sample period. The pollutants we examine also display strong seasonal patterns (not shown). In California, ambient levels of CO and PM10 tend to increase in cold weather when they are trapped by damp cold air. PM10 also spikes in cold weather because it is produced by combustion sources used for heating. In general, levels of CO and PM10 are highly correlated which may make it difficult to disentangle their effects. On the other hand, ozone forms at a higher rate in heat and sunlight, so that ozone levels spike during the summer. As we show below, the negative correlation of ozone with other pollutants can yield wrong-signed effects in single-pollutant models. Our richest models include zip code-month fixed effects (one also includes zip code-year effects) in order to control for seasonal effects that could be different in northern and southern California, for example. These effects also remove some of the variation in pollution, but a great deal of residual variation remains, as shown in the first panel of Table I.

Since weather is a key determinant of pollution levels but could also have independent effects on infant health, we include controls for maximum temperatures and average precipitation in our models. The weather data come from the Surface Summary of

TABLE I
LEVELS AND TRENDS IN POLLUTION AND INFANT HEALTH

Panel 1 Variable	Mean	Std. dev.	Between zip-month std. dev.	Within zip-month std.dev.
CO 8-hr ppm	1.998	1.169	1.018	0.447
PM10 24-hr μg/m3	39.448	14.755	12.899	7.869
O3 8-hr ppb	40.456	17.107	15.832	5.509
IMR per 1000	3.91	6.24	1.35	6.23
Low birth weight per 1000	48.35	21.45	3.45	21.40
Fetal deaths per 1000	3.58	5.97	0.84	5.96

Panel 2 year	CO (8 hour)	PM10	O3 (8 hour)
1989	2.458	49.651	46.139
1990	2.472	46.575	41.664
1991	2.288	46.377	43.516
1992	2.279	41.285	42.830
1993	1.974	37.040	41.089
1994	2.111	37.384	40.351
1995	1.857	34.256	40.037
1996	1.798	35.790	39.681
1997	1.608	34.052	36.630
1999	1.580	36.510	36.109
2000	1.376	33.572	35.657

Panel 3 year	IMR	Low birth weight	Fetal deaths	Number of births*
1989	5.33	51.02	4.10	388,097
1990	4.76	48.23	3.95	444,021
1991	4.46	47.41	3.79	454,902
1992	4.18	48.15	3.70	445,760
1993	4.08	48.59	3.55	449,374
1994	3.96	49.33	3.46	441,080
1995	3.56	48.42	3.59	419,948
1996	3.27	48.32	3.56	407,923
1997	3.21	48.31	3.20	386,137
1999	2.90	46.64	3.15	372,232
2000	2.96	47.39	3.21	383,527
Total				4,593,001

National ambient air quality standards		
O3	85 ppb	8-hr
CO	9.5 ppm	8-hr
PM10	155 μg/m3	24-hr

PPB refers to parts per billion. Ug/m3 refers to micrograms per cubic meter of air. PPM is parts per million. IMR is the infant mortality rate which is usually measured per 1000 live births.

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

Data on infant deaths come from the California Birth Cohort files for 1989 to 2000. These data are abstracted from birth and death certificates. Since there is no birth cohort file for 1998, this year is excluded from our analysis. We confine our analysis of infant deaths to infants with at least 26 weeks gestation so that we can define pollution exposure in the first, second, and third trimesters of the pregnancy.[9]

We have also used these data (and additional data from fetal death certificates) to examine the impact of pollution on low birth weight (defined as birth weight less than 2500 grams) and the incidence of fetal death.[10] The rationale for examining these additional outcomes is that low birth weight is a leading indicator of poor infant health that is much more common than death.[11] And if pollution has an effect on fetal deaths, then examining only the population of live births may yield biased estimates of its true effects. For example, if pollution causes a fetus that would have been born alive but low birth weight to be stillborn, then it could even appear that pollution increased birth weight. For these analyses, we combined live births and fetal deaths in order to create a sample of pregnancies lasting at least 26 weeks. Examination of the effects of pollution on this sample gave us estimates

9. This results in the exclusion of 127,189 live births with gestation less than 26 weeks.

10. The distinction between fetal and infant death 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.

11. Although Almond, Chay, and Lee [2002] argue that the effects of birth weight on infant mortality have been grossly overstated, low birth weight is still widely acknowledged to be the leading indicator of poor health at birth.

of the effects of pollution that are not biased by fetal selection that occurs after 26 weeks.[12]

Descriptive statistics for these outcome variables are shown in the third panel of Table I. Over the sample period 3.91 children per 1000 infants born alive (with gestation of 26 weeks or more) died in their first year. Although the infant mortality rate fell sharply over a relatively short time, trends in low birth weight and fetal death were much flatter. This part of the table suggests that declines in mortality were largely due to events occurring after the birth, rather than to improvements in prenatal health.

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 five-digit zip code of maternal residence, as well as information about use of prenatal care and whether the birth was covered by public health insurance. The rapid increase in the fraction of births covered by Medicaid is a potential confounding factor when examining birth outcomes because there is evidence that Medicaid coverage changed the way that at risk infants were treated (cf. Currie and Gruber [1996]), so it is fortunate that we can control for Medicaid coverage of the birth directly. Unfortunately, it is not possible to control for maternal smoking because this information is not included on California's birth certificate. To the extent that smoking is correlated with other variables included in our model, bias due to this omission will be reduced.

Finally, the last panel of Table I lists the federal standards for the pollutants we examine. A comparison of the first and last panels of the tables suggests that on an average day, pollution levels in California are well under the thresholds for these standards. However, the fact that Los Angeles is consistently found to be out of compliance for both ozone and CO (i.e., has at least one day in the year where average ambient levels over an eight-hour period are above these thresholds) indicates that there is substantial variability in pollution levels around these means.

Table II shows mean annual outcomes and pollution levels as

12. While pollution might also cause fetal deaths before 26 weeks, fetal deaths before 26 weeks are not accurately reported. Since we do not examine the effects of pollution on gestation or on infants with less than 26 weeks gestation, our results leave open the possibility that pollution could lead to premature termination of pregnancies or high rates of infant death in this population. Hence, our estimates will understate the total effect of pollution on infant health if it causes fetal losses before 26 weeks, or an increased probability of death in surviving infants with very short gestations.

TABLE II
POLLUTION LEVELS FOR INFANTS BORN IN HIGHEST AND LOWEST POLLUTION AREAS

Ranked by:	Pollution Level		Change in pollution between weeks 1&2		Change in pollution between weeks 2&3	
Variable	Lowest 1/3	Highest 1/3	Lowest 1/3	Highest 1/3	Lowest 1/3	Highest 1/3
CO 8-hr level	1.176	2.912	2.008	2.116	1.986	2.121
Weekly change in CO			−0.228	0.268	−0.237	0.249
PM10 24-hr level	25.647	54.139	39.479	41.067	39.685	40.662
Weekly change in PM10			−14.673	14.539	−14.594	14.417
O3 8-hr level	34.837	46.705	41.269	40.283	41.597	40.201
Weekly change in O3			−0.005	0.004	−0.005	0.004
IMR per 1000	3.583	4.406	3.976	3.997	3.976	3.965
Change in Deaths			901.000	929.000	235.000	242.000
Low BW per 1000	47.094	49.506	48.344	48.601	48.360	48.434
Fetal death per 1000	3.370	3.840	3.692	3.638	3.628	3.659
% Male	0.487	0.488	0.488	0.488	0.488	0.488
% Black	0.083	0.083	0.082	0.083	0.082	0.083
% Hispanic	0.317	0.550	0.473	0.471	0.472	0.472
% Asian	0.161	0.089	0.112	0.114	0.113	0.113
% Married	0.725	0.629	0.663	0.663	0.663	0.663
% Foreign mom	0.394	0.524	0.477	0.478	0.476	0.479
% Racial diff parents	0.189	0.139	0.158	0.159	0.158	0.158
% HS dropout	0.254	0.408	0.354	0.352	0.354	0.352
% HS grads	0.359	0.348	0.353	0.352	0.353	0.353
% AD degree	0.148	0.114	0.125	0.125	0.125	0.126
% College grads	0.239	0.130	0.168	0.170	0.168	0.169
% Teen mothers	−0.238	−0.225	−0.227	−0.227	−0.227	−0.226
% Age 19 to 25	0.304	0.366	0.344	0.343	0.344	0.343
% Age 26 to 30	0.281	0.281	0.282	0.281	0.282	0.281
% Age 31 to 35	0.233	0.187	0.203	0.204	0.203	0.204
% First born	0.419	0.391	0.398	0.398	0.398	0.398
% Second born	0.318	0.299	0.307	0.307	0.307	0.307
% Third Born	0.154	0.170	0.166	0.165	0.166	0.165
% Gov't insurance	0.384	0.495	0.462	0.460	0.462	0.461
% 1st tri. prenatal care	0.816	0.742	0.773	0.776	0.774	0.776

This table is calculated using the entire sample of births. A unit of observation is the birth, and the pollution levels in the first two columns are the pollution levels in the child's zip code in the week of birth. The change in pollution is the change that the child experienced between weeks x & y.

well as means of various control variables for the whole sample of births. The first two columns take all the births, and rank them by the level of pollution in the zip code at the time of the child's

birth. In order to come up with one pollution measure, we standardized all of the three pollution measures using a "z-score" and took the average of the three measures. While this is a rough way to rank areas, Table II indicates that it is informative—there are sharp differences in ambient pollution levels experienced by children born into the most polluted and the least polluted areas of the state. For example, the CO measure is almost three times higher in the most polluted areas compared with that in the least polluted ones.

These gradients in pollution levels correspond to gradients in birth outcomes: the most polluted areas have uniformly worse outcomes than the least polluted ones. But as the rest of the table shows, this association could be due to the fact that pollution levels are highly correlated with socioeconomic characteristics that are themselves predictive of poorer birth outcomes. For example, 73 percent of mothers are married in the least polluted areas compared with 63 percent in the most polluted areas; 25 percent are high school dropouts in the cleaner areas compared with 41 percent in the dirtiest; and the comparable figures for use of government insurance are 38 percent and 50 percent. These are very large differences in the average characteristics of mothers, and failure to adequately control for them could generate spurious relationships between pollution and birth outcomes. In our models, we will control for these important observable differences between locations.

The next two columns of Table II rank the births by the change in pollution faced by the infant between weeks 1 and 2 of life. In contrast to the first two columns of the table, these means suggest that there are virtually no differences on average between areas that experience short-run positive and negative changes in pollution levels. For example, both areas that experience positive changes and those that experience negative changes have similar levels of pollution in the birth week, similar infant mortality rates, and similar maternal characteristics (in terms of percent Black, etc.). However, despite the similarities in average characteristics, areas that experienced increases in pollution have more deaths than those that experienced decreases in pollution. The same is true if we rank individuals by the change in pollution experienced between weeks 2 and 3, as shown in the table. Hence, the table suggests that even if controls for observable characteristics are not sufficient to rule out confounders,

examining changes in weekly pollution within zip codes (as we do in our fixed effects models) will be informative.

Finally, Table II shows that the change in number of deaths between weeks 1 and 2 is much greater than the change in the number of deaths between weeks 2 and 3. This in turn is higher than the change in the number of deaths in any subsequent weeks. In other words, there is a very strong age effect on the probability of death, and probability of death is much higher in the first two weeks than thereafter. Uncontrolled, these age effects could lead us to either over- or underestimate the effect of pollution. To see this, consider a zip code that has both high pollution levels and high infant mortality rates, but infant mortality is high for reasons other than pollution. If many infants die in the first weeks, then there will be fewer older babies in this zip code. If older babies are less likely to die from pollution exposure than younger ones, we will overestimate the effect of pollution if we ignore this composition effect. Conversely, if older babies were more likely to die than younger ones, other things being equal, then failure to control for age would cause us to understate pollution effects. Clearly, it may be important to control for the age effect in our models.

III. Methods

Evidently, air pollution affects infants differently before and after birth. Before birth, pollution may affect infants either because it crosses the protective barrier of the placenta or because it has a systemic effect on the health of the mother. After birth, infants are directly exposed to inhaled pollutants. However, one might wonder whether effects observed after birth actually reflect the lingering effects of exposures before birth. In order to control for this possibility, we estimate models that include the infant's birth weight and gestation. These variables can be regarded as summary statistics for the infant's health at birth and hence will help to capture any effects of pollution before the birth. We also include separate controls for average pollution levels during the infant's first, second, and third trimesters of gestation, though as we show below, this has no effect on the estimated effect of postnatal pollution exposure.

The probability of death P_{izt} is specified as

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

where i indexes the individual, z indexes the zip code, t indexes the time period and $\alpha(t)$ is a measure of duration dependence and is specified as a linear spline in the weeks since the child's birth, with breaks after 1, 2, 4, 8, 12, 20, and 32 weeks. These break points reflect the fact that death is much more common in the first weeks than thereafter. The w_{iz} are time-invariant covariates measured at the individual level, such as the mother's demographic and background characteristics; the h_{iz} are time-invariant measures of the infant's health and pollution exposure at the time of the birth including indicators for low birth weight and short gestation and for pollution exposure in the first, second, and third trimesters; the x_{zt1} are time-varying measures of pollution exposure after the birth; the x_{zt2} are weather indicators; φ_{zt} is a vector of zip code-month specific fixed effects; and Y_t is a vector of year dummies that allows for statewide trends in these outcomes. We consider several variations on (1) including estimation of models that include both zip code-month and zip code-year fixed effects. The main coefficient of interest is β_1, the effect of postnatal 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, nonparametric duration dependence, and zip code-month level fixed effects. The model imposes little "structure" on the pattern of age coefficients, allowing the data to "speak for itself," a consideration that is particularly important given the lack of guidance in the literature regarding mechanisms and functional form. Allison [1982] shows that estimates from models of this type converge to those obtained from continuous time models, as discussed further in the appendix to Currie and Neidell [2004].[13]

In order to implement this estimation strategy, we treat an individual who lived for n weeks as if they contributed n person-week observations to the sample. The dependent variable is coded as 1 in the period the infant dies, and 0 in all other periods. Each time-invariant covariate is repeated for every period, while the time-varying covariates are updated each period. P_{izt} is then regressed on the covariates specified in (1) by ordinary least squares.

This procedure yields a very large number of observations. Most infants survive all 52 weeks of their first year, yielding a sample of 250 million weekly observations. Hence, we employ

13. Note that we have also estimated models using $f(P_{izt})$ as the dependent variable, where f is the logit transformation-the results were very similar.

case-control sampling to reduce the number of observations. First, we keep all individuals who died (the cases) in the week that they died. Then, in order to select controls, we choose randomly among all the observations on children who lived for at least as many periods as the index child and take the control child's observation for that week. That is, if a child died in week 3, the controls would be chosen from observations on all children who lived at least three weeks regardless of whether they later died. For each week, we randomly chose fifteen times as many nondeaths as deaths. This method greatly reduces computational burden while yielding unbiased estimates of the effects of pollution on the probability of death [Mantel 1973; Prentice and Breslow 1978; Lubin and Gail 1984].[14]

As discussed above, we chose a week as the unit of time in our base specification. A potential problem with choosing such a small interval is that children who die from exposure to high amounts of pollution in week t might have died at $t + 1$ in any case. This problem of mortality displacement is sometimes referred to as "harvesting" [Schwartz 2000]. If mortality displacement is an important phenomenon, then estimates based on weekly pollution measures will tend to overstate the loss of life caused by pollution. For example, the actual loss of life might be only one week rather than average life expectancy at birth.

In the literature, the usual response to the potential problem of "harvesting" is to estimate models using longer time units. But estimating models using longer time units, such as months, involves more measurement error because the measure of pollution is assigned more imprecisely. For example, if we use the month as the time unit, children who die in their first week of life are incorrectly assigned average pollution levels for all of the days in the month. Moreover, if it really was a sharp spike in a pollutant that caused death, these spikes would tend to be averaged out in more aggregate data. Hence, one might expect lower and more

14. Suppose, instead, that we took all children who died, and selected a control group by sampling all children who survived their first year. At any point in time during the year, we would have a sample that excluded infants who were at risk of death, but survived only to die later. We reproduce Mantel's discussion of why retaining individuals on the basis of their outcomes only adds a constant to the log odds ratio in the appendix to Currie and Neidell [2004]. Since we begin with the entire universe of births and can choose the sample to analyze, we have followed the case control literature that specifies the correct way to choose an analysis sample rather than the economics literature on "choice-based sampling" which suggests estimation methods to deal with samples that have already been chosen nonrandomly (cf. Manski and Lerman [1977] and Imbens [1992]).

imprecise estimates in monthly data even without harvesting. As we will show below, however, the point estimates are very stable when we use the month as the time unit, though the standard errors increase (as one would expect). We take this as strong evidence that our results are not driven by harvesting.

Models estimated using weekly pollution focus on the short-term effects of pollution exposure and the cumulative impacts of postnatal exposure might also be important. Hence, we also estimate models using cumulative pollution measures. We also present several additional specification checks, and find that our key results are insensitive to these considerations.

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

$$P_{iz} = w_{iz}\gamma + p_{z1}\eta_1 + p_{z2}\eta_2 + \varphi_{zt} + Y_t, \tag{2}$$

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

IV. Results

Table III shows estimates of model (1). For comparison with previous work we first estimate the model for each pollutant separately. The "basic" model shown in the first panel controls only for prenatal and postnatal pollution exposure, weather, year dummies, and month dummies. The pollution variables are estimated to have strong effects on mortality, although the overall

TABLE III
EFFECTS OF POLLUTION ON INFANT MORTALITY

	(1)	(2)	(3)	(4)
1. Controlling for pre- & postnatal pollution, weather, year dummies, month dummies				
CO	5.086			5.427
	[0.570]**			[0.762]**
PM10		0.211		0.037
		[0.035]**		[0.043]
O3			−0.074	0.144
			[0.050]	[0.058]*
R^2	0.004	0.004	0.003	0.004
2. Adding spline in child's age in weeks (1, 2, 3–4, 5–8, 9–12, 13–20, 21–32, >32 weeks)				
CO	2.867			2.566
	[0.512]**			[0.683]**
PM10		0.083		0.001
		[0.032]**		[0.039]
O3			−0.159	−0.054
			[0.045]**	[0.052]
R^2	0.21	0.21	0.21	0.21
3. Adding gender, race, maternal marital status & education, age of mother, parity, insurance, birth weight and gestation				
CO	2.458			2.466
	[0.488]**			[0.651]**
PM10		0.053		−0.026
		[0.031]		[0.037]
O3			−0.141	−0.038
			[0.043]**	[0.050]
R^2	0.27	0.27	0.27	0.27
4. Adding zip code * month fixed effects				
CO	2.631			2.89
	[0.977]**			[1.040]**
PM10		0.002		−0.036
		[0.039]		[0.042]
O3			−0.077	−0.046
			[0.065]	[0.067]
R^2	0.29	0.29	0.29	0.29
5. Magnitudes of the panel 4 effects in lives saved per unit pollution reduction				
CO	16.501			18.125
PM10		0.013		−0.226
O3			−0.483	−0.288

Robust standard errors are in brackets. All regressions include 206,353 observations. * denotes significant at the 90 percent level of confidence; ** indicate significance at the 95 percent level. The numbers in panel 5 are calculated as [coefficient * 100 * (overall IMR/sample IMR)]. The overall IMR in these data is 3.91 per 1000 live births as shown in Table I.

model explains relatively little of the variation in mortality.[15] In the single-pollutant models, both CO and PM10 are estimated to have statistically significant effects, while ozone has a puzzling wrong-signed (but insignificant effect). The last column shows, however, that when we control for the three pollutants simultaneously, both CO and ozone are estimated to increase mortality, while PM10 becomes statistically insignificant.

The case-control sampling makes the coefficients somewhat difficult to interpret. To determine the number of lives saved per unit of pollution reduction, it is necessary to multiply the coefficient by the ratio of the overall infant mortality rate to the sample infant mortality rate. Carrying out this calculation for the model in the fourth column suggests that a one-unit reduction in CO would prevent 34 deaths per 100,000 live births. This implies that the actual 1.1 unit reduction led to a total reduction in deaths over the period (given 4,593,001 births) of 1,719. Similarly, the estimate for ozone implies that the observed decline in ozone save 434 infant lives.

However, the second panel of Table III shows that controlling for the spline in the child's age reduces the estimated effect of pollution and increases the explanatory power of the model dramatically.[16] CO is still statistically significant, but now O3 is not. The third and fourth panels show the results of controlling for all of the observable child and mother characteristics (panel 3) and for those unobservable factors that can be controlled for using zip code∗month fixed effects. These additional controls are jointly significant but have remarkably little impact on the estimated coefficients on the pollutants.

The last panel of Table III presents estimates of the number of deaths per 100,000 births averted by a one-unit decrease in the pollutant in question. The estimates are based on the coefficients in panel 4 of Table III. For example, the estimated coefficient of 2.89 for CO in column (4) implies 18.125 less deaths per 100,000 births for each one-unit decline in CO. Therefore, the actual 1.1 unit decline in CO over the period studied saved 991 infant lives.

Coefficients on the other variables included in the model,

15. With case control sampling, the total variation to be explained is sensitive to the way the sample is selected. For instance, the R^2 will be different in a model with five "controls" per case compared with a model with fifteen controls per case. However, we can still use this statistic as a means to compare different models estimated on the same sample.

16. We have experimented with more flexible splines, but find that the key is to allow for the much higher death rate in the first and second weeks of life.

while suppressed to save space, are worth a brief discussion. We included nine measures of prenatal pollution exposure corresponding to exposure to each pollutant in the first, second, and third trimesters. We found that for each pollutant we could never reject the null hypothesis that the sum of the first, second, and third trimester coefficients was equal to zero. We investigated the sensitivity of these results by including controls only for pollution in the first trimester, the last three months of pregnancy, or the last month of pregnancy, with similarly insignificant results.

We also found that birth weight and gestation were significant predictors of mortality when they were added to the model, consistent with other research. Males, Hispanics, children of foreign-born mothers, and children whose mothers commenced prenatal care in the first trimester are all less likely to die, while children of high school dropouts, teen mothers, people on government insurance, and babies of high parity are more likely to die, consistent with our expectations. Maximum temperatures were estimated to reduce mortality in the cross-sectional models although they had little impact in the models that included zip code-month fixed effects, suggesting that the inclusion of the fixed effects helps to control for the effects of weather.

Table IV investigates the robustness of our results. The first two columns present results from models estimated on the subset of zip codes that belong to the Southern California Air Quality Monitoring District, as discussed above. The first column is our baseline specification with zip code∗month fixed effects, and shows that our results are not sensitive to the region of California we examine. The second column shows models that add zip code∗year fixed effects to the previous specification.[17] The coefficient on CO in column (2) is slightly higher than the corresponding estimate from column (1), but provides similar qualitative results.

The third column shows the effect of dropping infants who died in the first week. Many of these infants were probably very sick at birth, and if, for example, they were hospitalized, they would not have been exposed to much outdoor air. This change does not affect the results. Although the coefficient on CO rises, the implied number of deaths remains similar at 1148 because

17. Ideally, we would have estimated this specification for the entire sample, but it was computationally infeasible to include the necessary number of dummy variables. Estimating models with minor civil division∗year effects on the whole sample produced similar estimates to the main specification (a minor civil division is a cluster of two or three contiguous zip codes).

TABLE IV
ALTERNATIVE SPECIFICATIONS OF THE INFANT MORTALITY MODELS

	(1) SoCal	(2) Add zip-year SoCal only	(3) Drop 1st week	(4) Without prenatal pollution	(5) Without weather	(6) Monitors within 10 miles	(7) Time unit is month	(8) Including lags	(9) Including cumulative	(10) Including leads
CO	3.046	3.614	4.648	2.986	2.169	3.491	2.854	2.212	2.607	2.907
	[1.184]*	[1.226]**	[1.504]**	[1.017]**	[0.952]*	[0.920]**	[1.719]	[1.224]*	[1.021]**	[1.192]**
CO lead or cumulative or lag								1.226	1.048	−0.171
								[1.169]	[2.146]	[1.142]
PM10	0.047	0.014	−0.038	−0.035	−0.038	−0.061	0.128	−0.051	−0.063	−0.049
	[0.051]	[0.054]	[0.058]	[0.042]	[0.042]	[0.039]	[0.089]	[0.045]	[0.040]	[0.043]
PM10 lead or cumulative or lag								0.027	0.175	0.025
								[0.046]	[0.120]	[0.045]
O3	−0.116	−0.114	−0.141	−0.041	−0.099	0.001	−0.128	−0.036	−0.014	−0.026
	[0.082]	[0.085]	[0.095]	[0.065]	[0.061]	[0.065]	[0.118]	[0.078]	[0.068]	[0.076]
O3 lead or cumulative or lag								−0.01	−0.106	−0.047
								[0.076]	[0.125]	[0.076]
# Observations	125259	125259	131488	206352	206352	201990	205214	205958	206352	205981
R^2	0.29	0.27	0.16	0.29	0.29	0.29	0.23	0.29	0.29	0.29

Robust standard errors are in parentheses. ** indicates significance at the 99 percent level of confidence. * indicates significance at the 95 percent level of confidence. Aside from the variations noted in the column headings, the models all include variables similar to panel 4 of Table III. The columns (1) and (2) models include only zip codes in the Southern California Air Quality Monitoring District (Los Angeles, Orange, and parts of Riverside, and San Bernardino counties).

the mortality rate among infants falls after the first week. This suggests that the effects of CO are largely concentrated in infants who survived at least one week and would be more likely to be exposed to outdoor air.

The fourth column of Table IV shows that the coefficient on CO is not affected by dropping the prenatal pollution measures entirely. This is not surprising given the statistical insignificance of these controls. We also obtained similar results dropping birth weight and gestation. Column (5) shows estimates without the weather variables, which again are similar to those reported in panel 4 of Table III. Again, this is not surprising given that the effects of weather are largely absorbed by the included zip code*month effects. Column (6) shows the effect of excluding infants who lived more than ten miles from a monitor. Removing these infants increases the estimated effect of CO, which is consistent with the idea that pollution is more accurately measured among these individuals.

The next column addresses the issue of harvesting by estimating models using months instead of weeks as the time unit. That is, the unit of observation is still the individual, but we merge the infant's data to pollution measured at the calendar month level, and ask whether higher monthly pollution increased the probability that the infant died within the month. If most of the infants killed by high weekly levels of CO would have died within the month in any case, then the estimates from these monthly models should be much smaller than those from the weekly models. It is remarkable that the estimated effect of CO is almost identical to the weekly models, although the standard error rises. In models that did not include the zip code*month fixed effects (which are very demanding in models estimated using monthly pollution measures), we found an almost identical and significant effect of CO. These results provide evidence that the effect in the weekly models is not being driven by a harvesting phenomenon.

Column (8) of Table IV investigates the harvesting issue further by including a lagged pollution measure. In the presence of harvesting, one might expect the lag to be negative, since babies who died last week are not available to die this week. Thus, we would find a negative and significant effect of pollution last week, and a positive and significant effect of pollution this week. We find no evidence of this pattern, however.[18]

Column (9) investigates the timing issue further by including

18. We are grateful to a referee for this suggestion.

both the current week's pollution measure and a cumulative measure of the child's average cumulative exposure. In this specification, the contemporaneous exposure to CO remains significant, suggesting that exposure to this pollutant has a relatively instantaneous effect.

Finally, the last column of Table IV estimates models that include leads of weekly pollution levels. The inclusion of leads is a particularly strong test since an infant who dies in week t should not be affected by pollution in week $t + 1$. We find that the contemporaneous exposure remains unaffected by the inclusion of leads and that the leads themselves are not statistically significant. This finding is remarkable given the strong seasonal correlations in CO and suggests that we really are capturing the effect of exposure in a given week.

The results so far show that postnatal exposure to CO has a remarkably robust effect on infant mortality. We have estimated similar models of the effects of prenatal pollution exposure on birth weight and fetal death as discussed above, which are not shown. The richest model included nine coefficients, one for each pollutant in each trimester of the pregnancy. Only one coefficient was individually statistically significant, and it was wrong signed. F-tests indicated that we could not reject the null hypothesis that the coefficients on prenatal CO exposure (and on the other prenatal pollution exposures) summed to zero. This suggests that fetal selection is unlikely to bias our results. Of course, as discussed above, these estimates were based on a sample of pregnancies that lasted at least 26 weeks, so any fetal deaths that occurred earlier were not captured.

Several previous studies have used aggregate rather than individual-level data, so it is of interest to see what happens if we move to more aggregate data. We aggregate up to the zip code-quarter level and estimate models similar to (1) and (2) which include zip code-quarter fixed effects. In Tables III and IV the unit of observation is the individual child and the outcome is a death. In Table V the unit of observation is the zip code, quarter, and year, and the outcome is the death rate. Furthermore, in this aggregated specification we are not able to control for the age of the infant as we do using the individual data. Previous estimates using more aggregated data have not attempted to sort out the effects of postnatal and prenatal exposures, presumably because of the difficulty of precisely assigning such exposures in aggregate data, so we do not do so here either.

TABLE V
ESTIMATES USING DATA AGGREGATED TO QUARTERLY LEVEL

	(1)	(2)	(3)	(4)
CO, quarter of death	0.1112**			0.0925
	[0.0554]			[0.0616]
PM10, quarter of death		0.0041*		0.0028
		[0.0022]		[0.0026]
Ozone, quarter of death			0	−0.0005
			[0.0031]	[0.0033]
# Observations	29452	29452	29452	29452
R^2	0.17	0.17	0.17	0.17

The dependent variable in all cases is events per 1000, per quarter. To get rates per 100,000, multiply by 100. Robust standard errors in brackets. * and ** indicate significance at the 5 percent and 1 percent levels, respectively. Specifications are similar to those shown in Tables III and include zip code-quarter fixed effects.

The first panel of Table V shows that in the aggregate-level data, both CO and PM10 have statistically significant effects in the single-pollutant models. It is possible that the significant effects of PM10 in these models reflect cumulative effects (e.g., PM10 is thought to work through the immune system, and it may take time to provoke an immune system response). In Table IV we found larger point estimates for PM10 when we moved to the monthly time unit, or when we examined average weekly cumulative exposures (although these estimates were not statistically significant). Such a cumulative effect might account for the discrepancy between the quarterly models where we find a significant effect of PM10, and the weekly models, where we do not.

The coefficient of .1112 in column (1) indicates that there was a decline of 11.12 deaths per 100,000 births per unit of CO reduction, per quarter, which is a large effect. The point estimate of .0041 on PM10 in column (2) indicates that there was a decline of .41 deaths per 100,000 births per unit of PM10 reduction per quarter, which is smaller than the Chay and Greenstone estimate of the effects of TSPs.[19] The smaller estimate may reflect a nonlinear effect of particulates on infant health, the fact that TSPs are a broader measure than PM10, or a California-specific effect given that Chay and Greenstone use national data.

However, these estimates are not robust to changes in specification. Column (4) shows that when we enter all three pollut-

19. This is true even if we multiply our estimate by four to get an annual measure.

ants, none of them are statistically significant. When we estimate models including zip code fixed effects rather than zip code*quarter effects, only the effect of PM10 remains statistically significant (though the coefficient is of similar magnitude at .0034). We conclude that because responses to CO are acute, it is easier to detect them in models that use high frequency data and control carefully for the age of the child.

V. Discussion and Conclusions

Environmental policy continues to be contentious. For example, the EPA has responded to the threat posed by increased diesel emissions by proposing new rules that would require refiners to phase in cleaner diesel fuel between 2006 and 2010, but the American Petroleum Institute and the National PetroChemical and Refiners Association have filed suit in an effort to block implementation of these standards [Stafford 2001]. 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 have attacked the plan in part because it would not regulate CO production [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 control for many potential confounders, and our richest model is identified using only weekly variation in pollution within zip code, months, and years.

Our most interesting and novel finding is that high levels of postnatal exposure to CO have a significant effect on infant mortality. We believe that this effect has been overlooked because it is an acute effect that is hard to detect in aggregated data. This finding is remarkably robust to many changes in specification and suggests that decreases in CO levels over the 1990s saved about 1000 infant lives in California. These findings are clearly relevant to policy debates over automobile emissions and the Clear Skies Initiative, for example.

Our estimates of the effects of CO are likely to be underes-

timates. Unlike some of the latest epidemiological studies which use personal air quality monitors strapped to persons, we are using a crude proxy for individual exposures. The fact that we use noisy measures means that we are much more likely to falsely accept a null hypothesis of no effect than we are to detect an effect that is not there.[20] Hence, our estimates provide strong evidence of an effect of CO, but do not necessarily rule out effects of other pollutants such as ozone. Our estimates of PM10 are less robust, but given that CO and PM10 are highly correlated and come from many of the same sources, policies aimed at reducing one are likely to have significant effects on ambient levels of both.

Another factor that should be kept in mind is that these estimated effects do not take account of measures parents might have taken to shield infants from the effects of pollution exposure. For example, Neidell [2005a, 2005b] finds that people decrease the amount of time they spend outside in response to the "smog alerts" that are announced when ozone levels are forecasted to exceed a threshold. To the extent that parents of vulnerable infants practice avoidance behavior (e.g., by moving to less polluted areas,[21] keeping windows closed, keeping children inside, or using air filters) our estimates will understate the potential impact of pollution in the absence of such behaviors.

A complete evaluation of the costs and benefits of improvements in air quality is far beyond the scope of this paper.[22] It is likely that the costs of reducing pollution are greater at low levels of pollution than at higher levels. But there are also several reasons why conventional measures of the benefits of pollution abatement (such as the effects of pollution levels on housing prices) might understate them. First, the effects of pollution on infant health are not well-known—that is a starting point for this research. Second, CO is an odorless, colorless gas, and people may not be willing to pay for reductions in pollution that they do not

20. There are two other reasons that we may underestimate effects. First, we do not consider possible lives saved in areas without pollution monitors. However, if these areas did not have monitors because they had little pollution or were sparsely populated, then the impact of this bias is likely to be small. Second, we have excluded births in 1998 due to data problems, so we have eleven years instead of twelve. This would suggest inflating our estimates of the number of lives saved by a factor of 1.09.

21. See, e.g., Coffey [2003] for evidence on mobility because of pollution.

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

observe. Third, to the extent that parents place a lower value on infant health relative to other goods than infants would, the value of their health will not be fully captured by the parents' willingness to pay for pollution reduction.

What is the value, then, of improvements in infant health due to reductions in pollution? If, following Chay and Greenstone [2003a], we value a life at a very conservative $1.6 million, then the estimated reduction in infant deaths due to reduced air pollution in California over the 1990s would be valued at about $1.6 billion.[23] If we use the Environmental Protection Agency's [1999] value of $4.8 million, the benefit would grow to $4.8 billion. We are likely to have underestimated the number of lives saved, and these calculations ignore the value of improvements in the health in infants who are not at the life-death margin. Hence, we regard these estimates as lower bounds on the benefits to infants. But they may still provide a useful benchmark for assessing the benefits of further reductions in air pollution in terms of infant health.

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REFERENCES

Allison, Paul, "Discrete-Time Methods for the Analysis of Event Histories," *Sociological Methodology,* XII (1982), 61–98.
Almond, Douglas, Kenneth Chay, and David Lee, "The Cost of Low Birth Weight," xerox, Department of Economics, University of California, Berkeley, 2002.
Chay, Kenneth, and Michael Greenstone, "The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession," *Quarterly Journal of Economics,* CXVIII (2003a), 1121–1167.
Chay, Kenneth, and Michael Greenstone, "Air Quality, Infant Mortality, and the Clean Air Act of 1970," NBER Working Paper No. 10053, 2003b.
Coffey, Bentley, "A Reexamination of Air Pollution's Effect on Infant Health: Does Mobility Matter?" xerox, Department of Economics, Duke University, 2003.
Currie, Janet, and Jonathan Gruber, "Saving Babies: The Efficacy and Cost of Recent Expansions of Medicaid Eligibility for Pregnant Women," *Journal of Political Economy,* CIV 1996, 1263–1296.
Currie, Janet, and Matthew Neidell, "Air Pollution and Infant Health: What Can We Learn from California's Recent Experience?" NBER Working Paper No. 10251, 2004.
Environmental Defense, "President's Clear Skies Initiative Won't Clean Pollution without Changes," New Release, Jan 28, 2003, www.environmentaldefense.

23. Viscusi [1993] suggests that the value of a life was between $3.5 and $8.5 million, and Environmental Protection Agency [1999] values infant lives lost due to lead at $4.8 million, the same value that they used for adult lives.

org/pressrelease.cfm?ContentID=2629. *The Benefits and Costs of the Clean Air Act, 1990–2010.* Report to the U. S. Congress, November 1999.
——, Environmental Protection Agency, "Automobiles and Carbon Monoxide," EPA Office of Mobile Sources Fact Sheet, 400-F-92-005, January 1993.
——, "National Ambient Air Quality Standards," November 15, 2002, http://www.epa.gov/airs/criteria.html.
——, "Second Prospective Analytical Plan," May 12, 2003a (Environmental Protection Agency Scientific Advisory Board Advisory Council on Clean Air Compliance: Washington, DC).
——, "Criteria Pollutants," February 20, 2003b, http://www.epa.gov/oar/oaqps/greenbk/o3co.html.
Friedman, Michael, Kenneth Powell, Lori Hutwagner, LeRoy Graham, and W. Gerald Teague, "Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma," *Journal of the American Medical Association,* CCLXXXV (2001), 897–905.
Greenstone, Michael, "The Impacts of Environmental Regulations on Industrial Activity: Evidence from the 1970 and 1977 Clean Air Act Amendments and the Census of Manufactures," *Journal of Political Economy,* CX (2002), 1175–1219.
Huel, G., J. Godin, N. Frery, F. Girard, T. Moreau, C. Nessmann, P. Blot, "Aryl Hydrocarbon Hydroxylase Activity in Human Placenta and Threatened Preterm Delivery," *Journal of Exposure Analysis and Environmental Epidemiology,* III (1993), 187–189.
Imbens, Guido, "An Efficient Method of Moments Estimator for Discrete Choice Models with Choice-Based Sampling," *Econometrica,* LX (1992), 1187–1214.
Logan, W. P. D., and M. D. Glasg, "Mortality in London Fog Incident, 1952," *Lancet,* I (1953), 336–338.
Lubin, Jay, and Mitchell Gail, "Biased Selection of Controls for Case-Control Analyses of Cohort Studies," *Biometrics,* XL (1984), 63–75.
Lui, Shiliang, Daniel Krewski, Yuanli Shi, Yue Chen, and Richard Burnett, "Association between Gaseous Ambient Air Pollutants and Adverse Pregnancy Outcomes in Vancouver Canada," *Environmental Health Perspectives,* CXI (2003), 1773–1778.
Maisonet, Mildred, Timothy Bush, Adolfo Correa, and Jouni Jaakkola, "Relation between Ambient Air Pollution and Low Birth Weight in the Northeastern United States," *Environmental Health Perspectives,* CIX (2001), 351–356.
Manski, Charles, and Steven Lerman, "The Estimation of Choice Probabilities from Choice Based Samples," *Econometrica,* XLV (1977), 1977–1988.
Mantel, Nathan, "Synthetic Retrospective Studies and Related Topics," *Biometrics,* XXIX (1973), 479–486.
Neidell, Matthew, "Air Pollution, Health, and Socio-economic Status: The Effect of Outdoor Air Quality on Childhood Asthma," *Journal of Health Economics,* XXIII (2004), 1209–1236.
——, "Public Information and Avoidance Behavior: Do People Respond to Smog Alerts?" CISES Working Paper No. 24, 2005a.
——, "Information, Behavior, and Biology: Understanding the Health Effects of Ozone," CISES Working Paper No. 25, 2005b.
Pope, C. Arden, "Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley," *American Journal of Public Health,* LXXIX (1989), 623–628.
Pope, C. Arden, Joel Schwartz, and Michael Ransom, "Daily Mortality and PM10 Pollution in Utah Valley," *Archives of Environmental Health,* XLVII (1992), 211–216.
Prentice, R. L., and N. E. Breslow, "Retrospective Studies and Failure Time Models," *Biometrika,* LXV (1978), 153–158.
Ransom, Michael, and C. Arden Pope, "Elementary School Absences and PM10 Pollution in Utah Valley," *Environmental Research,* LVIII (1992), 204–219.
Ritz, Beate, and Fei Yu, "The Effects of Ambient Carbon Monoxide on Low Birth Weight among Children Born in Southern California between 1989 and 1993," *Environmental Health Perspectives,* CVII (1999), 17–25.
Ritz, Beate, Fei Yu, Guadalupe Chapa, and Scott Fruin, "Effect of Air Pollution on

Preterm Birth among Children Born in Southern California between 1989 and 1993," *Epidemiology,* XI (2000), 502–511.
Samet, Jonathan, Scott Zeger, Julia Kelsall, Jing Xu, and Laurence Kalstein, "Does Weather Confound or Modify the Association of Particulate Air Pollution with Mortality?" *Environmental Research Section A,* LXXVII (1997), 9–19.
Schwartz, Joel, "Harvesting and Long-Term Exposure Effects in the Relation between Air Pollution and Mortality," *American Journal of Epidemiology,* CLI (2000), 440–448.
Seaton, Anthony, et al., "Particulate Air Pollution and Acute Health Effects," *The Lancet,* CCCLIV (1995), 176–178.
Seig, Holger, V. Kerry Smith, H. Spencer Banzhaf, and Randy Walsh, "Estimating the General Equilibrium Benefits of Large Policy Changes: The Clean Air Act Revisited," NBER Working Paper No. 7744, 2000.
Spengler, John, Jonathan Samet, and J. F. McCarthy, *Indoor Air Quality Handbook* (New York, NY: McGraw Hill Book Co., 2000).
Stafford, Robert T, "An Uphill Drive," Washington, DC: Clean Air Trust, 2001.
Viscusi, W. Kip, "The Value of Risks to Life and Health," *Journal of Economic Literature.* XXXI (1993), 1912–1946.
Wilson, William, David Mage, and Lester Grant, "Estimating Separately Personal Exposure to Ambient and Nonambient Particulate Matter for Epidemiology and Risk Assessment: Why and How," *Journal of the Air Waste Management Association,* L (2000), 1167–1183.