Air Quality, Infant Mortality, and the Clean Air Act of 1970*

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Abstract

We examine the effects of total suspended particulates (TSPs) pollution on infant health using the air quality improvements induced by federal air pollution regulations. The Clean Air Act Amendment (CAAA) of 1970 marked an unprecedented attempt by the federal government to mandate lower levels of air pollution. If pollution concentrations in a county exceeded the federal ceiling, then the EPA designated the county as 'nonattainment'. Polluters in nonattainment counties faced greater federal regulatory oversight than their counterparts in attainment counties. We use county-level nonattainment status as an instrumental variable for TSPs changes after the 1970 CAAA. Since regulatory status is a discrete function of the previous year's TSPs levels, the discontinuity in its assignment can be used to gauge the credibility of the research design.

The county-level regulations are associated with sharp, large reductions in TSPs pollution in nonattainment counties soon after the 1970 CAAA. We estimate that a 1-$\mu g/m^3$ reduction in TSPs results in 5-8 fewer infant deaths per 100,000 live births at the county level (a 0.35 elasticity). Most of these effects are driven by a reduction in deaths occurring within one month of birth, suggesting that fetal exposure is a potential biological pathway. The estimated birth weight effects are consistent with this possibility. Also, there are discrete differences in TSPs and infant mortality changes between nonattainment and attainment counties near the federal ceiling, suggesting that the regulations are a causal factor. It appears that the 1970 Clean Air Act provided significant health benefits for infants.

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Introduction

The 1970 passage of the Clean Air Act Amendments (CAAA) 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 was required to meet.[1]

Figure 1 shows trends in average total suspended particulates pollution and infant mortality rates. Panel A of the figure presents trends for the entire United States from 1970-1990, while Panel B shows similar data for a fixed set of counties with continuous TSPs readings from 1969-1975.[2] From Panel A it is apparent that air quality improved dramatically in the 1970s and 1980s, with particulate concentrations falling from an average of 95 $\mu g/m^3$ to about 60 $\mu g/m^3$. Almost all of the reduction occurred in two punctuated periods, 1971-1973 and 1980-1982. The figure also shows the large fall in the infant mortality rate over the entire period. Although both TSPs pollution and infant mortality rates trended downward, their time-series correspondence does not provide conclusive evidence of a causal effect, since many other factors were also changing that may have affected infant mortality rates.

In previous work (Chay and Greenstone 1999), we established that most of the 1980-82 decline in TSPs can be attributed to the differential impacts of the 1981-82 recession across counties. We then used the substantial differences in air pollution reductions across sites to estimate the impact of TSPs on infant mortality. We find that a 1-$\mu g/m^3$ reduction in TSPs results in about 4-7 fewer infant deaths per 100,000 live births at the county level -- an elasticity of 0.35. Most of the reduction in infant mortality is driven by a decline in deaths occurring within one month of birth. Along with the estimated birth weight effects,

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[1] TSPs consist of all particles with diameters less than or equal to 40 micrometers (μm). The focus of federal regulation shifted to particulates with diameters less than or equal to 10 μm (PM_{10}) and 2.5 μm ($PM_{2.5}$) in 1987 and 1997, respectively.

[2] For Panel A, the TSPs pollution series is based on averages across 1,000-1,300 counties a year, while infant mortality rates are derived from the entire U.S. and are for all causes. Panel B is based on the same sample of counties used in the analysis below. The sample is described in Table 1. The infant mortality rate in Panel B is for internal causes of death (e.g., respiratory and cardiopulmonary) and excludes non-health related causes.

these findings suggest that fetal exposure to TSPs pollution during pregnancy is a pathophysiologic mechanism.

In view of the significance of a potential causal link between TSPs pollution and infant mortality, we believe it is important to validate the results from our analysis of the early 1980s. In this paper, we examine data on infant mortality and air pollution from the other major episode of substantial changes in TSPs levels in the early 1970s. Following Chay and Greenstone (2000), we argue that these changes were largely attributable to the regulations imposed by the 1970 CAAA. In particular, as shown in Panel B of Figure 1, over one-half of the 1970-75 reduction in TSPs concentrations in counties with continuous air monitor data occurred in the first year after passage of the law.

There are several ways in which this research design both complements and extends our earlier research. First, since it is plausible that regulatory pressure is orthogonal to county-level changes in infant mortality rates, except through its impact on air pollution, it is arguably a valid instrument. Second, regulatory status is a discrete function of the previous year's TSPs levels. This discontinuity in the assignment of regulations can be used to gauge the credibility of the research design. Third, our analysis provides direct and easily interpreted estimates of the air quality and health benefits of the 1970 Act.

Finally, this study provides a unique opportunity to cross-validate the results from our earlier work by examining a different design applied to a different era (the early 1970s). In contrast to the recession-driven TSPs reductions of the early 1980s, we use regulation-induced changes that occurred during an economic expansion (1971-1972). Thus, any potential biases due to economic shocks are likely to be mitigated. Also, particulates pollution levels were much higher in the early 1970s than in the early 1980s. As a result, we can examine a different range of the pollution-infant mortality function and analyze the health benefits at the federally mandated concentration ceiling.

We implement the design using the most detailed and comprehensive data available on infant births, deaths, and TSPs levels for the 1969-74 period. We find that the federally imposed county-level regulations are associated with sharp, large reductions in TSPs pollution in counties where TSPs levels were initially above the legislated maximum of the 1970 CAAA (so-called "nonattainment" counties).

We estimate that a 1-$\mu g/m^3$ reduction in TSPs results in 5-8 fewer infant deaths per 100,000 live births at the county level, which is a 0.35 elasticity. As in our earlier study, most of these effects are driven by a reduction in the number of deaths occurring within one month of birth, suggesting that fetal exposure is a potential biological pathway. Consistent with this hypothesis, we find significant effects of the TSPs reductions on deaths within 24 hours of birth and on infant birth weight.

Our attempts to probe the robustness of these conclusions suggest that instrumental variables estimates of the effect of TSPs pollution on infant mortality are far less sensitive to specification than conventional cross-sectional and fixed effects estimates. Importantly, there are discrete differences in TSPs and infant mortality changes between nonattainment and attainment counties near the federal ceiling. This suggests that the regulations are causally related to declines in both TSPs and infant deaths. As a test of internal validity, we also find that the regulation-induced TSPs reductions are uncorrelated with infant deaths due to accidents and homicides.

The evidence supports the conclusion that air pollution has a causal effect on infant health and that the 1970 Clean Air Act provided significant health benefits. Overall, the estimates imply that at least 1,300 fewer infants died from 1971-72 than would have in the absence of the Clean Air Act regulations. Also, the results correspond with the findings in Chay and Greenstone (1999).

Background on the 1970 Clean Air Act

Before 1970 the federal government did not play a significant role in the regulation of air pollution, a responsibility left primarily to state governments.[3] In the absence of federal legislation, few states acted to impose strict regulations on polluters within their jurisdictions. Concerned with the detrimental health effects of persistently high concentrations of suspended particulates pollution, and of other air pollutants, Congress passed the Clean Air Act Amendments of 1970.

[3] Lave and Omenn (1981) and Liroff (1986) provide more details on the CAAAs. In addition, see Greenstone (1999) and Chay and Greenstone (2000).

The centerpiece of the 1970 CAAA was the establishment of separate federal standards, known as the National Ambient Air Quality Standards (NAAQS), for six pollutants. The goal of the CAAA was to reduce local air pollution concentrations until all U.S. counties were in compliance with the NAAQS. As directed by the CAAA, the EPA annually determines the 'attainment-nonattainment' status of all U.S. counties for each of the regulated pollutants. If pollution concentrations in a county exceed the federal ceiling, then the EPA designates the county as being in nonattainment status for that pollutant in the following year. For TSPs pollution, a county is designated as nonattainment if either of two thresholds are exceeded in the previous year: 1) the annual geometric mean concentration exceeds 75 $\mu g/m^3$, or 2) the second highest daily concentration exceeds 260 $\mu g/m^3$. This standard prevailed from 1971 until 1987, when the EPA shifted its focus to the regulation of finer particles.

To achieve these standards, the fifty states were required to formulate and enforce local pollution abatement programs. In its nonattainment counties, each state had to develop plant-specific regulations for every major source of pollution. These local rules ordered that any substantial investment by a new or existing plant must be accompanied by the installation of state-of-the-art pollution abatement equipment and strict emissions ceilings. The 1977 Amendments to the CAAA added the requirement that any increase in emissions from a new investment had to be offset by a reduction in emissions from another source within the same county. The federally determined nonattainment designation also mandated that state authorities set emissions limits on existing plants.

In stark contrast to the oversite in nonattainment counties, the restrictions on polluters in attainment counties were considerably less stringent. First, large-scale investments, such as new plants and large expansions at existing plants, required the installation of less expensive (and less effective) pollution abatement equipment. Moreover, it was unnecessary to obtain offsets for increased emissions. Finally, existing plants and smaller investments were essentially left unregulated.

Both the states and the federal EPA were given substantial enforcement powers to ensure that the goals of the CAAAs were met. To limit variation in the intensity of regulation across states, the federal EPA had to approve all state regulation programs. On the compliance side, states initiated their own inspection programs and frequently fined non-compliers. Also, the 1977 legislation made the plant-

specific regulations both federal and state law. Thus, the EPA had legal standing to impose penalties on states that failed to aggressively enforce the regulations and on plants that failed to comply.

Henderson (1996) provides evidence that the regulations were successfully enforced. He finds that ozone concentrations declined more in counties that were nonattainment for ozone than in attainment counties. Chay and Greenstone (2000) find evidence that TSPs levels fell substantially more in counties that were nonattainment for TSPs than in attainment counties after the passage of the 1970 Clean Air Act and throughout the 1970s.[4]

Research Design

Previous research has documented a statistical association between TSPs concentrations and adult mortality.[5] However, 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 Dockery 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, the lifetime exposure of adults to air pollution is unknown. Many studies implicitly assume 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 the air quality improvements induced by the 1970 Clean Air Act Amendment to estimate the impact of TSPs on infant mortality. There are several reasons why this design may reduce the role of omitted variables bias. First, air pollution

[4] Greenstone (1999) provides further evidence on the effectiveness of the regulations. He finds that nonattainment status is associated with modest reductions in the employment, investment, and shipments of polluting manufacturers. Interestingly, the regulation of TSPs has little association with changes in employment. Instead, the overall employment declines are driven mostly by the regulation of other air pollutants.

[5] Studies of the adult mortality effects of particulates pollution include Lave and Seskin (1977), Pope and Dockery (1996), Dockery and Pope (1996), Dockery, et al. (1993), and Pope, et al. (1995).

regulations were associated with sharp variation in changes in TSPs across sites in the early 1970s. For example, Figure 2 shows the trends in TSPs levels from 1969 to 1974 in two groups of counties: those that were in compliance with the CAAA in 1970 (nonattainment for 1971), and those that were not.[6]

Before the Clean Air Act, TSPs concentrations were 40-$\mu g/m^3$ higher in nonattainment counties. While the pollution trends were similar in the two groups from 1969 to 1971, these trends were dramatically different after the passage of the 1970 CAAAs. From 1971-74 newly regulated counties had a stunning 20-$\mu g/m^3$ reduction in TSPs, while TSPs fell by only 3 $\mu g/m^3$ in attainment counties. These comparisons suggest that the entire national decline in TSPs from 1971-74 in Figure 1 was attributable to the regulations. In addition, two-thirds of the 1971-74 TSPs decline in nonattainment counties occurred from 1971-72, the first year after implementation of the CAAAs. Consequently, the analysis below focuses on this abrupt, one-year improvement in air quality. From Figure 2, TSPs fell 13 units more in nonattainment counties than in attainment counties from 1971-72.

Given CAAA rules, nonattainment status in 1971 was a discrete function of the annual geometric mean and second highest daily concentrations of TSPs in 1970. Thus, the assignment of regulatory status has the feature of a quasi-experimental regression-discontinuity design (Cook and Campbell 1979). If other factors affecting infant mortality are similar for counties just above and just below the regulatory thresholds, then comparing outcome changes in nonattainment and attainment counties with pre-regulation TSPs levels just around the threshold will control for all omitted factors correlated with TSPs. Under this assumption discrete differences in mean outcome changes between nonattainment and attainment counties near the federal ceilings can be attributed to the regulations. The discontinuity in the assignment of regulations provides a valuable opportunity to gauge the credibility of the research design and develop convincing specification tests.

Our focus on infant, rather than adult, mortality may circumvent the other important criticisms of previous research. The problem of unknown lifetime exposure to pollution is significantly mitigated, if

[6] The sample consists of the 401 counties with continuous monitor readings from 1969-1974. These counties account for almost 60 percent of all births in the U.S. in 1970.

not solved, by the low migration rates of pregnant women and infants. Thus, we assign TSPs levels 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). Also, since the mortality rate is higher in the first year of life than in the next 20 years combined (NCHS 1999), it is likely that infant deaths represent a large loss in life expectancy.

A final advantage of the design is that it permits a direct analysis of the health benefits of TSPs reductions at levels above and around the EPA's maximum allowable concentration. Clearly, the optimality of the design of the CAAAs depends crucially on the existence and relative magnitudes of the health effects above and near the federal regulatory threshold.

Econometric Methods

Our analysis compares changes in infant mortality rates and TSPs pollution from 1971-1972 for counties that were nonattainment and attainment for TSPs in 1971. Here, 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.

The cross-sectional model predominantly used in the literature on pollution and health is:

(1) $y_{ct} = X_{ct}'\beta + \theta T_{ct} + \varepsilon_{ct}, \qquad \varepsilon_{ct} = \alpha_c + u_{ct}$

(2) $T_{ct} = X_{ct}'\Pi + \eta_{ct}, \qquad \eta_{ct} = \lambda_c + v_{ct},$

where y_{ct} is the infant mortality rate in county c in year t, X_{ct} is a vector of observed characteristics, T_{ct} is the mean of TSPs across all monitors in the county, and ε_{ct} and η_{ct} are the unobservable determinants of infant mortality rates and TSPs levels, respectively. The coefficient θ is the 'true' effect of TSPs on infant mortality. For consistent estimation, the least squares estimator of θ requires $E[\varepsilon_{ct}\eta_{ct}]=0$. If there are omitted permanent (α_c and λ_c) or transitory (u_{ct} and v_{ct}) factors that covary with both TSPs and infant mortality, then the cross-sectional estimator will be biased.

With repeated observations over time, a "fixed-effects" model implies that first-differencing the data will absorb the county permanent effects, α_c and λ_c. This leads to:

(3) $y_{ct} - y_{ct-1} = (X_{ct} - X_{ct-1})'\beta + \theta(T_{ct} - T_{ct-1}) + (u_{ct} - u_{ct-1})$

(4) $T_{ct} - T_{ct-1} = (X_{ct} - X_{ct-1})'\Pi + (v_{ct} - v_{ct-1})$.

For identification, the fixed effects estimator of θ requires $E[(u_{ct} - u_{ct-1})(v_{ct} - v_{ct-1})]=0$. That is, there are no unobserved shocks to TSPs levels that covary with unobserved shocks to infant mortality rates.

Now, suppose there exists an instrumental variable, Z_c, that causes changes in TSPs without having a direct effect on infant mortality rate changes. One plausible instrument is the 1970 CAAA regulatory intervention for TSPs, measured by the attainment-nonattainment status of a county. Here, equation (4) becomes:

(5) $T_{c72} - T_{c71} = (X_{c72} - X_{c71})'\Pi_{TX} + Z_{c71}\Pi_{TZ} + (v_{c72} - v_{c71})$, and

(6) $Z_{c71} = 1(T_{c70} > \overline{T}) = 1(v_{c70} > \overline{T} - X_{c70}'\Pi - \lambda_c)$,

where Z_{c71} is the regulatory status of county c in 1971, $1(\bullet)$ is an indicator function equal to one if the enclosed statement is true, and $\overline{T}$ is the maximum concentration of TSPs allowed by the federal regulations. Regulatory status in 1971 is a discrete function of 1970 pollution levels.[7]

Two sufficient conditions for the instrumental variables (IV) estimator (θ_{IV}) to provide a consistent estimate of the effects of TSPs are $\Pi_{TZ} \neq 0$ and $E[v_{c70}(u_{c72} - u_{c71})] = 0$. The first condition requires that the regulations induced air quality improvements. The second condition requires that unobserved mortality rate shocks from 1971-72 are orthogonal to transitory shocks to 1970 TSPs levels.

In the simplest case, the IV estimator is consistent if $E[Z_{c71}(u_{c72} - u_{c71})] = 0$. Here, the IV estimate comes from comparing differences between nonattainment and attainment counties in 1971-72 changes in TSPs pollution and infant mortality rates. Even if $E[v_{c70}(u_{c72} - u_{c71})] \neq 0$, causal inferences on θ can be made by leveraging the regression discontinuity design implicit in the $1(\bullet)$ function determining regulatory status. If the relationship between v_{c70} and $(u_{c72} - u_{c71})$ is sufficiently smooth at the regulatory ceilings, then comparing changes in regulated and unregulated counties at the thresholds will control for any omitted variables.

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[7] For simplicity, we have written (6) as if regulatory status is a function of a single threshold crossing. If T_{c70}^{avg} and T_{c70}^{max} are the annual geometric mean and 2nd highest daily TSPs concentrations, respectively, then the actual regulatory instrument used is $1(T_{c70}^{avg} > 75\ \mu g/m^3$ or $T_{c70}^{max} > 260\ \mu g/m^3)$. Only six counties were nonattainment in 1971 for exceeding the 2nd highest daily concentration threshold, but not the annual geometric mean ceiling.

Data Sources

To implement our evaluation strategy, we use county level data on air pollution and infant health for the 1969-1974 period. Here, we describe the data used in this study. More details on the data sources and variables are contained in the Data Appendix.

The health 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 individual birth and infant death records at the county by demographic group level for each year to create infant age, race, and gender cells for each county. 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; 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 we discuss below, 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 this data, we calculated the mean TSPs concentrations in each county and year. The data also contain information on the annual geometric mean TSPs concentration for each monitor in a county and the number of daily monitor readings exceeding the federal standards in each year. This was used to determine the annual nonattainment status of each county (see Data Appendix).

In addition to the key infant health and pollution data, we use a variety of other county-level data

as controls. 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. This file also provides annual, county-level data on per-capita net earnings, the ratio of total employment to the total population, and the ratio of total manufacturing employment to the total population. 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.

Table 1 presents summary information from 1969-1974 for the 501 counties with continuously available TSPs pollution data in 1970, 1971, and 1972. This is the sample used in the analysis below. The top rows of the table show that these counties account for over 60% of the 3.3-3.7 million live births that occur in the United States in a given year. The next rows present infant fatalities per 100,000 live births, by cause of death. Internal causes are attributable to health complications (e.g., respiratory and cardiopulmonary deaths), while external causes are non-health related (e.g., accidents and homicides). The internal infant mortality rate is about 1.7-1.9 deaths within a year of birth per 100 births, while infant deaths due to external causes are much rarer. 45 and 75 percent of all internal infant deaths within a year of birth occur within the first 24 hours and 28 days of birth, respectively. Also, the black infant mortality rate is about 1.8 times greater than the white rate.

The table also shows national trends in infant mortality, average TSPs concentrations, and per-capita income across counties. Internal infant mortality rates decreased steadily from 1969-74. TSPs, on the other hand, fell by over 18 percent from 1971-74, but were relatively stable from 1969-71. Per-capita income increased by 8.5 percent from 1971-73, before falling slightly from 1973-74, presumably due to the recession.

The remaining rows of Table 1 present 1969-74 trends for several of the main control variables used in the analysis. Importantly, the magnitude of changes in these variables is small when compared to the TSPs pollution changes that occurred from 1971-72. Our measures of parental background

characteristics suggest that the average socioeconomic status (SES) of parents was declining from 1969-74, as the fractions of births to single, high school dropout, and black mothers increased. On the other hand, the likelihood of a mother receiving prenatal care or having a visit in the first trimester increased slightly. The percentage of births attributable to teenagers rose, while the share among women aged 35 and over fell.[8] Finally, the share of first-time births increased slightly while the fraction of mothers who had a prior fetal death remained stable.

Before proceeding, we examine whether the nonattainment instrumental variable is orthogonal to the observable predictors of infant mortality. While it is not a formal test of the exogeneity of the instrument, it seems reasonable to presume that research designs that meet this criteria may suffer from smaller omitted variables bias. First, designs that balance the observable covariates may be more likely to also balance the unobservables. For example, in experimental designs the orthogonality of the treatment to other observable variables is taken as evidence that the treatment was actually randomly assigned. Second, if the instrument balances the observables, then consistent inference will not depend on functional form assumptions on the relations between the observable confounders and infant mortality. Estimators that misspecify these functional forms (e.g., linear regression adjustment when the conditional expectations function is nonlinear) will be biased.

For the sample of counties used in the analysis below, Table 2 shows the association of TSPs levels and TSPs changes with other potential correlates of infant mortality. The first column of the table presents the p-values from F-statistics testing for significant differences in the covariate means across groups of counties with higher and lower TSPs levels in 1971. The counties are divided into three groups corresponding to those in the lowest and highest quartiles of TSPs levels, and a group consisting of the counties in the middle two quartiles. If TSPs levels were randomly assigned across counties, one would expect very few significant differences. However, there are significant differences across the groups for several key variables, including mother's race, education, age, use of prenatal care, father's education,

[8] 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.

and infant birth weight, which may be another index for socioeconomic status. This suggests that "conventional" cross-sectional estimates will be biased due to omitted variables.

Column 2 of the table performs a similar analysis for 1971-72 TSPs changes. The three county groups used correspond to the counties in the lowest and highest change quartiles, and a category consisting of the counties in the middle two quartiles. Changes in TSPs from 1971-72 are highly correlated with changes in mother's and father's education, marital status of the mother, and the fraction of mothers who had experienced a previous fetal death. Thus, fixed effects models may also lead to biased inference.

The final 3 columns of Table 2 show that the 1971 nonattainment instrumental variable is orthogonal to nearly all of the covariates. The p-values are from F-statistics testing the equality of mean 1971-72 changes in the variables by 1971 nonattainment status. Nonattainment and attainment counties experienced very similar changes in predictors of infant mortality, particularly as the sample is restricted to counties near the TSPs regulatory ceiling in Columns 4 and 5. Almost all of the p-values rise in value, most notably for mother's and father's education, mother's marital status and age, and infant birth weight. On the other hand, the first row shows that nonattainment status is associated with significant differences in TSPs reductions from 1971-72, even among counties close to the regulatory threshold. These results provide reassuring evidence on the quality of the instrumental variables research design used in this study.

Empirical Results

Cross-Sectional and Fixed Effects Results

First, we replicate the conventional cross-sectional approach to estimating the association between TSPs pollution and infant mortality across counties. For each cross-section from 1969-1974, Table 3 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 presents the unadjusted TSPs coefficient, 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 also across cross-sections. For example, the estimates in Column 2 vary from a significant positive association in 1970 and 1974 to an insignificant negative correlation in 1972. Including the basic control variables available in the Natality data in Column 2 has a noticeable effect on the point estimates in most years, even as the precision of the estimates increases due to the greatly improved fit of the regressions.[9]

Columns 3-5 present the results from specifications that include additional Natality variables and controls for per-capita income and earnings, employment, transfer payments, Medicaid receipt, and unrestricted state effects. None of the estimates in Columns 3 and 4 are significant at conventional levels. In Column 5, which includes state fixed effects, the estimated effect of TSPs varies from a significant positive correlation in 1974 to a significant negative correlation in 1972. The 1974 cross-section is the only year in which the estimated TSPs effects are significant and less sensitive to specification.

The largest estimates from the cross-sectional analyses imply that a 1-$\mu g/m^3$ reduction in mean TSPs results in 1.3 fewer internal infant deaths per 100,000 live births, which is a 0.06 elasticity. Overall, however, 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 4 presents the fixed effects estimates of the association between mean TSPs and internal infant mortality rates based on pooled data for the 1971-72 (first set of rows) and 1971-74 (second set of rows) periods. As with Table 3, the columns correspond to regression specifications that include additional sets of covariates. 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

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

and infant mortality rate changes.

Not surprisingly, including county-specific intercepts improves the fit of the regressions substantially when compared to the cross-sectional regressions in Table 3. However, for the pooled 1971-72 data, the estimated effects of TSPs on infant mortality are insignificant and sensitive to specification. While the estimated TSPs coefficient is significant in Columns 1 and 2 for the pooled 1971-74 period, it is greatly reduced when the analysis regression-adjusts for the Natality controls, economic variables, transfer payments, and year effects. From the table, it appears that the fixed effects association between TSPs and infant mortality is small and highly sensitive to specification. We conclude that conventional estimates of the infant health effects of TSPs may be plagued by difficulties of controlling for various unobserved factors.

Reduced-Form Effects of 1971 Nonattainment Status

The instrumental variables estimate of the effect of TSPs is a simple function of two reduced-form relations, the effect of 1971 nonattainment status on 1971-72 TSPs changes and on 1971-72 infant mortality rate changes. In particular, if the reduced form models for pollution and infant health are:

(7) $T_{c72} - T_{c71} = (X_{c72} - X_{c71})'\Pi_{TX} + Z_{c71}\Pi_{TZ} + (v_{c72} - v_{c71})$, and

(8) $y_{c72} - y_{c71} = (X_{c72} - X_{c71})'\Pi_{yX} + Z_{c71}\Pi_{yZ} + (u_{c72} - u_{c71})$,

then the IV estimator is $\theta_{IV} = \Pi_{yZ}/\Pi_{TZ}$. Here, we present evidence that our design plausibly identifies the causal effects of the regulation, Z_{c71}, and provides credible estimates of both Π_{yZ} and Π_{TZ}.

Table 5 contains the regression results from estimating equations (7) and (8). The first set of columns show the association between the 1971 nonattainment indicator and changes in mean TSPs from 1971-72. The second set of columns present the estimated effects of nonattainment on the number of infant deaths due to internal causes within a year of birth, per 100,000 live births. The columns contain both the unadjusted and regression adjusted effects of the regulation variable.

In the first three columns, the regulation indicator is associated with a 10-14-$\mu g/m^3$ reduction in TSPs from 1971-72, which is a 10-14% effect. This is identical to the effects shown in Figure 2. The estimates are highly significant with F-statistics suggesting that nonattainment status is the most

important (observable) determinant of TSPs changes. The estimates are relatively insensitive to the inclusion of the full set of Natality control variables and an unrestricted year effect. The first-stage impact of regulation is very powerful and appears to explain all of the overall reduction in mean TSPs in the sample.

The next three columns reveal another striking empirical regularity. The TSPs nonattainment variable is associated with 67-85 fewer infant deaths per 100,000 live births, which is a 3.7-4.7% effect. These estimates are also highly significant, although their precision decreases as more variables are added to the regression. Taken literally, this implies that the Clean Air Act regulations resulted in substantial reductions in infant mortality in regulated counties. The estimates change only slightly when the full set of controls are included. Overall, Table 5 shows that nonattainment counties had both larger declines in TSPs pollution and greater decreases in infant mortality immediately following the 1970 CAAAs.

Figure 3 provides more detailed evidence on the validity of using the TSPs nonattainment indicator as an instrument. The panels graph the bivariate relation between either the change in infant mortality or the change in pollution with the geometric mean of TSPs levels in 1970 (the regulation selection year). We estimated nonparametric regressions as a function of the 1970 geometric mean TSPs using a uniform kernel density regression smoother.[10] Counties that were nonattainment in 1971 for exceeding the daily concentration standard but not the annual geometric mean standard in 1970 are dropped from the analysis. Thus, all counties with 1970 geometric mean TSPs levels below (above) 75 $\mu g/m^3$ are attainment (nonattainment) in 1971, allowing for a sharp comparison at the vertical line in the figure.[11] Note that the comparisons in the figure do not adjust for the other covariates.

Panel A plots the conditional mean change in infant mortality rates and mean TSPs from 1971-72 as a function of 1970 mean TSPs. The figure shows that nonattainment counties experienced much larger reductions in mean TSPs from 1971-72 than their attainment counterparts that had 1970 mean TSPs less than 75 $\mu g/m^3$. Further, there is a clear "trend break" in TSPs changes at the regulatory threshold,

[10] The smoothed scatterplots are not very sensitive to the bandwidth choice.
[11] The sample contains 264 nonattainment counties and 230 attainment counties.

suggesting that regulation is a causal factor. Nonattainment counties also had larger reductions in infant mortality rates than attainment counties. There is a strong correspondence between the trend breaks in TSPs and infant mortality changes at the regulatory ceiling, again implying that nonattainment status has a causal role. On the other hand, there appear to be secular decreases in infant mortality rate reductions at 1970 TSPs levels well below (30-50 $\mu g/m^3$) and well above (100-150 $\mu g/m^3$) the 75-$\mu g/m^3$ threshold.

Panel B of Figure 3 narrows the focus to the set of counties with geometric mean TSPs between 50 and 100 $\mu g/m^3$ in 1970, based on the same kernel regression lines plotted in Panel A. For this range of initial pollution levels, there is a clear association between larger reductions in mean TSPs and greater decreases in infant mortality right at the EPA-mandated air quality standard. This suggests that near the regulatory ceiling, our research design identifies the casual relationship between air pollution and infant mortality through the mechanism of regulation.

Figure 4 provides two specification checks of the 1971 nonattainment research design. First, Panel A of the figure empirically examines the validity of the "smoothness" condition discussed above. It plots the infant mortality changes predicted by the observable covariates excluding TSPs changes, $E[(y_{c72}-y_{c71})|(X_{c72}-X_{c71})]$, along with the actual infant mortality changes from Figure 3. The predicted changes are from a linear regression that includes the full set of Natality control variables and an unrestricted time effect (i.e., final column of Table 5). The figure shows that the non-pollution determinants of infant mortality changes are smooth at the regulatory threshold, whereas actual infant mortality rates fall more due to greater TSPs reductions. The mortality predictors do appear to explain the decreasing reductions in infant mortality at low 1970 TSPs levels. But for counties with 1970 TSPs greater than 55-60 $\mu g/m^3$, there is little association between predicted mortality changes and pre-regulation TSPs levels. This finding is consistent with the results in Table 2. Thus, all else may be held fixed when comparing counties over this range of initial pollution levels.

Panel B of Figure 4 plots pre-regulation changes in infant mortality and mean TSPs from 1969-1970 by the geometric mean of TSPs in 1970. The graph shows that, if anything, mean TSPs were increasing more in nonattainment counties than in attainment counties before the passage of the 1970 CAAA. This corresponds with smaller infant mortality rate reductions in nonattainment counties in the

pre-regulation period. Importantly, pre-regulation changes in infant mortality and TSPs have a smooth relation with 1970 TSPs levels, particularly at the 75-$\mu g/m^3$ threshold. Taken together, the results in Table 5 and the plots in Figures 3 and 4 provide evidence of a causal link between 1971 nonattainment status and 1971-72 reductions in air pollution and infant mortality.

Instrumental Variables Results

Table 6 presents the instrumental variables estimates of the effect of TSPs pollution on internal infant mortality rates within one year of birth derived from two-stage least squares (2SLS). Here, the indicator for 1971 nonattainment status is used as an instrument for 1971-72 changes in TSPs. The first column presents the unadjusted estimate (i.e., the Wald estimate), while the remaining columns correspond to specifications that include additional sets of controls, as in Table 3.

In Column 1, the estimated TSPs effect is highly significant and implies that a 1-$\mu g/m^3$ decline in TSPs results in 4.8 fewer infant deaths per 100,000 live births. When the basic set of Natality controls are added in Column 2, the estimated TSPs coefficient rises to 6.1 and remains significant at well below the 1-percent level. The specifications in Columns 3 and 4 add the full set of Natality controls, an unrestricted year effect, and the per-capita income, earnings, and transfer payments variables. The point estimate rises to 7.8-8.5. Since the sampling errors are much larger, these estimates are significant at the 5-percent and 10-percent levels. Finally, the specification in Column 5 includes unrestricted state-year effects, which absorb all unobservables that vary across states from 1971-72. In this saturated model, the treatment effect is identified using only comparisons of changes between attainment and nonattainment counties within the same state. The TSPs estimate rises to 16.8 and is significant at conventional levels, although it is much less precise.[12]

In direct contrast to the cross-sectional estimates, the instrumental variables estimates of the TSPs effect are much larger in magnitude, significant, and fairly stable across specifications. The estimates

[12] For the same specifications and samples as those in the columns of Table 6, we calculated the first differences estimate of the association between 1971-72 changes in infant mortality and TSPs, without instrumenting the TSPs changes. The resulting estimates (standard errors) are 0.74 (0.71), 0.47 (0.71), -0.14 (0.67), -0.31 (0.67), and –0.54 (0.78). These are similar to the 1971-72 fixed effects estimates based on the larger sample of counties in Table 4.

suggest that a 1-$\mu g/m^3$ decline in TSPs is associated with 5-8 fewer infant deaths per 100,000 live births, which is an elasticity of 0.3-0.4. This is 4-6 times greater than the largest cross-sectional estimate in Table 3 and is similar to the estimated effects found in Chay and Greenstone (1999). For the counties in our sample, the internal infant death rate fell by an average of 54.6 per 100,000 births from 1971-72. Thus, the estimates also imply that the entire reduction in infant mortality from 1971-72 may be attributed to the 11-unit average reduction in TSPs in our sample of counties.

The relative stability of the estimates across specifications suggests that the design is revealing a causal effect of particulates pollution. We examine this further by focusing on sets of counties with geometric mean TSPs in 1970 near the nonattainment threshold. Table 7 presents the instrumental variables estimates of the TSPs effects for various subsamples of counties with 1970 mean TSPs near the threshold. The first two columns show the estimates for the entire sample of counties, unadjusted and adjusted for the basic Natality controls, respectively. The remaining columns present unadjusted and adjusted estimates for subsamples of counties with 1970 mean TSPs ranging from 30-150, 50-100, 60-90, and 65-85 $\mu g/m^3$, respectively.

The first row contains the results for internal infant mortality rates within one year of birth. The results are striking. As the sample is limited to counties with 1970 pollution levels closer to the regulatory threshold, the estimated effect of 1971-72 mean TSPs changes increases monotonically. This is consistent with the patterns depicted in Figure 3 and suggests that the estimates of the TSPs effects are biased down due to omitted variables. The adjusted estimates for counties with 1970 TSPs levels between 30-150, 50-100, and 60-90 $\mu g/m^3$ are all significant at conventional levels, even as the sample sizes fall to 428, 276, and 173, respectively. They imply that a 1-$\mu g/m^3$ TSPs reduction results in 7.2-11.6 fewer infant deaths per 100,000 live births. The estimates do not change when the sample is limited to the 120 counties with 1970 TSPs levels between 65-85 $\mu g/m^3$, although the sampling errors double.[13] Again, we conclude that TSPs pollution has a causal impact on infant survival rates.

[13] In each case, nonattainment counties account for about half of the sample. For example, there are 58 attainment and 62 nonattainment counties with 1970 geometric mean TSPs between 65-85 $\mu g/m^3$.

Evidence on the Pathophysiologic Mechanism

There exists little evidence in the biological literature on the causal pathways through which air pollution impacts human health (Utell and Samet 1996).[14] Despite this, we attempt to address whether fetal exposure to TSPs pollution has adverse health consequences. There is strong evidence that maternal cigarette smoking can retard fetal development and reduce the birth weight of infants (Sexton and Hebel 1984, Torelli 2000). Thus, 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.

The remaining rows of Table 7 contain the estimated effects of TSPs on early infant death based on the same specifications as before. For neonatal mortality, the estimates in the first two columns, based on all counties, are highly significant and imply that a 1-$\mu g/m^3$ decline in TSPs results in 4-4.6 fewer deaths within one month of birth (per 100,000 births). Remarkably, the results for the various subsamples imply that near the nonattainment discontinuity, all of the association between changes in TSPs and infant mortality is due to reductions in neonatal deaths. The adjusted estimates for the 50-100 and 60-90 $\mu g/m^3$ county ranges are highly significant and imply that a 1-$\mu g/m^3$ TSPs reduction leads to 9.6-12.2 fewer infant deaths within 28 days of birth, per 100,000 births. Again, given the large increase in sampling errors, the estimates change little when the sample is limited to counties with 1970 TSPs levels between 65-85 $\mu g/m^3$.

The next row of the table shows that infant deaths within 24 hours of birth also have a strong association with regulation-induced changes in TSPs. In the first two sets of columns, the instrumental variables estimate varies between 2.5-3.4 and is significant at conventional levels. The remaining columns show that as the sample is limited to counties close to the regulatory threshold, reductions in deaths within 24 hours of birth account for 55-80 percent of the overall decline in infant mortality.

[14] 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 non-experimental evidence for humans, it is not clear that these findings can be extrapolated to human adults or infants.

Particulates pollution seems to have a disproportionate impact on the probability of death immediately after birth.

It appears that maternal exposure to TSPs pollution during the gestation period may affect fetal development and health. To further address this possibility, Table 8 presents the instrumental variables estimates of the association between TSPs changes and the birth weight of infants. The first two columns present the estimated effects of TSPs on average infant birth weight, unadjusted and adjusted for the basic natality controls. The remaining columns contain the estimated effects on the probability of an infant being born in a specific birth weight category, adjusted for the basic natality controls.[15] 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 estimates in columns 1 and 2 imply that a 1-$\mu g/m^3$ reduction in TSPs is associated with a 0.2-0.4 gram increase in the average birth weight of infants in a county. While significant, this estimate is small in magnitude with an implied elasticity of only 0.01. When the sample is limited to counties with 1970 TSPs levels near the regulatory threshold, as in Table 7, the estimates of the birth weight effects of TSPs have the same magnitudes. These estimates are similar to those in Chay and Greenstone (1999).

The other columns in Table 8 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 births less than 2500 grams is positive but insignificant.[16] However, TSPs have a highly significant impact on the probability of "relatively" low weight births. A unit reduction in TSPs results in 28 fewer infants born with a weight less than 3000 grams, per 100,000 live births. This is an elasticity of 0.1. Taken literally, the results suggest that almost the entire effect of TSPs on average birth weight is due to fewer infants with birth weights between 2500-3000 grams. On the other hand, TSPs have an insignificant negative correlation with incidence of very low birth weight births less than 1500 grams.

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[15] The unadjusted results are qualitatively similar.

[16] Wang, et al. (1997) document a positive correlation between TSPs and low birth weight incidence in China, where TSPs levels are 4-5 times greater than the concentrations in this study.

Overall, it appears that TSPs pollution has a much larger impact on infant mortality than on birth weight. Thus, 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.[17] These findings, including the negative association between TSPs and incidence of very low birth weight, are identical to those in Chay and Greenstone (1999).[18]

Internal Validity of the Results

Finally, 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 effects of TSPs changes on external infant mortality rates 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 regulation-induced TSPs reductions from 1971-72.

Table 9 presents instrumental variables estimates of the impact of TSPs changes on external infant mortality rates. The first five columns show a small and insignificant positive correlation between changes in TSPs and external mortality rates. The final five columns of the table show a small and insignificant negative correlation. On the whole, these results provide no evidence that our estimates in Tables 6 and 7 are the result of spurious relations.

[17] 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.

[18] It should be noted that our estimates of the effects of TSPs 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. The birth weight estimates will be understated if a disproportionate number of the marginal fetuses that survive are in the low end of the birth weight distribution.

Discussion and Estimates of the Benefits of the Clean Air Act Regulations

As discussed in the introduction, the EPA's air quality standard of 75 $\mu g/m^3$ for annual geometric mean TSPs concentrations was chosen to "protect the public health". Clearly, the optimality of the design of the 1970 CAAA depends on the existence and magnitude of the health effects above and near the federal regulatory threshold. Our research design permits a direct analysis of the health benefits of TSPs reductions at levels above and around this maximum allowable concentration. The results suggest that TSPs have substantial effects on infant mortality rates at the 75-$\mu g/m^3$ standard. Specifically, Table 7 shows that a unit reduction in TSPs results in about 12 fewer infant deaths per 100,000 live births at pollution concentrations slightly above 75 $\mu g/m^3$.

It appears that the 1970 Clean Air Act resulted in substantial health benefits for infants. In 1972 there were over 1.5 million births in the nonattainment counties in our sample. Thus, our estimates imply that at least 1,300 fewer infants died in 1972 than would have in the absence of the Clean Air Act regulations. When a statistical life is valued at \$1.6 million to \$8.5 million (\$1997), this reduction in infant mortality is worth approximately \$2.1-11.1 billion (\$1997).[19] However, this calculation is likely to understate the total economic value of the regulation-induced TSPs reductions since it ignores the other health and aesthetic benefits associated with lower pollution levels. Chay and Greenstone (2000) use a hedonic analysis to value a similar reduction in mean TSPs associated with nonattainment status in the mid-1970s. We calculated a \$65 billion (\$1997) willingness-to-pay for this change.

Taken literally, Figure 3 implies that although there are significant health benefits to a reduction in TSPs at very high concentrations, the marginal benefit of a unit change is greater at TSPs levels below 100 $\mu g/m^3$. Chay and Greenstone (1999) also find that the marginal effect of a unit-reduction in TSPs on infant mortality appears to be greater at lower concentrations. Further, that study documents that TSPs have substantial effects on infant health at concentrations below the 75-$\mu g/m^3$ regulatory ceiling. These findings suggest that a federal air quality standard below 75 $\mu g/m^3$ could provide greater health benefits

[19] 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).

to the public. However, such a standard may be less optimal in a cost-benefit sense if the marginal cost of reducing pollution emissions also increases as concentrations fall. In any event, it should be noted that both studies find that a 1-percent reduction in TSPs results in a 0.3-0.4 percent decline in infant mortality rates over a wide range of TSPs concentrations.

Conclusion

We use the air quality improvements induced by the Clean Air Act Amendments of 1970 to estimate the effects of particulates pollution on infant mortality. The federal air pollution regulations are associated with sharp reductions in TSPs pollution soon after the 1970 CAAA. We estimate that a 1-$\mu g/m^3$ reduction in TSPs results in 5-8 fewer infant deaths per 100,000 live births at the county level -- an elasticity of 0.35. Most of the reduction in infant mortality is driven by a decline in deaths occurring within one month of birth, suggesting that fetal exposure during pregnancy is a biological pathway. Consistent with this, we also find significant effects of TSPs reductions on deaths within 24 hours of birth and on infant birth weight. Finally, there are discrete differences in TSPs and infant mortality changes between counties just above and just below the legislated air quality standard, suggesting that the regulations are a causal factor.

This study provides direct and easily interpreted estimates of the air quality and health benefits of the 1970 Clean Air Act. The results imply that TSPs have substantial effects on infant health at concentrations near the EPA-mandated air quality standard and that 1,300 fewer infants died soon after the 1970 Act than would have in the absence of the regulations. Importantly, this study cross-validates the findings of our earlier work examining the recession-induced pollution reductions of the early 1980s (Chay and Greenstone 1999). Both studies find that a 1-percent reduction in TSPs results in a 0.3-0.4 percent decline in infant mortality rates over a wide range of TSPs concentrations.

Staring in the late 1980s, the EPA shifted its focus away from the regulation of all particulates up to 40 μm in diameter (TSPs) and toward the regulation of finer particles less than 10 μm (PM_{10}) and 2.5 μm ($PM_{2.5}$) in diameter. Given the data constraints, we cannot determine the relative contributions of small and large particles to the decline in infant mortality found in this study. Future work will focus on

resolving this question. Any evidence on this could also be helpful in developing a credible theory of the biological pathways through which air pollution impacts infant health (e.g., how pollution may affect fetal development before birth).

DATA APPENDIX

Variables from 1969-1974 National Mortality Detail Files

The 1969-1974 National Mortality Detail Files are an annual census of deaths in the U.S, derived from the Standard Certificate of Death. 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 1-day, 28 days, and 1-year of birth) within a demographic-county cell is used as the numerator for the infant mortality rate.

Variables from 1969-1974 National Natality Detail Files

The 1969-1974 National Natality Detail Files are an annual census of births in the U.S, 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, socioeconomic 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 maternal 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

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
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

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 EPA air monitors that were located throughout the U.S. 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 TSPs 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.[20] 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.

In the 1971-1977 period, the EPA did not publicly release the names of the counties that were designated nonattainment. To learn the identity of these counties, we contacted the EPA but were informed that records from that period "no longer exist." However, the readings from the air pollution monitoring system were used by the EPA and the states to determine which counties were in violation of the federal air quality standards. Consequently, we use our pollution data to replicate the EPA's selection rule. Counties with monitor readings exceeding the NAAQS for TSPs were assigned nonattainment status; all other counties were designated attainment.

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

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 $1982-84). 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.

We also have annual, county-level data on per-capita net earnings, the ratio of total employment to the total population, and the ratio of total manufacturing employment to the total population.

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 insurance. The analysis controls for each of the following payment sub-categories, in terms of per-capita payments (in $1982-84):

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

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Table 1: Sample Statistics, 1969-1974

	1969	1970	1971	1972	1973	1974
Number of Counties in Sample	412	501	501	501	495	489
Total Births in Sample	2,265,764	2,340,590	2,196,180	1,994,530	1,907,132	1,912,205
Total Births in U.S.	3,600,206	3,737,800	3,563,548	3,266,235	3,146,125	3,170,631
Fatalites Per 100,000 Live Births						
Internal Causes						
At 1 Day	923.4	881.3	810.2	771.8	708.9	654.1
At 1 Month	1,547.3	1,483.2	1,386.2	1,335.9	1,268.6	1,201.0
At 1 Year	1,985.3	1,899.1	1,808.0	1,752.3	1,678.6	1,597.0
External Causes						
At 1 Year	65.6	61.1	64.5	57.7	58.6	49.7
At 1 Year All Causes, by Race						
Whites	1,756.5	1,680.6	1,602.9	1,533.9	1,490.9	1,401.0
Blacks	3,213.8	3,032.0	2,828.7	2,786.2	2,589.9	2,550.6
Mean County-Level Pollution, Income, and Employment-Population Ratio						
TSPs Concentration	93.1	94.2	92.4	81.3	78.3	75.6
Per Capita Income ($1982-84)	$11,276	$11,329	$11,442	$11,991	$12,413	$12,254
Total Employment/Population	48.6	48.1	47.3	48.2	50.0	50.4
Mean Parental Demographic and Socioeconomic Characteristics						
% Mother H.S. Dropout	17.7	18.5	18.8	18.9	19.5	19.0
Mother's Years of Education	12.0	12.1	12.1	12.1	12.1	12.2
Father's Years of Education	12.6	12.6	12.6	12.7	12.8	12.8
% Single Mother	7.0	7.5	8.2	9.1	9.6	9.7
% Black	16.1	16.6	17.5	18.0	18.1	17.8
% Foreign-born	-----	8.9	9.4	-----	10.6	11.2
Mean Medical Services Utilization						
% No Prenatal Care	1.60	1.49	1.40	1.36	1.35	1.29
% Prenatal Care in 1st Trimester	55.1	55.3	56.0	57.2	60.2	61.4
Number of Prenatal Visits	-----	-----	-----	9.5	9.8	10.0
Mean Maternal Health Endowment						
% Teenage Mother	16.5	16.9	17.3	18.4	18.8	18.4
% Mom >34 Years	6.8	6.2	5.8	5.5	5.1	4.7
% First Birth	36.4	37.2	37.4	38.2	38.4	39.4
% Prior Fetal Death	10.6	10.2	10.4	10.3	10.4	10.4
Mean Infant Health Endowment						
Weight	3253	3264	3273	3276	3285	3291
% Very Low Birth Weight	1.31	1.21	1.18	1.23	1.18	1.17
% Low Birth Weight	8.3	8.1	7.8	7.9	7.7	7.6

Notes: Very low birth weight is defined as a birth weight less than 1,500 grams; low birth weight is less than 2,500 grams. The sample is limited to the 501 counties with TSPs data in 1970, 1971 and 1972. All sample means are weighted by the total number of births in the county. See the Data Appendix for information on sources and more details on how the variables were calculated.

Table 2: P-Values from F-Tests of Equality of Determinants of Infant Mortality,
by 1971 TSPs Level Quartile Groups, 1971-72 TSPs Change Quartile Groups, and 1971 Nonattainment Status

	1971 TSPs Groups	1971-1972 TSPs Groups	1971 Attainment/Nonattainment Status		
Year of Covariates:	1971	1971 - 1972	1971 - 1972	1971 - 1972	1971 - 1972
Sample Restriction:	None	None	None	50 < 1970 TSPs < 100	65 < 1970 TSPs < 85
Number of Counties	501	501	501	279	120
	(1)	(2)	(3)	(4)	(5)
TSPs Concentration	**0.0001**	**0.0001**	**0.0001**	**0.0054**	**0.0067**
County-Level Economic Variables					
Per Capita Income (1982-4$s)	**0.0137**	**0.0008**	**0.0001**	**0.0001**	0.0179
Mean Parental Demographic and Socioeconomic Characteristics					
% Mother H.S. Dropout	0.3533	0.1922	0.6277	0.5957	0.0173
Mother's Years of Education	0.0435	**0.0119**	0.0862	0.7664	0.8876
Father's Years of Education	**0.0040**	0.0258	0.1009	0.7472	0.6635
% Single Mother	0.0607	**0.0129**	**0.0097**	0.1597	0.9638
% Black	**0.0001**	0.1075	0.0205	0.0176	0.1119
Mean Medical Services Utilization					
% No Prenatal Care	**0.0001**	0.5359	0.7486	0.5596	0.5872
% Prenatal Care in 1st Trimester	0.9256	0.7733	0.6604	0.4944	0.1327
Mean Maternal Health Endowment					
% Teenage Mother	**0.0018**	0.1237	0.9154	0.4787	0.7271
% Mom >34 Years	**0.0015**	0.9940	0.9565	0.6285	0.1601
% First Birth	0.1543	0.1690	0.2007	0.4463	0.1253
% Prior Fetal Death	0.0630	**0.0010**	0.7343	0.2731	0.0640
Mean Infant Health Endowment					
Weight	**0.0001**	0.0952	0.7896	0.5490	0.4248
% Very Low Birth Weight	**0.0001**	0.0348	0.6434	0.1030	0.2018
% Low Birth Weight	**0.0001**	0.2843	0.5985	0.9799	0.9642
% Twins	**0.0004**	0.7414	0.8627	0.9426	0.4279

Notes: See the notes to Table 1. The entries are p-values from F-tests that the weighted means of the determinants of infant mortality (where the weight is the total number of births) are equivalent across sets of counties. In column (1) there are three sets of counties: the quartile of counties with the lowest 1971 TSPs concentration, the quartile with the highest 1971 concentration, and the remaining counties. In column (2) there are also three sets of counties: the quartile of counties with the smallest decline in TSPs between 1972 and 1971; the quartile with the largest decline; and, the remaining counties. In columns (3) -(5) counties are divided into two groups based on 1971 TSPs nonattainment status. In column (1) the 1971 levels of the determinants are compared, while columns (2)-(5) report the result of tests of 1971-1972 changes. In columns (1)-(3), the sample includes all counties monitored for TSPs in 1970, 1971 and 1972. In columns (4) and (5), the sample is restricted based on the TSPs concentration in 1970, which determines nonattainment status in 1971. Bold text indicates that the null hypothesis of equality can be rejected at the 1% level.

Table 3: Cross-Sectional Estimates of the Association between Mean TSPs and Infant Mortality Rates
(estimated standard errors in parentheses)

	Infant Deaths Due to Internal Causes (per 100,000 Live Births)				
	(1)	(2)	(3)	(4)	(5)
1969 Cross-Section	**2.39**	0.66	0.19	-0.17	0.15
	(0.88)	(0.39)	(0.38)	(0.37)	(0.38)
	[484, .05]	[474, .53]	[471, .61]	[404, .66]	[471, .71]
1970 Cross-Section	**1.33**	**0.82**	0.49	0.24	0.20
	(0.68)	**(0.41)**	(0.31)	(0.27)	(0.26)
	[560, .02]	[540, .45]	[528, .54]	[477, .59]	[528, .61]
1971 Cross-Section	1.55	0.75	0.19	-0.02	0.55
	(0.84)	(0.46)	(0.43)	(0.41)	(0.37)
	[760, .02]	[749, .43]	[732, .51]	[676, .56]	[732, .58]
1972 Cross-Section	0.64	-0.24	-0.42	-0.86	**-1.71**
	(0.97)	(0.69)	(0.67)	(0.59)	**(0.66)**
	[1051, .00]	[1022, .24]	[998, .30]	[917, .34]	[998, .37]
1973 Cross-Section	2.12	0.94	0.06	-0.66	0.62
	(1.12)	(0.59)	(0.50)	(0.51)	(0.52)
	[1185, .01]	[1159, .37]	[1132, .45]	[1032, .48]	[1132, .49]
1974 Cross-Section	1.95	**1.32**	0.87	0.55	**1.33**
	(1.05)	**(0.65)**	(0.49)	(0.53)	**(0.56)**
	[1271, .01]	[1238, .34]	[1210, .41]	[1114, .44]	[1210, .48]
Basic Natality Vars.	N	Y	Y	Y	Y
Unrestricted Natality	N	N	Y	Y	Y
Income, Employment	N	N	N	Y	N
Income Assist. Sources	N	N	N	Y	N
State Effects	N	N	N	N	Y

Notes: See notes to Table 1. Numbers in brackets are the number of counties and R-squareds of the regressions, respectively. Sampling errors are estimated using the Eicker-White formula to correct for heteroskedasticity. 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 1. State Effects are separate indicator variables for each state.

Table 4: Fixed Effects Estimates of Association between Mean TSPs and Infant Mortality Rates
(estimated standard errors in parentheses)

	Infant Deaths Due to Internal Causes (per 100,000 Live Births)				
	(1)	(2)	(3)	(4)	(5)
1971-1972 Pooled					
Mean TSPs	0.91	0.65	0.11	-0.13	-0.17
	(0.77)	(0.74)	(0.71)	(0.72)	(0.71)
R-squared	0.78	0.79	0.81	0.82	0.82
Dep. Var. Mean	1784	1784	1783	1786	1786
Sample Size	1811	1771	1730	1593	1593
1971-1974 Pooled					
Mean TSPs	2.27	1.47	0.35	0.18	0.10
	(0.46)	(0.46)	(0.42)	(0.41)	(0.41)
R-squared	0.62	0.64	0.67	0.68	0.68
Dep. Var. Mean	1714	1713	1712	1715	1715
Sample Size	4267	4168	4072	3739	3739
County Fixed Effects	Y	Y	Y	Y	Y
Basic Natality Vars.	N	Y	Y	Y	Y
Unrestricted Natality	N	N	Y	Y	Y
Income, Employment	N	N	N	Y	Y
Income Assist. Sources	N	N	N	Y	Y
Year Effects	N	N	N	N	Y

Notes: Regressions are based on pooled data and include county indicator variables as controls. Standard errors are estimated using the Eicker-White formula to correct for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Table 5: Reduced-Form Estimates of the Impact of 1971 Nonattainment Status on 1971-1972 Changes in TSPs Pollution and Internal Infant Mortality Rates
(estimated standard errors in parentheses)

	1971-72 Change in Mean TSPs			1971-72 Change in Infant Mortality		
	(1)	(2)	(3)	(1)	(2)	(3)
Nonattainment in 1971	-14.1	-13.6	-10.0	-67.1	-83.3	-84.6
	(2.0)	(2.2)	(2.3)	(20.7)	(23.4)	(38.3)
County Fixed Effects	Y	Y	Y	Y	Y	Y
Basic Natality Vars.	N	Y	Y	N	Y	Y
Unrestricted Natality	N	N	Y	N	N	Y
Year Effects	N	N	Y	N	N	Y
F-stat. Nonattainment	48.1	39.5	19.5	10.5	12.7	4.9
(numerator d.o.f.)	(1)	(1)	(1)	(1)	(1)	(1)
F-stat. other variables		0.9	1.4		1.6	1.9
(numerator d.o.f.)		(11)	(43)		(11)	(43)
R-Squared	0.26	0.28	0.34	0.02	0.07	0.15
Dep. Variable Mean	-11.3	-11.3	-11.3	-54.6	-53.7	-51.2
Sample Size	501	489	474	501	489	474

Notes: The dependent variables are the 1971-72 first-differences of mean TSPs pollution and 1971-72 first-differences in infant deaths due to internal causes per 100,000 live births. Nonattainment in 1971 is an indicator variable equal to one if the county had TSPs concentrations above the federal air quality standard for TSPs in 1970. Standard errors are estimated using the Eicker-White formula to correct for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Table 6: Instrumental Variables Estimates of the Effect of Mean TSPs on Infant Mortality Rates, Based on 1971-72 Changes Using 1971 Attainment Status as Instrument
(estimated standard errors in parentheses)

	1971-1972 Change in Infant Deaths (per 100,000 Live Births)				
	(1)	(2)	(3)	(4)	(5)
Change in Mean TSPs	4.76	6.14	8.48	7.78	16.77
	(1.57)	(1.92)	(4.12)	(4.80)	(7.09)
County Effects	Y	Y	Y	Y	Y
Basic Natality Vars.	N	Y	Y	Y	Y
Unrestricted Natality	N	N	Y	Y	Y
Income, Employment	N	N	N	Y	N
Income Assist. Sources	N	N	N	Y	N
Year Effects	N	N	Y	Y	N
State-Year Effects	N	N	N	N	Y
Sample Size	501	489	474	449	474

Notes: Results are from two-stage least squares estimation using 1971-72 first-differences, with changes in mean TSPs instrumented by nonattainment status in 1971. Estimated standard errors allow for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Table 7: Instrumental Variables Estimates for Infant Deaths within 1-Year, 1-Month, and 1-Day of Birth
For Counties with1970 Geometric Mean TSPs near Regulatory Threshold
(estimated standard errors in parentheses)

	1971-72 Change in Infant Deaths Due to Internal Causes (per 100,000 Live Births)									
			1970 Geometric Mean TSPs in the Range of							
	All Counties		30-150 $\mu g/m^3$		50-100 $\mu g/m^3$		60-90 $\mu g/m^3$		65-85 $\mu g/m^3$	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Deaths w/in 1 year										
Mean TSPs	4.76	6.14	5.57	7.16	7.53	10.20	8.36	11.64	10.80	12.99
	(1.57)	(1.92)	(1.98)	(2.11)	(4.69)	(4.62)	(5.36)	(5.71)	(10.56)	(10.32)
Deaths w/in 28 days										
Mean TSPs	3.95	4.60	4.72	5.64	7.61	9.61	9.09	12.17	11.98	14.76
	(1.27)	(1.51)	(1.62)	(1.71)	(3.98)	(3.77)	(4.67)	(4.80)	(9.51)	(9.00)
Deaths w/in 1 day										
Mean TSPs	2.88	2.48	3.41	3.01	5.99	5.79	6.52	6.22	8.68	6.86
	(0.97)	(1.21)	(1.21)	(1.37)	(3.14)	(2.93)	(3.22)	(3.36)	(6.57)	(4.83)
Basic Natality Vars.	N	Y	N	Y	N	Y	N	Y	N	Y
Sample Size	501	489	435	428	279	276	176	173	120	117

Notes: The dependent variables are the 1971-72 first-differences in the number of infant deaths due to internal causes within one-year, 28-days, and 24-hours of birth (per 100,000 live births). The columns correspond to subsamples of counties with annual geometric mean readings of TSPs in 1970 in the specified range. Results are from two-stage least squares estimation, with 1971-72 changes in mean TSPs instrumented by nonattainment status in 1971. Estimated standard errors allow for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

Table 8: Instrumental Variables Estimates of Effects of Mean TSPs on Infant Birth Weight, 1971-72
(estimated standard errors in parentheses)

	Infant Birth Weight		Incidence in Birth Weight Categories (per 100,000 Live Births)					
	(1)	(2)	<1500g	<2000g	<2500g	<3000g	<3500g	<4000g
Mean TSPs	-0.19	-0.43	-1.77	-0.48	2.61	28.33	38.28	11.80
	(0.11)	(0.12)	(1.94)	(2.91)	(4.54)	(8.80)	(9.34)	(5.05)
Basic Natality Vars.	N	Y	Y	Y	Y	Y	Y	Y
1971 Dep. var. mean	3274	3273	1.2%	2.7%	7.9%	26.9%	65.6%	91.5%
Sample Size	501	489	489	489	489	489	489	489

Notes: The dependent variables in the 'Incidence in Birth Weight Categories' columns are the fraction of births with birth weight less than the specified amount. Results are from two-stage least squares estimation, with 1971-72 changes in mean TSPs instrumented by nonattainment status in 1971. Estimated standard errors allow for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

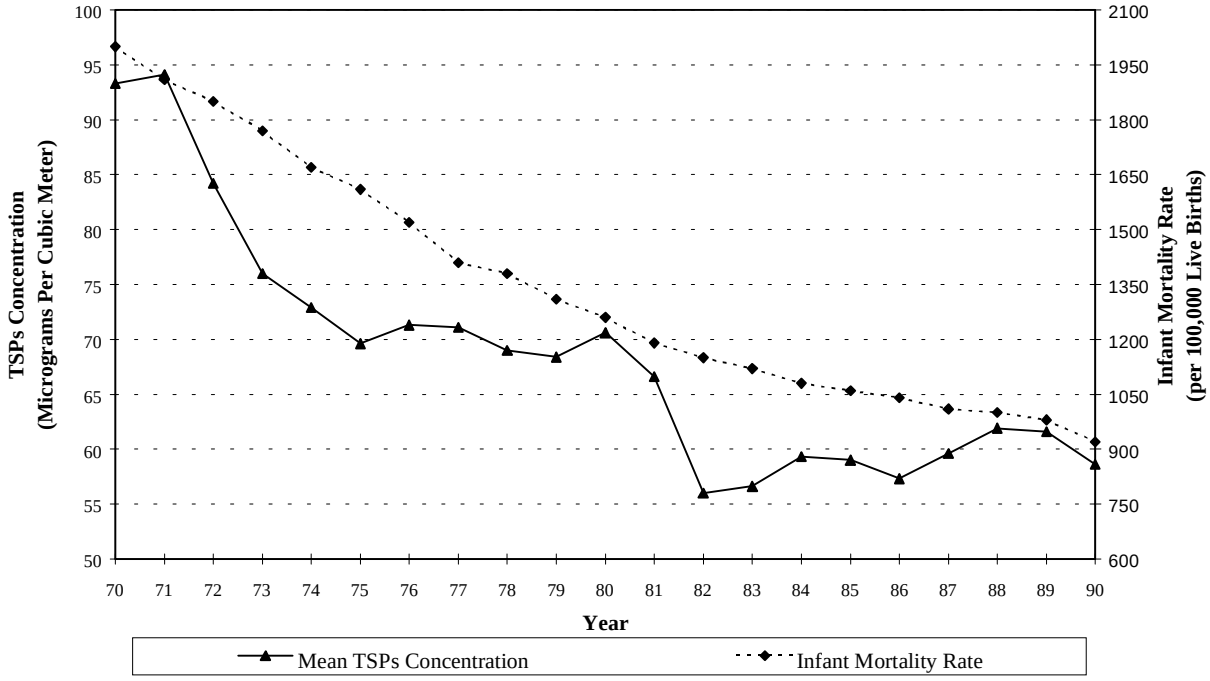
Table 9: Instrumental Variables Estimates of the Effect of Mean TSPs on Infant Deaths Due to External Causes, Based on 1971-1972 Changes
(estimated standard errors in parentheses)

	1971-72 Change in Infant Deaths Due to External Causes (per 100,000 Live Births)									
			1970 Geometric Mean TSPs in the Range of							
	All Counties		30-150 μg/m^3		50-100 μg/m^3		60-90 μg/m^3		65-85 μg/m^3	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Deaths w/in 1 year										
Mean TSPs	0.34	0.10	0.40	0.19	0.10	-0.36	-0.45	-0.31	-0.72	-0.39
	(0.24)	(0.31)	(0.29)	(0.34)	(0.87)	(0.95)	(0.99)	(1.10)	(1.73)	(1.74)
Basic Natality Vars.	N	Y	N	Y	N	Y	N	Y	N	Y
Sample Size	501	489	435	428	279	276	176	173	120	117

Notes: The dependent variable is the 1971-72 first-differences in number of infant deaths due to external causes, such as accidents and homicides, within one-year of birth (per 100,000 live births). The columns correspond to subsamples of counties with annual geometric mean readings of TSPs in 1970 in the specified range. Results are from two-stage least squares estimation, with 1971-72 changes in mean TSPs instrumented by nonattainment status in 1971. Estimated standard errors allow for heteroskedasticity. Regressions are weighted by numbers of births in each county and year.

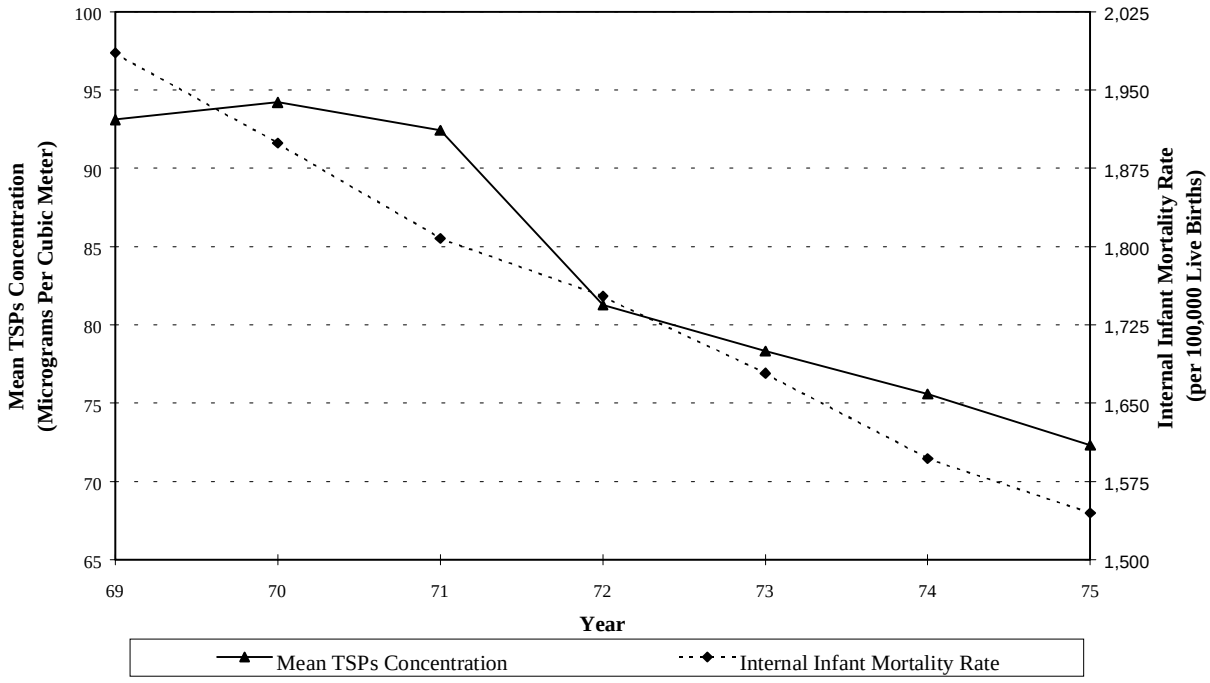
Figure 1: Trends in Total Suspended Particulates Pollution and Infant Mortality Rates

A: National Trends from 1970-1990



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

B: Trends for Fixed Set of Counties with TSPs Data, 1969-1975



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

Figure 2: Trends in Total Suspended Particulates Pollution 1969-1974, by 1971 Nonattainment Status

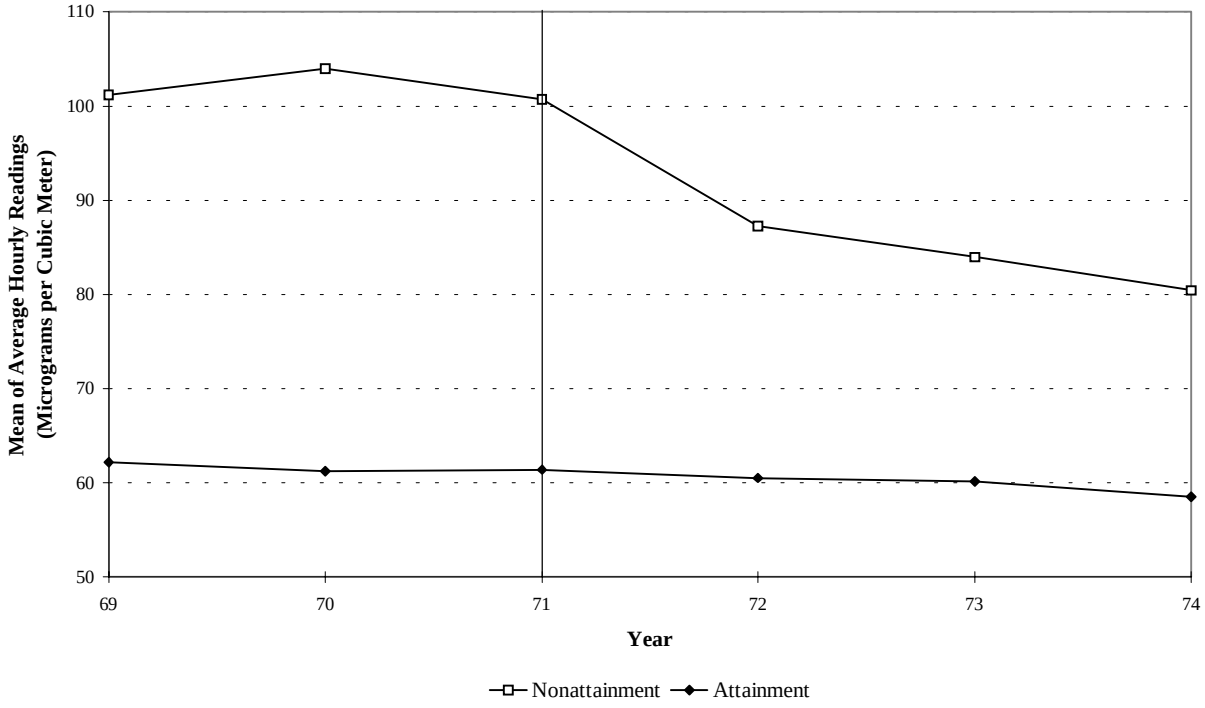
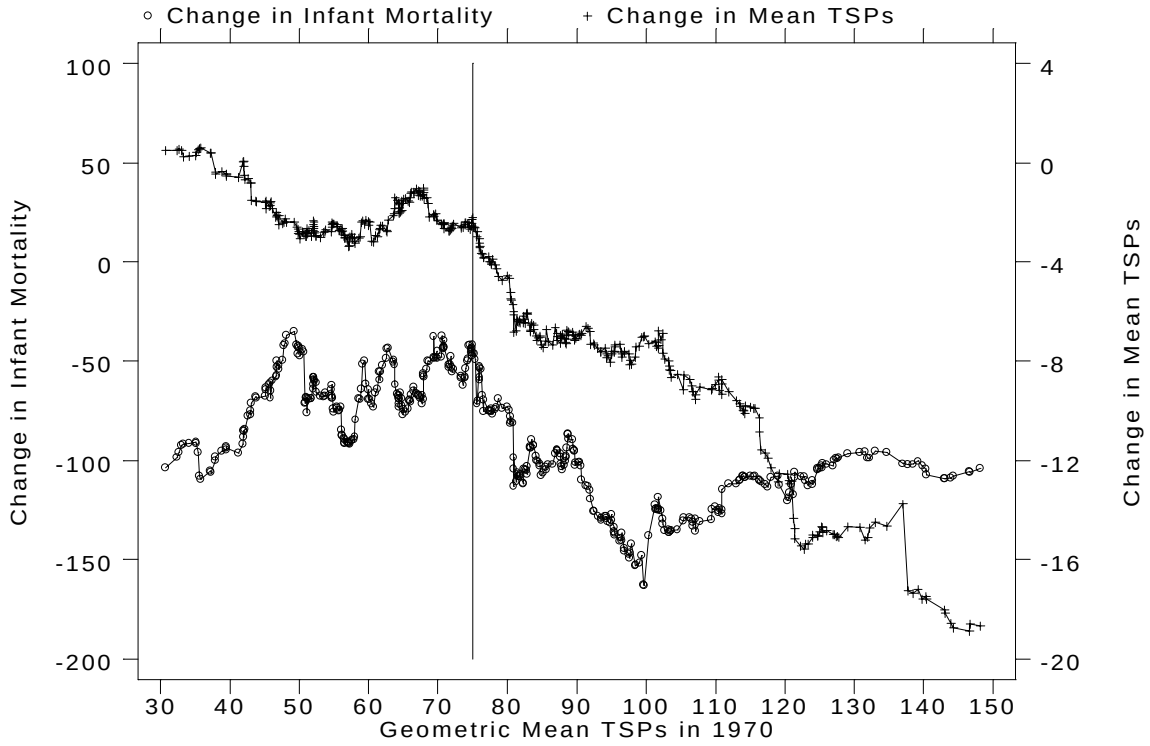


Figure 3: 1971-72 Post-Regulation Changes in Mean TSPs and Internal Infant Mortality Rates By Geometric Mean TSPs in Regulation Selection Year, 1970

A. 1971-72 Post-Regulation Changes in Mean TSPs and Infant Mortality Rates



B. Changes for Counties with 1970 Geometric Mean TSPs between 50-100 μg/m^3

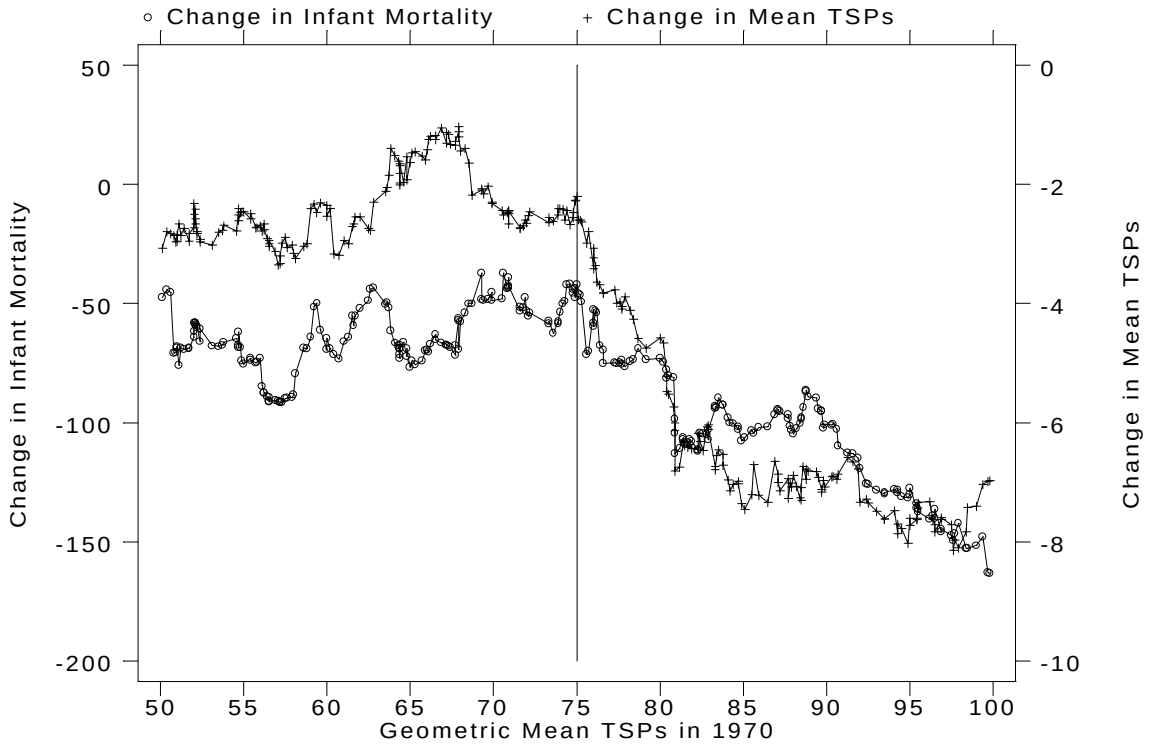
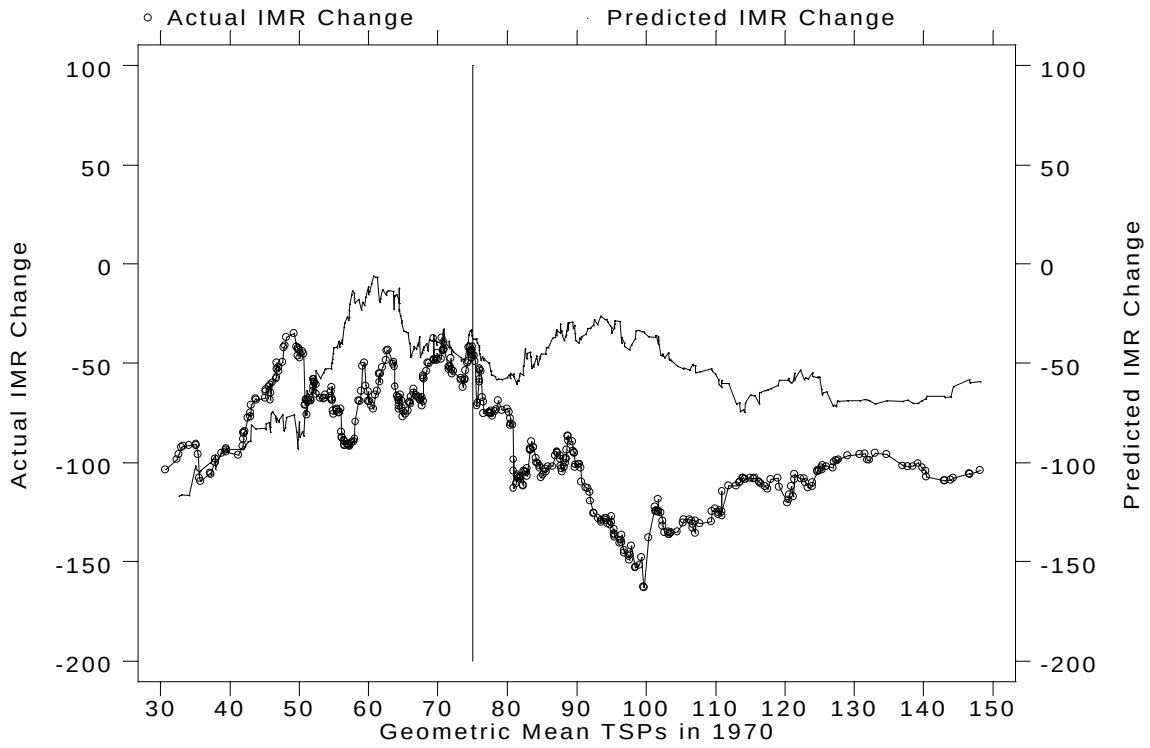


Figure 4: Checks of Robustness of the Effects of 1971 Nonattainment Status

A. 1971-72 Post-Regulation Changes in Actual and Predicted Infant Mortality Rates



B. 1969-70 Pre-Regulation Changes in Mean TSPs and Infant Mortality Rates