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Estimating Causal Effects of Particulate Matter Regulation on Mortality

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Abstract

Background: Estimating the causal effect of pollution on human health is integral for evaluating returns to pollution regulation, yet separating out confounding factors remains a perennial challenge.

Methods: We use a quasi-experimental design to investigate the causal relationship between regulation of particulate matter smaller than 2.5 micrograms per cubic meter (PM_{2.5}) and mortality among those 65 years of age and older. We exploit regulatory changes in the Clean Air Act Amendments (CAAA). Regulation in 2005 impacted areas of the United States (U.S.) differentially based on pre-regulation air quality levels for PM_{2.5}. We use county-level mortality data, extracted from claims data managed by the Centers for Medicare & Medicaid Services, merged to county-level average PM_{2.5} readings and attainment status as classified by the EPA.

Results: Based on estimates from log-linear difference-in-differences models, our results indicate after the CAAA designation for PM_{2.5} in 2005, PM_{2.5} levels decreased 1.59 micrograms per cubic meter (95% CI 1.39-1.80) and mortality rates among those 65 and older decreased by 0.93% (95% CI 0.10%-1.77%) in nonattainment counties, relative to attainment ones. Results are robust to a series of alternate models, including nearest-neighbor matching based on propensity score estimates.

Conclusion: This analysis suggests large health returns to the 2005 PM_{2.5} designations, and provides evidence of a causal association between pollution and mortality among the Medicare population.

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Data are restricted usage. Code available for replication upon request.

Keywords

particulate matter; mortality; environmental policy; quasi-experimental studies

Introduction

Estimating the causal effect of pollution on human health is integral for assessing costs and benefits of pollution regulation. Pollution correlates with many factors that affect both economic and health outcomes independent of pollution, complicating the isolation of causality. For example, pollution can reduce the amenity value of living in a particular area and therefore affect housing prices [1]. As a result, people may select into high and low pollution areas in non-random ways. Simply comparing health outcomes of populations in more polluted places to other populations would capture not only differences in pollution levels, but differences in a set of other hard-to-measure factors.

Quasi-experimental designs can help isolate causal relationships by leveraging an event that effectively assigns populations into either “treatment” or “control” groups exposed to differing levels of pollution (or, in our case, changes in levels of pollution). A key feature is that the event designating treatment status is determined by policy, nature, or some other external factor such that being exposed to the treatment – which usually varies by both geography and time – is ideally as good as randomly assigned. Applied research uses a variety of quasi-experimental designs, particularly in the social sciences, to estimate causal effects in contexts such as pollution regulations [2].

Here we use a quasi-experimental design in an effort to isolate the causal relationship between regulation of particulate matter smaller than 2.5 micrograms per cubic meter ($PM_{2.5}$) and reductions in $PM_{2.5}$ and associated mortality. We exploit changes in air quality standards from the Clean Air Act Amendments (CAAA), beginning in 2005, that impacted areas of the United States (U.S.) differentially based on prior air quality levels. As we demonstrate below, this led to substantial reductions in pollution in some areas, but not others. In particular, the National Ambient Air Quality Standards (NAAQS) for $PM_{2.5}$ stipulate an acceptable threshold for the annual mean of $PM_{2.5}$, based on a 3-year average, of 15 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) [3] (Current standards stipulate a threshold of 12 $\mu\text{g}/\text{m}^3$). If $PM_{2.5}$ levels in a county exceed the threshold, the federal government classifies the county in violation of the CAAA and applies a status of “nonattainment”. When this occurs, the state where the county resides must develop a State Implementation Plan (SIP), requiring approval from the US Environmental Protection Agency (EPA), detailing measures the state will take to lower $PM_{2.5}$ levels in nonattainment counties [3]. Although the NAAQS for annual $PM_{2.5}$ occurred in 1997, the federal government designated attainment status in 2005, assigning attainment status on $PM_{2.5}$ values from 2001 through 2003. Once the EPA began enforcing the CAAA in 2005, it caused a stark reduction in pollution in nonattainment counties relative to attainment counties.

Our empirical model exploits both the timing of the policy and its differential effect across counties depending on attainment status. We compare changes in mortality over time within treatment counties (in violation of the NAAQS in 2005) to the changes in mortality over

time within control counties (in compliance with NAAQS in 2005). For valid causal inference, this quasi-experimental “difference-in-differences” approach assumes the policy-induced changes in pollution over time are unrelated to other factors determining health once we factor out baseline differences across counties and nationwide secular trends. We compare results from this difference-in-differences model with an associational model estimating the relationship between changes in pollution over time, regardless of attainment status.

We build on a large literature documenting a sizable relationship between pollution and health outcomes. Some of the earliest examples of quasi-experimental designs on air pollution are studies by Pope and co-authors ([4]; [5]; [6]), which examine changes in pollution resulting from a labor strike-driven closure of a steel mill, and showed local improvement on a range of morbidity measures. Other quasi-experimental papers focus on the NAAQS for total suspended particles from the 1970 and 1977 CAAs. [7] focus on adult mortality; [8] on fetal mortality; and [9] on exposure during early childhood and changes in adult earnings. We build on the successful design of these previous studies, while furthering the research on environmental policy and health outcomes by exploiting the more recent NAAQS with respect to $PM_{2.5}$.

Data

Our raw data are at various degrees of time aggregation. Given attainment status, population, and mortality data are annual and at the county level, we aggregate all other data to the county-by-year level as well. All data span the years 2000-2013. The online appendix table shows how missing values across the various data sets affect the sample size by year.

Mortality data.—We use mortality data extracted from the claims managed by the Centers for Medicare & Medicaid Services. Among Medicare beneficiaries, we identify all deaths along with information on year of death, age, gender, and Federal Information Processing Standard (FIPS) code of residence. Our primary outcome is the log of deaths per 100,000 population 65 years or older in a given county/year. Some models examine deaths by age and gender subgroups; there, we use the relevant population in the denominator to construct mortality rates (e.g., 65 and older males).

Pollution data.—We calculate daily 24-hour average $PM_{2.5}$ (in micrograms/cubic meter) for each county as a simple average of all monitors within a county (if multiple monitors exist). After aggregating to the annual level, we merge these data to the Medicare data using the FIPS code of residence.

Age denominator.—To construct a mortality rate, we divide death counts by the estimated relevant population in a given county and year. The population data come from National Cancer Institute’s Surveillance, Epidemiology, and End Results (SEER) program. We also include log of the relevant population as a control in our main regressions. This addresses the possibility that population growth might correlate with unobservable confounders.

Attainment status.—Using the “Green Book Nonattainment Areas” hosted on the EPA’s website, we obtained the attainment status of each county in the US to identify them as treatment or control counties as of 2005 (Obtained from <https://www.epa.gov/green-book> as of May 20th, 2019.) The definition of the law allows for counties to be in “partial attainment,” where only certain geographic areas of the county are classified as in nonattainment. For our purposes, we consider these partial attainment counties as treated as they were still subject to regulation.

Weather data.—We control for temperature and rainfall to address the concern that attainment status spuriously correlates with weather, which might affect mortality risk independent of pollution [10]. We use data from the Global Historical Climate Network Daily (GHCND) data, maintained by the National Oceanic and Atmospheric Administration [11]. The GHCND data have information on (daily) maximum and minimum temperature and total precipitation for over 4,000 weather stations throughout the United States during our time period. We calculate weather conditions at the county level using weather stations within 100 miles of the county centroid and inverse distance weights.

Economic data.—We use income per capita and share of population employed, available annually at the county level from the Bureau of Economic Analysis (BEA) Regional Economic Accounts (<http://bea.gov/regional/index.htm>), as controls to mitigate potential confounder bias.

Migration data.—We bring in migration data to assess whether populations moved as a result of improvements in air quality. We use county-level migration data from the Internal Revenue Service (IRS) (Available at <https://www.irs.gov/statistics/soi-tax-stats-migration-data>, accessed May 20th, 2019). Although the IRS data do not measure population per se, they report the number of returns and exemptions claimed on tax returns, which serve as a proxy for the number of households and the population, respectively. Data record the number of tax returns for movers changing county of residence in a given year, both in terms of inflows and outflows. We calculate net changes in migration using the number of inflows minus outflows. The IRS data do not allow for us to test for differential migratory responses by certain subpopulations. Nonetheless, observing no migratory response across the whole population could help rule out the possibility that people with differential health characteristics were more or less likely to migrate, which would bias estimates.

Methodology

We have two main estimating equations: a standard difference-in-differences estimator and a more flexible “event study” design. Equations (1) and (1’) show the standard difference-in-differences models for $PM_{2.5}$ and mortality:

$$PM_{2.5ct} = \alpha(\text{Treatment}) * (\text{Year} \geq 2005) + \delta_c^p + \lambda_t^p + \epsilon_{ct}^p \quad (1)$$

$$\log(\text{mortality rate})_{ct} = \pi(\text{Treatment}) * (\text{Year} \geq 2005) + \mu * \log(\text{population})_{ct} + \delta_c^m + \lambda_t^m + \varepsilon_{ct}^m \quad (1')$$

Equation (1) tests if the policy reduced pollution in treatment counties relative to control counties and equation (1') tests if the policy reduced the log of the mortality rate in treatment counties relative to control counties. Observations are at the county-by-year level, where the subscript c and t indicate county and time, respectively. Superscript p indicates the $PM_{2.5}$ regression model, while m indicates the mortality regression model. "Treatment" is the nonattainment status indicator, taking a value of 1 if the county is in nonattainment and 0 otherwise. "Year 2005" is the post-regulation indicator, taking a value of 1 for the years 2005 and beyond and 0 otherwise. α and π are difference-in-differences estimates: the change in outcomes for treatment counties, as compared to control counties, after the NAAQS regulations come into effect. δ_c is a vector of county fixed effects (controlling for baseline differences between treatment and control counties) and λ_t is a vector of year fixed effects (controlling for common changes in outcomes across all counties before and after the policy). Although population is used in the denominator of the dependent variable, μ mitigates confounders related to population growth over time.

To better trace out the pre- and post-policy differences between treatment and control counties, equations (2) and (2') show a more flexible "event study" design:

$$PM_{2.5ct} = \sum_{t \neq 2005} \alpha_t (\text{Treatment}) * (\text{Year} = t) + \delta_c^p + \lambda_t^p + \varepsilon_{ct}^p \quad (2)$$

$$\log(\text{mortality rate})_{ct} = \sum_{t \neq 2005} \pi_t (\text{Treatment}) * (\text{Year} = t) + \mu^m * \log(\text{population})_{ct} + \delta_c^m + \lambda_t^m + \varepsilon_{ct}^m \quad (2')$$

These equations are similar to (1) and (1'), but here α_t and π_t flexibly capture the effects of treatment over time by using separate dummy variables for each year. We interact these dummy variables with attainment status for $PM_{2.5}$ in 2005 ("Treatment"). We omit 2005 in the yearly interactions, making α_t and π_t the difference between treatment and control counties in year t as compared to the difference between treatment and control counties in the year 2005.

In all mortality regressions, we weight by the relevant group population to account for the accuracy of the rate as the dependent variable. We use no weights in regressions with pollution as the outcome. We cluster all error terms at the county level to flexibly account for the group nature of data [12]. All individuals in the same county face the same pollution and attainment status, and clustering allows for serial correlation in the error term within a given county over time. The cluster estimator also allows for heteroskedastic errors.

We use these empirical models to address two questions: (1) did attainment status under the NAAQS affect $PM_{2.5}$ levels, and (2) did attainment status reduce mortality? The results reveal the causal effect of the policy on $PM_{2.5}$ and mortality. Our difference-in-differences

model plausibly mitigates concerns of confounders (e.g., general health or macroeconomic trends) that spuriously correlate with the policy.

To assess the internal validity of our difference-in-differences model (i.e., evaluate how well attainment counties serve as controls for nonattainment counties), we provide two tests. First, we assess whether adding additional control variables (meteorologic elements and economic variables) to our equation affects estimates of the coefficients of interest. If adding these variables does not affect our coefficients of interest, this suggests the change in $PM_{2.5}$ is uncorrelated with other factors correlated with the included controls. Second, we estimate models using migration variables as the outcome in equation (2). If we find attainment status is not statistically related to migration, this provides evidence consistent with the hypothesis that compositional shifts in the population are not an important confounder of the effects of the CAAs on mortality.

With difference-in-differences models, a common concern is differential trending in treatment and control groups even absent treatment. Our event study design allows us to explore if such trends exist pre-treatment, and, as we demonstrate below, we find suggestive evidence of some differential trends in mortality. To address this issue, as a robustness check we use nearest neighbor matching based on a propensity score estimate of the probability of attainment status. The independent variables used to estimate the propensity score are the mortality counts and population for each year from 2000 to 2005, where we sample without replacement.

To identify the causal effect of $PM_{2.5}$ on mortality itself requires further assumptions. To that end, we also perform an instrumental variables (IV) analysis by dividing the mortality coefficient (π) by the $PM_{2.5}$ coefficient (α), to obtain an estimate of the effect of $PM_{2.5}$ on mortality. This is equivalent to a Wald IV estimator [13], where the standard error of the estimate is calculated via two stage least squares [14]. The two assumptions for a valid IV are: (1) the policy has an effect on $PM_{2.5}$, and (2) the only route through which the policy affects mortality is through $PM_{2.5}$. While we can directly test the first assumption ($\alpha > 0$), there is no formal test for the second. A specific concern is that NAAQS regulation affected mortality through other channels, such as co-pollutants. We interpret our IV estimates with this caveat in mind and generally focus our mortality discussion around the causal effects of the policy (and not $PM_{2.5}$).

For comparison purposes, we also estimate a model that compares the within-county changes in $PM_{2.5}$ with the within-county changes in mortality rates according to a standard ordinary least squares (OLS) model:

$$\log(\text{mortality rate}_{ct}) = \beta * PM_{2.5} + \mu^w * \log(\text{population})_{ct} + \delta_c^w + \lambda_t^w + \varepsilon_{ct}^w \quad (3)$$

The term β gives the percent change in the mortality rate from a $1 \mu\text{g}/\text{m}^3$ change in $PM_{2.5}$. All terms are defined as before, where the superscript w indicates the “within” regression model for mortality. This model provides a useful benchmark to compare to our IV estimates.

We perform all statistical analyses using the computer program Stata, version 15.0 (StataCorp LP, College Station, TX). We make use of several user-written Stata commands: `psmatch2`, `ftools`, `reghdfe`, and `outreg2`. Each of these commands are available on the Boston College Statistical Software Components (SSC) archive. An independent IRB review performed by Chesapeake IRB (now Advarra) determined that the project did not constitute human subjects research.

Results

Figure 1 shows a map of treatment (nonattainment) and control (attainment) counties for our final analysis sample. There are 137 nonattainment counties and 467 control counties in 2006, though this varies by year because we do not restrict our sample to a balanced panel of counties in order to preserve sample size. The included counties represent around 200 million people in the US (shown in the eAppendix).

Table 1 shows summary statistics for our data. The annual mortality rate is 4.843 per 1,000 population over 65; annual average $PM_{2.5}$ is $10.84 \mu\text{g}/\text{m}^3$. The lower panel of Table 1 shows these values separately for attainment and nonattainment areas before and after the NAAQS. In both attainment and nonattainment areas, the mortality rate fell by approximately 0.5 per 1,000. $PM_{2.5}$ levels decreased from 11.0 to 9.3 in attainment areas and 15.3 to 12.0 in nonattainment areas.

Table 2 presents results from the difference-in-differences estimate based on equation (1). Column 1 shows the estimate for particulates, which implies $PM_{2.5}$ in treatment counties decreased by $1.591 \mu\text{g}/\text{m}^3$ (95% CI $-1.797, -1.386$) relative to control counties after the AQS for $PM_{2.5}$. Column 2 shows the change in log mortality rates, estimated at .009 (95% CI $-.018, -.001$), or an approximate 1% decrease for treatment counties relative to control counties.

We combine these two estimates to calculate the IV effect of $PM_{2.5}$ on mortality, shown in Table 3. Assuming the policy exclusively affects mortality via changes in $PM_{2.5}$, the IV estimate indicates that a $1 \mu\text{g}/\text{m}^3$ change in $PM_{2.5}$ causes a .006 change in the over-65 log mortality rate (95% CI .001-.011), which is a 0.59% change. This assumption is strong, and as such we favor the causal interpretation for a link between the policy and mortality. The within estimates, based on equation (3), suggest a .002 increase in mortality (95% CI .000, .003) from a $1 \mu\text{g}/\text{m}^3$ increase in $PM_{2.5}$, which is a .16% change.

Our next results illustrate the effect of the policy over time using our event study design based on equation (2). Figure 2 graphs the coefficient estimates and 95% confidence intervals for estimates for $PM_{2.5}$. Levels decreased after the $PM_{2.5}$ regulation took hold in 2005; the coefficient for the year 2006 implies $PM_{2.5}$ levels dropped by 1.05 micrograms per cubic meter in nonattainment areas relative to attainment counties just one year after the policy went into effect. The difference in pollution between nonattainment and attainment counties increases in the years since the policy's implementation, while differences in the years preceding the policy are close to zero, suggesting pre-existing trends in $PM_{2.5}$ are not systematically different in attainment vs. nonattainment counties.

Figure 3 presents the event study estimates based on equation (2') for mortality. We see mortality for all individuals over age 65 decreased in nonattainment counties, relative to attainment counties, after the PM_{2.5} regulation took hold. Results suggest that by 2013, mortality dropped by approximately 1% in nonattainment counties relative to attainment counties. Health improvements appear gradual in the early years after the policy, growing over time. We do not observe differential trends in mortality prior to the introduction of the policy in 2005, suggesting pre-existing trends in mortality rates are unlikely to be a source of bias. Combined with the results in Figure 2, these results show abatement of PM_{2.5} moves in line with the decreases in mortality, suggesting a contemporaneous effect of PM_{2.5} on health.

Table 4 presents a series of robustness checks for the mortality results. Column 1 adds the weather variables and economic controls. Consistent with the exogeneity of the policy change, adding these variables has minimal impact on our coefficients of interest or standard errors, though it does change our sample size slightly due to occasional missing values in the additional covariates. Column 2 shows results using a nearest-neighbor matching approach to better control for any trends in mortality before the AQS. Estimates are again quite similar, though with the substantial reduction in observations our confidence intervals widen. Column 3 replaces nearest-neighbor matching with restricting to a common propensity score support, and results are again quite similar.

The results in Figure 4 further probe the role of pre-existing trends by reproducing the mortality results in the event study framework using nearest neighbor matching. Compared with Figure 3, pre-trends look even more similar when we perform matching, with all coefficient estimates closer to zero. The magnitudes of the effects of the regulations in the post-treatment period is comparable to those shown in Figure 3, suggesting any possible differences in pre-trends do not explain our main findings.

Table 5 presents results exploring heterogeneity of mortality estimates by gender and age (note our sample changes slightly by subgroups given the occasional cells with zero deaths, which are undefined for the log function). The estimates in columns 1 and 2 show that the effects on mortality are smaller for males than females, though confidence intervals are large. Column 3 shows the mortality effects for only those over age 75 are larger than those over age 65 (including those over 75), though again confidence intervals are broad.

We next explore the effect of attainment status on migration. If people respond to improved pollution levels by migrating, changes in mortality may be due to changes in population composition rather than a direct effect of PM_{2.5}. Table 6 shows attainment status has a small estimated relationship with migration as measured by tax returns filed or tax exemptions claimed. We do not use the log function here, as net returns and exemptions can both be negative.

Discussion

Our study has attempted to isolate the causal effect of PM_{2.5} regulation through the NAAQS on PM_{2.5} concentrations and mortality using a quasi-experimental difference-in-differences

research design. As a point of comparison, [15] also examined mortality rates among the Medicare population using particulate matter NAAQS regulations. In a comparison of mortality rates across different counties between 2010 and 2012, they found nonattainment designation was associated with a decrease in mortality rates of approximately 1.251 per 1,000 (95% CI $-2.631, 0.108$). Using our average mortality rate of 48 per 1,000, our estimate finds counties in nonattainment saw 0.436 fewer deaths per 1,000, putting our estimates within prior estimate confidence intervals. In similar research, [16] find nonattainment for particulate matter smaller than 10 micrometers (PM₁₀) reduced mortality by 1.08 deaths per 1,000 among Medicare beneficiaries.

Our analysis uses a log-linear regression rather than a Poisson regression because the mortality data are annual aggregates and are far from zero; the mean number of counts is 48.4 and in only two instances do we observe zero deaths in a county-year. Poisson regression is most appropriate when counts are low; otherwise they approximate a linear regression model. The log-linear regression yields a coefficient with a comparable interpretation as the coefficient from a Poisson regression.

We find large estimated decreases in both pollution and mortality risk. Several caveats remain in interpreting our results. First, changes in attainment status could lead to other changes in population characteristics that correlate with mortality, suggesting a potential source of unobserved confounding. We provide two pieces of evidence that diminish this concern: 1) estimates are robust to the inclusion of various economic and weather controls; and 2) estimates show no detectable migratory response to NAAQS.

Second, in our IV estimate, the AQS for PM_{2.5} may have affected other pollutants as well, hindering the interpretation of this estimate as the effect of PM_{2.5} on mortality. For example, emission control strategies that limit PM_{2.5} may also limit ozone, which impacts mortality [17]. While these other pollutants likely have a smaller effect on mortality, we cannot rule out that some of the IV estimate may be driven by changes in the other pollutants.

Third, our estimates may violate the stable unit treatment value assumption—reduced emissions in a treatment county may affect pollution concentrations and therefore health outcomes in neighboring counties. Atmospheric modeling suggests particulate emissions can travel hundreds of miles with predominant wind patterns [18]. If such spillovers contaminate our control group, they bias both our PM_{2.5} and mortality estimates downward. Table 1 hints at this possibility, as PM_{2.5} decreased in attainment areas (though by less than in nonattainment areas). Our nearest neighbor matching algorithm limits some of this concern—the control group focuses on counties most similar in mortality rates, which may or may not be geographically proximate to treatment counties. To the extent our control counties benefit from pollution reductions in neighboring treated counties, our estimates understate the true effects.

Conclusion

Using a quasi-experimental technique that exploits change in ambient air quality standards for PM_{2.5}, this paper presents two main findings. First, air standards reduced ambient PM_{2.5}

levels in counties classified in nonattainment relative to attainment. Second, there was an accompanying reduction in mortality rates for those age 65 years and older. Several robustness checks support a causal interpretation of these results. Calculating an instrumental variables estimate, we find that a 1 $\mu\text{g}/\text{m}^3$ reduction in $\text{PM}_{2.5}$ reduces over 65 mortality rates by 0.6%. Our focus on policy variation, rather than per-unit pollution levels, provides more robust evidence on the causal link between pollution policy and human health. As many important reductions in pollution result from policy intervention, this analysis provides an important step forward in pollution-health science.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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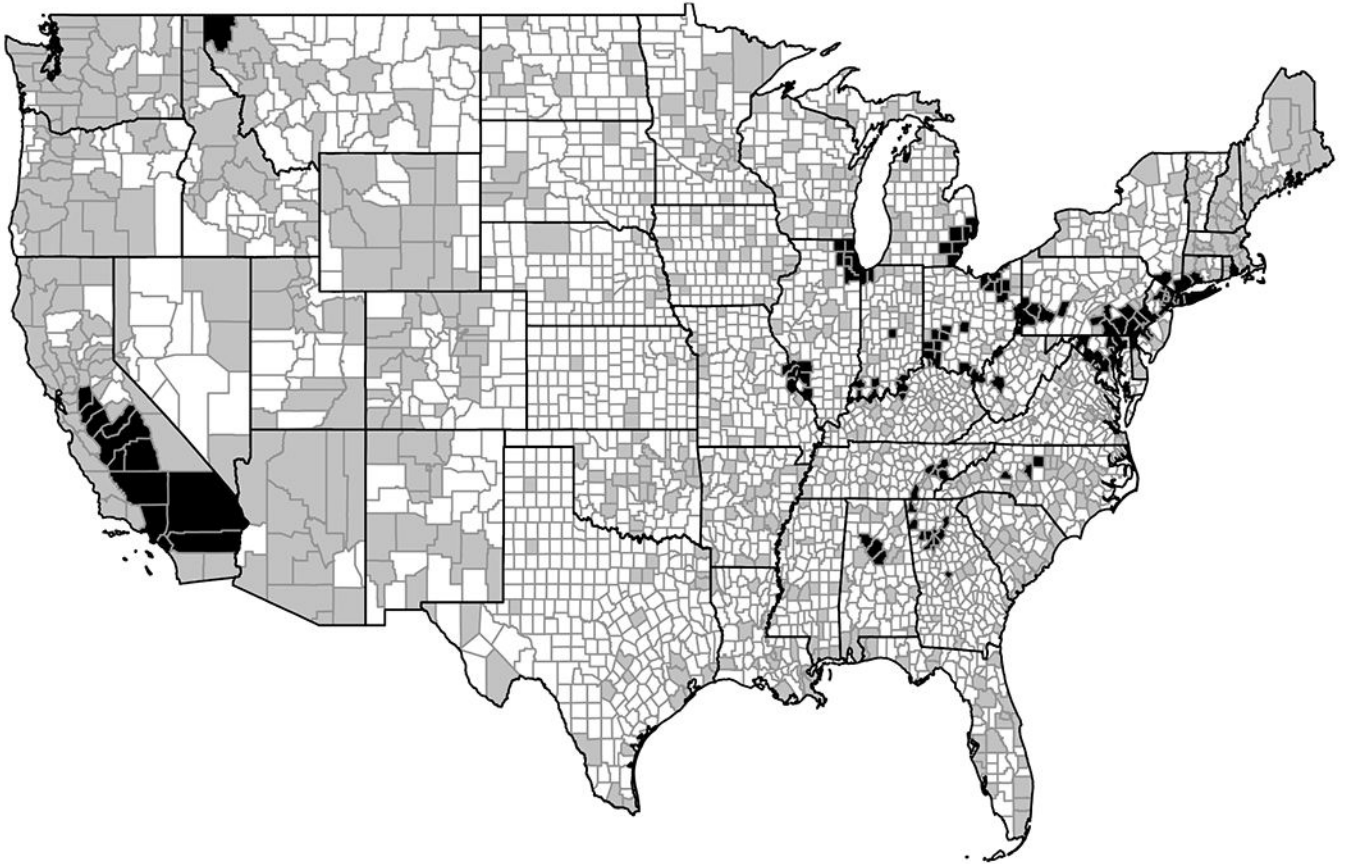


Figure 1.
Map of Nonattainment Counties

Notes: Black and gray shaded areas represent counties in our sample. The black shade are the nonattainment counties as of 2005, while the gray are the attainment counties.

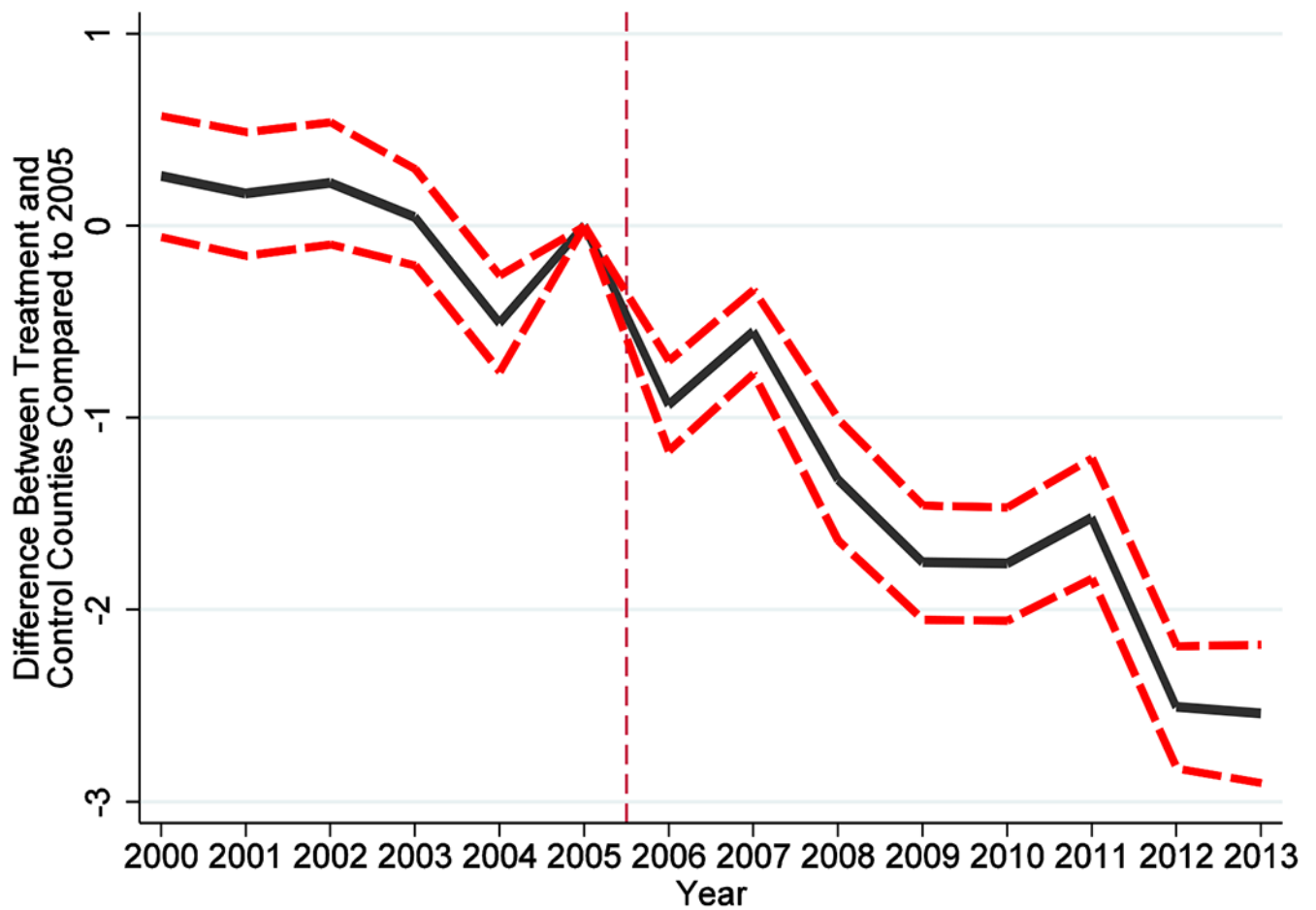


Figure 2.
 Event Study Estimates for PM_{2.5}
 Notes: Estimates represent the difference in PM_{2.5} concentrations for nonattainment counties vs. attainment counties. Dotted lines represent 95% confidence interval based on standard errors clustered on county. All regressions include year dummy variables, county fixed effects, income per capita, share employed, mean temperature, maximum temperature, and precipitation. The vertical line represents when the air quality standard for PM_{2.5} began. 2005 is the reference category.

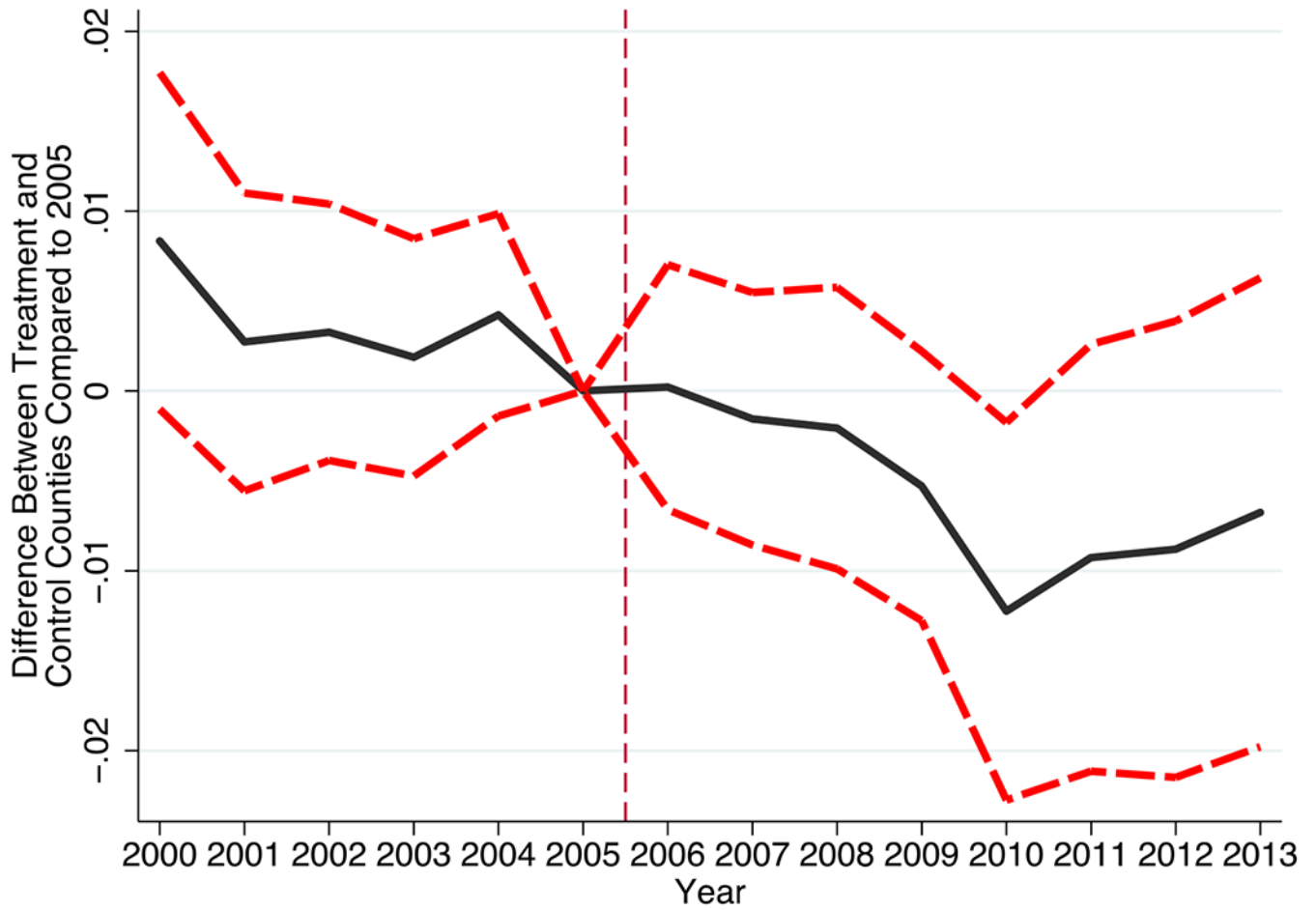


Figure 3.

Event Study Results for Mortality Rate

Notes: Estimates represent the difference in log mortality per 100,000 population for nonattainment counties vs. attainment counties. Dotted lines represent 95% confidence interval based on standard errors clustered on county. All regressions include year dummy variables, county fixed effects, log of population, income per capita, share employed, mean temperature, maximum temperature, and precipitation. We weight regressions by population. The vertical line represents when the air quality standard for PM_{2.5} began. 2005 is the reference category.

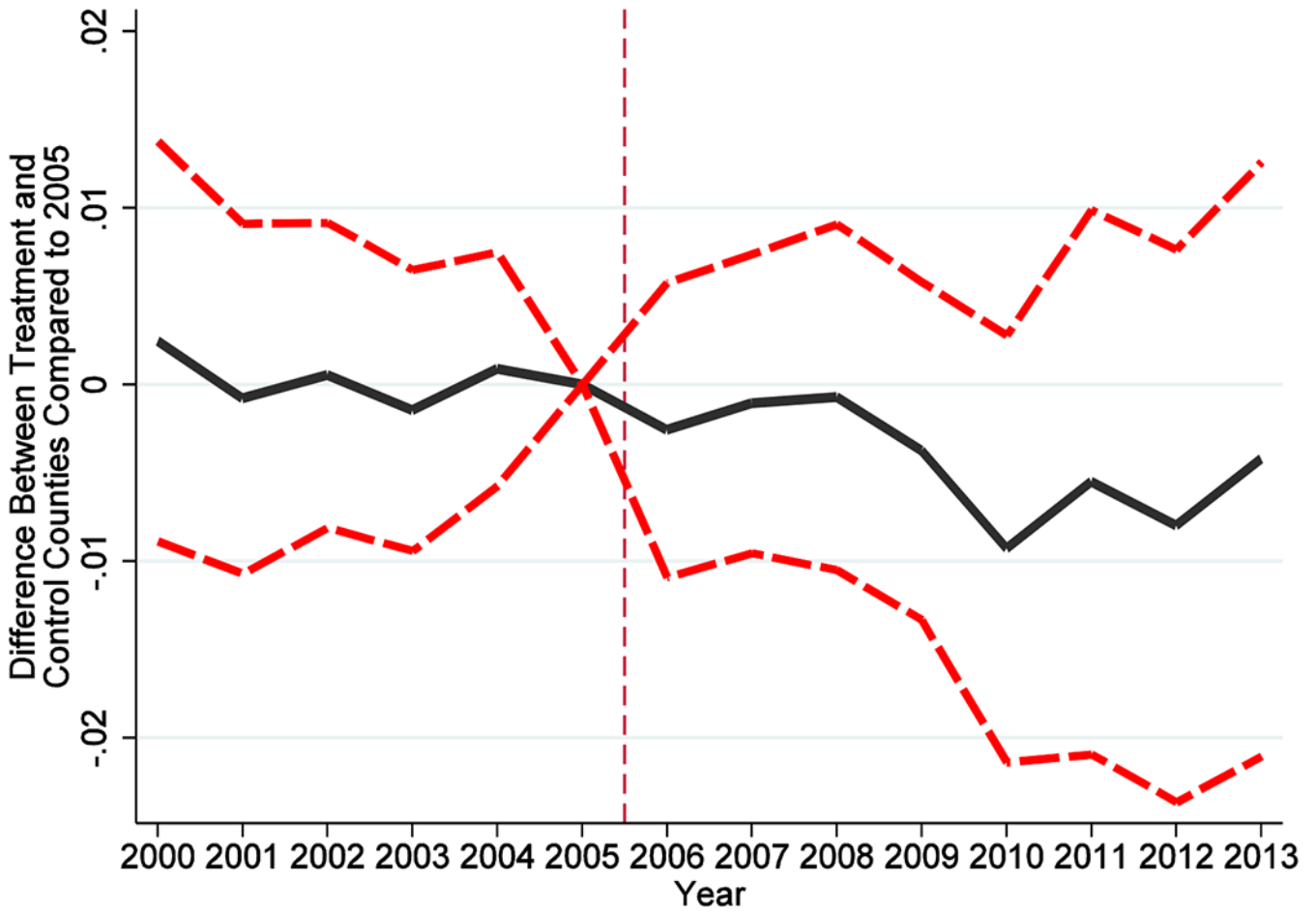


Figure 4. Event Study Results for Mortality Rate using Nearest Neighbor Matching
 Notes: Estimates represent the difference in log mortality per 100,000 population for nonattainment counties vs. attainment counties based on nearest neighbor matching using the estimated propensity score. The propensity score is a logit model where the dependent variable is attainment status and the independent variables are raw over 65 mortality and population over 65, separately for the years 2000-2005. Dotted lines represent 95% confidence interval based on standard errors clustered on county. All regressions include year dummy variables, county fixed effects, log of population, income per capita, share employed, mean temperature, maximum temperature, and precipitation. We weight regressions by population. The vertical line represents when the air quality standard for PM_{2.5} began. 2005 is the reference category.

Table 1.

Summary Statistics

Variable	Mean	Std. Dev.
mortality rate (per 1,000)	4.843	7.500
PM _{2.5} (µg/m ³)	10.84	3.060
income per capita (\$)	41,349	11,909.75
share employed	0.58	0.148
mean temperature	56.91	7.970
maximum temperature	67.84	8,123
Precipitation	11.15	4.308
net returns (per 100,000)	25.20	570.290
net exemptions (per 100,000)	91.85	1242.387
mortality rate (per 1,000)	non-attainment	attainment
year < 2006	5.131	5.084
year 2006	4.662	4.617
PM _{2.5} (µg/m ³)	non-attainment	attainment
year < 2006	15.29	10.99
year 2006	11.96	9.33

Notes: Number of observations = 8275 for all variables in Panel A except for net returns and exemptions, which = 8272.

Table 2.Difference-in-Differences (DiD) Estimates for PM_{2.5} and Mortality

	1	2
Dependent variable	PM_{2.5}	Log Mortality Rate
DiD estimate	-1.591 [-1.797, -1.386]	-0.009 [-0.018, -0.001]
Observations	8275	8275
R-squared	0.873	0.951

Notes: 95% confidence intervals based on standard errors clustered on county in brackets. All regressions include year dummy variables and county fixed effects. Regression in column 2 also control for log of population and is weighted by population.

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Table 3.Instrumental variable and within estimates of PM_{2.5} on Mortality

Regression model	Instrumental variables	Within county
PM _{2.5}	0.006 [0.001, 0.011]	0.002 [0.000, 0.003]
Observations	8275	8275
R-squared	0.951	0.951

Notes: 95% confidence intervals based on standard errors clustered on county in brackets. All regressions include year dummy variables, county fixed effects, log of population and are weighted by population. Instrumental variables regression uses attainment status*I(year = 2006) as an instrument for PM_{2.5}.

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Table 4.

Sensitivity Analysis for Difference-in-Differences (DiD) Estimates for Mortality

	1	2	3
Dependent variable	Additional Covariates	Nearest Neighbor Matching	Common Support
DiD estimate	-0.008 [-0.016, -0.000]	-0.004 [-0.014, 0.006]	-0.008 [-0.0169, 0.000]
Observations	7828	3372	6906
R-squared	0.953	0.96	0.953

Notes: 95% confidence intervals based on standard errors clustered on county in brackets. All regressions include year dummy variables, county fixed effects, log of population, income per capita, share employed, mean temperature, maximum temperature, and precipitation. The regressions in columns 2 and 3 are based on nearest neighbor matching and a common support, respectively, using the estimated propensity score. All regressions are weighted by population.

Table 5.

Heterogeneity of Difference-in-Differences (DiD) Estimates for Mortality

	1	2	3
Dependent variable	Log Male Mortality Rate	Log Female Mortality Rate	Log Mortality Rate Over 75
DiD estimate	-0.004 [-0.012, 0.005]	-0.012 [-0.021, -0.002]	-0.011 [-0.019, -0.002]
Observations	8272	8274	8275
R-squared	0.933	0.930	0.916

Notes: 95% confidence intervals based on standard errors clustered on county in brackets. All regressions include year dummy variables, county fixed effects, log of the relevant subgroup population, income per capita, share employed, mean temperature, maximum temperature, and precipitation. All regressions are weighted by relevant subgroup population.

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Table 6.

Difference-in-Differences (DiD) Estimates for Net Tax Returns and Exemptions

	1	2
Dependent variable	Net Returns	Net Exemptions
DiD estimate	9.394 [-85.975, 104.763]	15.058 [-210.922, 241.038]
Observations	8274	8274
R-squared	0.495	0.542

Notes: 95% confidence intervals based on standard errors clustered on county in brackets. All regressions include year dummy variables, county fixed effects, and economic and weather controls. Net returns and exemptions are defined as (inflows – outflows) / (population/100,000).

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