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Author(s): Kenneth Y. Chay and Michael Greenstone

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THE IMPACT OF AIR POLLUTION ON INFANT MORTALITY: EVIDENCE FROM GEOGRAPHIC VARIATION IN POLLUTION SHOCKS INDUCED BY A RECESSION*

KENNETH Y. CHAY AND MICHAEL GREENSTONE

The 1981–1982 recession induced substantial variation across sites in air pollution reductions. This is used to estimate the impact of total suspended particulates (TSPs) on infant mortality. We find that a 1-percent reduction in TSPs results in a 0.35 percent decline in the infant mortality rate at the county level, implying that 2500 fewer infants died from 1980–1982 than would have in the absence of the TSPs reductions. Most of these effects are driven by fewer deaths occurring within one month of birth, suggesting that fetal exposure is a potential pathophysiologic mechanism. The analysis also reveals nonlinear effects of TSPs pollution and greater sensitivity of black infant mortality at the county level. Importantly, the estimates are stable across a variety of specifications.

I. INTRODUCTION

Since at least 1307, when King Edward I banned the burning of coal to protect his citizens' "bodily health," governments have regulated the release of pollution into the air. The 1970 passage of the Clean Air Act Amendments (CAAs) and the establishment of the Environmental Protection Agency (EPA) marked an unprecedented attempt by the U. S. government to mandate lower levels of air pollution. The stated goal of these efforts was to achieve air quality standards that "protect the public health." In the case of total suspended particulates (TSPs), widely thought to be the most pernicious form of air pollution, the EPA set maximum allowable concentrations that every county is required to meet.¹ The health effects of TSPs were presumed to be highly nonlinear and very small below certain thresholds.

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1. The TSPs standard required counties to have annual geometric mean TSPs concentrations less than 75 micrograms-per-cubic meter ($\mu\text{g}/\text{m}^3$) and to have no more than one daily reading that exceeded $260 \mu\text{g}/\text{m}^3$. TSPs consist of all particles with diameters less than or equal to 40 micrometers (μm). The arithmetic mean TSPs concentration in the United States was $93.3 \mu\text{g}/\text{m}^3$ in 1970 and had declined to $69.4 \mu\text{g}/\text{m}^3$ by 1978. For the 1978–1984 period examined in this study, the standard deviation of annual changes in mean TSPs across counties was $9.8 \mu\text{g}/\text{m}^3$. The focus of federal regulation shifted to particulates with dia-

A consensus exists on the link between human health and radically elevated concentrations of TSPs pollution [Holland et al. 1979; Wilson 1996; Wang et al. 1997]. However, the association between health and TSPs at the lower concentrations regulated by the EPA is much more controversial. Evidence on this relationship comes from three types of studies: 1) cross-sectional analyses of the correlation between adult mortality rates and pollution levels across U. S. cities [Lave and Seskin 1977; Pope and Dockerty 1996]; 2) time-series analyses of the correlation between daily adult mortality rates and pollution levels within a given site [Dockery and Pope 1996]; and 3) cohort-based longitudinal studies of adults that suggest that particulates pollution results in excess mortality [Dockery et al. 1993; Pope et al. 1995].

Although this research documents a statistical association between more moderate pollution concentrations and *adult* health outcomes, the reliability of the evidence has been seriously questioned for several reasons. First, since air pollution is not randomly assigned across locations, previous studies may not be adequately controlling for a number of potential confounding determinants of adult mortality [Pope and Dockerty 1996; Fumento 1997]. For example, areas with higher pollution levels also tend to have higher population densities, different economic conditions, and higher crime rates, all of which could impact adult health. Second, in all three types of studies, the lifetime exposure of adults to air pollution is unknown. The analysis implicitly assumes that the current pollution concentration observed at a site accurately measures each resident's lifetime exposure. Third, it is possible that the excess adult deaths that are attributed to changes in air pollution occur among the already sick and represent little loss in life expectancy [Spix et al. 1994; Lipfert and Wyzga 1995].

In the absence of randomized clinical trials, this study uses an event that caused sharp variation in changes in TSPs across sites within a narrow time frame to estimate the impact of air pollution on *infant* mortality. Specifically, we exploit differential changes in TSPs from 1980 to 1982 attributable to geographic variation in the effects of the 1981–1982 recession. The analysis compares changes in infant mortality rates in counties that had

ters less than or equal to $10 \mu\text{m}$ (PM_{10}) and $2.5 \mu\text{m}$ ($\text{PM}_{2.5}$) in 1987 and 1997, respectively.

large reductions in TSPs with the changes in counties with small or no pollution reductions.

The 1980–1982 period provides a compelling setting for estimating the impact of particulates pollution while potentially reducing the role of omitted variables bias. While there is substantial variation across counties in changes in TSPs pollution from 1980–1982, there are few confounding changes in other observed factors. This is notable since, to implement our approach, we bring together the most detailed and comprehensive data available on the determinants of infant health. This includes information on parents' characteristics, the health endowment and medical history of the mother, utilization of medical services (e.g., prenatal care), transfer payments from programs such as Medicaid, and per capita incomes. The 1980–1982 period is unique in this respect, as there appears to be greater potential for confounding in cross-sectional analyses and analysis of changes in the surrounding nonrecession years.

Further, our focus on infant, rather than adult, mortality may circumvent the second and third criticisms of previous research. The problem of unknown lifetime exposure to pollution is significantly mitigated, if not solved, by the low migration rates of pregnant women and infants. Here, TSPs levels are assigned to infants based on the exposure of the mother during pregnancy and the exposure of the newborn during the first few months after birth (we focus on infant deaths within 24 hours, 28 days, and 1 year of birth).² Also, since the mortality rate is higher in the first year of life than in the next twenty years combined [NCHS 1993], it is likely that infant deaths represent a large loss in life expectancy.³

A final advantage of the 1980–1982 period is that the wide variation in the size of the TSPs reductions allows us to examine nonlinearities or thresholds in the effects of pollution on infant mortality. Clearly, the optimality of the EPA's maximum allowable concentration policy depends crucially on the existence and relative magnitudes of the health effects of TSPs above and below the federal regulatory threshold.

2. It is possible that a mother's lifetime exposure to air pollution, and not just exposure while pregnant, may also impact infant health. One might expect any bias arising from unknown lifetime exposure of the mother to be small.

3. Relative to the adult mortality studies, another advantage of an analysis which focuses on infant deaths within one day, one month, and one year of birth is that it circumvents the measurement problem of unpredictable time delays ("delayed causation") in the impact of pollution exposure on eventual death.

The evidence suggests that this quasi-experimental research design provides a more credible basis for evaluating the health impact of TSPs pollution than those used in previous studies. Conventional cross-sectional estimates are very sensitive to specification and provide little evidence of a systematic association between TSPs and infant mortality. Based on our research design, however, we find a significant effect of TSPs reductions on decreases in infant mortality rates at the county level, with a $1\text{-}\mu\text{g}/\text{m}^3$ reduction resulting in about 4–7 fewer infant deaths per 100,000 live births (a 0.35 elasticity). Most of these effects are driven by fewer deaths occurring within one month of birth, suggesting that fetal exposure is a potential pathophysiologic mechanism. Consistent with this hypothesis, we find significant effects of the TSPs reductions on deaths within 24 hours of birth and on infant birth weight.

Importantly, the estimates from our design are robust across a variety of specifications. For example, they are insensitive to the inclusion of many, detailed control variables. As a test of internal validity, we also find that the TSPs reductions are uncorrelated with infant deaths due to accidents and homicides. Since county-level income changes appear to be the most relevant confounder, we further refine the treatment and control groups by matching counties with similar income shocks. This reveals non-linear effects of TSPs on infant mortality at the county level, and large mortality effects at TSPs concentrations well below the EPA mandated ceilings. There is also evidence of greater sensitivity of black infant mortality to the county-level TSPs reductions.

The timing and location of the abrupt trend breaks in both TSPs and infant mortality rates in 1980–1982 provide evidence that air pollution may have a causal effect on infant health. Overall, the estimates imply that about 2500 fewer infants died from 1980–1982 than would have in the absence of the dramatic reductions in particulates pollution.

II. BACKGROUND ON THE LINK BETWEEN AIR POLLUTION AND INFANT MORTALITY

Strong theory and evidence in the biological literature on the pathophysiological mechanism linking air pollution to human health would increase the credibility of an empirical finding of a statistical association. Unfortunately, the underlying biological

pathways are largely unknown.⁴ As one review article notes, "Neither clinical experience nor review of the literature identify a direct pathophysiologic mechanism that can be used to explain the relationship between inhaled particles and mortality" [Utell and Samet 1996, p. 187]. The evidence on the mechanism for infant mortality is even scarcer.

Consequently, it is not surprising that the few previous studies that have examined pollution and infant mortality have used different outcome variables. For example, while one study limits its analysis to neonatal deaths [Joyce, Grossman, and Goldman 1989], another argues that postneonatal mortality is more likely to be affected by environmental factors [Woodruff, Grillo, and Schoendorf 1997]. Similarly, these studies disagree on which causes of death are more likely to be related to air pollution.

As a result of this uncertainty, we examine infant deaths within 24 hours, 28 days (neonatal), and 1 year of birth. In addition, we separately examine infant deaths due to internal health reasons and deaths due to external "nonhealth"-related causes such as accidents and homicides.⁵ Internal infant deaths are not disaggregated further, since coroners assign the vast majority of them to two vague categories. That is, about 70 percent of these deaths are classified as resulting from either "certain conditions originating in the perinatal period" or from "congenital anomalies." Since there is no obvious causal pathway linking air pollution to external causes of death, the estimated association between external mortality rates and TSPs pollution is used as a check on the internal consistency of our findings.⁶

Finally, despite the lack of evidence on causal pathways in the biological literature, we attempt to address whether fetal exposure to pollution has adverse health consequences. There

4. Controlled experiments on animals ranging from guinea pigs to monkeys provide the strongest evidence on the mechanism. These studies find that air pollution exposure can cause a constriction of the bronchial system that impairs lung functioning [Amdur 1996]. Although consistent with some nonexperimental evidence for humans, it is not clear that these findings can be extrapolated to human adults or infants.

5. "Internal" and "external" deaths span all possible causes of death. Deaths with ninth International Classification of Diseases (ICD) codes from 001 to 799 are classified as internal, while those with ICD codes from 800 to 999 are in the external category.

6. That is, the association between TSPs and external infant deaths may result from unobserved secular factors that affect all types of infant death.

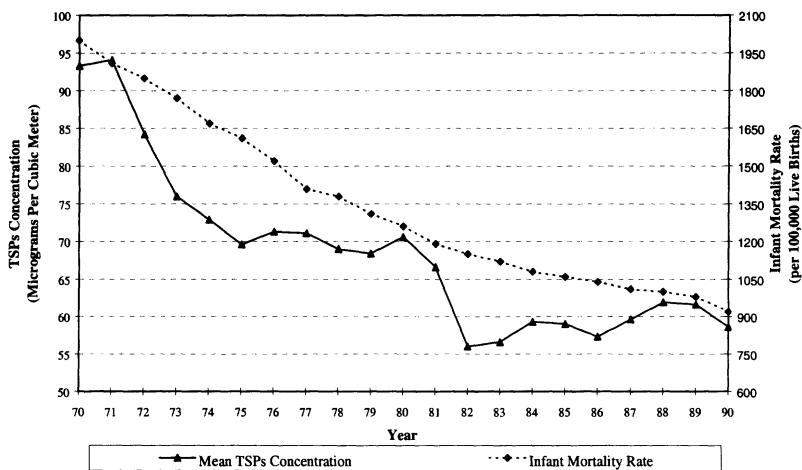


FIGURE I

National Trends in Total Suspended Particulates Air Pollution and Infant Mortality Rates

Source: Authors' tabulations from EPA's *Quick Look Reports* data file and U. S. National Center for Health Statistics.

is strong evidence that maternal cigarette smoking can retard fetal development and reduce the birth weight of infants [Sexton and Hebel 1984; Torelli 2000]. Since air pollution may work through a similar mechanism, we examine the effects of TSPs changes on infant birth weight. In addition, fatalities that occur soon after birth are thought to reflect poor fetal development. As a result, the estimated effects of particulates reductions on death rates within 24 hours and 28 days of birth may also provide evidence on this potential pathophysiologic pathway.

III. CHANGES IN INFANT MORTALITY RATES AND TSPs FROM 1980 TO 1982

To estimate the effects of TSPs pollution on infant mortality, we focus on the 1980–1982 period, a narrow two-year window with one of the most striking changes in air pollution over the last 30 years. Figure I presents trends for the United States in average total suspended particulates pollution and rates of infant mortality within one year of birth due to all causes from 1970–

1990.⁷ It is apparent that air quality improved dramatically in the 1970s and 1980s, with particulate concentrations falling from an average of 95 $\mu\text{g}/\text{m}^3$ to about 60 $\mu\text{g}/\text{m}^3$. However, all of these improvements occurred in two punctuated periods, 1971–1975 and 1980–1982.

Chay and Greenstone [2000] document that most of the improvements in the early 1970s are attributable to federal enforcement of the 1970 CAAAs. For the 1980–1982 period, on the other hand, most of the TSPs reductions can be attributed to the differential impacts of the 1981–1982 recession across counties.⁸ Remarkably, the air quality improvement in this two-year period is nearly as large as the improvement occurring immediately after the 1970 CAAAs. The second series in the figure documents the large fall in the infant mortality rate over the entire period. Although both TSPs concentrations and infant mortality rates trend downward, their time-series correspondence does not provide conclusive evidence of a direct relationship.

Figures IIA, B, and C provide an overview of trends from 1978–1984 in TSPs concentrations, internal infant mortality rates, and per capita income, by TSPs change groups. The counties are divided into three groups with “Big, Middle, and Small” 1980–1982 changes in TSPs based on change quartiles.⁹ Figure IIA illustrates the striking differences across counties in pollution reductions from 1980–1982. The quartile of counties with the largest reduction had a 35 percent decline in TSPs from 1980–1982, on average, while the county group with the smallest reduction had very little change in TSPs. The “Middle Change” set of counties experienced a 20 percent reduction in TSPs. Also note that the “Big Change” group exhibits the most reversion to the mean in TSPs levels in 1983 and 1984.

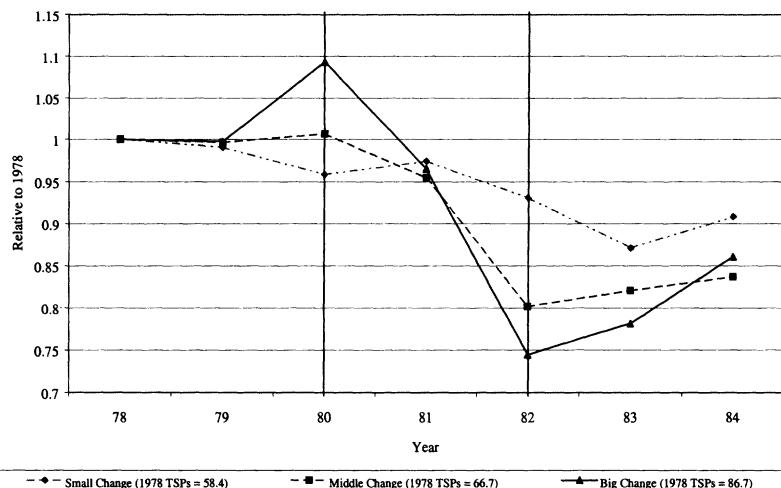
Figure IIB shows that the group of counties that had the largest TSPs reductions also had the biggest declines in internal

7. The TSPs pollution series is based on averages across 1000–1300 counties a year, while infant mortality rates are derived from the entire United States. The counties with TSPs data account for upwards of 80 percent of the U. S. population. Unless otherwise noted, infant mortality refers to death within the first year after birth.

8. For example, there is a strong across-county correlation between 1980–1982 reductions in TSPs and declines in per capita incomes and increases in unemployment rates. Interestingly, both carbon monoxide and ozone pollution concentrations are relatively insensitive to the 1980–1982 recession.

9. The Small and Big Change groups consist of the quartiles of counties with TSPs changes greater than -7.3 units and less than -20.5 units, respectively. The Middle Change group consists of all other counties.

A. Trends in Mean TSPs Concentrations, by 1980-1982 Change in TSPs Concentration



B. Trends in Internal Infant Mortality Rate, by 1980-1982 Change in TSPs Concentration

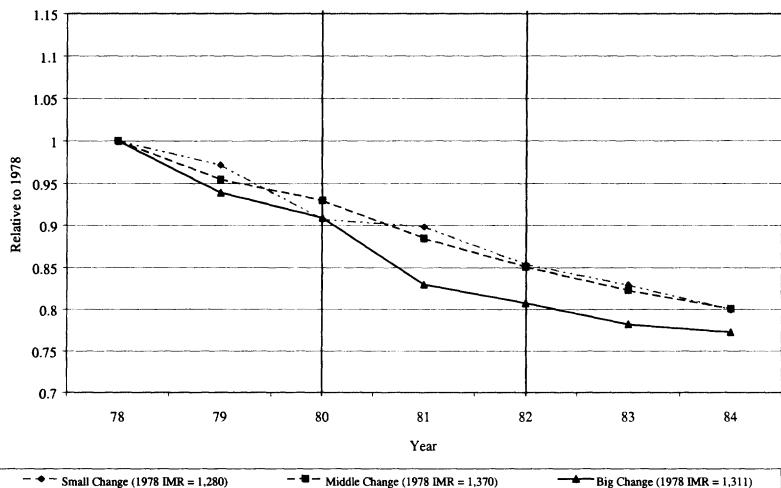


FIGURE II

infant mortality within one year of birth in the narrow 1980–1982 period. While the infant mortality rates of the three groups track each other reasonably well before 1980, there is a clear relative shift down in infant mortality in the largest pollution

C. Trends in Per Capita Income, by 1980-1982 Change in TSPs Concentration

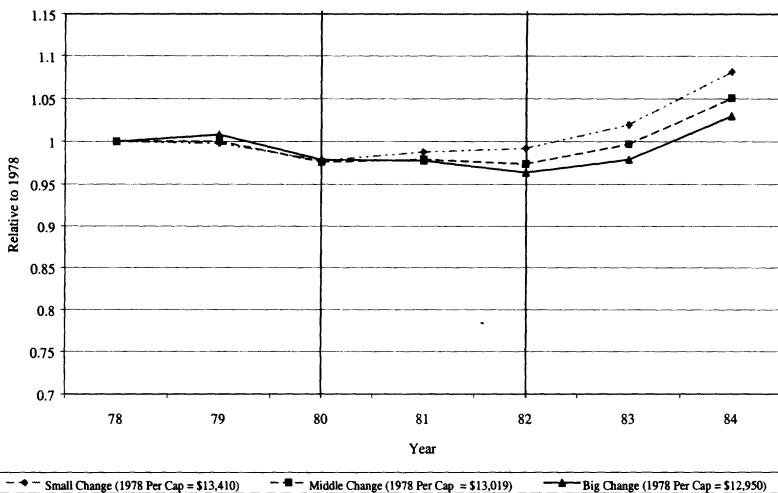


FIGURE II
Trends in TSPs Pollution, Infant Mortality, and Income per Capita,
By 1980–1982 Change in TSPs Groups

decline group in the key period. There is also a trend break in the infant mortality rates of the Middle Change county group relative to the Small Change group during 1980–1982.¹⁰ Taken together, these plots provide suggestive evidence of a direct link between particulates pollution and infant mortality. Based on these raw numbers, the elasticity of infant mortality rate changes with respect to TSPs changes varies between 0.2–0.5, depending on the groups and years used for comparison.

In our analysis, per capita income changes appear to be the most important observable confounder. Figure IIC plots per capita income for each of the three TSPs change groups in Figure IIA. While there is a systematic covariance between changes in TSPs and income, the differences in income shocks across the county

10. The relative trends in TSPs and infant mortality have a strong correspondence for the entire 1978–1984 period. In the postrecession period, for example, the Big Change county group exhibits a reversion back in both TSPs levels and infant mortality rates relative to the other county groups. However, it has greater declines in both TSPs and infant mortality rates over the entire 1980–1984 period. It should also be noted that the Small and Big county groups have similar baseline infant mortality rates in 1978 (1.3 deaths per 100 births) and have nearly identical 4 percent increases in the number of live births from 1980–1982.

groups are small in magnitude during the 1980–1982 evaluation period. From 1980–1982, income fell by only 2.9 percent more in the Big Change group than in the Small Change group. However, since the Big Change group rebounded from the recession at a noticeably slower rate, it had a 5.2 percent smaller increase in per capita income from 1980–1984. As a result, our analysis focuses on the narrow 1980–1982 period in order to reduce the role of confounding biases.

Comparing changes in infant mortality rates across counties that had large and small TSPs reductions from 1980–1982 may provide credible estimates of the health impact of air pollution. The across-county differences in TSPs changes in this narrow time frame are both dramatic and abrupt. For example, the mean 1980–1982 reduction in TSPs among the Big Change counties was $30.3 \mu\text{g}/\text{m}^3$, while the mean reduction in the Small Change group was only $1.6 \mu\text{g}/\text{m}^3$. This difference is 1.5 times greater than the standard deviation of TSPs *levels* during the 1978–1984 period ($19 \mu\text{g}/\text{m}^3$). Given these discontinuous differences in pollution reductions, it seems unlikely that other factors changed as sharply or abruptly in this short period. Below, we find little evidence that any observed factors changed in a way leading to a spurious correlation between TSPs and infant health.

IV. EMPIRICAL METHODOLOGY

Now, we discuss the econometric models used to estimate the infant mortality-TSPs association. For simplicity, it is assumed that the “true” effect of exposure to particulates pollution is homogeneous across infants and over time. As discussed above, the predominant approach used in the literature is cross-sectional analysis. A general cross-sectional model of infant mortality can be written as

$$(1) \quad y_{jt} = f(x_{jt}, z_{jt}, w_{jt}) + \epsilon_{jt},$$

where y_{jt} is the infant mortality rate in county j in year t , x_{jt} is the average TSPs reading across all monitors in the county, z_{jt} is per capita income, w_{jt} is a vector of the other determinants of county-level infant mortality rates (parents' attributes and behavior, county characteristics, etc.), and f is the “nonparametric” conditional mean function of y_{jt} . Thus, $f(\cdot)$ and w_{jt} are defined so that $E(\epsilon_{jt}|x_{jt}, z_{jt}, w_{jt}) = 0$.

Empirical implementation of equation (1) is not practical for

two reasons. First, w_{jt} could be of extremely high dimension, and many of its elements may not be observable to the researcher (e.g., maternal behaviors). Second, $f(\cdot)$ is a general conditional expectation that is a function of the joint distribution of y_{jt} , x_{jt} , z_{jt} , and w_{jt} . Consequently, estimation of the effects of interest is practically infeasible due to the "curse of dimensionality."

A common approach to reducing the dimensionality of the problem is to assume that the effects of the covariates are additive and linear. This results in the linear regression model:

$$(2) \quad y_{jt} = x_{jt}\beta + z_{jt}\theta + w'_{jt}\Pi + \epsilon_{jt}, \quad \epsilon_{jt} = \alpha_j + u_{jt}.$$

The coefficient β is the true effect of TSPs on infant mortality. For consistent estimation, the least squares estimator of β requires that $E[x_{jt} \cdot \epsilon_{jt}] = 0$. If there are omitted permanent (α_j) or transitory (u_{jt}) factors that covary with TSPs levels, then the cross-sectional estimator will be biased. Any nonlinearities or interactions in the true effects will also lead to bias.

To determine the likely reliability of the cross-sectional approach, we examined the across-county association of TSPs levels with other correlates of infant mortality (details in Chay and Greenstone [1999]). We found that TSPs levels systematically covary with almost every variable, including mother's race, marital status, prenatal care usage, education, and age. We also examined the correlation between TSPs levels and *predicted* internal infant mortality rates from a regression of mortality on the other covariates, excluding TSPs. This may be a more direct measure of the importance of observable sources of bias. In the cross sections there is a strong and significant association between predicted mortality rates and TSPs. This suggests that "conventional" cross-sectional estimates could be severely biased.

The ideal research design is a controlled experiment in which different levels of pollution exposure are randomly assigned across mothers/infants. Random assignment ensures that variation in TSPs exposure is independent of other factors (i.e., z_{jt} and w_{jt}) that impact infant mortality. This reduces the dimensionality of the inference problem. However, using randomized clinical trials to study the effects of TSPs exposure on infant health would be unethical.

Our "quasi-experimental" approach uses the sharp variation across sites in TSPs reductions from 1980–1982. There is evidence that this design may significantly reduce the omitted variables problem. For example, there is little systematic covariance

between 1980–1982 TSPs changes and changes in the control variables, with associations that are substantially smaller than the cross-sectional correlations. Not surprisingly, since recessionary shocks drive some of the TSPs reductions, per capita income changes do covary with TSPs changes. However, the correlation between 1980–1982 TSPs changes and the infant mortality rate changes predicted by the other covariates is small and insignificant. Also, this correlation is smaller in the 1980–1982 period than in the periods immediately before (1978–1980) and after (1982–1984) the recession (see Chay and Greenstone [1999]). At least with respect to the observable controls, this research design appears to emulate the dimensionality reduction ensured by controlled experiments.

Assuming linearity of the effects of TSPs, the quasi-experimental model in the ideal case is

$$(3) \quad dy_{jt} = y_{j82} - y_{j80} = x_{j82}\beta - x_{j80}\beta + \epsilon_{j82} - \epsilon_{j80} = dx_{jt}\beta + d\epsilon_{jt}.$$

Below, we estimate this model using a fixed-effects estimator applied to the pooled three years of data from 1980–1982.

In the “almost ideal” situation, TSPs changes may be weakly correlated with changes in other factors, requiring the use of linear regression adjustment. Further, Figure IIB suggests that there are some differences across counties in infant mortality trends before the recession. Thus, we also estimate the following model:

$$(4) \quad dy_{jt} = dx_{jt}\beta + dz_{jt}\theta + dw'_{jt}\Pi + d\epsilon_{jt}, \quad d\epsilon_{jt} = \lambda_{st} + du_{jt},$$

where λ_{st} are state fixed effects in infant mortality changes. Define $T_j^t = (x_{j1}, \dots, x_{jt}, z_{j1}, \dots, z_{jt})$; then the assumption $E(u_{jt}|T_j^t) = 0$ implies that the lag levels of TSPs and income can be used as instrumental variables for dx_{jt} and dz_{jt} . This condition, which presumes that the treatments are predetermined, is robust to differential trends and dynamic feedback from y to T of an unrestricted form. Consequently, it is much weaker than the strict exogeneity condition underlying identification of equation (3).¹¹

Since equations (3) and (4) are based on first-differences, any permanent unobserved differences across counties, α_j , are controlled for. Also, since the key variables are measured at the

11. Chay and Greenstone [1999] also estimate specifications that control for lag mortality rate changes. We find that the estimates are insensitive to this.

county level, in some specifications we control for unrestricted state-time effects in infant mortality levels, λ_{st} . This nonparametrically absorbs all unobservables that vary across states over time. Here, the treatment effect is identified using only comparisons across counties within the same state. We find that the estimated effects of TSPs changes are insensitive to the inclusion of detailed controls and similar across this wide variety of specifications. This provides reassuring evidence on the credibility of the research design.

Before proceeding, we note the potential for censoring bias in our estimates of the effects of TSPs. The analysis is based on the population of live births. Since air pollution may damage the fetus before birth, it may also affect the likelihood of a miscarriage or stillbirth. Consequently, the large pollution reductions during the recession may have resulted in a reduction in fetal deaths. In this case, our analysis will *understate* the impact of TSPs on infant mortality by conditioning on fetuses that survive to a live birth. If a disproportionate number of the "marginal" fetuses that are born alive are in the low end of the birth weight distribution, estimates of the birth weight effects of TSPs will also suffer from these selection biases. We find evidence below that is consistent with this potential selection mechanism.¹²

V. DATA SOURCES

To implement our evaluation strategy, we bring together an unprecedented amount of unique and comprehensive data for the 1978–1984 period. This database allows for a much broader examination, both across sites and over time, than has previously been conducted. Here, we describe the data used in this study. More details on the data sources and variables are contained in the Data Appendix.

The outcome variables and a number of the control variables come from the National Mortality Detail Files and National Natality Detail Files, which are derived from censuses of death and birth certificates. We merge the microdata on infant births and deaths at the county by demographic group level for each year to create infant age at death, race, and gender cells for each county.

12. Since machine-readable data on fetal deaths at the county level are not available for the period of interest, a direct examination of the impact of TSPs on fetal deaths is left for future research.

For each cell, the infant mortality rate is calculated as the ratio of the total number of infant deaths of a certain type (age and cause of death) in a cell in a given year to the total number of births in that cell in the same year. The cell-level rates of death within 24 hours and 28 days of birth are also computed for each year.

The microdata on the control variables available in the Natality Files are also aggregated into annual demographic-county cells. The detailed Natality variables fall into four categories: socioeconomic and demographic characteristics of the parents [Meara 2001]; medical system utilization, including prenatal care usage [Rosenzweig and Schultz 1983; Institute of Medicine 1985]; maternal health endowment, such as mother's age and pregnancy history [Rosenzweig and Schultz 1983; Rosenzweig and Wolpin 1991]; and infant health endowment. As discussed above, since TSPs may affect fetal development, infant birth weight is used as another outcome variable. The Natality variables used in the analysis are described in detail in the Data Appendix.

Annual, monitor-level data on TSPs concentrations were obtained by filing a Freedom of Information Act request with the EPA. This yielded the information collected by the EPA's nationwide network of pollution monitors, including the location of each monitor and their readings. From these data we calculated the mean TSPs concentrations for about 1200 counties per year (see the Data Appendix).

Accounting for across-county differences in income changes during the 1980–1982 period is particularly important [Ettner 1996]. Per capita income data come from the Bureau of Economic Analysis' annual series, which provides the most comprehensive measure of income available at the county level. County-level unemployment rates come from the Employment and Unemployment for State and Local Areas publications of the BLS [Ruhm 2000]. Annual data on manufacturing and nonmanufacturing employment levels in each county come from the County Business Patterns files.

The Regional Economic Information System file provides annual, county-level data on several different categories of transfer payments. These include separate series for total transfer payments; total medical care payments; public expenditures on medical care for low-income individuals (primarily Medicaid and local assistance programs); income maintenance benefits; family assistance payments, including AFDC; Food Stamps payments; and Unemployment Insurance benefits. State-level data on the num-

ber of Medicaid recipients, the cost per recipient, and Medicaid payments to medical vendors come from the Health Care Financing Administration.¹³ Finally, since weather conditions may be a potential confounder [Samet et al. 1998], we obtained monthly temperature and precipitation data for each state from the National Weather Service.

Table I presents summary information from 1978–1984 on the 1050 counties with nonmissing TSPs pollution and per capita income data in both 1980 and 1982. This is the sample used in Figure II and in the matched comparisons analysis below. The top rows of the table show that these counties account for about 80 percent of the 3.3–3.7 million live births that occur in the United States in a given year, making this the most comprehensive study to date. While trending up from 1978–1982, the number of births declined in 1983. This suggests that birthrates in the entire country responded to the 1981–1982 recession with a lag, but were not strongly affected during the 1980–1982 evaluation period.

The next set of rows presents infant fatalities per 100,000 live births. The internal infant mortality rate is about 1.1–1.3 deaths within a year of birth per 100 births, while infant deaths due to external causes are much rarer. Respectively, 40 and 70 percent of all internal infant deaths within a year of birth occur within the first 24 hours and 28 days of birth. In addition, the black infant mortality rate is nearly two times greater than the white rate.

The table also shows national trends in infant mortality, average TSPs concentrations, and per capita income. Internal infant mortality rates decreased steadily from 1978–1984, with a slightly larger percentage decline in 1980–1982 than in the other periods. TSPs, on the other hand, fell by over 20 percent in 1980–1982 but were relatively steady in the “pre” period and exhibit some mean reversion in the “post” period. Per capita income decreased about 2.5 percent from 1978–1980 and remained relatively stable during the key 1980–1982 period before rebounding in 1983 and 1984.

13. Currie and Gruber [1996] find that expansions of state Medicaid programs are associated with improved infant health outcomes. Since these expansions did not begin in earnest until 1984, they are unlikely to be a source of confounding in this study. Notably, counties with large and small TSPs reductions from 1980–1982 had nearly identical changes in public expenditures on medical care for low-income individuals.

TABLE I
SAMPLE STATISTICS, 1978-1984

	1978	1979	1980	1981	1982	1983	1984
Number of counties in sample	1003	1019	1050	1043	1050	988	952
Total births in sample	2,599,010	2,737,663	2,842,817	2,862,625	2,909,385	2,820,255	2,825,214
Total births in U. S.	3,338,300	3,499,795	3,617,981	3,635,515	3,685,457	3,642,821	3,673,568
Fatalities per 100,000 live births							
Internal causes							
At 1 day	516.6	490.4	470.2	452.3	448.0	431.9	419.2
At 1 month	950.9	895.4	853.2	809.6	775.5	733.9	709.4
At 1 year	1336.4	1274.7	1226.2	1166.4	1121.6	1087.4	1059.8
External causes							
At 1 year	41.5	35.3	38.1	33.7	33.8	30.6	28.5
At 1 year all causes, by race							
Whites	1159.5	1111.6	1066.5	1019.9	978.2	946.8	923.8
Blacks	2217.8	2091.5	2059.4	1947.8	1900.2	1844.5	1789.2
Mean county-level pollution, income, and unemployment rate							
TSPs concentration	69.4	69.1	71.1	66.9	56.4	57.0	59.8
Income per capita (\$1982-1984)	\$13,117	\$13,126	\$12,794	\$12,850	\$12,781	\$13,091	\$13,825
Unemployment rate (%)	6.2	5.9	7.2	7.7	9.7	9.6	7.6
Mean parental demographic and socioeconomic characteristics							
% Mother H.S. dropout	22.7	18.0	17.4	16.7	16.2	15.6	14.9
Mother's years of education	12.2	12.4	12.4	12.5	12.5	12.6	12.6
Father's years of education	12.8	12.9	12.9	13.0	12.9	13.1	13.1
% Single mother	17.4	18.1	19.6	20.1	20.5	21.6	22.3
% Black	17.5	17.5	17.2	17.0	16.9	17.0	17.1
% Foreign-born	10.8	11.2	11.6	12.4	12.5	12.8	13.1

<u>Mean medical services utilization</u>						
% No prenatal care	1.42	1.29	1.33	1.38	1.53	1.63
% Prenatal care in 1st trimester	72.0	73.7	74.6	74.8	74.9	75.2
Number of prenatal care visits	9.6	8.8	9.2	9.2	9.3	9.2
<u>Mean maternal health endowment</u>						
% Teenage mother	16.0	15.5	15.0	14.2	13.7	13.2
% Mom >34 years	4.6	4.6	4.7	4.8	5.4	5.9
% First birth	37.2	37.1	36.6	36.8	36.3	35.9
% Prior fetal death	16.1	17.1	17.6	18.8	19.2	19.8
<u>Mean infant health endowment</u>						
Birth weight	3321	3328	3335	3335	3340	3345
% Very low birth weight	1.19	1.17	1.18	1.18	1.20	1.22
% Low birth weight	7.2	7.1	6.9	6.9	6.9	6.8

The following states do not report mother's marital status until 1980: California, Connecticut, Georgia, Maryland, Michigan, Montana, New York, Nevada, Ohio, and Texas. These states are excluded from the calculation of "% Single mother" in 1978 and 1979. Very low birth weight is defined as a birth weight less than 1500 grams; low birth weight is less than 2500 grams. See the Data Appendix for information on sources and more details on the variables. The sample is limited to the 1050 counties with nonmissing data on TSPs concentrations and per capita incomes in both 1980 and 1982. The total number of births in each county is used as weights in calculating the sample means.

The rest of Table I presents 1978–1984 trends for a subset of the large number of control variables used in the analysis. Importantly, the magnitude of changes in these variables is small when compared with the TSPs pollution changes that occurred in the 1980–1982 period. Our measures of parents' demographic and SES characteristics suggest that the SES status of parents rose slightly from 1978–1984, although the fraction of births to single and immigrant mothers increased during the period. Also, while the average number of prenatal care visits was stable during the period, the likelihood of a mother receiving no prenatal care or having a visit in the first trimester increased slightly. The percentage of births attributable to teenagers declined, while the share among women aged 35 and over rose.¹⁴ Finally, the share of first-time births fell slightly while the fraction of mothers who had a prior fetal death rose.

VI. BASIC EMPIRICAL RESULTS

VI.A. Cross-Sectional and Fixed Effects Results

We begin by replicating the conventional cross-sectional approach to estimating the association between TSPs pollution and infant mortality across counties. For each cross section from 1978–1984, Table II presents the regression estimates of the effect of TSPs on the number of internal infant deaths within a year of birth per 100,000 live births. Column (1) is based on a specification that adjusts only for per capita income, while the remaining columns correspond to specifications that include additional sets of controls. The sample sizes and R^2 's of the regressions are shown in brackets.

There is wide variability in the estimated effects of TSPs, both across specifications for a given cross section and across cross sections for a given specification. For example, the estimates in column (1) vary from a significant positive association in 1978 to an insignificant negative correlation in 1984. In column (2) the inclusion of the basic control variables available in the Natality data has a noticeable effect on the point estimates in

14. Rees et al. [1996] find that much of the correlation between poor infant health and mother's age is due to the higher incidence of low birth weight births among teenagers and women 35 and older.

TABLE II
CROSS-SECTIONAL ESTIMATES OF THE ASSOCIATION BETWEEN MEAN TSPS
AND INFANT MORTALITY RATES

	Infant deaths due to internal causes (per 100,000 live births)				
	(1)	(2)	(3)	(4)	(5)
<u>1978 Cross section</u>	1.51 (0.73)	0.44 (0.52)	0.77 (0.58)	0.53 (0.58)	0.76 (0.60)
	[1201, .02]	[1188, .41]	[1180, .48]	[1188, .53]	[1120, .48]
<u>1979 Cross section</u>	0.67 (0.77)	0.37 (0.50)	0.22 (0.50)	0.05 (0.55)	0.16 (0.53)
	[1188, .02]	[1173, .42]	[1163, .51]	[1173, .53]	[1126, .50]
<u>1980 Cross section</u>	0.44 (0.65)	0.36 (0.54)	1.08 (0.48)	1.04 (0.51)	1.06 (0.49)
	[1174, .02]	[1164, .47]	[1154, .54]	[1162, .57]	[1129, .54]
<u>1981 Cross section</u>	−0.19 (0.71)	−0.92 (0.58)	0.23 (0.65)	0.36 (0.68)	0.08 (0.67)
	[1122, .01]	[1112, .42]	[1104, .47]	[1111, .51]	[1077, .47]
<u>1982 Cross section</u>	0.39 (1.06)	−0.20 (0.69)	1.14 (0.87)	1.52 (0.86)	1.14 (0.93)
	[1104, .02]	[1098, .41]	[1091, .49]	[1098, .52]	[1062, .49]
<u>1983 Cross section</u>	1.72 (1.23)	0.64 (0.61)	1.89 (0.65)	2.15 (0.67)	2.03 (0.67)
	[1076, .02]	[1067, .46]	[1060, .50]	[1067, .53]	[1036, .49]
<u>1984 Cross section</u>	−0.30 (0.78)	−0.09 (0.53)	−0.24 (0.68)	0.23 (0.73)	−0.41 (0.70)
	[1029, .02]	[1023, .42]	[1016, .49]	[1023, .52]	[991, .47]
Income per capita	Y	Y	Y	Y	Y
Basic natality variables	N	Y	Y	Y	Y
Unrestricted natality	N	N	Y	Y	Y
Weather	N	N	Y	N	Y
State Medicaid	N	N	N	N	Y
Income assistance sources	N	N	N	N	Y
State effects	N	N	N	Y	N

Estimated standard errors are in parentheses and are based on the Eicker-White formula to correct for heteroskedasticity. Numbers in brackets are the number of counties and R^2 's of the regressions, respectively. Regressions are weighted by numbers of births in each county. Internal causes of death arise from common health problems, such as respiratory and cardiopulmonary deaths. The control variables are listed in the Data Appendix and in Table I. State effects are separate indicator variables for each state.

most years.¹⁵ While the estimates are more precise due to the greatly improved fit of the regressions, none are significant at conventional levels.

Columns (3)–(5) present the results from specifications that include additional Natality variables and controls for weather,

15. The control variables included in the "Basic Natality" and "Unrestricted Natality" sets of variables are listed in the Data Appendix.

TABLE III
FIXED EFFECTS AND INSTRUMENTAL VARIABLES ESTIMATES OF THE EFFECT
OF MEAN TSPs ON INFANT MORTALITY RATES

	Infant deaths due to internal causes (per 100,000 live births)							
	1978–1980 data		1982–1984 data		1980–1982 data		1978–1984 data	
	FE	IV	FE	IV	FE	IV	FE	IV
Mean TSPs	-0.96 (0.67)	4.68 (4.00)	0.66 (0.87)	0.54 (2.87)	3.51 (0.52)	5.21 (1.99)	5.27 (0.40)	3.75 (1.46)
Income per capita (1/10)	0.85 (0.15)	-1.69 (1.01)	-0.47 (0.09)	-0.21 (0.29)	0.00 (0.19)	-2.42 (1.26)	-0.31 (0.07)	-0.78 (0.40)
County fixed effects	Y	Y	Y	Y	Y	Y	Y	Y
Year effects	N	Y	N	Y	N	Y	N	Y
R ²	0.70	0.00	0.69	0.00	0.71	0.00	0.58	0.00
Depend. var. mean	1276	-53.4	1088	-30.4	1170	-49.7	1179	-44.5
Sample size	3563	2172	3209	1994	3400	2099	7894	6265

Regressions are based on the pooled data for the referenced years. The "FE" results are from levels regressions that include county indicator variables as controls. The "IV" results are from two-stage least squares estimation using stacked first-differences, with changes in mean TSPs and per capita income instrumented by first and second lags of TSPs and income levels. The "Year effects" are indicators that allow for unrestricted differences in year-to-year changes. Estimated standard errors are in parentheses and are based on the Eicker-White formula to correct for heteroskedasticity in the FE column and further correct for county-level correlation in the stacked first-differenced errors in the IV column. Regressions are weighted by numbers of births in each county and year.

transfer payments, Medicaid receipt, and unrestricted state effects. Again, the estimated effects vary from a significant positive correlation to an insignificant negative correlation. For the 1980 and 1983 cross sections, the estimated TSPs effects are significant and less sensitive to specification. For the other years, however, the estimates are imprecise or small in magnitude.

The largest estimates from the cross-sectional analyses imply that a 1- $\mu\text{g}/\text{m}^3$ reduction in mean TSPs results in 1–2 fewer internal infant deaths per 100,000 live births, which is a 0.06–0.10 elasticity. However, the modal estimate suggests an imprecise, zero impact of TSPs. Overall, there is little evidence of a systematic association between particulates pollution and infant survival rates in these cross-sectional regressions. In addition, the results are very sensitive to the year analyzed and the set of variables used as controls, suggesting that omitted variables may play an important role.

Table III presents fixed effects (FE column) and instrumental variables (IV column) estimates of the association between mean

TSPs and internal infant mortality rates based on pooled data for the 1978–1980, 1982–1984, 1980–1982, and 1978–1984 periods. The fixed effects results are from levels regressions that include county indicator variables as controls. Including county fixed effects in the regressions eliminates the bias in the cross-sectional estimates attributable to time-invariant omitted factors that vary across counties. However, this approach will be biased if there are unobserved shocks that are correlated with both TSPs and infant mortality rate changes.¹⁶ The instrumental variables results come from estimating equation (4) using first-differences in internal infant mortality rates and TSPs pollution, where indicators are included to allow for unrestricted differences in year-to-year changes (i.e., year effects).¹⁷ The year effects absorb aggregate trends in TSPs and infant mortality. Further, as discussed above, using the first and second lags of TSPs levels as instruments for TSPs changes accounts for differential trends and dynamic feedback—nonstationary sources of bias that could contaminate fixed effects and first-differences estimates. The results are also adjusted for per capita income in the FE columns and income changes, which are instrumented by the first and second lags of income levels, in the IV columns.

Focusing first on the FE columns, including county-specific intercepts improves the fit of the regressions substantially when compared with the cross-sectional regressions in Table II. For the years before the recession (1978–1980), there is an insignificant, negative correlation between changes in TSPs and infant mortality rates. The fixed effects estimate in the postrecession years (1982–1984) is also small in magnitude and imprecise.

The fixed effects results for the 1980–1982 recession period are strikingly different. Changes in TSPs concentrations now have a large and significant correlation with changes in internal infant mortality rates, with a 1- $\mu\text{g}/\text{m}^3$ decline in TSPs associated with 3.5 fewer infant deaths per 100,000 live births. Further, the TSPs coefficient is very precisely estimated due to the sharp variation in TSPs changes during the recession. For the entire 1978–1984 period, the TSPs fixed effects estimate is again precise

16. The pooled cross-sectional estimates (standard errors) of the TSPs effect for the 1978–1980, 1982–1984, 1980–1982, and 1978–1984 periods, are 0.50 (0.68), 0.24 (0.96), 0.67 (0.71), and 1.54 (0.78), respectively. The sampling errors are corrected for county-level correlation in the residuals.

17. For the 1980–1982 period, results from the specification that allows for unrestricted state-year effects are presented in column (4) of Table IV.

and highly significant, implying that a 1- $\mu\text{g}/\text{m}^3$ TSPs reduction results in about 5.2 fewer infant deaths per 100,000 live births. It appears that most of the fixed effects association between TSPs and infant mortality from 1978–1984 is driven by their correlation during the 1980–1982 recession.

The instrumental variables results show that these conclusions remain after accounting for year effects and other sources of dynamic and time-varying omitted variables biases. In contrast to the pre- and postrecession periods, the instrumental variables estimate of the TSPs effect for 1980–1982 is precise and significant. Although larger in magnitude, it is statistically indistinguishable from the 1980–1982 fixed effects estimate due to its larger sampling error.¹⁸ Again, the significant IV estimate of the TSPs effect for the entire 1978–1984 period seems to be solely due to the 1980–1982 period.¹⁹

VI.B. Quasi-Experimental Results

The association between changes in TSPs and infant mortality rates is strong during the 1980–1982 recession and nonexistent in the periods before and after the recession. Further, when compared with the nonrecession years, the 1980–1982 period has much greater variation in TSPs changes that is less correlated with the relatively small changes in the observed covariates. This suggests that the omitted variables problem may be reduced by narrowly focusing on the 1980–1982 period.

Table IV presents the results from estimating equation (4) using first-differences in internal infant mortality rates and TSPs pollution from 1980–1982. The estimates are from two-stage least squares estimation applied to the stacked 1980–1981 and 1981–1982 first-differences of the data.²⁰ To account for nonsta-

18. The greater magnitude of the instrumental variables estimate suggests that the fixed effects estimate is biased down by not accounting for differential trends. For example, Figure IIB shows that the prerecession (i.e., 1979–1980) reductions in infant mortality in the “Middle Change” and “Big Change” counties were smaller than the reduction in the “Small Change” counties. To the extent that the TSPs data imperfectly measure individual exposure to pollution, the instrumental variables estimates may also suffer from less attenuation bias than the fixed effects estimates.

19. The IV estimates for the 1978–1980 and 1982–1984 periods are never significant and are very sensitive to adjustment for additional controls (the same as those used in Table IV). The IV estimates for the entire 1978–1984 period are always significant and relatively stable across specifications.

20. The sampling errors for the instrumental variables estimates allow for county-level correlation in the stacked first-differenced residuals. Note that if u_{jt} is independent over time, $\text{cov}(du_{jt}, du_{jt-1}) < 0$.

TABLE IV
INSTRUMENTAL VARIABLES ESTIMATES OF THE EFFECT OF MEAN TSPS ON INFANT
MORTALITY RATES, BASED ON 1980–1982 CHANGES

	Infant deaths due to internal causes (per 100,000 live births)				
	(1)	(2)	(3)	(4)	(5)
<u>Deaths w/in 1 year</u>					
Mean TSPs	5.21	5.44	4.72	5.21	4.93
	(1.99)	(1.96)	(1.70)	(1.68)	(1.73)
Income per capita (1/10)	−2.42	−2.56	−1.99	−2.88	−2.37
	(1.26)	(1.24)	(1.38)	(1.36)	(1.32)
<u>Deaths w/in 28 days</u>					
Mean TSPs	3.92	4.24	3.83	3.81	3.87
	(1.69)	(1.75)	(1.46)	(1.49)	(1.52)
<u>Deaths w/in 24 hours</u>					
Mean TSPs	3.49	3.84	2.97	2.53	3.01
	(1.32)	(1.41)	(1.07)	(1.08)	(1.11)
Basic natality variables	N	Y	Y	Y	Y
Unrestricted natality	N	N	Y	Y	Y
Weather	N	N	Y	N	Y
State Medicaid	N	N	N	N	Y
Income assistance sources	N	N	N	N	Y
Year effects	Y	Y	Y	N	Y
State-year effects	N	N	N	Y	N
Sample size	2099	2078	2061	2075	2011

Results are from two-stage least squares estimation using the stacked 1980–1981 and 1981–1982 changes, with changes in mean TSPs and per capita income instrumented by first and second lags of TSPs and income levels. The state-year effects in column (4) are unrestricted state fixed effects in year-to-year changes (i.e., separate state fixed effects for both the 1980–1981 and 1981–1982 first differences). Estimated standard errors are in parentheses and allow for heteroskedasticity and county-level correlation in the stacked first-differenced errors. Regressions are weighted by numbers of births in each county and year.

tional sources of bias, the first and second lags of TSPs and income levels are used as instruments for changes in TSPs and income. The columns correspond to specifications that include additional sets of controls, as in Table II. All specifications further adjust for year effects, with the specification in column (4) including separate state fixed effects in both the 1980–1981 and 1981–1982 first differences, i.e., unrestricted state-year effects in infant mortality levels.

The first set of rows shows the results for infant mortality within one year of birth. The specification in column (1) is the most parsimonious and identical to the one used in the IV column of Table III. The estimated TSPs effect is significant at conventional levels and implies that a $1\text{-}\mu\text{g}/\text{m}^3$ decline in TSPs results in

5.2 fewer infant deaths per 100,000 live births. The instrumental variables estimate of the income effect is marginally significant and implies that counties that had larger declines in per capita income experienced greater increases in infant mortality rates during the recession.

In columns (2)–(5) the estimated TSPs coefficient is insensitive to adding the Natality variables, controls for weather, Medicaid receipt, transfer payments, and state-year effects. The estimates are significant and imply that a $1\text{-}\mu\text{g}/\text{m}^3$ TSPs reduction leads to 4.7–5.4 fewer infant deaths per 100,000 live births. Further, their precision improves as more variables are added to the analysis, for example, when unrestricted state-year effects are included in column (4). This provides reassuring evidence on the credibility of the analysis and estimates. The estimated effect of per capita income is more sensitive to specification and insignificant at the 5 percent level in columns (3) and (5).

In direct contrast to the cross-sectional estimates, the estimated TSPs effect based on 1980–1982 changes is much larger in magnitude, significant at the 1-percent level, and stable across specifications. Further, in columns (2)–(5) only the coefficients on the indicators for twins or greater birth and no prenatal care have *t*-ratios similar in magnitude to the *t*-ratio of the TSPs coefficient.²¹ This suggests that the “signal-to-noise” ratio for evaluating the effect of TSPs on infant mortality is high in this design.

Overall, the estimates suggest that a $1\text{-}\mu\text{g}/\text{m}^3$ decline in TSPs is associated with five fewer infant deaths per 100,000 live births, which is an elasticity of 0.3. This is 2.5 times greater than the largest cross-sectional estimate in Table II. It implies that an area experiencing a $25\text{-}\mu\text{g}/\text{m}^3$ reduction in TSPs during the recession (e.g., Pittsburgh) had 125 fewer infant deaths per 100,000 births than would have otherwise occurred. This is a 10 percent decrease in the average infant mortality rate. For the counties in our sample, the internal infant death rate fell by an average of 101.5 per 100,000 births from 1980–1982. Thus, the estimates also imply that over 70 percent of the overall reduction in infant mortality from 1980–1982 may be attributed to the fifteen-unit average reduction in TSPs.

21. Receiving no prenatal care and having a twins or greater birth are both associated with higher rates of infant death. The estimated coefficients on almost all of the other variables (e.g., parents' demographic and socioeconomic characteristics) are statistically insignificant.

VI.C. Evidence on the Pathophysiologic Mechanism

As discussed above, fetal exposure to particulates pollution may be one biological mechanism leading to lower infant survival rates. Since deaths occurring soon after birth are thought to reflect poor fetal development, we estimate the effects of the 1980–1982 TSPs changes on internal infant mortality rates within 28 days and 24 hours of birth.

The remaining rows of Table IV contain the estimation results for early infant death based on the same specifications as before. For neonatal mortality the estimates are highly significant and imply that a 1- $\mu\text{g}/\text{m}^3$ decline in TSPs results in 3.8–4.2 fewer deaths within one month of birth (per 100,000 births). As before, the estimated TSPs coefficient is stable across specifications and its precision increases as variables are added to the analysis. Infant deaths within 24 hours of birth also have a strong association with 1980–1982 changes in TSPs. While slightly more sensitive to specification, the instrumental variables estimate varies between 2.5–3.8 and also has greater precision as more controls are added.

These results suggest that particulates pollution has a disproportionate impact on the probability of death soon after birth. The estimates imply that 80 percent of the effect of TSPs on infant mortality is due to reductions in neonatal mortality. Similarly, 60–70 percent of the TSPs effect is driven by fewer deaths within 24 hours of birth. By contrast, Table I shows that less than 70 and 40 percent of all internal infant deaths occur within the first month and day after birth, respectively. For the counties in our sample, the neonatal mortality rate fell by an average of 75.2 per 100,000 births from 1980–1982, while the 24-hour mortality rate fell by 20.3. Thus, the results imply that 80 percent of the overall 1980–1982 decrease in neonatal mortality rates can be attributed to the fifteen-unit average reduction in TSPs. Further, they suggest that the overall 24-hour mortality rate would have increased from 1980–1982 in the absence of the TSPs reduction.

It appears that maternal exposure to TSPs pollution during the gestation period may affect fetal development and health. To further address this possibility, Table V presents the 1980–1982 instrumental variables estimates of the association between TSPs changes and the birth weight of infants, based on the specification that adjusts for unrestricted state-

TABLE V
INSTRUMENTAL VARIABLES ESTIMATES OF EFFECTS OF MEAN TSPS ON INFANT
BIRTH WEIGHT, 1980–1982

	Birth weight	Incidence in birth weight categories (per 100,000 live births)					
		<1500 g	<2000 g	<2500 g	<3000 g	<3500 g	<4000 g
Mean TSPs	−0.317 (0.121)	1.54 (1.80)	0.17 (2.42)	1.96 (4.69)	19.70 (8.05)	16.40 (10.87)	8.05 (5.61)
Income per capita (1/10)	0.242 (0.083)	−1.51 (1.24)	−1.74 (1.59)	−6.20 (3.02)	−14.70 (5.15)	−12.80 (7.19)	−6.62 (4.14)
Basic natality variables	Y	Y	Y	Y	Y	Y	Y
Unrestricted natality	Y	Y	Y	Y	Y	Y	Y
State-year effects	Y	Y	Y	Y	Y	Y	Y
Dependent var. mean	3337	1.2%	2.5%	6.9%	23.3%	60.4%	89.2%
Sample size	2075	2075	2075	2075	2075	2075	2075

Results are from two-stage least squares estimation using the stacked 1980–1981 and 1981–1982 changes, with changes in mean TSPs and per capita income instrumented by first and second lags of TSPs and income levels. The dependent variables in the "Incidence in birth weight categories" columns are the fraction of births with birth weight less than the specified amount. The dependent variable means are for the *levels* of the outcome variables. Estimated standard errors are in parentheses and allow for heteroskedasticity and county-level correlation in the stacked first-differenced errors. Regressions are weighted by numbers of births in each county and year.

year effects (column (4) of Table IV).²² The first column presents the estimated effect of TSPs on average infant birth weight. The remaining columns contain the estimated effects on the probability that a birth occurs in a specific birth weight category. In these columns the dependent variables are the fractions of births in the county with birth weight less than 1500 grams to 4000 grams, respectively.

The estimate in column (1) implies that a 1- $\mu\text{g}/\text{m}^3$ reduction in TSPs is associated with a 0.3 gram increase in the average birth weight of infants in a county. While significant at the 1-percent level, this estimate is small in magnitude. For example, in counties experiencing a 25-unit TSPs decline during the recession, the estimate implies that infant birth weights increased by

22. The fixed effects estimates and instrumental variables estimates based on the other specifications lead to the same conclusions.

7.5 grams, on average. This is an elasticity of only 0.006. Per capita income growth is associated with increasing birth weight at the county level. It is worth noting that in both the pre-(1978–1980) and postrecession (1982–1984) periods, the estimated association between TSPs changes and birth weight is small and insignificant.

The other columns in Table V provide a more detailed picture of the effect of TSPs on the entire distribution of birth weights. The association between TSPs and the incidence of low birth weight (i.e., less than 2500 grams) is positive but not significant.²³ However, TSPs have a significant impact on the probability of “relatively” low weight births. A unit reduction in TSPs results in twenty fewer infants born with a weight less than 3000 grams, per 100,000 live births. This is an elasticity of 0.07. Taken literally, the results suggest that the entire effect of TSPs on average birth weight is due to fewer infants with birth weights between 2500–3000 grams.

Overall, it appears that TSPs pollution has a much larger impact on infant mortality than on birth weight.²⁴ Further, the estimated effect of TSPs on infant mortality falls very little when flexible controls for birth weight are included in the instrumental variables specifications.²⁵ This implies that most of the effect of TSPs on infant mortality is independent of their effect on birth weight. This finding contrasts sharply with the documented effects of maternal cigarette smoking on infant health and suggests that particulates pollution works through a different causal pathway.²⁶

It should be noted that our estimates of the effects of TSPs

23. Wang et al. [1997] document a positive correlation between TSPs and low birth weight incidence in China, where TSPs levels are six times greater than the concentrations in this study.

24. Interestingly, the estimated association between TSPs changes and 5-minute APGAR scores is significant and an order of magnitude larger than the estimated birth weight effects (results available from the authors). Almond, Chay, and Lee [2002] provide evidence that the 5-minute APGAR—a diagnostic test that scores newborn's heart rate, respiratory effort, muscle tone, reflex irritability, and color and is administered immediately after birth—may be a more reliable proxy for health at birth than birth weight.

25. For example, we added controls for average birth weight and eight birth weight category dummies to instrumental variables specification 4 in Table IV, which includes state-year effects. The resulting estimates of the effect of TSPs on one-year, one-month, and one-day mortality are 4.83 (1.66), 3.27 (1.43), and 2.30 (0.99), respectively.

26. Torelli [2000] provides convincing evidence that while maternal smoking has a strong association with reduced birth weight, it is uncorrelated with infant mortality after adjustment for confounding factors. Cigarette smoking during pregnancy is thought to slow fetal growth by depriving the fetus of oxygen.

on birth weight may be biased down due to censoring. Specifically, if declines in TSPs also reduce the likelihood of a miscarriage or stillbirth, then conditioning on fetuses that survive to birth will lead to selection bias. For example, the estimates for the very low birth weight category (i.e., less than 1500 grams) will be severely biased if a disproportionate number of the marginal fetuses that survive are in the low end of the birth weight distribution.²⁷ Further, our estimates of the mortality effects may also be understated, since the sample of live births could include a higher fraction of infants with low survival probabilities.

VII. ROBUSTNESS OF FINDINGS

VII.A. Internal Validity of the Results

As discussed earlier, there is no obvious causal pathway linking air pollution to external causes of infant death, such as accidents and homicides. Thus, we use the estimated association between 1980–1982 changes in external infant mortality rates and TSPs to evaluate the internal consistency of the findings above. While a weak association does not prove that the estimated effects of TSPs on infant mortality are causal, a significant, positive relation would suggest that the analysis is biased by omitted factors that coincided with the TSPs reductions during the recession.

Table VI presents fixed effects (first column) and instrumental variables (remaining columns) estimates of the impact of 1980–1982 TSPs changes on external infant mortality rates. In the first column the fixed effects correlation is small and insignificant at conventional levels. The instrumental variables estimates in the next four columns show a small and insignificant negative correlation between changes in TSPs and external mortality rates from 1980–1982. On the whole, these results provide

27. Experimental studies find similar patterns for the birth weight effects of maternal smoking. Sexton and Hebel [1984] find that the incidence of low birth weight births is lower in the treatment group, which is encouraged to stop smoking, than in the control group, which is not. However, the probability of a very low birth weight birth is slightly *higher* in the treatment group. They also find that the treatment group has a lower incidence of still births than the control group. However, their sample size (935 women) is too small for a reliable conclusion.

TABLE VI
ESTIMATES OF THE EFFECT OF MEAN TSPS ON INFANT DEATHS DUE TO *EXTERNAL CAUSES*, BASED ON 1980–1982 CHANGES

Fixed effects	Infant deaths due to external causes (per 100,000 live births)				
	Instrumental variables applied to first differences				
	(1)	(2)	(3)	(4)	
Mean TSPs	0.14 (0.08)	-0.48 (0.35)	-0.43 (0.32)	-0.46 (0.36)	-0.34 (0.36)
Income per capita (1/10)	-0.03 (0.03)	0.32 (0.21)	0.34 (0.19)	0.76 (0.28)	0.51 (0.27)
Basic natality variables	N	N	Y	Y	Y
Unrestricted natality	N	N	N	Y	Y
Weather	N	N	N	Y	N
Year effects	N	Y	Y	Y	N
State-year effects	N	N	N	N	Y
Sample size	3400	2099	2078	2061	2075

The dependent variable is number of infant deaths attributable to external causes, such as accidents and homicides, per 100,000 live births. The fixed-effects results are from a regression of pooled 1980, 1981, and 1982 data with county indicators as controls. Results in the "Instrumental variables" columns (1)–(4) are from two-stage least squares estimation using the stacked 1980–1981 and 1981–1982 changes, with changes in mean TSPs and per capita income instrumented by first and second lags of TSPs and income levels. Estimated standard errors are in parentheses and allow for heteroskedasticity and county-level correlation in the stacked first-differenced errors. Regressions are weighted by numbers of births in each county and year.

no evidence that our estimates in Table IV are the result of spurious relations.

VII.B. Matching on Income Shocks

Of all of the observable covariates, county-level income shocks have the greatest systematic correlation with 1980–1982 TSPs changes. If higher incomes lead to improved infant health outcomes, then these confounding shocks may bias our estimates of the TSPs effects downward.

We use two methods to further purge the estimates of the influence of changing economic conditions. First, we refine the treatment and control groups by matching counties with similar income changes in the 1980–1982 period. This matching scheme permits a nonlinear association between income shocks and infant mortality. It also allows us to analyze interactions in the effects of TSPs and income changes and the size of the TSPs

effects below the EPA mandated air quality standard. Second, we limit the sample to counties with low employment levels in manufacturing that do and do not neighbor counties with high manufacturing employment levels. The idea is to isolate the counties that had a TSPs reduction solely because of their location, and not due to an economic shock within their borders.

Chay and Greenstone [1999] find that 1980–1982 changes in per capita income provide a reasonable single-index summary of county-level changes in most of the control variables.²⁸ However, the functional forms of the relations between income changes and changes in infant mortality rates and TSPs pollution are unknown. Thus, we match groups of counties with similar changes in per capita income from 1980–1982 but different changes in TSPs. We then compare infant mortality rate changes across the matched groups. This approach is more robust to confounding due to income shocks. Further, if the change in income is a valid single index for other confounding changes, adjustment by subclassification on this variable will remove most sources of bias [Cochran 1968; Rosenbaum and Rubin 1984].

To implement the matching estimator, we classify counties into “Small, Medium, and Big” TSPs change categories based on their 1980–1982 changes. The Small and Big categories consist of the counties in the bottom and top quartiles of TSPs changes, while the Medium category contains the 25–75 interquartile range of counties. Counties are also given “Small, Medium, and Big” income change designations based on the quartiles of 1980–1982 income changes.²⁹ The intersection of these two sets of subclassifications yields nine cells (e.g., Big TSPs shock/Small income shock, Small TSPs shock/Small income shock, etc.). We then match county groups in different TSPs change cells but identical income change cells, and regress 1980–1982 changes in infant mortality on changes in TSPs for each matched subsample of counties.³⁰

The first three rows of Table VII present the estimated effects

28. For example, matching counties with similar income changes eliminates most of the differences in changes in the observables between counties with large and small (and medium and small) TSPs reductions [Chay and Greenstone 1999].

29. The Small and Big TSPs change groups consist of the quartiles of counties with 1980–1982 changes greater than -7.3 units and less than -20.5 units. The Small and Big income change groups consist of the quartiles of counties with 1980–1982 changes greater than \$200 per capita and less than \$(-350) per capita.

30. Focusing on total changes over the 1980–1982 period may reduce the ambiguity concerning the exact timing of the changes. For example, it is not clear whether per capita income in 1982 is a “pre- or postexposure” variable. Using the

TABLE VII
 MATCHED GROUP ESTIMATES OF EFFECTS OF 1980–1982 MEAN TSPs CHANGES
 ON INFANT MORTALITY RATES, MATCHING COUNTIES WITH SIMILAR
 1980–1982 INCOME CHANGES

	Infant deaths due to internal causes (per 100,000 live births)	
	Big vs. small TSPs change	Medium vs. small TSPs change
Small income shock	4.61 (1.59)	5.97 (2.02)
Medium income shock	4.46 (0.81)	6.65 (1.19)
Big income shock	2.51 (0.96)	6.91 (2.06)
Overall	4.16 (0.58)	6.57 (0.95)
Black infants	10.06 (2.18)	9.59 (2.53)
White infants	3.28 (0.62)	5.56 (0.97)

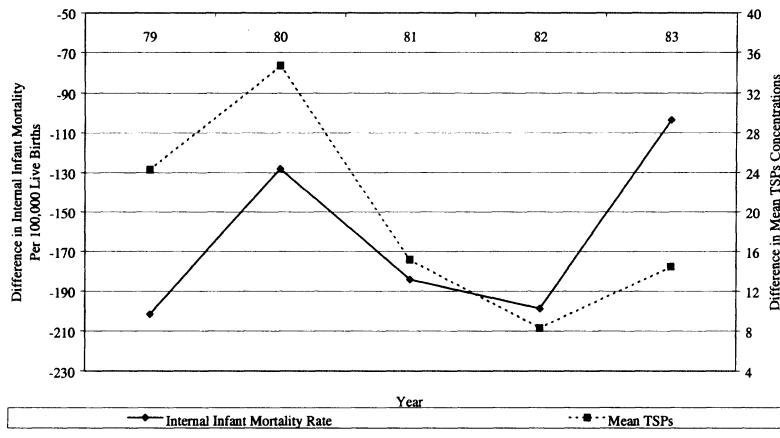
Entries are from regressions of 1980–1982 changes in infant mortality rates on 1980–1982 changes in mean TSPs. For "Small," "Medium," and "Big income shock" rows, regressions do not adjust for 1980–1982 per capita income changes. For remaining rows, results are regression-adjusted for income changes. For the "Black" and "White infants" columns, dependent variables are the race-specific infant mortality rates within one year of birth. Estimated standard errors are in parentheses and are based on the Eicker-White formula to correct for heteroskedasticity. Regressions are weighted by (race-specific) numbers of births in each county and year.

of TSPs on internal infant mortality rates based on this matching procedure. We show the estimates derived from matching "Big and Small" (first column) and "Medium and Small" (second column) TSPs change groups, who are in identical Small, Medium, and Big income shocks groups (three rows). While the estimated effects of TSPs vary from about 4 to 7, they are similar across the different income shock categories within both sets of TSPs change comparisons. There appears to be little interaction effect between TSPs changes and income changes. Also, the estimates are highly significant and do not change when controls for per capita income are included, suggesting that the crude subclassification scheme is accurate.

Figure III depicts the 1979–1983 time-series patterns of the

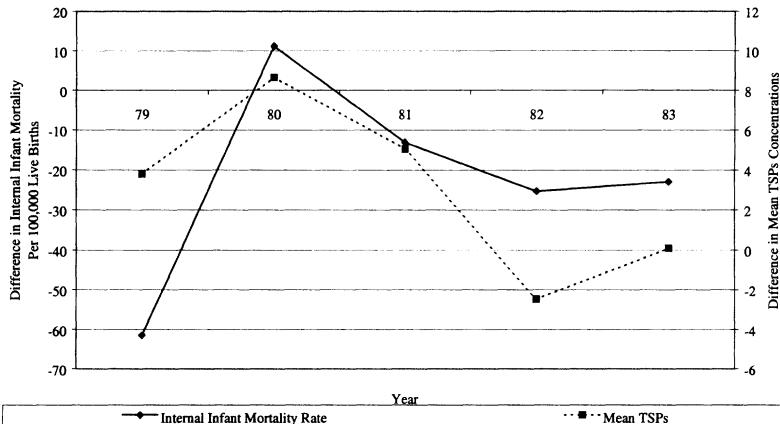
accumulated 1980–1982 changes as a proxy for true changes circumvents some of these timing issues.

A. Differences Between the Groups with Big and Small TSPs Changes
and Small Income Shocks during 1980-82¹



¹ See text for a description of how the Big/Medium/Small categories were determined for TSPs and Per Capita Income.

B. Differences Between the Groups with Medium and Small TSPs Changes
and Small Income Shocks during 1980-82¹



¹ See text for a description of how the Big/Medium/Small categories were determined for TSPs and Per Capita Income.

FIGURE III
Annual Differences in Internal Infant Mortality and Mean TSPs Pollution
between Comparison Groups in Table VII (Row 1)

raw differences in TSPs levels and internal infant mortality rates between the big and small (Panel A) and medium and small (Panel B) TSPs change groups that had small income shocks from

1980–1982 (first row of Table VII). These comparisons are particularly interesting since counties experiencing income growth from 1980–1982 may have been less affected by recessionary economic shocks. In both graphs, the correspondence of the two series is striking, especially the timing of the abrupt trend breaks centered in 1980–1982. The “sawtooth” patterns that emerge in both series provide evidence consistent with a causal relationship between changes in TSPs pollution and infant mortality. It seems unlikely that any unobserved factors changed differentially in these comparison groups in the same sawtooth manner during the key period. Also, differences in neonatal mortality rates exhibit identical patterns.

The estimated effect of TSPs appears to be systematically higher in the comparisons between medium and small TSPs change county groups than in the comparisons between big and small change groups. The next row of Table VII presents the pooled estimates of the TSPs effects for the two sets of comparisons, based on the 1980–1982 first-differences regression adjusted for per capita income. For a 1- $\mu\text{g}/\text{m}^3$ reduction in mean TSPs, there are 4.2 and 6.6 fewer infant deaths per 100,000 live births in counties with big and medium declines in TSPs, respectively, when compared with counties with no average change in TSPs.³¹ The difference between these estimates is significant at conventional levels. Due to the differences in initial 1980 TSPs levels, the implied elasticities of 0.33 and 0.35 are similar.

This analysis reveals a nonlinear effect of TSPs reductions on internal infant mortality rates at the county level.³² From 1971 to 1987 the EPA required all counties to achieve annual geometric mean concentrations of TSPs less than 75 $\mu\text{g}/\text{m}^3$. Almost all of the counties in the medium TSPs change group had 1980 TSPs levels below this threshold. Thus, taken literally, the results imply that TSPs pollution has large effects on infant mortality at concentrations below the EPA mandated ceilings.

There are several possible explanations for the nonlinear response observed at the county level. First, individual infants may have nonlinear reactions to TSPs reductions that depend on

31. The overall first-differences estimate (standard error) of the TSPs effect, adjusted for per capita income, is 4.88 (0.54). The first-differences estimate of the income effect is -0.19 (0.19).

32. Using a uniform kernel regression smoother to estimate the nonparametric relation between 1980–1982 changes in infant mortality and TSPs leads to similar findings (results available from the authors).

the initial level of exposure. Second, if there are heterogeneous responses to TSPs declines, then differences in average population characteristics across counties could generate variation in the estimated TSPs effects. Third, since TSPs pollution is measured at the county level, one could observe different responses across counties if different populations are benefiting from the pollution reductions due to residential segregation. Finally, non-linear responses could also result from differences across counties in the within-county distribution of maternal health endowments and other predictors of infant mortality.

Given the data constraints, we cannot plausibly differentiate these competing hypotheses. However, since the births and deaths microdata provide the race of the infant, we can separately calculate infant mortality rates by race within a county, which allows us to stratify the analysis by race. This is interesting for two related reasons: 1) the black infant mortality rate is almost two times greater than the white rate; and 2) race is correlated with other important factors, such as parents' socioeconomic status and access to health care services.

The final two rows of Table VII present the 1980–1982 first-differences estimates of the effect of TSPs separately for black and white infants. It is clear that black infant mortality is more sensitive to county-level TSPs reductions than white infant mortality.³³ For the medium and small TSPs change group comparisons, the elasticities for black (0.31) and white (0.35) infants are similar. However, for the big and small TSPs change group comparisons, the black elasticity (0.46) is much larger than the white elasticity (0.29). Two potential reasons for this are 1) racial differences in the health endowment of pregnant women and their access to medical care may interact with the effects of TSPs changes; and 2) since TSPs levels are measured at the county level, residential segregation may lead to racial differences in the benefits of TSPs reductions within a county. Thus, response heterogeneity may be one factor underlying the nonlinear effect of TSPs at the county level. However, given that the nonlinearity persists for white infants, a nonlinear infant response function may also play a role.

33. The overall first-differences estimates are 9.56 (1.67) for black infants and 3.95 (0.57) for white infants. Fixed effects and instrumental variables estimation leads to similar ratios for the black-white TSPs coefficients, although the instrumental variables estimates are imprecise.

VII.C. "Neighboring" Counties

Another approach to reducing bias due to confounding economic shocks is to match counties with little activity in manufacturing before the recession. If manufacturing activity in 1980 is a reasonable proxy for the economic impact of the recession, counties with low levels of manufacturing employment may have experienced relatively small economic shocks.³⁴ Thus, we compare the changes in TSPs and infant mortality rates of counties with low manufacturing employment levels that do and do not border counties with high manufacturing employment levels. Counties that neighbor areas where the recession had a heavy impact may have experienced air quality improvements due to local wind patterns instead of an economic downturn within their borders.³⁵ Conversely, sites that did not contain pollution-intensive manufacturing and did not neighbor such counties may have experienced minimal TSPs changes.

To implement this approach, we use the Contiguous County data file [U. S. Census Bureau 1991], which provides information on the locational relationship between counties. From the file we can determine all counties that are physically adjacent to any base county.³⁶ We match two sets of counties with low employment in manufacturing: 1) counties with at least one neighbor that has high employment in manufacturing; and 2) counties that are surrounded only by areas that also have low employment in manufacturing. Counties are defined to have high or low manufacturing employment levels if they are in the top (over 14,000) or bottom (under 1800) quartiles of manufacturing employment in 1980.

We estimate the effect of TSPs on infant mortality rates using the 1980–1982 first-differences specification. The estimated TSPs coefficient (standard error) for this subsample of counties is 4.72 (2.42), which is nearly identical to the estimates in Table IV. Also, it is significant at the 5 percent level even

34. For example, we find that counties with low employment levels in manufacturing in 1980 had relatively small decreases (increases) in per capita income (unemployment rates) from 1980–1982.

35. Cleveland et al. [1976] and Cleveland and Graedel [1979] provide evidence on the "trans-boundary" nature of air pollution (e.g., wind patterns often transport air pollution hundreds of miles).

36. In our analysis, "neighboring" areas are defined to be all counties that either share a common land border with or meet at a corner of a base county.

though the sample consists of only 142 counties.³⁷ While this approach may be more arbitrary than directly matching on income shocks, it leads to similar findings.

It is instructive to consider an identification strategy that isolates the recession-induced variation in TSPs reductions by instrumenting 1980–1982 changes in TSPs with the preexisting level (or share) of manufacturing employment. We find that a higher level of 1980 manufacturing employment is associated with a greater reduction in TSPs from 1980–1982. However, it is also strongly correlated with a greater decline in per capita income, suggesting that this instrument is picking up the economic shock component of the TSPs changes. Thus, this approach is likely to provide downwardly biased estimates of the TSPs effects due to confounding economic shocks. Indeed, we find evidence consistent with this possibility.³⁸

VII.D. Alternative Explanations

Now we discuss alternative explanations for our findings. First, there are several medical interventions that may affect an infant's health. For example, the presence of a local specialized neonatal intensive care unit (NICU) or a public clinic is correlated with a decrease in infant mortality rates [Institute of Medicine 1985; Menard et al. 1998]. However, most of the diffusion of NICUs occurred after the early 1980s [Baker and Phibbs 2001]. In addition, the two most notable changes in birth technology during the last 30 years were the advent of respiratory therapy techniques and improvements in mechanical ventilation in 1974 and the introduction of surfactant therapy in October 1989. The impact of the former innovation had largely been diffused by the late 1970s [OTA 1988, p. 41]. Nevertheless, these medical interventions and their diffusion are potential sources of bias in our analysis.

The literature on the “production” of infant health emphasizes the importance of a mother’s ability to support a fetus for

37. Among the counties with low manufacturing employment, 90 counties only have neighbors with low manufacturing employment, and 52 counties have at least one neighbor with high manufacturing employment. Using only the sample of 100 counties with 1980 manufacturing employment less than 1000 resulted in an estimated TSPs coefficient of 4.76 (2.97).

38. We implemented the prerecession manufacturing activity instrument using similar specifications to those used in Table IV. The resulting estimates of the TSPs coefficient varied between 2.5–3.8, but were imprecise due to the much weaker first-stage.

nine months. This ability, also known as the maternal health endowment, is believed to affect an infant's survival probabilities. In addition to its direct effects, the maternal health endowment may interact with the local availability of abortion services to affect the distribution of health outcomes for fetuses brought to term [Grossman and Joyce 1990]. It is possible that the recession affected pregnancy and abortion decisions and the migration patterns of pregnant women, thereby changing the distribution of *unobserved* maternal health endowments.

Interestingly, 1980–1982 is one of the few periods in which both pregnancy and abortion rates were stable among all females and among teenage women in the United States.³⁹ While some states changed Medicaid funding for abortions in 1981, these policies may not have impacted pregnancy and abortion rates until 1984 [Levine, Trainor, and Zimmerman 1995]. Further, Table I shows that while the total number of births in the United States fell slightly between 1982 and 1984, it increased from 1980 to 1982. This suggests that births responded to the recession with a lag, which is not surprising given the nine-month pregnancy period associated with a birth. Also, we find that counties experiencing large and small TSPs reductions during the recession had statistically identical percentage changes in the number of births from 1980–1982 [Chay and Greenstone 1999]. We conclude that the biases in our estimates due to changes in the distribution of births within a county are likely to be small.

If the recession caused other air quality indices to improve, then our estimates represent the combined effect of changes in TSPs and other air pollutants. However, the declines in ozone, carbon monoxide, and sulfur dioxide concentrations between 1980 and 1982 were much smaller (4.7, 9.5, and 12.1 percent, respectively) than the TSPs reduction. Moreover, the reductions in carbon monoxide and sulfur dioxide started well before 1980 and do not appear to be due to the recession. Notably, ambient lead concentrations declined by 33.7 percent in the 143 counties that were continuously monitored for lead during this period. Due to the small number of monitored counties, we do not include lead measurements in the analysis. However, the decline in lead was

39. The OTA [1988, p. 41] states, "Among adolescents, the percentage of pregnancies ended by abortion remained virtually unchanged between 1980 and 1982."

presumably the result of the *national* program to reduce the lead content of gasoline that started in 1975, and thus, it began before 1980 and was largely uniform across counties. Nonetheless, our analysis may be identifying the effect of all air quality improvements during the recession and not just the effect of reductions in TSPs.

More generally, Figure III shows that there were abrupt trend breaks in *differences* across counties in TSPs pollution and infant mortality rates centered in 1980–1982. Therefore, any alternative explanation for the observed infant mortality-TSPs association must exhibit similar trend breaks in differences in order to be viable. This would seem to rule out many competing hypotheses, such as the diffusion of birth technology. Further, the estimated TSPs effects were insensitive to including unrestricted state-time effects, which rule out any omitted factors that vary across states over time, such as state-level policies and population characteristics.

Finally, a criticism of previous research on air pollution and *adult* mortality is that pollution may have caused those who were already very ill to die slightly earlier than they would have otherwise, implying minimal loss in life expectancy [Spix et al. 1994; Lipfert and Wyzga 1995]. This phenomenon is referred to as “harvesting” by epidemiologists. If harvesting is important, then the association between reductions in TSPs and infant mortality should disappear as one examines longer periods after birth. Table IV showed that the estimated effects of TSPs on infant mortality *increase* as the period since birth lengthens from one day to one month to one year. This is inconsistent with the harvesting theory.

We also examined whether the harvesting phenomenon becomes evident over longer periods. Using the Mortality Detail Files, we find that counties with large and small TSPs reductions from 1980–1982 had similar changes in cumulative mortality rates among children aged 1–4 years olds from 1981–1983, 1982–1984, 1983–1985, and 1984–1986. The strength of this finding is mitigated by two related factors: 1) the migration rates of mothers and children may be greater several years after birth; and 2) other factors are likely to change differentially across counties after the recession (recall Figure IIC). Nonetheless, these results suggest that the decline in TSPs during the recession had long-run effects on life expectancy.

VIII. POLICY IMPLICATIONS

After the passage of the Clean Air Act Amendments in 1970, the EPA required all counties to achieve annual geometric mean concentrations of TSPs below $75 \mu\text{g}/\text{m}^3$. This standard was chosen to "protect the public health." The optimality of this regulatory ceiling depends on the existence and magnitude of the health effects of TSPs pollution above and below it. Our results suggest that TSPs have substantial effects on infant mortality rates both above and below the $75 \mu\text{g}/\text{m}^3$ standard. In fact, the estimates derived from matching counties with similar income shocks imply that the infant mortality rate effects of TSPs may be greater below the regulatory threshold. They do not support the view that $75 \mu\text{g}/\text{m}^3$ is a threshold below which there are no effects on human health.

Our estimates can also be used to calculate the benefits of regulating TSPs pollution. Currently, there are about 4 million births per year in the United States. Thus, our estimates imply that a $1 \mu\text{g}/\text{m}^3$ reduction in TSPs is associated with approximately 200 additional infants surviving to one year of age. When a statistical life is valued at \$1.6 million, the reduction in infant mortality associated with a $1 \mu\text{g}/\text{m}^3$ decline in TSPs is worth approximately \$320 million (\$1997).⁴⁰ However, this calculation ignores other health and aesthetic benefits and is likely to underestimate the total benefits of a reduction in TSPs pollution.

In principle, a hedonic analysis of the housing market can provide a measure that captures the economic value of all health benefits and improved aesthetics. Estimating such a model, Chay and Greenstone [2000] find that a $1 \mu\text{g}/\text{m}^3$ decline in TSPs is associated with a 0.4–0.5 percent increase in housing prices during the 1970s. The 1999 American Housing Survey reports that the value of the owner-occupied housing stock in the United States is approximately \$7.1 trillion (\$1997).⁴¹ Assuming that our estimate of the marginal willingness to pay is correct and that the hedonic price function has not changed since the 1970s, a $1 \mu\text{g}/\text{m}^3$

40. Viscusi's [1993] review of the literature suggests that the value of a statistical life ranges from \$3.5–\$8.5 million (\$1997), but recent research by Ashenfelter and Greenstone [2001] indicates that it may be less than \$1.6 million (\$1997).

41. This figure is calculated as the median value of owner-occupied homes in 1999 (\$108,300) multiplied by the number of such homes (68,796,000) and is deflated by the shelter component of the CPI. It understates the total value of the housing stock, because the American Housing Survey does not collect value information on the 46,419,000 housing units that are not owner occupied.

TSPs reduction would increase the value of the housing stock by \$32 billion (\$1997).

A complete analysis of a TSPs regulatory policy requires information on costs. A recent EPA evaluation of the Clean Air Act Amendments found that compliance costs were roughly \$32 billion (\$1997) in 1990, the last year for which these data are available [EPA 1997]. Since the regulation of particulates is only one part of this legislation, this is surely an overestimate of the costs of compliance with TSPs-specific regulations. Greenstone [2001] provides the only estimates of the costs associated with the regulation of TSPs. He finds that the TSPs regulation specified by the CAAAs caused TSPs-emitting manufacturers to reduce their employment, investment, and shipments in the areas of the country that faced stringent regulation. However, his estimates do not provide measures of the increased costs faced by manufacturers or the adjustment costs resulting from workers switching industries and the redeployment of existing capital into new industries.

On the whole, the regulation of TSPs appears to pass a cost-benefit test if the estimated benefits from the hedonic study are used. However, the results of a cost-benefit analysis are less clear if the benefits are limited to those associated with reductions in infant mortality.

IX. CONCLUSION

We use the substantial variation in air quality changes during the 1981–1982 recession to estimate the effects of particulates pollution on infant mortality. We find that a 1- $\mu\text{g}/\text{m}^3$ reduction in TSPs is associated with 4–7 fewer infant deaths per 100,000 live births at the county level, which is an elasticity of 0.35. Most of these effects are driven by fewer deaths occurring within one month of birth, suggesting that fetal exposure during pregnancy is a biological pathway. Consistent with this, we find significant effects of TSPs reductions on deaths within 24 hours of birth and on infant birth weight. The analysis also reveals nonlinear effects of TSPs and large infant mortality effects at TSPs concentrations below the EPA-mandated air quality standard. Overall, the estimates imply that about 2500 fewer infants died from 1980–1982 than would have in the absence of the reductions in air pollution.

Starting in the late 1980s, the EPA changed the focus of its particulates regulatory program. In 1987 and 1997 the regulation of TSPs, defined as particles up to 40 μm in diameter, was

replaced with regulation of particulates less than 10 μm (PM_{10}) and 2.5 μm ($\text{PM}_{2.5}$) in diameter. The relative health effects of fine and large particles have become a controversial topic in the public debate on federal air pollution regulation. We cannot determine whether the decreases in infant mortality found in this study are due to reductions in small or large particles. The only indication comes from the fact that the 1980–1982 recession was concentrated in the industrial sector and fuel combustion-related emissions are disproportionately comprised of small particles. However, this anecdotal evidence is speculative at best. Future work should resolve this question and focus on developing credible theory and evidence on the causal pathways through which air pollution affects infant health.

DATA APPENDIX

Variables from 1978–1984 National Mortality Detail Files

The 1978–1984 National Mortality Detail Files are an annual census of deaths in the United States, derived from the Standard Certificate of Death.⁴² The data contain the universe of deaths and information on the deceased's county of residence, race, gender, age at death, and cause of death. Since infant deaths cannot be directly linked to individual births for this period, the microdata are aggregated into race-gender-age at death by county of residence cells. The total number of infant deaths (deaths within one day, 28 days, and one year of birth) within a demographic-county cell is used as the numerator for the infant mortality rate.

Variables from 1978–1984 National Natality Detail Files

The 1978–1984 National Natality Detail Files are an annual census of births in the United States, derived from the Standard Certificate of Live Birth. Each file contains the universe of births and information on the county of residence of the mother, socio-economic and demographic characteristics of the parents, the mother's usage of medical services, the health endowment and medical history of the mother, and the infant health endowment. The microdata on births are aggregated into race-gender by ma-

42. Both the National Mortality Detail and Natality Detail files are published by the National Center for Health Statistics in the U. S. Department of Health and Human Services. Detailed descriptions of the files are available at http://www.cdc.gov/nchs/products/elec_prods/subject/mortud.htm and http://www.cdc.gov/nchs/products/elec_prods/subject/nativity.htm, respectively.

ternal county of residence cells. The total number of births within a demographic-county cell is used as the denominator for the infant mortality rate.

The following variables are included as controls in specifications labeled "*Unrestricted natality*" in the tables. The word "indicator" refers to an indicator variable at the individual level. The cell observation contains the fraction of individual observations for which the indicator is one.

Socioeconomic and Demographic Characteristics

Mother:

county of residence

continuous years of education

indicators for years of education <12, =12, 13–15, 16+

marital status indicator

immigrant indicator

Father:

continuous years of education

indicators for years of education <12, =12, 13–15, 16+

age

Infant:

racial indicators for white, black, and other birth

indicator for gender of birth

not a singleton indicator (twins or greater birth)

Medical System Utilization

month of first prenatal care visit

first prenatal care visit in months 1 or 2 of pregnancy, indicator

first prenatal care visit in month 3, indicator

first prenatal care visit in months 4, 5, or 6, indicator

first prenatal care visit in months 7, 8, or 9, indicator

no prenatal care, indicator

total number of prenatal care visits

delivery outside of hospital, indicator

physician present at delivery, indicator

Maternal Health Endowment and Medical History

age of mother

indicators for mother's age 10–14, 15–19, 20–24, 25–29, 30–34, 35–39, 40+

1 previous live birth, indicator

2 or more previous live births, indicator

- 1 previous fetal death, indicator
- 2 or more previous fetal deaths, indicator
- last pregnancy resulted in live birth, indicator
- last pregnancy resulted in fetal death, indicator
- indicators for 1–11, 12–17, 18 or more months since termination of last pregnancy
- indicators for 1–11, 12–17, 18 or more months since last live birth
- indicators for 1–11, 12–17, 18 or more months since last fetal death
- 1 previous termination before week 20 of gestation, indicator
- 2 or more previous terminations before week 20 of gestation, indicator
- 1 previous termination after week 20 of gestation, indicator
- 2 or more previous terminations after week 20 of gestation, indicator

The following variables are included in specifications labeled "*Basic natality variables*" in the tables: indicators for race and gender of infant, mother's education, father's education, age of mother, marital status of mother, indicators for month of first prenatal care visit, indicator for no prenatal care.

We also use the Natality data to examine the following birth weight outcomes for infants:

continuous birth weight

indicators for birth weight < 1000 grams, 1000–1499, 1500–1999, 2000–2499, 2500–2999, 3000–3499, 3500–3999, 4000–4499, 4500+

Total Suspended Particulates (TSPs) Pollution Data

The data on suspended particulates pollution levels in each county were obtained by filing a Freedom of Information Act request with the EPA. This yielded the *Quick Look Report* data file, which is derived from the EPA's *Air Quality Subsystem* (AQS) database. This file contains the universe of recordings of TSPs pollution concentrations from each of the approximately 4000 EPA air monitors that were located throughout the United States during the period of interest, as well as the location of each monitor. For each county the annual concentration of TSPs used in this study is the weighted average of the annual arithmetic means of each monitor in the county, with the number of observations per monitor used as weights.

Our analysis relies on the presumption that the pollution

concentration readings used accurately reflect the "true" exposure of individuals to TSPs. The readings from the TSPs monitors are used to determine which counties are heavily regulated under the Clean Air Act Amendments. To preclude the possibility that counties or states place monitors to fabricate the appearance of favorable pollution concentrations, the Code of Federal Regulations contains very precise criteria that govern the siting of a monitor.⁴³ Among the most important criteria is that the monitors capture representative pollution concentrations in high population areas. Moreover, the EPA must approve the location of all monitors and requires documentation that the monitors are actually placed in the approved locations.

County-Level Per Capita Income Data

Ideally, data would be available on the incomes of women giving birth in each county. In its absence, the analysis uses the Bureau of Economic Analysis' annual county-level series on per capita income (deflated to \$1982–1984). It is "the sum of wage and salary disbursements, other labor income, proprietors' income with inventory valuation and capital consumption adjustments, rental income of persons with capital consumption adjustment, personal dividend income, and transfer payments to persons, less personal contribution for social insurance" (Bureau of Economic Analysis 1994). This is the most comprehensive measure of income available at the county level and is superior to less broad measures, such as labor income, which do not capture all of the resources available to individuals.

Some specifications also control for the annual, civilian unemployment rate in each county, which comes from the *Employment and Unemployment for State and Local Areas*, published annually by the U. S. Bureau of Labor Statistics. We have matched and compared counties with similar levels of manufacturing activity before the recession. For this, we use data on employment levels in manufacturing in each county from the annual *County Business Patterns* data files.

County-Level Data on Transfer Payments

From the Regional Economic Information System (REIS), we obtained county-level information on medical transfer payments and payments for income maintenance and unemployment insur-

43. This discussion results from the Code of Federal Regulations (CRF) 1995, title 40, part 58 and a conversation with Manny Aquilania and Robert Palorino of the EPA's District 9 Regional Office.

ance. The analysis controls for each of the following payment subcategories, in terms of per capita payments (in \$1982–1984):

Total transfer payments per capita

Total medical care payments per capita

Public assistance medical care payments for low-income individuals (primarily Medicaid and state and local government general assistance medical programs)

Income maintenance benefit payments per capita

Family assistance payments per capita (includes AFDC)

Food stamps payments per capita

Unemployment Insurance (UI) benefit payments per capita

State-Level Medicaid Data

County-level data on Medicaid recipiency are unavailable for the period of interest. Instead, from the Health Care Financing Administration we obtained annual, state-level information on the number of Medicaid recipients, the average cost per Medicaid recipient, and payments to medical vendors from the Medicaid program. Data for Arizona are missing in all years.

State-Level Temperature and Precipitation Data

We obtained data on precipitation and temperature from the National Weather Service, which is a subdivision of the National Oceanic and Atmospheric Administration. The National Weather Service has weather stations located throughout the United States that record local precipitation and temperature. The publicly available annual data contain state-level averages of precipitation and temperature in each month of the year. These variables are used as controls in the analysis since temperature and precipitation covary with pollution levels, and it is thought that they may also affect health [Samet et al. 1998].

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