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ABSTRACT

We estimate the effect of acute air pollution exposure on mortality, life-years lost, and health care utilization among the US elderly. We address endogeneity and measurement error using a novel instrument for air pollution that strongly predicts changes in fine particulate matter (PM 2.5) concentrations: changes in the local wind direction. Using detailed administrative data on the universe of Medicare beneficiaries, we find that an increase in daily PM 2.5 concentrations increases three-day county-level mortality, hospitalizations, and inpatient spending, and that these effects are not explained by co-transported pollutants like ozone and carbon monoxide. We then develop a new methodology to estimate the number of life-years lost due to PM 2.5. Our estimate is much smaller than one calculated using traditional methods, which do not adequately account for the relatively low life expectancy of those killed by pollution. Heterogeneity analysis reveals that life-years lost due to PM 2.5 varies inversely with individual life expectancy, indicating that unhealthy individuals are disproportionately vulnerable to air pollution. However, the largest aggregate burden is borne by those with medium life expectancy, who are both vulnerable and comprise a large share of the elderly population.

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I. INTRODUCTION

It is widely accepted that air pollution negatively affects human health, leading many countries to regulate air pollution. Accurately quantifying the health effects of marginal pollution reductions matters greatly for determining optimal environmental policy, especially for countries like the United States where current pollution levels are relatively low and further reductions may be very costly. However, estimating the causal effect of pollution on health is complicated due to well-documented challenges, including separately identifying the effects of different pollutants, endogeneity, and measurement error. Quasi-experimental studies that use a plausibly exogenous source of pollution variation are typically confined to narrow geographic and temporal scales, raising questions of external validity. Such studies also lack power to detect changes in important but rare outcomes like adult mortality due to relatively small sample sizes and thus may overlook an important component of the social cost of pollution. Even if mortality effects are detected, estimating the life-years lost due to pollution is difficult because those who die prematurely from pollution may have shorter life expectancies than those who survive.

This paper presents the first large-scale, quasi-experimental investigation of the effects of acute fine particulate matter exposure on mortality and medical costs among the elderly. We overcome the identification and statistical power challenges described above by exploiting daily variation in fine particulate matter (PM 2.5) concentrations caused by changes in daily wind direction to estimate the causal effect of pollution on three-day county-level elderly mortality rates, life-years lost, hospitalizations, and medical spending.

A key innovation of our study relative to previous quasi-experimental designs is that our approach does not require identifying the source of pollution to estimate its effects. This allows us to harness variation in pollution across a broad geographic scale and over a long time period, enabling us to estimate effects on rare health outcomes such as mortality and to explore effect heterogeneity across subpopulations. We do this by combining data on the universe of elderly Medicare beneficiaries, comprising approximately 97 percent of the US population aged 65 and older, with pollution and weather data from 1999 through 2011. The many locations and time periods spanned by our dataset coupled with our novel methodology also allow us to separately identify the effects of different pollutants on mortality, which has proven to be extremely challenging in prior studies.

The identifying assumption of our instrumental variables (IV) approach is that, after flexibly controlling for a large number of fixed effects and climatic variables, changes in a county's average wind direction are unrelated to changes in the county's mortality or health care utilization except through air pollution. Wind may affect pollution measured by a particular monitor either by redistributing locally-produced pollution (e.g., from traffic or local power plants) or by transporting pollution that is produced elsewhere into the county. As we describe below, we construct our empirical specification to exploit only

the wind-induced variation in pollution exposure that affects the whole county in a similar manner. Using this variation, which is more likely to arise from long-range transport of pollutants, reduces the potential for measurement error in residents' pollution exposure due to within-county transport.

We find that a 1 microgram per cubic meter ($\mu g/m^3$) (about 10 percent of the mean) increase in PM 2.5 exposure for one day causes 0.61 additional deaths per million elderly individuals over the three-day window consisting of the day of the increase and the following two days. The effect is largest for the oldest beneficiaries in absolute terms. However, the relative mortality risk changes non-monotonically with age, suggesting that age alone is a noisy predictor of vulnerability to air pollution. The IV estimate is over six times larger than the corresponding ordinary least squares (OLS) estimate, demonstrating the potential for substantial bias in observational studies of pollution exposure. Moreover, the IV estimate is robust to simultaneously instrumenting for PM 2.5, carbon monoxide, and ozone, which is feasible because different wind directions transport varying amounts of each pollutant. We also find that increases in PM 2.5 lead to more emergency room (ER) visits, more hospitalizations, and higher inpatient spending, driven almost entirely by ER admissions. Each 1-$\mu g/m^3$ increase in PM 2.5 increases three-day ER visits by 2.3 per million beneficiaries and ER spending by over $15,000 per million. OLS estimates are much smaller and, in the cases of total inpatient spending and the total admission rate, significantly *negative*.

A central concern that arises when estimating mortality effects is whether those who die from pollution exposure would have died soon anyway, a phenomenon referred to as "mortality displacement" or "harvesting." If the mortality effect of pollution is concentrated among relatively old or sick individuals, then the mortality cost, as measured by the number of life-years lost, is likely to be much smaller than if the effect was concentrated among individuals randomly chosen from the population. Failing to account for the lower life expectancy of those who die can substantially bias estimates of the social cost of mortality, which depends closely on life-years lost. Some studies address this issue by using lags of the independent variables of interest to investigate whether mortality effects decrease as the length of time under consideration increases, as would be the case under harvesting (Schlenker and Walker 2016), or by averaging pollution fluctuations over longer time periods. However, these approaches cannot account for displacement that occurs outside the time window spanned by the independent variable.

We develop a novel, direct approach to estimate the number of life-years lost due to pollution exposure. Using the rich individual-level data on health and health utilization available in the Medicare data, we estimate several survival models and use the results to predict remaining life expectancy for each individual in our sample. We then aggregate the estimates of decedents' counterfactual life expectancies up to the county level to provide daily measures of life-years lost per capita and use them to directly estimate the life-years lost due to pollution exposure.

Our life-years lost analysis reveals that accounting for decedents' age and gender reduces estimates of life-years lost by 31 percent compared to a naïve estimate that controls for neither age nor gender. We then employ machine-learning techniques to incorporate information from over one thousand individual- and neighborhood-level variables derived from Medicare histories and the American Community Survey. Doing so reduces the life-years lost estimate by an additional 55 percent. Our preferred estimate is that a 1-$\mu g/m^3$ increase in PM 2.5 causes the loss of 2.7 life-years per million beneficiaries over three days. Due to their high mortality rates, those with a life expectancy of less than one year lose the largest number of life years per capita in both absolute (11.3 per million) and relative terms. Whereas the relative mortality effects of PM 2.5 are non-monotonic with respect to age, both our mortality and life-years lost estimates decrease steeply with counterfactual life expectancy. This suggests that an individual's life expectancy, as predicted by our model, identifies vulnerability to pollution shocks more effectively than age alone.

Although individuals with a life expectancy of less than one year bear the largest mortality and life-years lost burden in per capita terms, they are not the primary contributors to the aggregate social burden because they comprise less than one percent of all beneficiaries. The social cost of PM 2.5 is concentrated among the elderly with 5-10 years of remaining life expectancy, followed by those with 2-5 years remaining, because these groups represent a large fraction of the Medicare population. Using the conventional value of $100,000 per statistical life year, our estimates of life-years lost imply that the social mortality cost of a 1-$\mu g/m^3$ increase in PM 2.5 is $270,000 per million beneficiaries, which is an order of magnitude larger than our corresponding medical cost estimate of $15,000 per million beneficiaries.

To put these results into perspective, consider the national reduction in average PM 2.5 concentrations of 3.65 $\mu g/m^3$ that occurred during our study period, 1999-2011 (see Figure 1). Scaling our estimates linearly, we calculate that by 2011 this reduction reduced the number of elderly deaths nationwide by 55,000 per year and the number of life-years lost by 150,000 per year. Assuming a standard value of $100,000 per statistical life-year implies a corresponding benefit of $15 billion per year, which represents a large fraction of the estimated annual costs of complying with air pollution regulations (EPA 2011).[1] By comparison, estimating life-years lost using an average life expectancy for the population increases this estimate by 220 percent, to $47 billion. Accounting for gender and age of the decedents mitigates this upward bias, but still causes the benefits to be overestimated by 120 percent. This contrast demonstrates the importance of our technique for placing an accurate value on the social benefits of reducing mortality.

[1] The EPA's calculation of the annual costs of meeting the 1990 Clean Air Act Amendment air quality standards (which include standards for all criteria pollutants, not just PM 2.5) increased from $19.9 billion in 2000 to $43.9 billion in 2010 (EPA 2011). Standards for PM 2.5 were first implemented in 1997, and then tightened in 2006.

For the past several decades, the Environmental Protection Agency (EPA) has been tightening its regulation of particulates, focusing increasingly on fine particulates. It has regulated particulate matter smaller than 100 micrometers in diameter (total suspended particulates or TSP) since 1971. Concerned with growing epidemiological evidence that smaller particulate matter was especially harmful, in 1987 the EPA set a standard for particulate matter less than 10 micrometers in diameter (PM 10). Similar concerns prompted the EPA in 1997 to set a daily and an annual limit for fine particulate matter (PM 2.5), defined as particles less than 2.5 micrometers in diameter. The daily limit for PM 2.5 was tightened in 2006, and the annual limit was tightened in 2012.[2] The evidence supporting these regulations has come primarily from associational studies that have consistently demonstrated a relationship between PM 2.5 and increased morbidity and mortality, even after controlling for various confounding factors (e.g., Dockery et al. 1993, Pope et al. 1995, Laden et al. 2000, Samet et al. 2000, Pope and Dockery 2006, EPA 2009). However, concerns about bias in these and other associational estimates have caused both the scientific community and regulators to question how many deaths are avoided from reductions in particulate matter (OMB 2012; Dominici et al. 2014). While randomized controlled laboratory trials have shown that healthy volunteers exposed to ambient pollution concentrations for as little as one or two hours have worse cardiovascular performance than those exposed to very clean air, these studies face issues of external validity and are too small to draw conclusions about mortality effects (Brook et al. 2009, Langrish et al. 2013). Thus, significant uncertainty remains about the causal effects of PM 2.5 on human health.

Our study addresses this uncertainty by providing the first quasi-experimental estimates of the causal effect of acute PM 2.5 exposure on adult mortality, hospitalizations, and medical costs. Our work contributes to the recent literature in economics that uses quasi-experimental approaches to estimate the effects of pollution on health. Much of this work has focused on the effect of pollutants other than fine particulate matter, such as TSP, ozone, sulfur dioxide, or nitrous oxides (Chay et al. 2003; Chay and Greenstone 2003; Currie and Neidell 2005; Currie et al. 2009; Moretti and Neidell 2011; Chen et al. 2013; Schlenker and Walker 2016; Deryugina et al. 2016; Deschenes et al. 2016). Of these studies, only four consider non-infant mortality (Chay et al. 2003; Chen et al. 2013; Deryugina et al. 2016; Deschenes et al. 2016). In one of the few studies to focus on fine particulate matter, Knittel et al. (2016) only consider infant mortality rates. Anderson (2015) uses variation in wind direction across a highway in Los Angeles to proxy for changes in air pollution, but does not directly measure which pollutants are changing and focuses on chronic (long-run) rather than acute pollution exposure.

[2] See https://www3.epa.gov/ttn/naaqs/standards/pm/s_pm_history.html.

Our study moves beyond these papers in three important ways. First, our approach allows us not only to estimate the causal impact of PM 2.5 on mortality, but also to separately identify the causal impact of other pollutants on mortality. We find that the PM 2.5-mortality relationship is more robust than that of other pollutants. Second, our study includes elderly mortality as an outcome, which few previous studies have considered. We find that the mortality costs of PM 2.5 among the elderly are an order of magnitude larger than the health care costs, demonstrating that ignoring mortality can cause researchers to overlook a primary social cost of pollution.

Third, we estimate mortality costs more precisely than previous studies by developing and applying a novel method to estimate the life-years lost associated with pollution exposure. Our estimates suggest that traditional methods for estimating life-years lost are prone to significant upward bias. Moreover, our approach allows for an unprecedented investigation of the distributional effects of the mortality costs of air pollution by health status. For instance, we show that unhealthy individuals (as measured by life expectancy) bear a disproportionate share of the life-years lost burden of PM 2.5 despite their low life expectancies. Our methodology is general and can be applied to investigate mortality costs across a wide variety of contexts. For example, whether health insurance reduces mortality is an important question in health economics (Finkelstein and McKnight 2008; Card et al. 2009; Huh and Reif 2016). As in our study, estimating the social value of that mortality reduction depends on the number of life-years saved. Using our approach, this quantity can be estimated with administrative datasets such as Medicare or other, more easily accessible, surveys that contain information on demographics, health status, and mortality, such as the Health and Retirement Study or the Panel Study of Income Dynamics.

The rest of the paper is organized as follows. Section II summarizes how air pollution is transported by the wind and gives a preview of our estimation strategy. Section III describes our data. Section IV describes our econometric strategy in detail, including how we estimate the life-years lost. Section V presents results, and Section VI concludes.

II: WIND TRANSPORT OF POLLUTION

Fine particulate matter, PM 2.5, refers to particles with diameters of 2.5 microns or less. Rather than having a single chemical composition, PM 2.5 is a mixture of various compounds including nitrates, sulfates, ammonium, and carbon (Kundu and Stone 2014). In addition to natural sources, PM 2.5 is created from atmospheric conversion of power plant and auto emissions. While not themselves particulates, sulfur dioxide and nitrogen dioxide, two “criteria” pollutants regulated by the EPA under the Clean Air Acts, are precursors to sulfates and nitrates, which are components of PM 2.5. The PM 2.5 present in a given location will consist of both locally-produced pollution and pollution produced elsewhere that is transported into the region by the wind. The amount of transported pollution is significant. For example, the EPA estimates that

most of the PM 2.5 in the Eastern United States was not produced locally, but instead was transported from hundreds of miles away (EPA 2004).

PM 2.5 is not unique in its ability to traverse considerable distances; other pollutants, including carbon monoxide, sulfur dioxide, nitrous oxide, and ozone precursors, can also be carried by the wind. Pollution transport patterns depend on a host of factors, including the pollutant, the location of the pollution source, wind direction and speed, precipitation, the height of the planetary boundary layer, and the presence of other airborne molecules, which can react with the windborne pollutant. One way to exploit variation in pollution transport is to employ a sophisticated atmospheric science model (e.g., Muller and Mendelsohn 2007) to simulate daily pollution transport across the United States and use the resulting estimates as instruments. However, this is computationally infeasible at the daily level.

An instrumental variables approach, by contrast, makes use of only some of the factors involved in pollution transport and thus is much simpler to implement. Such an approach only requires that the instrument (a) be sufficiently correlated with the endogenous variable of interest and (b) not be correlated with any unobserved determinants of the outcome of interest. We instrument for changes in a county's daily average PM 2.5 concentrations using changes in the county's daily average wind direction, which we shall show is by itself an important determinant of pollution levels. Our approach does not use variation due to prevailing wind directions because the predictability of prevailing winds may cause individuals to sort into locations that are upwind or downwind of the pollution, thereby biasing the estimates. Employing variation attributable to changes in daily wind direction eliminates this concern, but also means that our method is most useful for examining acute, rather than chronic, exposure.

We now illustrate the type of variation used to estimate the causal effects of PM 2.5, relegating the details to Section IV. Figure 2 shows the relationship between the estimated daily wind direction at pollution monitors, in 10-degree bins, and PM 2.5 concentrations measured by these monitors in and around the Bay Area, CA. Figure 3 shows the same relationship for pollution monitors in and around Greater Boston, MA. All estimates are relative to 260-270 degrees, where 270 degrees corresponds to a "Westerly" (blowing *from* the West) wind direction. The figures display results from a regression that controls for county, month-by-year, and state-by-month fixed effects, as well as a flexible set of controls for maximum and minimum temperatures, precipitation, wind speed, and the interactions between them.

In both figures, the change in local wind direction is a very strong predictor of changes in local pollution levels. Moreover, the patterns are consistent with what we would expect given the geographic placement of the monitors. In and around the Bay Area, PM 2.5 levels are highest when the wind is blowing from the Southeast and lowest when the wind is blowing from the West and the North. In other words, more pollution is blown in from Southeast California than from the ocean and the Northern states like Oregon and Washington. In and around Boston, MA, pollution is highest when the wind is blowing from the

Southwest, where New York City is located, and lowest when it is blowing from the East, North, Northeast and Northwest, where the ocean and sparsely populated areas are dominant.

III: DATA

III.A. Air pollution

We obtain air pollution data from the EPA's Air Quality System database, which provides hourly data at the pollution-monitor level for pollutants that are regulated by the Clean Air Act. Comprehensive data for PM 2.5 are available beginning in 1999. We focus on PM 2.5, but we also obtain data on two other criteria pollutants: ozone (O_3) and carbon monoxide (CO).[3] As with PM 2.5, past literature has linked these air pollutants to infant mortality and other adverse health outcomes (Currie and Neidell 2005; Moretti and Neidell 2011; Schlenker and Walker 2016). We aggregate monitor readings to the daily level by averaging across hourly observations and then construct county-level pollution measures by averaging all available pollution readings on a given day across all monitors located within the county.

Figure 1 displays aggregate trends in PM 2.5 over time. Average concentrations of PM 2.5 have been steadily falling from 13.0 micrograms per cubic meter ($\mu g/m^3$) in 1999 to 9.37 $\mu g/m^3$ in 2011.[4] One unit of PM 2.5 thus represents about 10 percent of the average concentration during our time period. Figure 1 also shows that the number of PM 2.5 monitors has remained fairly constant since 2001. However, the set of monitored counties does change over time, and Grainger et al. (2016) find evidence that counties strategically place their pollution monitors in relatively clean areas. To avoid this compositional bias problem, we perform all analyses at the county level with county fixed effects. Moreover, our instrumental variables approach exploits variation in pollution that is independent of monitor placement, resulting in unbiased estimates.

III.B. Atmospheric conditions

Wind speed and wind direction data for the years 1999-2011 are obtained from the North American Regional Reanalysis (NARR) daily reanalysis data published by the National Centers for Environmental Information (NCEI).[5] Wind conditions are reported on a 32 by 32 kilometer grid, and consist of vector

[3] We ignore sulfur dioxide and nitrogen dioxide because they are precursors to PM 2.5. Lead is also a criteria pollutant and can in principle be transported by the wind. However, there are only about 64,000 county-day level observations for lead between 1999 and 2011, and only 52,000 of these also contain observations of PM 2.5.

[4] By comparison, in China the population-weighted average of PM 2.5 concentrations across 190 cities in 2014 was 61 $\mu g/m^3$ (Zhang and Cao 2015).

[5] Available from https://www.ncdc.noaa.gov/data-access/model-data/model-datasets/north-american-regional-reanalysis-narr. The NCEI was formerly the National Climatic Data Center (NCDC).

pairs, one for the East-West wind direction (u-component) and one for the North-South wind direction (v-component). We first interpolate between grid points in the original dataset to estimate the daily u- and v-components at the location of each pollution monitor, using simple linear interpolation. We then use trigonometry to convert the average u- and v-components into wind direction and wind speed. Specifically, the wind speed is calculated as $ws = \sqrt{u^2 + v^2}$, where u and v are the county-day-level vectors. To calculate the wind angle, we first calculate $\theta = \frac{180}{\pi} Arctan\left(\frac{|v|}{|u|}\right)$ and then translate θ into a 0-360 scale depending on the signs of u and v. Specifically, given θ, the wind angle, wa, is calculated as follows:

$$wa = \begin{cases} 180 - \theta & \text{if u} < 0 \text{ and v} > 0 \\ \theta + 180 & \text{if u} < 0 \text{ and v} < 0 \\ 360 - \theta & \text{if u} > 0 \text{ and v} < 0 \\ \theta & \text{if u} > 0 \text{ and v} > 0 \end{cases}$$

We average the estimated monitor-day-level wind direction and speed to the county-day level.

Finally, we obtain daily temperature and precipitation data from Schlenker and Roberts (2009), who produce a detailed weather grid at the daily level using data from PRISM and weather stations.[6] These data include total daily precipitation, and daily maximum and minimum temperatures for each point on a 2.5 by 2.5 mile grid covering the contiguous United States for the years 1999-2011. To aggregate the gridded data to the county level, we average the daily measures across all grid points in a particular county.

III.D. Mortality, morbidity, and health care costs

Our data on mortality, morbidity, and health care costs come from Medicare administrative data. We focus on elderly beneficiaries aged 65-100, a sample that includes over 97 percent of elderly living in the U.S. Dates of death, age, sex, and county of residence are obtained for all beneficiaries from the 1999-2011 Medicare enrollment files. Health care utilization and costs are derived from the Medicare Provider Analysis and Review (MEDPAR) File, which includes an observation for each inpatient stay in a hospital or skilled nursing facility for any beneficiary enrolled in Original (fee-for-service or FFS) Medicare. MEDPAR observations are derived from the accumulation of service claims corresponding to that stay, and include the date of admission, length of stay, and total cost of the stay.[7] All county-level daily measures of hospital utilization and costs are aggregates of the MEDPAR records based on the patient's county of residence and the admission date.

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[6] See http://www.prism.oregonstate.edu/ for the original PRISM dataset and http://www.wolfram-schlenker.com/dailyData/dataDescription.pdf for a more detailed description of the daily data.

[7] Specifically, our measure of cost is the total allowed charges due to the provider. This amount includes payments made by Medicare, the beneficiary, or another payer.

Individual-level indicators for the presence of 27 chronic conditions, which we use when estimating life-years lost, are obtained from the Chronic Conditions segment of the Master Beneficiary Summary File. Chronic conditions include heart disease, COPD, diabetes, and depression, among others. Professional medical coders infer these conditions from detailed claims data, which are only available for beneficiaries enrolled in FFS Medicare. Because it may take some time for a relevant claim to appear in the data, information about chronic conditions will be most reliable for those who have been enrolled in FFS Medicare for multiple years.

Table 1 presents summary statistics for our main estimation sample, which consists of 1,600,846 observations at the county-day level. Our sample does not encompass the entire U.S. due to limitations in the EPA's pollution monitor coverage. In particular, PM 2.5 pollution measures are available for only 902 counties during our sample period (see Figure 4). However, because pollution monitors tend to be placed in more populated counties, our main regression estimates still capture about 70 percent of the elderly Medicare population.

Table 1 reports that the mean daily concentration of PM 2.5 in our estimation sample is 10.86 micrograms per cubic meter, with a standard deviation of 7.34. There are on average 49,486 Medicare beneficiaries in each county, with close to half of these aged between 65 and 74. Because we focus on the elderly, the 3-day death rate in our sample is fairly high, ranging from 138 per million for those aged 65-69 to nearly 1,200 per million for those aged 85 and over.

We observe hospital spending only for beneficiaries who are enrolled in fee-for-service Medicare (FFS); these make up about 80 percent of the population in our sample. For the life-years lost analysis, we focus on the subset of beneficiaries who have been continuously enrolled in FFS for at least two years (67 percent of the people in our sample) to ensure well-measured chronic conditions, as we described earlier. On average, there are 27,716 such individuals in each county, and their 3-day mortality rate is higher than the overall mortality rate in the Medicare population. There are at least two reasons for this. First, because of the continuous enrollment restriction, individuals in this population are at least 67 years old and thus older than average. Second, conventional wisdom and empirical evidence suggest that the fee-for-service population is generally sicker than the average Medicare beneficiary (McGuire et al. 2011).

Finally, the average 3-day hospital spending for the entire FFS population is about $34 per beneficiary, in nominal terms. About 40 percent of this inpatient spending originates from emergency room (ER) admissions. On average, there are 3,370 hospital admissions per million FFS beneficiaries over any given 3-day period, and 47 percent (1,579) of these admissions happen through the emergency room. There are also many ER visits that do not result in hospital admissions: the overall ER visit rate is 4,159 per million FFS beneficiaries.

IV: EMPIRICAL STRATEGY

IV.A. Effects of PM 2.5 on mortality and health care utilization

The key causal relationship we would like to estimate is the effect of short-run fluctuations in particulate matter on mortality, health, and health care spending, net of any potentially confounding factors. This relationship can be represented by the following regression equation:

$$Y_{cdmy} = \beta \text{PM2.5}_{cdmy} + f\left(Temp_{cdmy}, Prcp_{cdmy}, WS_{cdmy}\right) \quad (1)$$
$$+ \sum_{t=d+1}^{d+2} \left[\gamma_t \text{PM2.5}_{ctmy} + f_t\left(Temp_{ctmy}, Prcp_{ctmy}, WS_{ctmy}\right)\right]$$
$$+ \sum_{t=d-1}^{d-2} \gamma_t \text{PM2.5}_{ctmy} + \alpha_c + \alpha_{sm} + \alpha_{my} + \epsilon_{cdmy},$$

where the dependent variable is one of several possible outcomes in county c on day d in month m and year y. The parameter of interest is β, the coefficient on daily PM 2.5 levels. We first examine the effect of PM 2.5 on the death rate, measured in deaths per million Medicare beneficiaries. The other outcome variables measure health care utilization. We observe all hospitalizations and the inpatient spending associated with them; we also observe all ER visits, but only see spending for ER visits that resulted in hospitalizations. Our main outcomes of interest are total hospital spending and admissions per million beneficiaries. We calculate these measures for all hospital admissions and also for the subset of hospital admissions that originate through the emergency room (ER) admissions. Finally, we estimate the effect of PM 2.5 on the total ER visit rate per million beneficiaries, which also includes visits that did not result in a hospital admission.

The dependent variable Y_{cdmy} is calculated using a 3-day total, based on the day *d* and the following two days. For example, we estimate the effect of pollution on January 1st on the death rate calculated across January 1-3. This avoids including short-run mortality displacement in our estimates (because dying on January 1 instead of January 2 does not count as a death in our measure) and also allows for delayed effects (e.g., being exposed to pollution on January 1 might cause death on January 2). To ensure that β is not capturing the effects of pollution and weather fluctuations over the following two days, which may be correlated with contemporaneous variation, we include two leads of PM 2.5 concentrations and weather conditions. To ensure that β is not capturing any effects from *past* pollution variation, we also include two lags of PM 2.5 concentrations.[8]

[8] We do not have enough power to also include lags of the weather variables, which add about 9,300 regressors per lagged day, as discussed below.

The high granularity and comprehensive scope of our data allow us to estimate this regression with multiple sets of high-dimensional fixed effects. We control for weather, geography, time, and seasonality far more flexibly than previous studies have done. Specifically, we generate indicators for daily maximum temperatures falling into one of 17 bins, ranging from -15 degrees Celsius (5°F) or less to 30 degrees Celsius (86°F) or more, with 15 intermediate bins each spanning 3 degrees Celsius (5.4°F). We do the same for minimum temperatures. These variables are represented by $Temp_{cdmy}$ in the equation above. For daily precipitation and wind speed ($Prcp_{cdmy}$ and WS_{cdmy}), we generate indicators for deciles of these variables. We then generate a set of indicators for all possible interactions of these temperature, precipitation, and wind speed variables and include it in all our regressions.[9] We also estimate a series of alternative specifications to demonstrate that our estimates are robust to less flexible weather controls or omitting weather controls entirely. Those results reinforce the assumption that our identifying variation is exogenous.

The regressions also include county (α_c), state-by-month (α_{sm}), and month-by-year (α_{my}) fixed effects. The county fixed effects control for underlying differences in health and pollution that vary by geography. State-by-month fixed effects control for potential seasonal correlation between pollution, wind direction, and population health, allowing this correlation to vary by state. Finally, month-by-year fixed effects control flexibly for common time-varying shocks, such as those induced by any Medicare or environmental policy changes during our sample period. As with weather controls, we estimate alternative specifications with varying fixed effects to demonstrate the robustness of our results.

OLS estimates of equation (1) are prone to bias because exposure to PM 2.5 is not randomly assigned and is likely to be measured with error. We address this by employing an instrumental variables (IV) strategy, using daily wind direction in the county as an instrument for pollution. Because the effect of wind direction on PM 2.5 levels varies by geography, as illustrated by Figures 1 and 2, we allow the effect of the wind instruments in our first stage to also vary according to geography. The specification for our first stage is:

[9] Thus, we have up to 28,899 ($=17 \times 17 \times 10 \times 10 - 1$) weather indicators included in our regression for each of the three days we control for. In practice, not all possible combinations are realized in the data, so the actual number of included weather controls is about 9,300 per day (i.e., about 27,900 weather indicators per regression).

$$\begin{aligned}\text{PM2.5}_{cdmy} = &\sum_{g=1}^{100}\sum_{b=0}^{2}\beta_b^g 1[G_c = g] \times WINDDIR_{cdmy}^{90b} + f\left(Temp_{cdmy}, Prcp_{cdmy}, WS_{cdmy}\right) \\ &+ \sum_{t=d+1}^{d+2}\left[g_t\left(1[G_c = g] \times WINDDIR_{ctmy}\right) + f_t\left(Temp_{ctmy}, Prcp_{ctmy}, WS_{ctmy}\right)\right] \\ &+ \sum_{t=d-1}^{d-2} g_t(1[G_c = g] \times WINDDIR_{ctmy}) + \alpha_c + \alpha_{sm} + \alpha_{my} + \epsilon_{cdmy}.\end{aligned} \tag{2}$$

The excluded instruments are the variables $1[G_c = g] \times WINDDIR_{cdmy}^{90b}$. Each variable in the set $WINDDIR_{cdmy}^{90b}$ is equal to 1 if the daily average wind direction in county c falls in the 90-degree interval $[90b, 90b + 90)$ and 0 otherwise. The omitted category corresponds to the interval [270,360). The variable $1[G_c = g]$ is an indicator for county c being classified into monitor group g. The coefficient on the interaction between these two variables, β_b^g, is thus allowed to vary across 100 different geographic regions, as explained below. Because our outcomes of interest are measured over three days, we include two leads of the instrumental variables as controls (represented by $g_t\left(WINDDIR_{ctmy}\right) = \sum_{g=1}^{100}\sum_{b=0}^{2}\gamma_{b,t}^g 1[G_c = g] \times WINDDIR_{ctmy}^{90b}$).[10] To capture any autocorrelation in wind direction, we also control for two lags of the instruments. Our results are robust to the inclusion of more (or fewer) lags, as shown in Table 9. The other control variables are defined as in equation (1). We cluster all standard errors at the county level and weight all estimates by the relevant population in cases where the dependent variable is in per capita terms.[11]

Equation (2) estimates a common effect of county wind direction on pollution for all monitors within each of the 100 geographic areas, all of which span multiple counties. We use the *k*-means cluster algorithm to classify all the pollution monitors in our dataset into 100 spatial groups based on their location.[12] Cluster analysis is a standard tool used to assign observations (in our case, pollution monitors) into a pre-specified number of groups based on their characteristics (in our case, longitude and latitude). The resulting groups are displayed in Figure 4. Intuitively, monitors that are close to each other are more likely to be assigned to the same group than monitors that are far apart. On average, each geographic area

[10] In principle, we could follow the specification of equation (1) and instrument for leads of PM 2.5 instead. However, instrumenting for PM 2.5 leads significantly raises the computational burden of the estimation, and those coefficients are not of primary interest.

[11] For example, if the dependent variable is the elderly mortality rate, then we weight by the number of Medicare beneficiaries in the county at the beginning of the reference day; if the dependent variable is the mortality rate for those 85 and older, then we weight by the number of beneficiaries who are 85 and older.

[12] If a county has monitors belonging to more than one group, we assign the larger integer group number to the county, which is effectively random assignment.

(group) contains 21 monitors with PM 2.5 readings and 9 counties. As we discuss later, our results are robust to using more or fewer monitor groups.

Grouping monitors together in this way is appealing because it removes pollution variation emitted by local sources from our estimates. Instead, the pollution variation captured by equation (2) consists primarily of pollution emitted by distant sources that are systematically located to one side or another of the entire monitor group.[13] This is beneficial for two reasons. First, pollution emitted by local sources within the group (e.g., daily traffic patterns) might be correlated with other factors that affect health (e.g., traffic-related stress). Second, pollution from local sources is unlikely to reach everybody within the group, thereby generating measurement error.

For example, consider a power plant with a short smokestack located in the center of a county. Suppose an air pollution monitor is located just to the east of the plant. When the wind blows from the west, the monitor will record high levels of pollution, and when it blows from the east, it will record low levels of pollution. Yet, in either case, pollution exposure increases for only one half of the county; in the other half of the county, pollution exposure actually decreases. On net, there might not be any observable health effects at the county level. Thus, this measurement error can cause the researcher to underestimate the effect of air pollution on health. By contrast, the pollution variation we employ comes primarily from long-range transport, so exposure will be more constant across a county.

Equation (2) restricts the effect of wind direction on pollution levels to be constant within each of the four $WINDDIR$ bins because it is computationally very burdensome to increase the number of instruments. The specification presented in (2) includes hundreds of instruments and tens of thousands of control variables and fixed effects, and is estimated using over one million observations. While the fixed effects and controls are partialled out prior to estimation, the instruments are not, making it more difficult to increase the number of instruments than the number of control variables. The main cost of restricting the number of bins is the loss of potentially useful variation in wind direction. We have investigated the effect of increasing the number of $WINDIR$ bins on our estimates; those results, shown in the robustness section, are very similar to our preferred specification.

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[13] We cannot test for this directly. However, in order for our approach to pick up emissions from local sources within a county, those sources would have to be located consistently in the same direction away from all the monitors in their cluster group, which on average spans 9 counties. Because the monitors are fairly dispersed geographically (see Figures 1, 2, and 4), we think this is unlikely to be the case. Furthermore, we have also estimated alternative specifications that include 50 cluster groups instead of 100, so that each group spans an even larger portion of the country. Those estimates are reported in our robustness section and are similar to estimates from our primary specification.

The large number of instruments employed in our analysis raises the concern that our two-stage least squares (2SLS) IV estimates may suffer from weak instrument bias. However, as illustrated by Figures 1 and 2, wind direction is a strong predictor of air pollution levels, and this is confirmed by the large first-stage F statistics presented in our tables.[14] Moreover, estimating our IV model using the limited information maximum likelihood (LIML) estimator, which is approximately median-unbiased yields results similar to 2SLS. As a robustness check, we also estimate our model using placebo instruments and obtain very small F-statistics.

IV.B. Effect of PM 2.5 on life-years lost

The previous section detailed how we estimate the effect of PM 2.5 on the number of lives lost, as measured by the mortality rate in deaths per million Medicare beneficiaries. To monetize the social cost of this mortality, we can simply multiply the estimated number of lost lives by some value of a statistical life (VSL). However, such an exercise may overstate the cost if the individuals who die as a result of pollution exposure are sicker than the general population and would not have lived for much longer anyway. While this is a concern with any population, including infants, it may be particularly relevant for the elderly.

Thus, in cases where the decedents may not be representative of the population they come from, it is more appropriate to calculate the social cost of mortality based on the number of statistical *life-years* lost rather than *lives* lost. According to this metric, extending an individual's life by four years is twice as valuable as extending her life by two years. A common estimate of the value of a statistical life-year is $100,000 (Cutler 2004). However, because the mortality cost scales linearly in the value of a statistical life-year, the key conceptual challenge is accurately estimating the number of life-years lost.

In practice, estimating life-years lost is challenging because counterfactual life expectancy is unobserved. The standard in the health and environmental literatures is to multiply the estimated number of lives lost by an assumed value of counterfactual life expectancy per life lost. This counterfactual life expectancy is typically derived from population life tables (Deschenes and Greenstone 2011) or is set equal to a value estimated from changes in cause- and age-specific mortality over time (Finkelstein and McKnight 2008; Huh and Reif 2016). A general concern with this approach is that it overstates life-years lost if individuals affected by pollution have shorter life expectancies than average (Deschenes and Greenstone 2011). For example, frail individuals with advanced heart or lung disease may be more susceptible to the

[14] Our tables present first-stage F statistics that are computed assuming errors are homoskedastic. This means they can be compared to the well-known Stock and Yogo (2005) critical values, which are valid only under homoskedasticity. We have also computed first-stage F statistics assuming serially correlated errors. In every specification we have run, those statistics are significantly larger than the first-stage F statistics computed assuming homoskedastic errors.

adverse effects of air pollution, but have lower life expectancies than observationally similar individuals who do not have such conditions, even absent a pollution event.

We propose a new methodology that exploits the detailed data derived from Medicare claims to generate an estimate of life-years lost that is less prone to bias than previous methods.[15] We first present a framework that illustrates why the traditional method of estimating life-years lost is likely to produce upwardly biased estimates. We outline the specific assumptions required to eliminate this bias and explain how our approach is more likely to meet those assumptions than prior approaches. We then use our new methodology to produce estimates of the number of life-years saved due to a reduction in PM 2.5 pollution.

Let L_{it} be the number of statistical life-years lost due to death by individual *i* in period *t*. For individuals who do not die, $L_{it} = 0$, while for individuals who do die at time *t*, L_{it} is equal to the number of years that individual *i* would have lived conditional on being alive at the start of period *t*. For simplicity, we first assume that exposure to PM 2.5 is assigned randomly and affects all individuals equally. If L_{it} was observable, then the researcher could estimate the effect of PM 2.5 on the number of life-years lost in period t by estimating the following regression equation:

$$L_{it} = \alpha + \gamma \text{PM2.5}_{it} + e_{it} \tag{3}$$

The error term e_{it} represents factors other than pollution that affect life-years lost and, by assumption, is uncorrelated with PM2.5_{it}. Under these conditions, equation (3) consistently estimates γ, the causal effect of PM2.5_{it} on life-years lost.

In practice a researcher does not observe L_{it}, but observes only whether an individual dies. Counterfactual life expectancy must therefore be estimated. For example, one could model it as a function of age, which is a strong predictor of remaining life expectancy. Let $\hat{L}_{it}$ be the estimate of life expectancy generated by some model, and let $u_{it} \equiv L_{it} - \hat{L}_{it}$ describe the measurement error in this estimate. Then the analog of equation (3), which the researcher can estimate with observable data, is

$$\hat{L}_{it} = \alpha + \gamma \text{PM2.5}_{it} - u_{it} + e_{it} \tag{4}$$

Bias arises when estimating equation (4) in the presence of heterogeneous treatment effects, such as if pollution exposure is deadlier for those who are sicker. To see this, let the effect of air pollution on individual mortality be equal to γ_i, and decompose this effect into $\gamma_i = \gamma + v_i$. The estimating equation can then be written as

$$\hat{L}_{it} = \alpha + \gamma \text{PM2.5}_{it} + (v_i \text{PM2.5}_{it} - u_{it} + e_{it}) \tag{5}$$

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[15] As in other studies, we focus on estimating the *immediate* effects of pollution exposure on life-years lost. It is also possible that exposure reduces an individual's remaining life expectancy without immediately killing her. In that case, our estimates are lower bounds.

The error term in (5) contains a third component, $v_i \text{PM2.5}_{it}$, which represents the portion of the individual's treatment effect not accounted for by the average treatment effect, γ. Bias arises if the heterogeneous treatment effect, v_i, is correlated with the measurement error in counterfactual life expectancy, so that $Cov(v_i \text{PM2.5}_{it}, u_{it}) \neq 0$. For example, suppose that the researcher does not account for pre-existing heart conditions when estimating $\hat{L}_{it}$ and does not include it as a control variable in (5). Then the estimation of γ will be biased if people with and without heart conditions have both different life expectancies and different probabilities of dying following exposure to PM 2.5.

Equation (5) summarizes the key challenge researchers face when estimating the effect of pollution exposure on life-years lost. Even if pollution is as good as randomly assigned, estimation of γ is upwardly biased in the presence of an unobserved factor that is positively correlated with both remaining life expectancy and the probability of dying following exposure to pollution. This is problematic because populations with low levels of remaining life expectancy, such as the elderly, are often more vulnerable in general and may be more susceptible to dying from pollution exposure than populations with high levels of remaining life expectancy. The individual-level framework above also holds for more aggregated levels of analysis, such as the county level.

We address this challenge by harnessing the comprehensive health and demographic information available in the Medicare dataset to generate relatively precise predictions of counterfactual life expectancy. In other words, we minimize the magnitude of the measurement error represented by u_{it} in equation (4). Note that it is not necessary to eliminate all measurement error to remove the bias in estimating life-years lost; it suffices to eliminate just the portion of the measurement error that is correlated with the heterogeneous treatment effect, v_i. To our knowledge, no previous study has addressed this bias by using variables other than age to predict life expectancy (e.g., Deschenes and Greenstone 2011). By contrast, we incorporate information on chronic conditions, medical spending, health care utilization, and geographic location, among others. We show that this matters: using average life expectancy or estimating life expectancy using only basic demographic variables such as age and gender causes significant upward bias in regression estimates of life-years lost due to air pollution.

A challenge with estimating counterfactual life expectancy is that not everybody dies during the period we observe them. We therefore employ the semi-parametric Cox proportional hazards survival model, which assumes that the hazard rate of death for individual *i* can be factored into two functions:[16]

$$h(t_i|x_i, \beta) = h_0(t_i)\exp[x_i'\beta]$$

[16] Employing fully parametric models that assume survival rates are governed by either the Gompertz or Weibull distributions yields very similar results.

The hazard rate at time t_i, $h(t_i|x_i, \beta)$, depends on the baseline hazard rate, $h_0(t_i)$, and on a vector of individual characteristics, x_i. The parameter vector β is estimated by maximizing the log partial likelihood function:

$$\ln L(\beta) = \sum_{i=1}^{N} \delta_i \left[x_i'\beta - \ln \sum_{j \in R(t_i)} \exp[x_j'\beta] \right] \tag{6}$$

where the indicator variable δ_i is equal to one for individuals whose deaths we observe (uncensored observations) and equal to zero otherwise, and the risk set $R(t_k) = \{l: t_l \geq t_k\}$ is the set of observations at risk of death at time t_k. We then nonparametrically estimate the baseline hazard function, $h_0(t_i)$, following Breslow (1972). See the appendix for details.

We estimate this Cox proportional hazards model using data from the 2002 cohort of Medicare beneficiaries.[17] We observe all deaths that occur among this cohort between January 1, 2002 and December 31, 2011. During this 10-year time period, 50 percent of the sample dies; the remaining deaths are censored. To ensure accurate measures of beneficiaries' chronic conditions, we limit the sample to Medicare beneficiaries who as of January 1, 2002 had been continuously enrolled in fee-for-service Medicare for at least two years.[18] For computational ease, we further limit the analysis to a random 5 percent sample of these beneficiaries. The final estimation sample for our survival analysis includes 1,211,585 individuals.

To assess the importance of accounting for decedents' remaining life expectancy, we estimate the survival model several times, using increasingly large sets of characteristics. First, we use no individual characteristics; that is, we assume a homogeneous survival function. A second specification controls for age and sex, and then a third specification additionally controls for the presence of 27 different chronic conditions. Our final and preferred specification incorporates over one thousand variables, derived from individual-level Medicare data and ZIP code-level data from the American Community Survey. These variables contain information on prior medical spending; outpatient and inpatient visits; length of stay for inpatient, skilled nursing facility, and hospice events; number of hospital readmissions; and average commute times, median income, median housing values, and employment in the beneficiary's 5-digit ZIP code of residence.[19] Including so many control variables creates two challenges. First, some variables may

[17] Although earlier cohorts are observable for a longer period of time, we do not use them because the Medicare variables denoting the presence of pre-existing chronic conditions, which are strong predictors of survival, are nonexistent or unreliable in earlier years.

[18] Because our analysis focuses on beneficiaries who are eligible for Medicare based on their age being 65 or older, this restriction means that no one in this sample is under age 67.

[19] There are 1,062 variables in total, detailed in the appendix.

be significant predictors of survival for the 2002 cohort just by chance, even if they are not good predictors of survival in general. This may cause bias due to overfitting (Harrell et al. 1996). Second, computational limitations prevent us from including a large set of regressors when performing conventional maximum likelihood estimation on a large sample using standard numerical procedures.

Recent advances in machine learning techniques help us overcome these challenges and use all 1,062 variables when predicting individual-level life expectancies (Athey and Imbens 2016). One popular method is the Least Absolute Shrinkage and Selection Operator (LASSO) estimator (Tibshirani 1997).[20] LASSO can be implemented by maximizing a penalized version of objective function (6):

$$\ln L(\beta) = \left(\sum_{i=1}^{N} \delta_i \left[x_i'\beta - \ln \sum_{j \in R(t_i)} \exp[x_j'\beta] \right] \right) - \lambda \sum_{i=1}^{k} |\beta_i| \tag{7}$$

where $|\beta_i|$ is the absolute value of β_i (where β_i is element i of the vector β) and k is the number of included regressors. We select the optimal penalty parameter λ using 5-fold cross validation.[21] We then use estimates of β and observable characteristics x_i to predict the life expectancy of each Medicare beneficiary who was continuously enrolled in fee-for-service for at least two years at some point during our sample period.

Integrating these estimates into the county-level empirical strategy presented in Section IV.A is straightforward: we simply aggregate life-years lost over all individuals in the county and replace the dependent variable in equation (1) with the estimated daily number of life-years lost per capita in county c, $\hat{L}_{cdmy}$. The variable $\hat{L}_{cdmy}$ is equal to the sum of the estimated counterfactual life expectancies for all decedents divided by the total number of beneficiaries in the county, and thus is analogous to how to we calculate the mortality rate.

Figure 5 demonstrates the increase in explanatory power that accompanies the inclusion of additional demographic and health variables in estimating life-years lost. This figure plots the average counterfactual life expectancy for all Medicare beneficiaries who died between 2001 and 2011, as predicted by models with increasing numbers of explanatory variables. We focus on beneficiaries who died because this group forms the basis of our mortality estimates. The green bar, "Medicare FFS average", reports the average life expectancy for all Medicare fee-for-service beneficiaries (11.56 years) and serves as a baseline. This value would be an accurate measure of counterfactual life expectancy if Medicare beneficiaries died randomly.

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[20] We also used other machine learning techniques like ridge regression and elastic net. The results are similar.

[21] See Simon et al. (2011) for a detailed discussion of the algorithm we employ to implement the Cox proportional hazards estimator with a LASSO penalty term. The efficiency of this algorithm allows us to estimate a survival model with both many regressors and a large number of observations.

In reality, the individuals who died between 2001 and 2011 are on average older than the typical Medicare beneficiary, as one would expect. The red bar, "Cox (age sex)" adjusts life expectancy for age and sex and predicts an average life expectancy of 7.85 years. The blue bar additionally controls for 27 different chronic conditions, reducing predicted life expectancy by over two years. This happens because decedents are sicker than the average Medicare beneficiary, even after controlling for age and gender. Finally, the black bar in Figure 5 displays average predicted life expectancy based on a model that incorporates data from all 1,062 variables in our dataset. This reduces counterfactual life expectancy by yet another half year, to 4.86 years per decedent.

Note that these results do not necessarily mean that it is important to control for these additional variables. As shown by our model, generating more precise estimates of life expectancy is only necessary if the existing measurement error is correlated with heterogeneous treatment effects. For example, if air pollution kills people at random, then one does not need to have precise individual-level estimates of life expectancy; the population mean will suffice. The only way to know whether it matters is to see how results differ when using these different predictions. Those results are presented in the next section.

V. RESULTS

V.A. Mortality and health care utilization

Panel A of Table 2 reports OLS estimates describing the relationship between daily PM 2.5 and 3-day mortality rates per million beneficiaries for different age groups. As reported in Column (1), each 1-$\mu g/m^3$ increase in daily PM 2.5 exposure is associated with 0.098 additional deaths per million elderly over the following three days, or a 0.025 percent increase relative to the average 3-day mortality rate. Columns (2)-(6) report results estimated separately for each of five age groups. The absolute and relative increases in mortality are non-monotonic across age groups, with those aged 70-79 experiencing lower (and insignificant) increases in death rates than those aged 64-69 despite having higher mean death rates.

Panel B of Table 2 presents the corresponding IV estimates of the causal effect of daily PM 2.5 on 3-day mortality. The IV estimates are about five times larger than the corresponding estimates in Panel A, suggesting that OLS estimation suffers from significant bias. The IV estimates imply that each 1-$\mu g/m^3$ increase in daily PM 2.5 exposure corresponds to 0.605 additional deaths per million elderly over the following three days, or a 0.15 percent increase relative to the average 3-day mortality rate.[22] The

[22] As described in our empirical strategy, we focus on 3-day mortality in order to avoid capturing short-run mortality displacement and to allow for pollution to have lagged effects. The comparable estimate from IV estimation of (1) for 1-day mortality yields a coefficient of 0.382 additional deaths resulting from a 1-$\mu g/m^3$ increase in daily PM 2.5 exposure, suggesting that the mortality impact of PM 2.5 exposure grows over time due to lagged effects.

corresponding estimate for a one standard deviation increase in daily PM 2.5 is a 1.1 percent increase in 3-day mortality. Columns (2)-(6) show a largely monotonic relationship between the mortality effect of PM 2.5 and age, with each 1-$\mu g/m^3$ increase in daily PM 2.5 causing 0.263 additional deaths per million among the 65-69 population but 2.050 additional deaths per million among the 85 and over population. However, because the average mortality rate is also much higher for the older elderly, the *relative* mortality effects across age groups follow a U-shaped pattern: each 1-$\mu g/m^3$ increase in daily PM 2.5 exposure increases 3-day mortality by 0.20 percent among ages 65-69, by 0.10 percent among ages 75-79, and by 0.18 percent among ages 85 and over. This pattern is somewhat unexpected, since, if sicker individuals are more vulnerable to pollution shocks, and if age is a good proxy for health, then we would expect relative mortality to increase monotonically with age. We return to this point when discussing our estimates of life-years lost due to PM 2.5, where we will find that relative mortality does increase monotonically with counterfactual life expectancy.

Next, we estimate the effect of daily PM 2.5 on 3-day hospitalization rates and associated medical spending per million beneficiaries enrolled in fee-for-service Medicare. As discussed earlier, the change in sample from all Medicare beneficiaries to those enrolled in traditional, fee-for-service Medicare is necessary because spending information is only available for this subsample. For reference, we show the all-age mortality response to PM 2.5 for this population in Column (1) of Table 3; it is very similar to what we find for the overall Medicare population. Panel A of Table 3 shows that the association between PM 2.5, hospitalization, and medical spending is mixed: each 1-$\mu g/m^3$ increase in daily PM 2.5 exposure is associated with significantly *less* inpatient spending and *fewer* hospital admissions, is not associated with spending on ER admissions, and is associated with significantly *more* ER admissions and visits. A more consistent story emerges from our IV approach (Panel B), which shows that increases in daily PM 2.5 increase both hospitalizations and inpatient spending, driven primarily by encounters that originate in the ER. The IV estimates imply that each 1-$\mu g/m^3$ increase in daily PM 2.5 causes a highly significant increase in ER inpatient spending of over $15 thousand per million beneficiaries (relative to a mean of $13.7 million). This increase is almost as large as the increase in total inpatient spending, and we cannot reject that the latter is driven entirely by increases in ER spending. The overall admissions rate increases by 2.03 per million beneficiaries, an increase which also can be almost entirely explained by the 1.96 additional admissions originating through the ER. Finally, we estimate that PM 2.5 increases total ER visits, including visits that do not result in a hospital admission, by 2.29 per million beneficiaries.

Comparing the OLS estimates to the IV estimates in Tables 2 and 3 provides strong evidence that observational studies of the relationship between air pollution and health outcomes suffer from significant bias: virtually all our OLS estimates are smaller than the corresponding IV estimates. If the only source of bias were measurement error, which causes attenuation, we would not expect to see significantly *negative*

OLS estimates. Thus, biases that are not driven by measurement error, such as changes in economic activity that are correlated with both hospitalization patterns and pollution, appear to be a concern even when working with high-frequency daily data.

Finally, it may be of interest to compare the magnitudes of our IV estimates to those from the epidemiological literature, which are often used for calculating the benefits of various environmental policies. This is not straightforward because many of these studies differ from ours in terms of the setting, the population of interest, the outcome variables, and the included control variables. For example, many studies are confined to a particular set of cities, do not control for weather, or only examine hospital admissions associated with a particular disease. Overall, our IV mortality estimates are generally smaller than observational studies of chronic exposure, but, perhaps surprisingly given the aforementioned caveats, in line with estimates from studies of acute exposure.[23] Our hospitalization results are also similar to what has previously been estimated by the epidemiological literature.[24] However, as we next demonstrate with our life-years lost analysis, knowing the mortality rate increase caused by pollution is insufficient for determining the mortality costs of acute pollution exposure.

V.B. Life-years lost and the value of mortality reductions

Having established the causal effect of PM 2.5 on mortality, we next turn to estimating the cost of that mortality, using the method of estimating life-years lost described in Section IV.B. Table 4 displays estimates of equation (1) when the outcome variable is the estimated 3-day life-years lost per million beneficiaries ($\hat{L}_{cdmy}$). As discussed in Section III, this estimation sample is limited to those beneficiaries continuously enrolled for at least two years in fee-for-service (FFS) Medicare so that chronic conditions are well-measured. For reference, Column (1) shows the estimated effect of PM 2.5 on the 3-day mortality rate among the 2-year FFS population. This estimate is larger in absolute terms than the IV estimate from Table 2 in part because the 2-year FFS restriction mechanically excludes individuals ages 65-66, causing this sample to be older on average. The effects relative to average 3-day mortality are very similar for both populations.

Column (2) displays results when every decedent's counterfactual life expectancy is set equal to the mean for the 2-year FFS population (11.6 years). This estimate implies that each 1-μg/m^3 increase in

[23] Pope et al. (2002) find that a 10-μg/m^3 increase in long-run PM 2.5 exposure is associated with a 4 percent increase in the annual risk of all-cause mortality. Our coefficient implies that the same magnitude *daily* increase in PM 2.5 increases *daily* mortality by 1.5 percent of the average daily mortality rate. Pope and Dockery (1999) review the literature on short-term effects and report that a 10-μg/m3 PM 10 increase raises daily mortality rates by 0.5 percent to 1.5 percent (see their Figure 31.3, p. 681). Using a standard conversion of PM 10 to PM 2.5 (1.67:1, Dockery and Pope 1994), our estimated effect from PM 2.5 is within this range.

[24] See Dai et al. (2014), Pope and Dockery (1999), Schwartz (1994a), and Zanobetti et al. (2009).

daily PM 2.5 increases life-years lost by 8.6 years per million beneficiaries. This same effect can also be obtained directly by multiplying the mortality effect of 0.746 in Column (1) by the mean life expectancy of 11.6. However, this estimate is accurate only if beneficiaries killed by PM 2.5 are representative of the overall 2-year FFS population. If decedents have a lower counterfactual life expectancy than those who remain alive, then the estimate in Column (2) will be biased upward.

Columns (3)-(5) of Table 4 illustrate this bias by progressively expanding the set of covariates used to predict counterfactual life expectancy. Those covariates are reported in the column headers. Column (3) displays estimates when a decedent's counterfactual life expectancy is modeled solely as a function of age and sex. This approach is comparable to studies that estimate age- and sex-specific mortality effects and multiply them by the corresponding life expectancies from population life tables (e.g., Deschenes and Greenstone 2011). In our setting, accounting for decedents' age and sex reduces the estimated impact of PM 2.5 on life-years lost by 31 percent, to 5.9 life-years per million beneficiaries. This decrease is consistent with the results presented in Table 2: older beneficiaries, who have lower life expectancies, are also more likely to be killed by PM 2.5. The estimate decreases by another 40 percent when the counterfactual life-years estimates account for previously diagnosed chronic conditions (Column 4), implying significant heterogeneity in the mortality effect of PM 2.5 even among individuals of the same age and sex.

Finally, we estimate counterfactual life expectancy using the LASSO machine learning algorithm, which allows us to optimally incorporate over 1,000 additional predictors, as described earlier. This final estimate, reported in Column (5), is 24 percent smaller than estimates that account only for age, sex, and chronic conditions and implies that each 1-$\mu g/m^3$ increase in daily PM 2.5 increases life-years lost by 2.7 years per million beneficiaries. The fact that the estimate from Column (5), with over 1,000 control variables, is only modestly smaller than the prior estimate, based only on age, sex, and chronic conditions, suggests that this final estimate may be close to the true value.

Table 4 illustrates that adding additional predictors when estimating life expectancy can substantially reduce the estimate of life-years lost due to pollution. This reduction can occur for two reasons. First, better survival models should predict lower remaining life expectancy for decedents *on average*. Table 4 (and Figure 5) report that the mean life-years lost per decedent ("LYL per decedent") decreases from 11.56 in the model with no predictors to 4.86 in the LASSO model. Second, a better survival model should also predict a more accurate *distribution* of predicted life expectancies among decedents. This matters if air pollution selectively kills individuals in this population who are systematically healthier (or sicker) than the average decedent. Indeed, Table 4 demonstrates that this second channel also plays a role in reducing the estimated life-years lost from improved survival modeling. While the average LYL per decedent decreases by only 0.43 per million when moving from LYL estimates based on age, sex, and chronic conditions to those based on the LASSO model, the estimated effect of PM 2.5 on LYL drops by

nearly twice as much (0.85 per million). This indicates that the mortality effects of PM 2.5 tend to be larger among individuals with characteristics that LASSO associates with lower life expectancy, even after conditioning on age, sex, and chronic conditions.

The estimates in Table 4 can also be used to describe the estimated counterfactual life-years lost among "compliers": those individuals who died because of increases in wind-driven PM 2.5. This estimate can be compared to the average life-years lost among all decedents to shed light on whether those dying from increased pollution appear to be differentially healthy or frail compared to those who die on a typical day. The LYL per complier is calculated by dividing the estimated effect of increased PM 2.5 on life-years lost by the estimated mortality effect (the coefficient reported in Column 1).[25] When life expectancy is modeled as a function of age and sex alone, those dying from pollution appear to have slightly longer life expectancies (7.9 years) compared to the average decedent (7.8 years). However, estimates that rely on chronic conditions or the LASSO model show the opposite pattern. In Column (5), those dying from pollution appear to have somewhat shorter life expectancies (3.6 years) compared to the average decedent (4.9 years).

Pursuing this point further, we next estimate the effects of PM 2.5 on mortality and life-years lost separately for groups of beneficiaries with different life expectancies. Because the results from Table 4 showed that the typical complier has a low life expectancy, we focus this exercise on individuals in the bottom half of the life expectancy distribution (i.e., less than 10 years), although we show estimates for those in the top half of the distribution as well. This analysis sheds light on whether it is only the very sick who are killed by air pollution or whether people in fair health are vulnerable as well.

Table 5 reports mortality rate and life-years lost estimates separately for five groups of beneficiaries: those with a predicted life expectancy of less than 1 year, 1-2 years, 2-5 years, 5-10 years, and over 10 years. The column headers report the percent of all beneficiaries falling into each group: 55 percent of our sample has a life expectancy that exceeds 10 years, while only 0.7 percent has a life expectancy of less than 1 year. Panel A of Table 5 illustrates that the mortality rate effect of PM 2.5 decreases monotonically with life expectancy. A 1-$\mu g/m^3$ increase in daily PM 2.5 increases deaths among those with life expectancy of less than one year by 18.9 per million. By contrast, the effect on those with life expectancies of 5-10 years is only 0.53 deaths per million, and the mortality rate effect for those with life expectancies exceeding 10 years is even smaller and not statistically different from zero. This pattern parallels the estimates reported in Table 2, which showed that the mortality effect is largest among the oldest beneficiaries (who generally have low life expectancies). However, unlike the pattern in Table 2, we

[25] For example, in Column (3), a one-unit pollution increase causes 0.746 deaths per million and causes an increase in LYL of 5.925 years per million. Thus, the LYL per person killed by pollution is 5.925/0.746 = 7.94.

find that *relative* mortality also decreases monotonically with life expectancy, which is consistent with the notion that the sickest individuals are most vulnerable to pollution shocks.

Table 5 shows that, although beneficiaries with a life expectancy of less than one year are the most likely to be killed by air pollution, beneficiaries with a life expectancy of up to 10 years are also vulnerable. Beneficiaries with a life expectancy of less than one year make up less than 1 percent of our sample, while those with life expectancies of 5-10 years make up almost 30 percent. Therefore, the absolute number of deaths caused by PM 2.5 varies less across these two groups than does the relative number of deaths.

Panel B shows the effects of PM 2.5 on life-years lost in each of these five groups. Although beneficiaries in Column (1) have less than one year of life expectancy, their high mortality rate causes their number of life-years lost due to pollution to exceed that of any other group: 11.3 life years per million beneficiaries. Thus, even among the group where the "harvesting critique" is most likely to apply, there are still significant benefits to reducing pollution on a per-capita basis. By contrast, among beneficiaries with a life expectancy of 5-10 years (Column 4), the life-years lost from pollution is only equal to 3.7. Although their life expectancy is high relative to those in Column (1), their mortality rate is much lower, resulting in a smaller loss of life years. Those with 1-2 or 2-5 years of life expectancy (Columns 2 and 3) fall somewhere in between, losing 8.2 and 6.7 life years per million beneficiaries, respectively, when PM 2.5 increases by 1 $\mu g/m^3$.

Weighting the life-years lost coefficients from Table 5 by the respective sizes of the groups, we see that the largest portion of the social cost of pollution is borne by those with a life expectancy of 5-10 years (30 percent of sample, 43 percent of burden), followed by those with a life expectancy of 2-5 years (12.7 percent of sample, 33 percent of burden). While the per capita burden is highest for those with the lowest life expectancy, the majority of the aggregate social burden falls on those with intermediate life expectancy (2 to 10 additional years).

We also note that our approach, which involves predicting life expectancy, identifies vulnerable populations better than an approach that uses age alone. In Table 2, we found significant mortality effects of pollution across all age groups, but there was no clear relationship between age and the relative mortality effect. In contrast, our life expectancy based approach has identified a group of beneficiaries (i.e., those with life expectancies of more than 10 years) who do not appear to be vulnerable to pollution shocks (i.e., the mortality impact is a precisely estimated zero). In addition, the health-vulnerability gradient is much stronger in both absolute and relative terms when health is measured by life-years remaining rather than life-years lived (i.e., age). Overall, this suggests that a precise measure of life expectancy may be useful not only for characterizing mortality costs, but also in identifying which populations are particularly vulnerable to those shocks.

A simple numerical exercise helps to illustrate the policy implications of our results. The average level of PM 2.5 decreased by 3.65-μg/m^3 nationwide between 1999 and 2011, as shown in Figure 1. The estimate reported in Column (5) of Table 4 implies that such a decrease saved 147,098 life-years annually among the 41 million Medicare beneficiaries alive in 2011.[26] If we assign each life year a standard value of $100,000 each, the mortality reduction benefits of this decrease added up to about $15 billion in 2011. The EPA's calculation of the annual costs of meeting the 1990 Clean Air Act Amendment air quality standard increased from $19.9 billion to $43.9 billion between 2000 and 2010 (EPA 2011). Thus, the estimated $15 billion in annual mortality benefits represents a large fraction of the estimated annual costs of complying with air pollution standards during this period. By contrast, the reduction in hospitalization costs implied by our estimates is an order of magnitude smaller – about $0.93 billion annually.

Finally, our estimate of the mortality reduction benefits is nearly 70 percent lower than the estimate of $47 billion obtained from ignoring heterogeneity in the effect of pollution on elderly mortality. Estimated benefits that account for age and sex alone are $32 billion, still more than double the estimated benefit of $15 billion based on our most comprehensive model. This demonstrates the importance of properly accounting for "harvesting" when calculating the mortality benefits of reductions in air pollution.

V.C. Other pollutants and robustness checks

One concern with interpreting our estimates as the causal effects of PM 2.5 is that other air pollutants like ozone (O_3) and carbon monoxide (CO) can be co-transported with fine particulate matter (PM 2.5). However, because these pollutants are not perfectly co-transported (they can be produced by sources located in different places and are carried differently by the wind), our empirical strategy allows us to instrument separately for each pollutant. Two other pollutants, sulfur dioxide (SO_2) and nitrogen oxide (NO_2) are precursors to PM 2.5 and are also thought to have independent health effects. SO_2 converts to SO_4^{2-}, an important component of particulate matter, on the order of several percent per hour (Luria et al. 2001). NO_2 converts to particulate nitrate at a similar rate (Lin and Cheng 2007). Recall that we are considering the effect of a 1-day change in average pollution concentrations on 3-day outcomes. Because the majority of SO_2 and NO_2 converts to particulate matter within 2-3 days, it is impossible to distinguish their effects from those of PM 2.5 with a 3-day specification.[27] We therefore focus our investigation on

[26] The exact calculation is $2.693 \times 365 \times 41 \times 3.65$. This calculation assumes that our daily mortality effects can be linearly scaled to the annual level. The epidemiological literature generally finds larger effects from long-run exposure than from short-run exposure (Pope and Dockery 1999), suggesting that linear scaling is a conservative assumption.

[27] For example, a conservative conversion rate of 3 percent per hour implies that over half (three-quarters) of the SO_2 and NO_2 would have converted to particulate matter after 24 (48) hours. A 4 percent hourly conversion rate implies that over 60 (85) percent is converted after 24 (48) hours. An earlier version of this paper considered 1-day mortality

whether our previous estimates change after controlling for CO and O_3. We restrict the sample to county-days where readings for these pollutants and PM 2.5 are simultaneously available, and then sequentially add the CO and O_3 endogenous variables to our main estimating equation.

The results are shown in Table 6. The estimated effects of PM 2.5 are always significant and fairly stable across the different specifications. This suggests that the mortality effects we found are indeed primarily attributable to PM 2.5 and not these other pollutants. The coefficient on ozone is negative in column (3), reflecting the well-known finding that it is negatively correlated with other pollutants (Currie and Neidell 2005). When we also add carbon monoxide as a control (column 4), we get slightly different O_3 and CO results depending on whether we consider the entire Medicare population or just fee-for-service beneficiaries (Panel A versus B). Nevertheless, our conclusions about the impacts of PM 2.5 are the same for both populations.

Our main empirical specification employs 300 instruments. Although our reported F-statistics are generally quite large, and our IV and OLS estimates are quite different, we nevertheless undertake two different sets of robustness exercises to ensure that our estimates are not driven by weak instrument bias. First, we estimate our IV model using LIML, which is approximately median unbiased even in the presence of weak instruments, rather than 2SLS. Those estimates, presented in Table 7, are very similar to the 2SLS estimates presented in Table 2. Second, we conduct a placebo exercise where we generate a set of random wind directions and use those in our first stage instead of the actual wind direction. Those results, shown in Table 8, are largely insignificant. Moreover, the first-stage F-statistics for those estimates are very small, which provides strong evidence that our wind direction instrument is picking up meaningful rather than spurious variation in PM 2.5 levels.

Finally, Tables 9-11 shows the robustness of our primary empirical specification to (1) including more or fewer instrument lags (Table 9); (2) including different types of fixed effects and weather controls (Table 10); and (3) varying the width of the angle bins (60 or 90 degrees) as well as the number of monitor groups (50, 100, or 200) (Table 11). Our estimates are not sensitive to these choices.

VI. CONCLUSION

Understanding how air pollution affects health and health care spending is essential for crafting efficient environment policy, such as Pigouvian pricing based on health externalities. Causal effects of pollution are difficult to identify because of endogeneity and measurement error. Valuation of mortality

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and instrumented for all five pollutants (PM 2.5, SO_2, NO_2, CO, and O_3) simultaneously, showing that controlling for other pollutants did not change our conclusions about the effects of PM 2.5. These results are available upon request.

reductions is also difficult because of the likely heterogeneity of pollution's effects. If deaths caused by pollution occur disproportionately among the least healthy, then ignoring this heterogeneity will lead to upward bias when estimating the social cost of pollution.

This paper sheds light on these issues by estimating the causal effect of acute fine particulate matter exposure on mortality, life-years lost, and hospitalizations using a novel identification strategy based on changes in wind direction. This is accomplished by linking daily pollution and climatic variables to detailed administrative records on all Medicare beneficiaries from 1999-2011. We find significant effects of pollution on mortality, health care spending, and hospitalizations. Finally, we calculate that the reduction in PM 2.5 experienced nationwide between 1999 and 2011 generated $15 billion annually in mortality benefits among the elderly alone by the end of that period.

Our life-years lost analysis shows that the least healthy individuals are more vulnerable to pollution than the average elderly person, whether vulnerability is measured by total mortality risk, relative mortality risk, or expected number of life-years lost. Our estimate of the total number of life-years lost, which is based on health information gleaned from detailed Medicare data, is less than half the magnitude of an estimate that accounts for age and sex alone, and less than a third of the magnitude of an estimate based solely on the life expectancy of an average Medicare beneficiary. This suggests that failure to adjust for the health of those who die can result in substantial overvaluation of the mortality benefits of pollution reduction. Finally, we note that although we present our life-years lost analysis in the context of air pollution, our methodology could be used to estimate mortality costs or identify vulnerable populations in other studies that estimate health effects.

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FIGURES

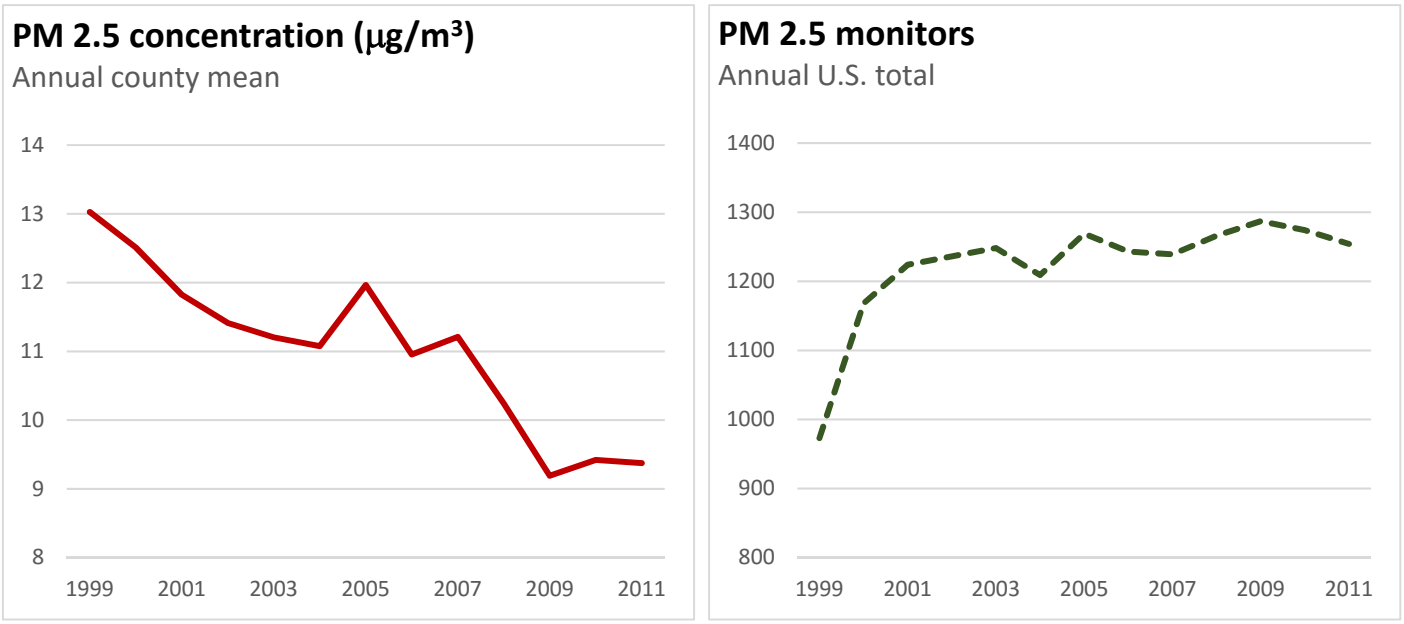


Figure 1. Trends in PM 2.5 air pollution and monitoring, 1999-2011. Figure displays annual county means for PM 2.5 concentration (left panel), and the nationwide total number of PM 2.5 monitors (right panel).

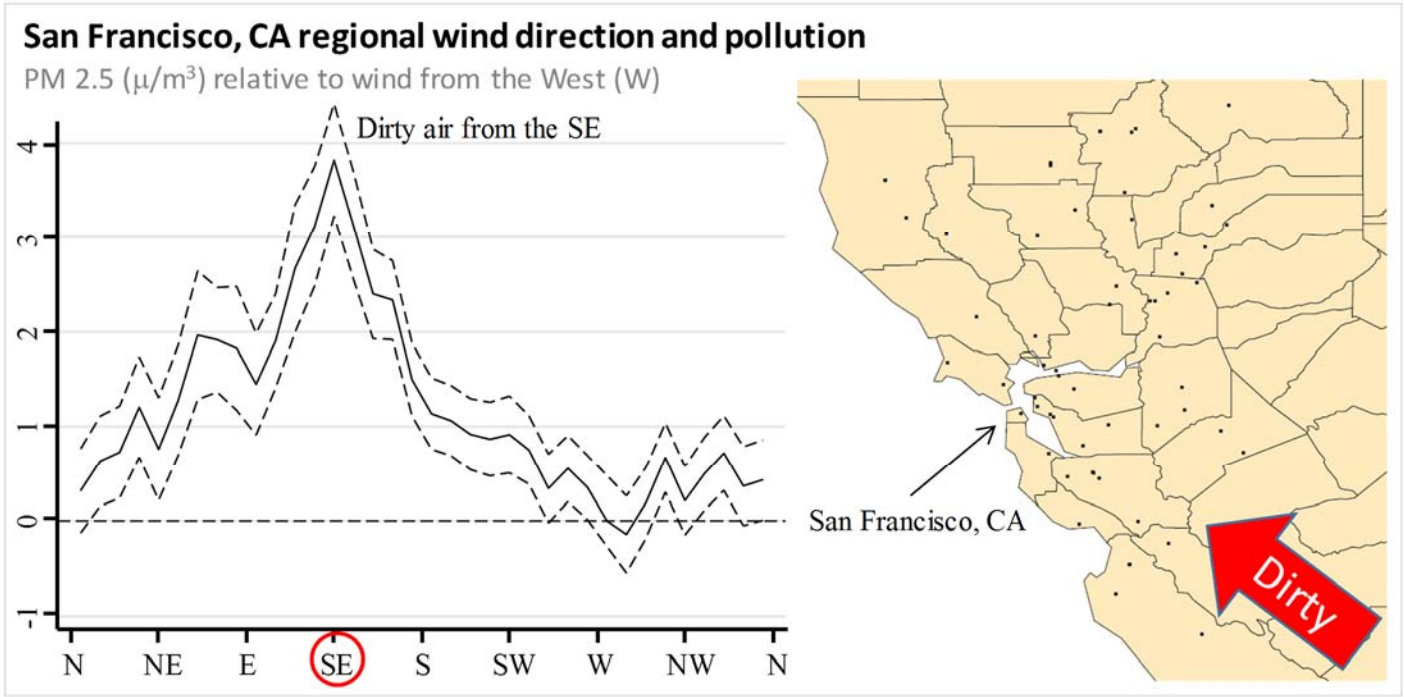


Figure 2. Relationship between daily average wind direction and PM 2.5 concentrations for counties in and around the Bay Area, CA. The left panel shows regression estimates of equation (A1) from the appendix, where the dependent variable is the county average daily PM 2.5 concentration and the key independent variables are a set of indicators for the daily wind direction falling into a particular 10-degree angle bin. Controls include county, month-by-year, and state-by-month fixed effects, as well as a flexible function of maximum and minimum temperatures, precipitation, wind speed, and the interactions between them. The dashed lines represent 95 percent confidence intervals based on robust standard errors. The right panel shows the location of the PM 2.5 pollution monitors (black dots) in the Bay Area that provided the pollution measures for this regression.

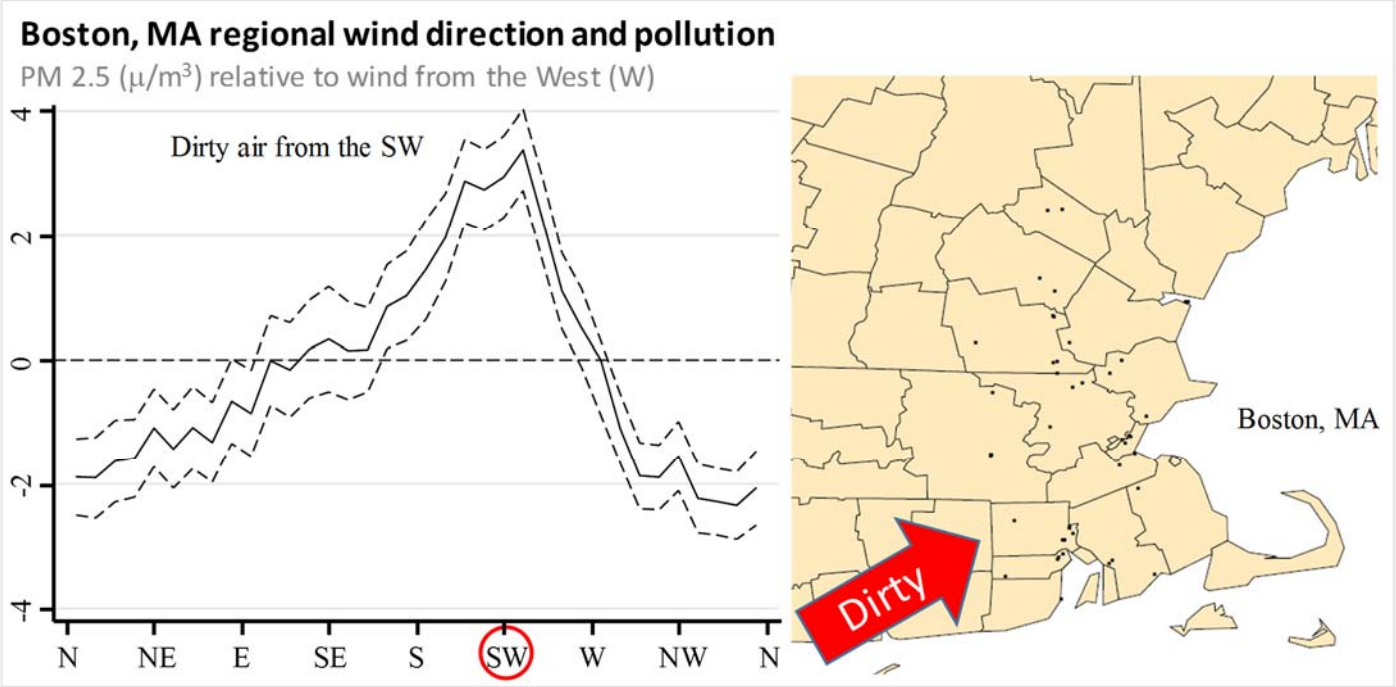


Figure 3. Relationship between daily average wind direction and PM 2.5 concentrations for counties in and around the Boston Area, MA. The left panel shows regression estimates of equation (A1) from the appendix, where the dependent variable is the county average daily PM 2.5 concentration and the key independent variables are a set of indicators for the daily wind direction falling into a particular 10-degree angle bin. Controls include county, month-by-year, and state-by-month fixed effects, as well as a flexible function of maximum and minimum temperatures, precipitation, wind speed, and the interactions between them. The dashed lines represent 95 percent confidence intervals based on robust standard errors. The right panel shows the location of the PM 2.5 pollution monitors (black dots) in the Boston Area that provided the pollution measures for this regression.

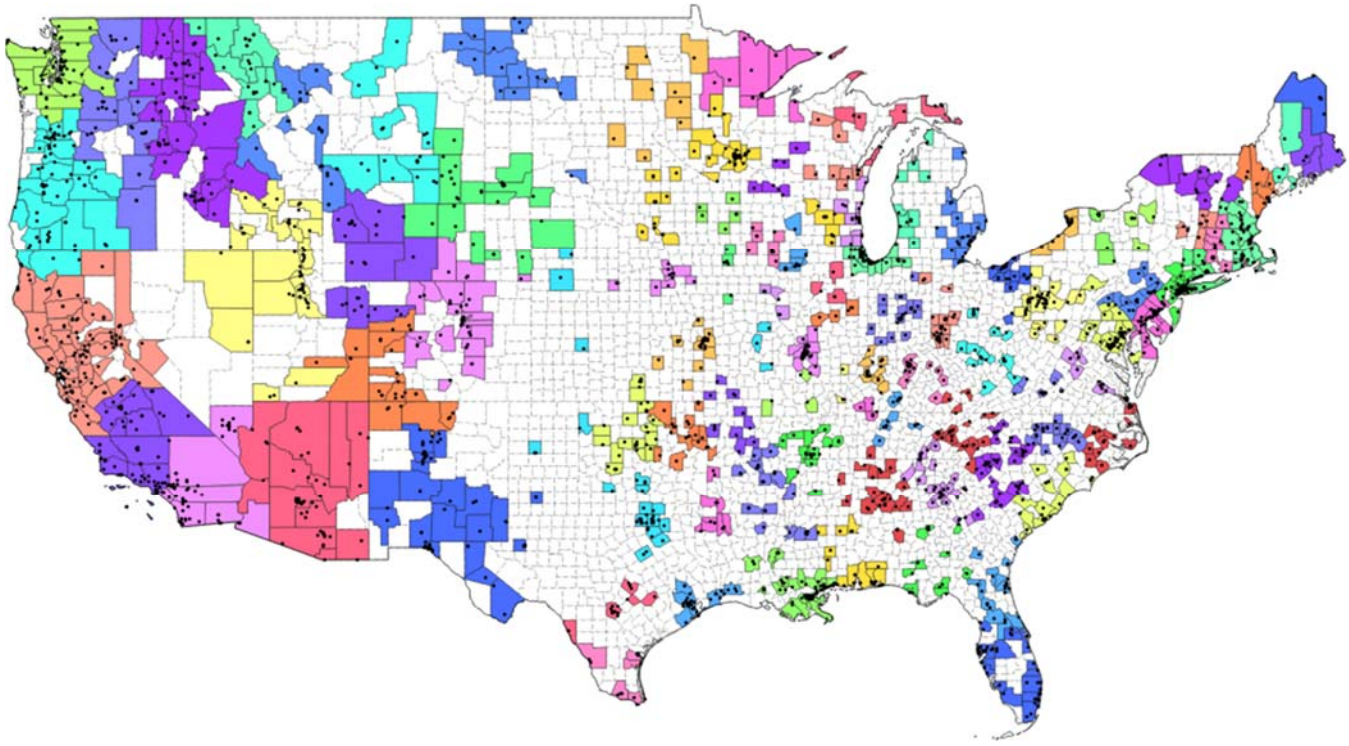


Figure 4. Counties assigned to each monitor group. Different colors correspond to different monitor groups. White corresponds to counties not assigned to any monitor group due to lack of monitors. Black dots represent PM 2.5 pollution monitors.

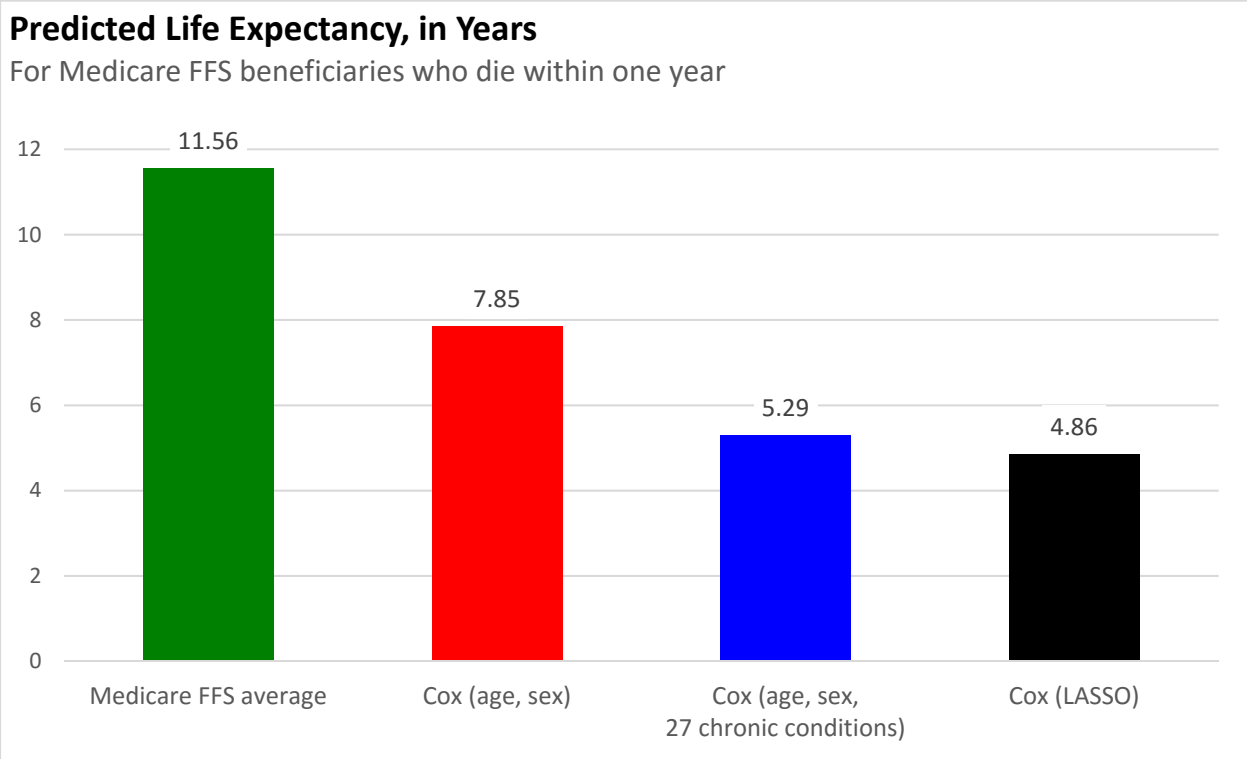


Figure 5. Average life expectancy for continuously enrolled fee-for-service Medicare beneficiaries who later die within one year, 1999-2011. Life expectancy for each beneficiary is estimated as of January 1 of the calendar year of death. Estimates for "Medicare FFS average" are produced by MLE estimation of survival model (6) with no covariates. Estimates for "Cox (age sex)" and "Cox (age sex cc) are produced by estimating the survival model (6) using age and gender, and age, gender and 27 chronic conditions, as predictors, respectively. Estimates for "Cox (LASSO)" are produced by machine learning estimation of the survival model (7) with 1,062 included regressors.

TABLES

Table 1: Summary statistics, 1999-2011

	Mean	Standard deviation	Observations
PM 2.5 ($\mu g/m^3$)	10.86	7.34	1,600,846
Number of beneficiaries, all ages	49,486	78,795	1,600,846
Number of beneficiaries, 65-69	12,923	20,262	1,600,846
Number of beneficiaries, 70-74	11,726	18,731	1,600,846
Number of beneficiaries, 75-79	9,960	16,088	1,600,846
Number of beneficiaries, 80-84	7,695	12,437	1,600,846
Number of beneficiaries, 85+	7,181	11,708	1,600,846
Number of FFS beneficiaries	34,911	52,748	1,518,623
Continuously enrolled FFS beneficiaries	27,716	40,090	1,518,623
3-day mortality rate, all ages	393.49	249.46	1,600,846
3-day mortality rate, 65-69	137.56	269.47	1,600,846
3-day mortality rate, 70-74	205.25	379.71	1,600,846
3-day mortality rate, 75-79	325.52	486.45	1,600,846
3-day mortality rate, 80-84	530.92	742.12	1,600,846
3-day mortality rate, 85+	1,169.86	1,119.82	1,600,846
3-day mortality rate, all FFS	409.02	274.61	1,518,623
3-day mortality rate, continuously enrolled FFS	458.21	315.98	1,518,623
3-day inpatient spending, planned and ER	34,463,288	14,976,401	1,518,623
3-day inpatient ER spending	13,659,622	7,693,555	1,518,623
3-day admissions rate, planned and ER	3,370	1,210	1,518,623
3-day ER admissions rate	1,579	709	1,518,623
3-day ER (inpatient and outpatient) visit rate	4,159	1,198	1,518,623

Unit of observation is county-day. All rates are per million Medicare beneficiaries in the relevant group. Spending and admissions variables are only available for fee-for-service (FFS) beneficiaries. Life-years lost analysis uses variables only available for continuously enrolled FFS beneficiaries.

Table 2: OLS and IV estimates of effect of PM 2.5 on elderly mortality, by age group

	(1) 65+	(2) 65-69	(3) 70-74	(4) 75-79	(5) 80-84	(6) 85+
Panel A: OLS estimates						
PM 2.5 ($\mu g/m^3$)	0.098***	0.042***	0.022	0.033	0.137***	0.423***
	(0.021)	(0.015)	(0.019)	(0.023)	(0.037)	(0.074)
Dep. var. mean	393	138	205	326	531	1,170
Effect relative to mean, percent	0.025	0.030	0.011	0.010	0.026	0.036
Observations	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846
Adjusted R-squared	0.249	0.080	0.086	0.084	0.081	0.115
Panel B: IV estimates						
PM 2.5 ($\mu g/m^3$)	0.605***	0.263***	0.312***	0.307***	0.775***	2.050***
	(0.065)	(0.071)	(0.075)	(0.106)	(0.177)	(0.264)
F-statistic	241.115	232.367	236.416	241.909	247.716	256.311
Dep. var. mean	391	134	201	318	514	1,132
Effect relative to mean, percent	0.155	0.196	0.155	0.097	0.151	0.181
Observations	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports OLS and IV estimates of equation (1) from the main text. Standard errors (in parentheses) clustered by county. Dependent variable is the 3-day mortality rate per million beneficiaries in the relevant age group. All regressions include county, month-by-year, and state-by-month fixed effects; flexible controls for temperatures, precipitation, and wind speed; and two leads of these weather controls. OLS (IV) estimates also include two lags and two leads of PM 2.5 (instruments). Estimates are weighted by the number of beneficiaries in the relevant age group.

Table 3: OLS and IV estimates of effect of PM 2.5 on Medicare hospitalization outcomes

	(1) FFS all-age mortality	(2) All inpatient spending	(3) Inpatient E.R. spending	(4) Inpatient admissions rate	(5) Inpatient E.R. admissions rate	(6) Inpatient + outpatient E.R. rate
Panel A: OLS estimates						
PM 2.5 ($\mu g/m^3$)	0.137***	-8439***	877	-0.560***	0.127**	0.406***
	(0.023)	(1993)	(790)	(0.156)	(0.062)	(0.094)
Dep. var. mean	407	34,463,220	13,659,597	3,370	1,579	4,159
Effect relative to mean, percent	0.034	-0.024	0.006	-0.017	0.008	0.010
Observations	1,518,549	1,518,549	1,518,549	1,518,549	1,518,549	1,518,549
Adjusted R-squared	0.236	0.518	0.685	0.515	0.695	0.651
Panel B: IV estimates						
PM 2.5 ($\mu g/m^3$)	0.628***	17074*	15446***	2.034***	1.960***	2.290***
	(0.075)	(10182)	(4151)	(0.714)	(0.336)	(0.394)
F-statistic	237.338	237	237	237.338	237.338	237.338
Dep. var. mean	407	37,861,232	16,645,971	3,463	1,783	3,960
Effect relative to mean, percent	0.154	0.045	0.093	0.059	0.110	0.058
Observations	1,518,549	1,518,549	1,518,549	1,518,549	1,518,549	1,518,549

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports OLS and IV estimates of equation (1) from the main text. Standard errors (in parentheses) clustered by county. All dependent variables are 3-day measures per million fee-for-service (FFS) beneficiaries. All regressions include county, month-by-year, and state-by-month fixed effects; flexible controls for temperatures, precipitation, and wind speed; and two leads of these weather controls. OLS (IV) estimates also include two lags and two leads of PM 2.5 (instruments). Estimates are weighted by the number of FFS beneficiaries.

Table 4: IV estimates of effect of PM 2.5 on elderly life-years lost, using different survival models

		Life-years lost regressions			
	(1) All-age mortality	(2) None	(3) Age, sex	(4) Age, sex, chronic conditions	(5) LASSO
PM 2.5 ($\mu g/m^3$)	0.746***	8.625***	5.925***	3.539***	2.693***
	(0.085)	(0.978)	(0.757)	(0.562)	(0.521)
F-statistic	239	239	239	239	239
Dep. var. mean	462	5,338	3,624	2,444	2,245
Effect relative to mean, percent	0.162	0.162	0.163	0.145	0.120
LYL per decedent	NA	11.557	7.847	5.292	4.861
LYL per complier	NA	11.557	7.939	4.742	3.608
Observations	1,518,549	1,518,549	1,518,549	1,518,549	1,518,549

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text. Standard errors (in parentheses) clustered by county. The dependent variable in column 1 is the 3-day mortality rate per million continuously enrolled fee-for-service (FFS) Medicare beneficiaries. The dependent variable in columns 2-5 is life-years lost (LYL) over 3 days for the same group. The headings in columns 2-4 display the variables used to predict life expectancy when using a traditional Cox proportional hazards model. Column 5 displays results when life expectancy is predicted using a Cox proportional hazards model that is estimated using a LASSO machine learning algorithm with over one thousand predictors. LYL per decedent is calculated by dividing the average LYL in the sample by the average mortality rate. LYL per complier is calculated by dividing the columns estimate by the mortality effect reported in column 1. All regressions include county, month-by-year, and state-by-month fixed effects, as well as flexible controls for temperatures, precipitation, and wind speed; two leads of the weather controls; and two leads and lags of the instruments. Estimates are weighted by the number of continuously enrolled FFS beneficiaries.

Table 5: IV estimates of effect of PM 2.5 on elderly life-years lost, by remaining life expectancy

	(1) <1 year (0.69%)	(2) 1-2 years (2.24%)	(3) 2-5 years (12.7%)	(4) 5-10 years (29.8%)	(5) >10 years (54.6%)
Panel A: mortality					
PM 2.5 ($\mu g/m^3$)	18.885***	5.267***	2.160***	0.533***	0.041
	(3.441)	(1.409)	(0.426)	(0.140)	(0.049)
F-statistic	238	254	249	241	234
Dep. var. mean	4,593	2,955	1,425	421	89
Effect relative to mean, percent	0.411	0.178	0.152	0.127	0.047
Observations	1,482,554	1,515,728	1,518,549	1,518,549	1,518,549
Panel B: life-years lost					
PM 2.5 ($\mu g/m^3$)	11.281***	8.202***	6.727***	3.700***	0.688
	(2.233)	(2.239)	(1.439)	(0.973)	(0.633)
F-statistic	238	254	249	241	234
Dep. var. mean	2,711	4,517	4,773	2,958	1,132
Effect relative to mean, percent	0.416	0.182	0.141	0.125	0.061
Observations	1,482,554	1,515,728	1,518,549	1,518,549	1,518,549

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text. Standard errors (in parentheses) clustered by county. The dependent variable is either deaths (Panel A) or life-years lost (Panel B) over 3 days per million continuously enrolled fee-for-service beneficiaries for those with remaining life expectancy in the range given by the column heading. Column headings also display (in parentheses) the percent of beneficiaries falling into each range. Life expectancy is predicted using a Cox proportional hazards model that is estimated using a Cox machine learning algorithm with over one thousand predictors. All regressions include county, month-by-year, and state-by-month fixed effects, as well as flexible controls for temperatures, precipitation, and wind speed; two leads of the weather controls; and two leads and lags of the instruments. Estimates are weighted by the number of continuously enrolled FFS beneficiaries.

Table 6: IV estimates of effect of PM 2.5 on elderly mortality when controlling for other pollutants

	(1)	(2)	(3)	(4)
		Panel A: all beneficiaries		
PM 2.5 ($\mu g/m^3$)	0.437***	0.298***	0.568***	0.346***
	(0.101)	(0.098)	(0.097)	(0.123)
CO		0.023***		0.021***
		(0.007)		(0.008)
Ozone			-0.290***	-0.084
			(0.109)	(0.121)
F-statistic	118	33	49	27
Dep. var. mean	391	391	391	391
Observations	552,412	552,412	552,412	552,412
		Panel B: fee-for-service beneficiaries		
PM 2.5 ($\mu g/m^3$)	0.663***	0.568***	0.859***	0.799***
	(0.113)	(0.122)	(0.125)	(0.170)
CO		0.013		0.005
		(0.009)		(0.010)
Ozone			-0.443***	-0.393**
			(0.149)	(0.180)
F-statistic	111	31	45	25
Dep. var. mean	462	462	462	462
Observations	490,413	490,413	490,413	490,413

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Standard errors (in parentheses) clustered by county. Table reports IV estimates of equation (1) from the main text, with the addition of the endogenous variables CO and/or ozone, which are instrumented for using wind speed. Dependent variable is the 3-day mortality rate per million beneficiaries (Panel A) or per million fee-for-service (FFS) beneficiaries (Panel B). The sample is restricted to county-days where readings for CO, ozone, and PM 2.5 are simultaneously available. All regressions include county, month-by-year, and state-by-month fixed effects; flexible controls for temperatures, precipitation, and wind speed; two leads of the weather controls; and two leads and lags of the instruments. Estimates are weighted by the number of Medicare beneficiaries in Panel A and by the number of FFS beneficiaries in Panel B.

Table 7: LIML IV estimates of effect of PM 2.5 on elderly mortality, by age group

	(1) 65+	(2) 65-69	(3) 70-74	(4) 75-79	(5) 80-84	(6) 85+
PM 2.5 ($\mu g/m^3$)	0.607***	0.264***	0.313***	0.308***	0.777***	2.055***
	(0.066)	(0.071)	(0.075)	(0.107)	(0.178)	(0.265)
F-statistic	241	232	236	242	248	256
Dep. var. mean	393	138	205	326	531	1,170
Observations	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text when using the LIML estimator instead of the 2SLS estimator. Standard errors (in parentheses) clustered by county. All dependent variables are 3-day mortality rates per million beneficiaries in the relevant age group. All regressions include county, month-by-year, and state-by-month fixed effects; flexible controls for temperatures, precipitation, and wind speed; two leads of these weather controls; and two lags and two leads of the instruments. Estimates are weighted by the number of beneficiaries in the relevant age group.

Table 8: Placebo IV estimates of effect of PM 2.5 on elderly mortality, by age group

	(1) 65+	(2) 65-69	(3) 70-74	(4) 75-79	(5) 80-84	(6) 85+
PM 2.5 ($\mu g/m^3$)	-0.674	0.509	0.697	2.279	-2.736	-7.554**
	(0.672)	(0.840)	(1.056)	(1.384)	(1.898)	(3.260)
F-statistic	1.511	1.455	1.516	1.552	1.548	1.550
Dep. var. mean	393	138	205	326	531	1,170
Observations	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846	1,600,846

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text when using randomly generated placebo instruments. Standard errors (in parentheses) clustered by county. All dependent variables are 3-day mortality rates per million beneficiaries in the relevant age group. All regressions include county, month-by-year, and state-by-month fixed effects; flexible controls for temperatures, precipitation, and wind speed; two leads of these weather controls; and two lags and two leads of the instruments. Estimates are weighted by the number of beneficiaries in the relevant age group.

Table 9: Robustness of all-age mortality and life-years lost estimates to including fewer or more instrument lags

	(1) No lags	(2) 1 lag	(3) 3 lags	(4) 4 lags	(5) 5 lags
Panel A: mortality					
PM 2.5 ($\mu g/m^3$)	0.525***	0.655***	0.614***	0.611***	0.616***
	(0.078)	(0.065)	(0.066)	(0.065)	(0.065)
F-statistic	316	247	242	241	239
Dep. var. mean	394	394	393	393	393
Observations	1,624,689	1,612,384	1,590,074	1,579,878	1,570,025
Panel B: life-years lost					
PM 2.5 ($\mu g/m^3$)	2.589***	3.043***	2.751***	2.713***	2.714***
	(0.536)	(0.535)	(0.554)	(0.548)	(0.546)
F-statistic	309	244	238	237	236
Dep. var. mean	2,211	2,211	2,210	2,210	2,210
Observations	1,537,668	1,527,835	1,509,758	1,501,350	1,493,170

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text. Column headings report the number of instrument lags included in the regression. (The specification reported in other tables includes 2 lags.) Standard errors (in parentheses) clustered by county. Dependent variable in Panel A is the 3-day mortality rate per million beneficiaries. Dependent variable in Panel B is the life-years lost over 3 days per million continuously enrolled fee-for-service (FFS) beneficiaries. Estimates are weighted by the number of Medicare beneficiaries in Panel A and by the number of FFS beneficiaries in Panel B.

Table 10: Robustness of mortality IV estimates to including different fixed effects and weather controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PM 2.5 ($\mu g/m^3$)	0.382***	0.571***	0.244***	0.295***	0.372***	0.615***	0.649***	0.583***
	(0.043)	(0.066)	(0.046)	(0.047)	(0.042)	(0.065)	(0.066)	(0.066)
Type of weather controls	None	Separate	None	None	None	Full	Full	Full
County f.e.	X	X	X	X		X	X	
Month f.e.			X			X		
Year f.e.			X			X		
Year-by-month f.e.	X	X		X	X		X	X
State-by-month f.e.	X	X						
County-by-month f.e.					X			X
F-statistic	374	269	355	363	385	228	231	247
Dep. var. mean	394	394	394	394	394	393	393	393
Observations	1,602,889	1,602,889	1,602,889	1,602,889	1,602,860	1,600,846	1,600,846	1,600,817

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text when varying the inclusion of different weather controls and fixed effects. Standard errors (in parentheses) clustered by county. The dependent variable is the 3-day mortality rate per million Medicare beneficiaries. Estimates are weighted by the number of beneficiaries.

Table 11: Robustness of mortality IV estimates to different levels of aggregation for pollution monitors and wind angles

	(1)	(2)	(3)
PM 2.5 ($\mu g/m^3$)	0.615***	0.646***	0.612***
	(0.058)	(0.062)	(0.063)
Size of wind angle bins (degrees)	60	90	90
Number of monitor groups	100	50	200
F-statistic	158	452	142
Dep. var. mean	393	393	393
Observations	1,600,846	1,600,846	1,600,844

Significance levels: * 10 percent, ** 5 percent, *** 1 percent. Table reports IV estimates of equation (1) from the main text. The baseline specification reported in other tables aggregates pollution monitors into 100 groups and wind angles into 90-degree intervals. This table demonstrates that our estimates are not sensitive to the chosen level of aggregation. Standard errors (in parentheses) clustered by county. The dependent variable is the 3-day mortality rate per million Medicare beneficiaries. Estimates are weighted by the number of beneficiaries.

APPENDIX FOR ONLINE PUBLICATION ONLY

Relationship between wind direction and PM 2.5

We illustrate the variation that drives our results by estimating the following regression separately for each of the 100 monitor groups described in the main text:

$$\text{PM2.5}_{cdmy} = \sum_{b=0}^{34} \beta_b WINDDIR_{cdmy}^{10b} + f(Temp_{cdmy}, Prcp_{cdmy}, WindSpeed_{cdmy}) + \alpha_c + \alpha_{sm} + \alpha_{my} + \epsilon_{cdmy} \quad \text{(A1)}$$

The variables are defined as in equation (2) of the main text. The estimates $\hat{\beta}_b$ are plotted in Appendix Figure A1. The Bay Area in California (cluster 88) and the Boston, MA area (cluster 9) are reproduced in Figures 1 and 2 from the main text.

These regression equation (A1) differs slightly from the first stage of the instrumental variable regressions estimated in the main text: (1) it employs 10-degree bins for $WINDDIR$ instead of 90-degree bins; (2) it does not include any leads or lags; and (3) it does not employ county weights. In addition, the large number of control variables included in equation (A1) causes estimation to be impossible for a small number of the monitor groups (see notes in Appendix Figure 1). This does not occur in the instrumental variable regressions we estimate in the main text because the first stage in those regressions is estimated simultaneously, not separately, for each monitor group. (This forces the estimated coefficients on the control variables to be constant across all groups, which increases statistical power.)

Medicare sample and mortality data

The baseline sample used in our analysis consists of all Medicare beneficiaries aged 65-100 and is derived from 100% Medicare enrollment information files for years 1999-2011.[1] These annual files include an observation for each beneficiary enrolled in Medicare for at least one day in that calendar year, whether enrolled in Traditional Medicare (fee-for-service) or Medicare Advantage. The enrollment files report a variety of demographic and enrollment variables, including unique beneficiary identifiers that can be used to link individuals over time; monthly indicators for Medicare eligibility; state, county, and ZIP code of residence based on the mailing address for official correspondence; and exact date of birth, date of death, and gender.

The vast majority of elderly living in the United States are enrolled in Medicare. The *Left Panel* of Appendix Figure 2 compares the size of our baseline Medicare sample to Census estimates of the U.S.

[1] The Research Data Assistance Center (ResDAC) provides a helpful overview of the Medicare enrollment information files at http://www.resdac.org/training/workshops/intro-medicare/media/3.

population age 65 and over. To aid comparison, we use Census estimates of the resident population on July 1 each year and limit the Medicare sample to beneficiaries who reside in the 50 states and the District of Columbia and who turned 65 before July 1. Over the period 1999-2011, the Census estimates an average of 37.3 million elderly individuals each year, compared to 36.2 million elderly in Medicare. Thus, the Medicare sample covers over 97% of elderly living in the U.S., a share which remains roughly constant over the sample period.

The mortality variables used in our analysis are based on dates of death recorded in the Medicare enrollment files. Medicare's death data come primarily from the Social Security Administration but are augmented based on reviews triggered by hospitalization claims indicating patient death. The annual mortality rates in the Medicare data align closely with mortality rates based on National Vital Statistics death records and Census population estimates, as shown in the *Right Panel* of Appendix Figure 2. While all recorded deaths in the Medicare data are validated, some death *dates* in the data are not validated, in which case they are assigned the last date in the month of death. Because much of our analysis is performed at the daily level, we drop individuals who die at any point in the year and who do not have a validated death date flag. This restriction affects less than 2% of the deaths in our sample, and the share of deaths with unvalidated dates diminishes over time (see Appendix Figure 2).

Estimating counterfactual life expectancy

We model counterfactual life expectancies for Medicare beneficiaries by estimating a semi-parametric Cox proportional hazards model.[2] This model assumes that the hazard rate of death for individual i can be factored into two separate functions:

$$h(t_i|x_i,\beta) = h_0(t_i)\exp[x_i'\beta]$$

The hazard rate at time t_i, $h(t_i|x_i,\beta)$, depends on the baseline hazard rate, $h_0(t_i)$, and on a vector of individual characteristics, x_i. The parameter vector β is estimated by maximizing the log partial likelihood function:

$$\ln L(\beta) = \sum_{i=1}^{N} \delta_i \left[x_i'\beta - \ln \sum_{j \in R(t_i)} \exp[x_j'\beta] \right] \quad \text{(A2)}$$

where the indicator variable δ_i is equal to one for individuals whose deaths we observe (uncensored observations) and equal to zero otherwise. The risk set $R(t_k) = \{l: t_l \geq t_k\}$ is the set of observations at risk of death at time t_k and consists of all individuals who are alive at that time. Thus, individuals whose deaths

[2] We have also estimated fully parametric models that assume survival rates are governed by either the Gompertz or Weibull distributions. Those results are very similar.

we do not observe (censored observations) affect the partial likelihood function only through the terms indexed by j in equation (A2).

Once $\hat{\beta}$ has been obtained by maximizing the log partial likelihood, we nonparametrically estimate the baseline hazard function following Breslow (1972):

$$\hat{h}_0(t_i) = \frac{d_{t_i}}{\sum_{j \in R(t_i)} \exp[x_j' \hat{\beta}]} \tag{A3}$$

The numerator, d_{t_i}, is the number of deaths that occur at t_i. The corresponding baseline survival function is calculated as

$$\hat{S}_0(t_i) = \exp[-\hat{H}_0(t_i)]$$

where $\hat{H}_0(t_i)$ is the cumulative hazard function, calculated as $\hat{H}_0(t_i) = \sum_{\tau=1}^{t_i} \hat{h}_0(\tau)$. The individual-specific survival function, which allows us to calculate life expectancy, can then be estimated as:

$$\hat{S}(t_i | x_i, \hat{\beta}) = \hat{S}_0(t_i)^{\exp[x_i' \hat{\beta}]}$$

In practice, the nonparametric estimate of the baseline hazard function is limited to the ten years of Medicare data we have available for this survival analysis. We extrapolate the baseline hazard function to future years by assuming it follows a log-linear form. As shown in Appendix Figure 3, this appears to be a very reasonable assumption.

We estimate the Cox proportional hazards model (A2) using data from the 2002 cohort of Medicare beneficiaries, which we observe beginning on January 1st, 2002. We observe all deaths that occur among this cohort on or before December 31, 2011. During this 10-year time period, 50 percent of our sample dies; the remaining deaths are censored.[3] To ensure that we have accurate measures of beneficiaries' chronic conditions, we limit the sample to Medicare beneficiaries who as of January 1, 2002 had been continuously enrolled in fee-for-service Medicare for at least two years. For computational ease, we further limit the analysis to a random 5 percent sample of these beneficiaries. The final estimation sample consists of 1,211,585 individuals.

The life-years lost analysis presented in the main text varies the set of individual characteristics included in the vector x_i in order to understand whether they affect the results. As described in the text, we estimate the survival model several times, using increasingly large sets of characteristics. Column (2) of Table 4 includes no characteristics; column (3) includes age and sex, and column (4) includes age, sex, and

[3] Although earlier cohorts are observable for a longer period of time, we do not use them because the Medicare variables denoting the presence of pre-existing chronic conditions, which are strong predictors of survival, are nonexistent or unreliable in earlier years.

indicators for 27 different chronic conditions. As we describe in detail below, column (5) utilizes a machine learning algorithm to optimally incorporate information from 1,062 variables. Including so many control variables creates two challenges. First, some variables may be significant predictors of survival for the 2002 cohort just by chance, even if they are not good predictors of survival in general. This may cause bias due to overfitting (Harrell et al. 1996). Second, computational limitations prevent us from including a large set of regressors when performing conventional maximum likelihood estimation on a large sample using standard numerical procedures.

Recent advances in machine learning techniques help us overcome these challenges and use all 1,062 variables when predicting individual-level life expectancies (Athey and Imbens 2016). One popular method is the Least Absolute Shrinkage and Selection Operator (LASSO) estimator (Tibshirani 1997).[4] LASSO can be implemented by maximizing a penalized version of objective function (A2):

$$\ln L(\beta) = \left(\sum_{i=1}^{N} \delta_i \left[x_i'\beta - \ln \sum_{j \in R(t_i)} \exp[x_j'\beta] \right] \right) - \lambda \sum_{i=1}^{k} |\beta_i| \qquad \text{(A4)}$$

where $|\beta_i|$ is the absolute value of β_i (where β_i is element i of the vector β) and k is the number of included regressors. We select the optimal penalty parameter λ using 5-fold cross validation.[5] We include the following 1,062 regressors (not including omitted categories) when estimating this model of life expectancy:[6]

1. Age in days as of January 1, 2002
2. Indicator variables for sex and for 7 different races
3. Indicator variables for the presence of the following 27 different chronic conditions as of December 31, 2001: acute myocardial infarction, Alzheimer's disease, senile dementia, atrial fibrillation, cataracts, chronic kidney disease, chronic obstructive pulmonary disease (COPD), heart failure, diabetes, glaucoma, hip/pelvic fracture, ischemic heart disease, depression, osteoporosis, rheumatoid arthritis, stroke, breast cancer, colorectal cancer, prostate cancer, lung cancer, endometrial cancer, anemia, asthma, hyperlipidemia, benign prostatic hyperplasia, hypertension, and hypothyroidism

[4] We also used other machine learning techniques like ridge regression and elastic net. The results are similar.
[5] See Simon et al. (2011) for a detailed discussion of the algorithm we employ to implement the Cox proportional hazards estimator with a LASSO penalty term. The efficiency of this algorithm allows us to estimate a survival model with both many regressors and a large number of observations.
[6] Variable names correspond to the descriptions given by ResDAC: http://www.resdac.org/cms-data/files/mbsf/data-documentation

    a. Indicator variables for all pairwise interactions of these 27 chronic conditions
4. Indicator variables for the interaction of 27 chronic conditions with 7 race indicators
5. Indicator variables for the interaction of 27 chronic conditions with sex
6. Indicator variables for 12 quantiles (10, 20, 30, 40, 50, 60, 70, 80, 90, 95, 99, 99.9) of the *beneficiary's* total prior year spending (i.e., spending that excludes payments made by Medicare)
    a. Indicator variables for the same 12 quantiles for each of the following 17 different categories of *total* prior year medical spending: hospice, home health care, hospital outpatient, acute inpatient, other inpatient, skilled nursing facility, ambulatory surgery center, Part B drugs, evaluation and management, anesthesia, dialysis, other procedures, imaging, tests, durable medical equipment, other Part B carrier, Part B physician
7. Indicator variables for various quantiles (listed in parentheses) of the total annual number of:
    a. Dialysis events (10, 30, 50, 70, 90)
    b. Home health visits, hospital outpatient emergency room visits (10, 30, 50, 70, 90, 95)
    c. Anesthesia events, hospital outpatient visits, other Part B carrier events, acute inpatient stays, durable medical equipment (10, 30, 50, 70, 90, 99)
    d. Part B drug events (10, 50, 70, 90, 99, 99.5)
    e. Other procedures events, evaluation and management events, imaging events, hospital outpatient emergency room visits, tests events, Part B physician events (10, 30, 50, 70, 90, 99, 99.5)
8. Fourth-order polynomials in each of 37 different variables that have been merged to the respondent's 5-digit ZIP code of residence. All variables are standardized so that they follow a normal distribution with mean 0 and variance 1. These zipcode-level data are obtained from the 2007-2011 and 2008-2012 American Community Surveys. The variables include data on the following categories (number of variables in parentheses if more than one): travel time to work (2), fraction below the poverty line (3), median household income, aggregate household income, aggregate household social security income, aggregate household retirement income, fraction in labor force, heating fuel sources (3), aggregate number of vehicles, median home value, fraction immigrant, gini index of household income, fraction with less than high school education, median year housing built, fraction on disability (2), fraction with hearing difficulties (2), fractions with vision difficulty (2), fraction with cognitive difficulty (2), fraction with ambulatory difficulty (2), fraction with self-care difficulty (2), fraction with independent-living difficulty (2), fraction with any health coverage, (2)and fraction with private health coverage (2).

The counterfactual life expectancy that forms the basis of the estimate in Column (5) of Table 4 is based on estimating (A4) when including the 1,062 regressors listed above.

The dashed lines in Appendix Figure 4 show the distribution of estimated counterfactual life expectancies for the subsample of Medicare beneficiaries who were used to estimate our survival model. The range of the distribution is wider when the model includes all 1,062 predictors (the dashed black line) than when it includes only age and gender as predictors (the dashed red line). The model based on age and gender corresponds to a typical life table and consists of only 68 $(= (100 - 67 + 1) \times 2)$ values. The maximum and minimum values in this table correspond to life expectancies for a 67-year-old female and a 100-year-old male, respectively. By contrast, the LASSO model generates a much larger set of predictions, some of which lie outside of the range of a basic life table.

The solid lines in Appendix Figure 4 show how the distribution of predicted values changes when it is limited to the subset of beneficiaries who died during the 2002 calendar year. The distribution produced by the model that includes only age and gender—given by the solid red line—shifts to the left because these decedents are older than the average Medicare beneficiary and thus have below-average life expectancies. The distribution for the LASSO model—given by the solid black line—shifts to the left even more. This indicates that beneficiaries who died within one year of January 1, 2002 were not only older than the average beneficiary in that year, but also they were less healthy than average, as captured by variables like prior medical spending and prior chronic conditions. Accounting for these additional variables reduces (on average) the prediction of the counterfactual life expectancies for these Medicare beneficiaries. This demonstrates that the Cox LASSO model that incorporates data from many variables generates predictions that are more accurate than a simple Cox model that accounts only for age and gender.

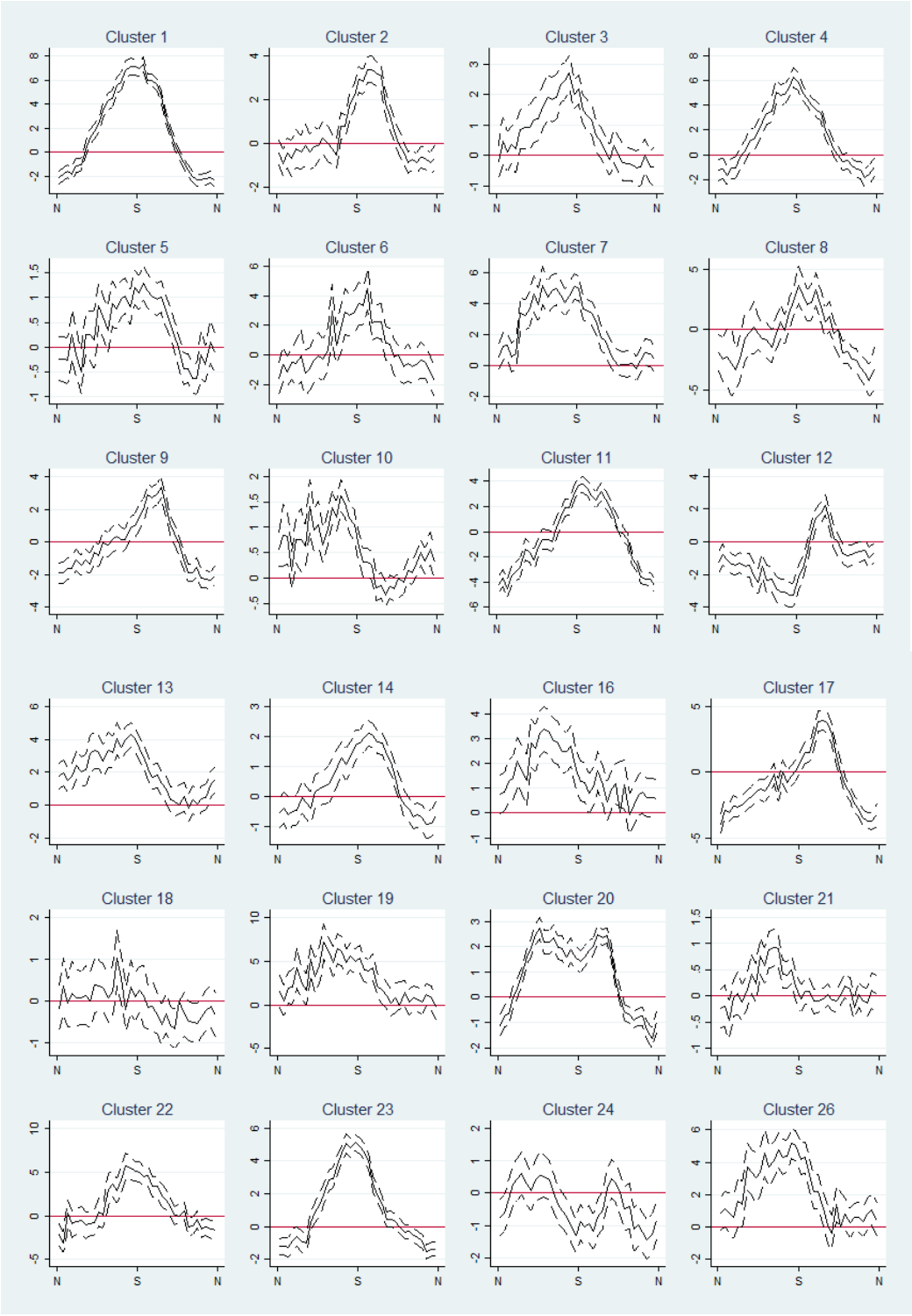
To further validate these estimates, we perform a similar exercise that incorporates Medicare data from individuals not included in our estimation sample. We first use the estimates from our model to predict life expectancy for Medicare beneficiaries as of January 1 of each calendar year. For each of these years, we then calculate the average life expectancy for all fee-for-service beneficiaries who die during that year ("decedents"). We focus on this group because these decedents form the basis of the life-years lost estimates reported in Table 4.

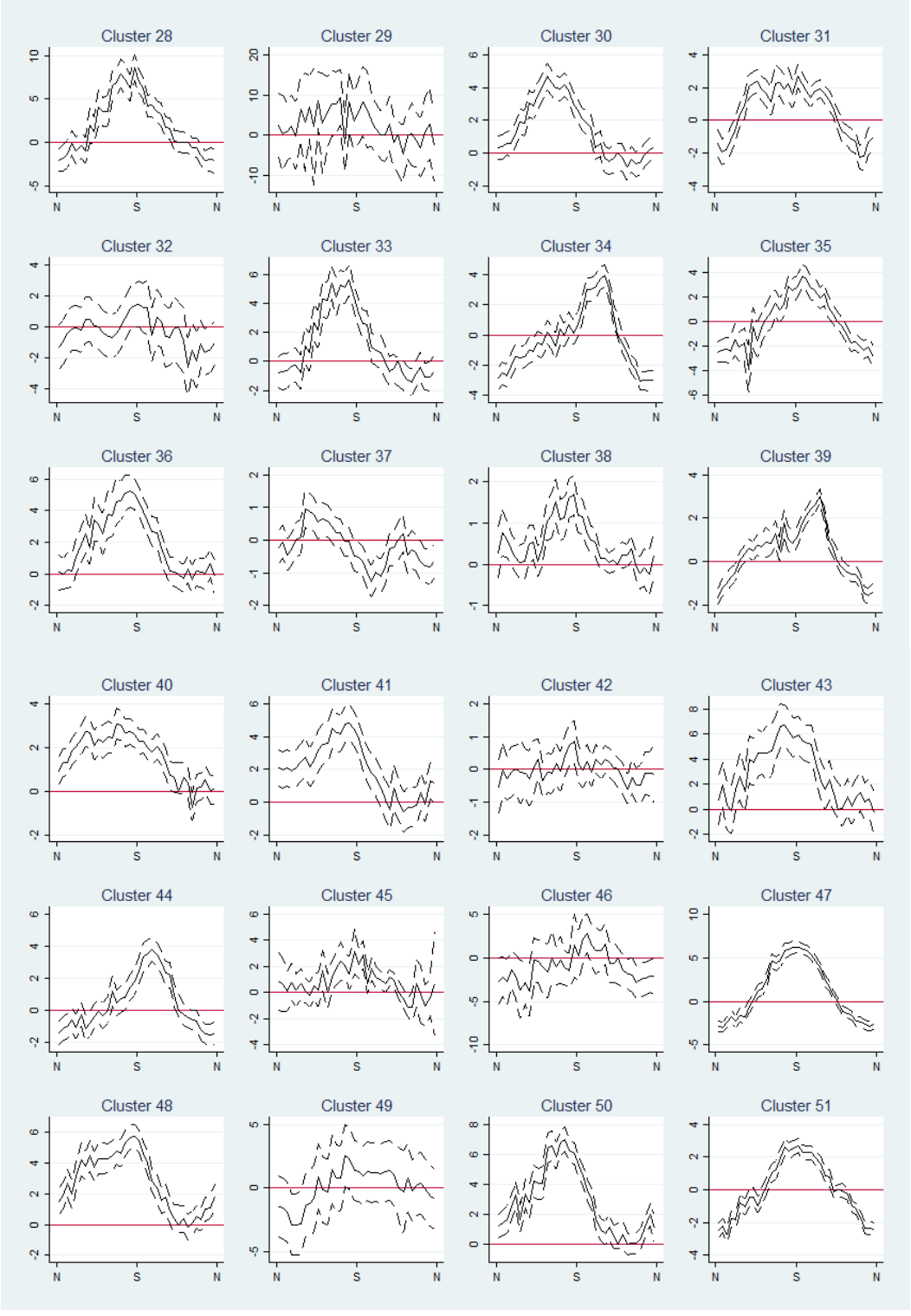
Appendix Figure 5 displays the results of this exercise. The solid green line, which serves as a baseline, displays our estimate of the unconditional life expectancy (11.6 years) for all Medicare beneficiaries. The solid red line displays the average life expectancy among decedents, as predicted by a Cox proportional hazards model that conditions on age and gender. Because the typical decedent is older than the average beneficiary, the predictions from this model are about 2.5 years lower than the baseline. This is clearly a more accurate prediction, since these decedents by definition died within one year of when their life expectancy was estimated. For the sake of comparison, we also include predictions based on a

period life table published by the Social Security Administration. Because that life table also conditions on age and sex, its predictions are nearly identical to those of the Cox model. Finally, the solid black line displays estimates based on the LASSO estimation of the Cox proportional hazards model with 1,062 regressors. This reduces the prediction by yet another 2.5 years. The estimates decline slightly over time, which likely reflects the improvement in the recording of chronic conditions in later years.[7]

[7] Medicare data on chronic conditions become increasingly incomplete in earlier years beginning in 2006. Because beneficiaries in these earlier years are less likely to have their chronic conditions recorded in the data, their estimated life expectancy is higher than beneficiaries in later years, who are more likely to have chronic conditions.

APPENDIX FIGURES

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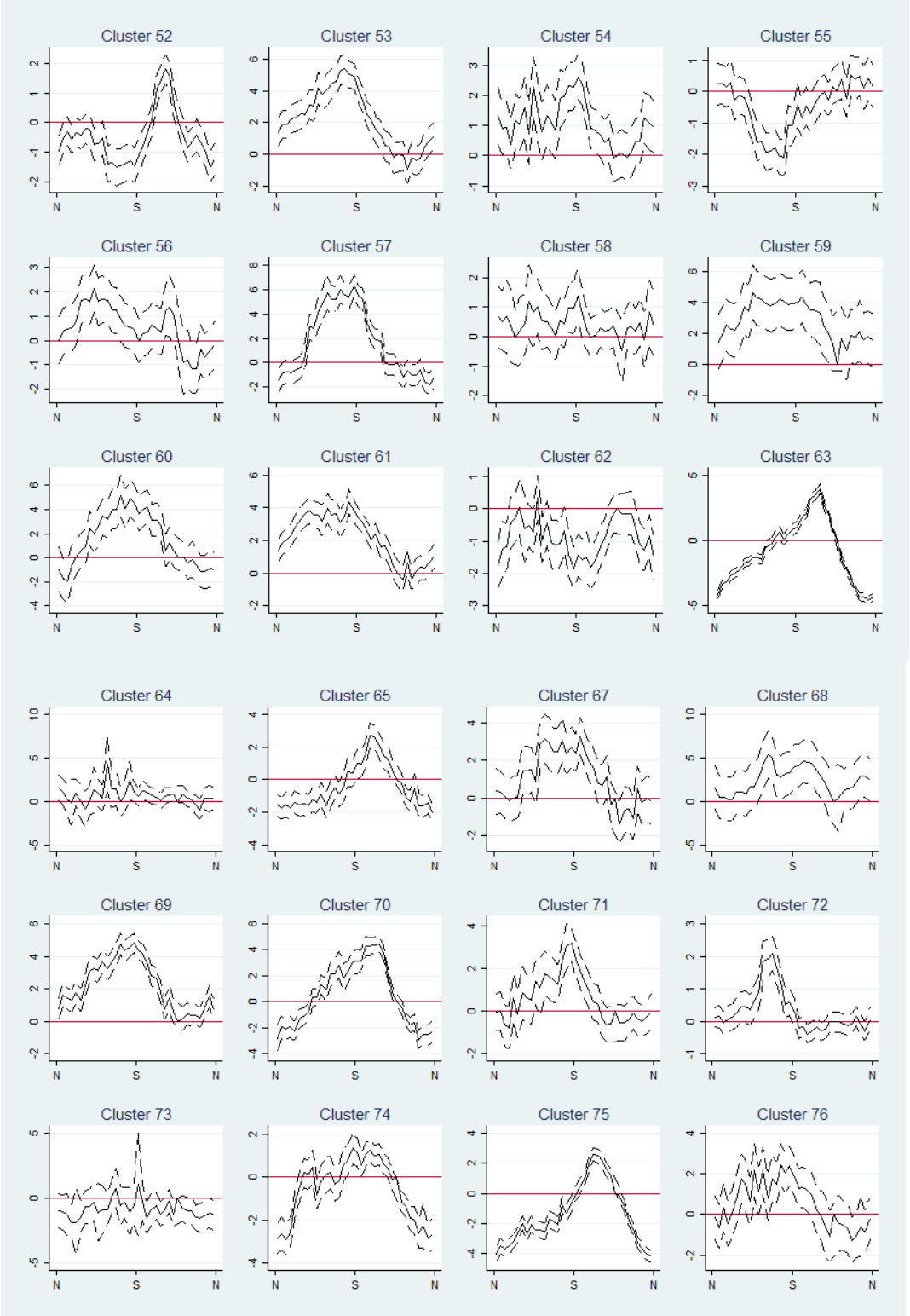
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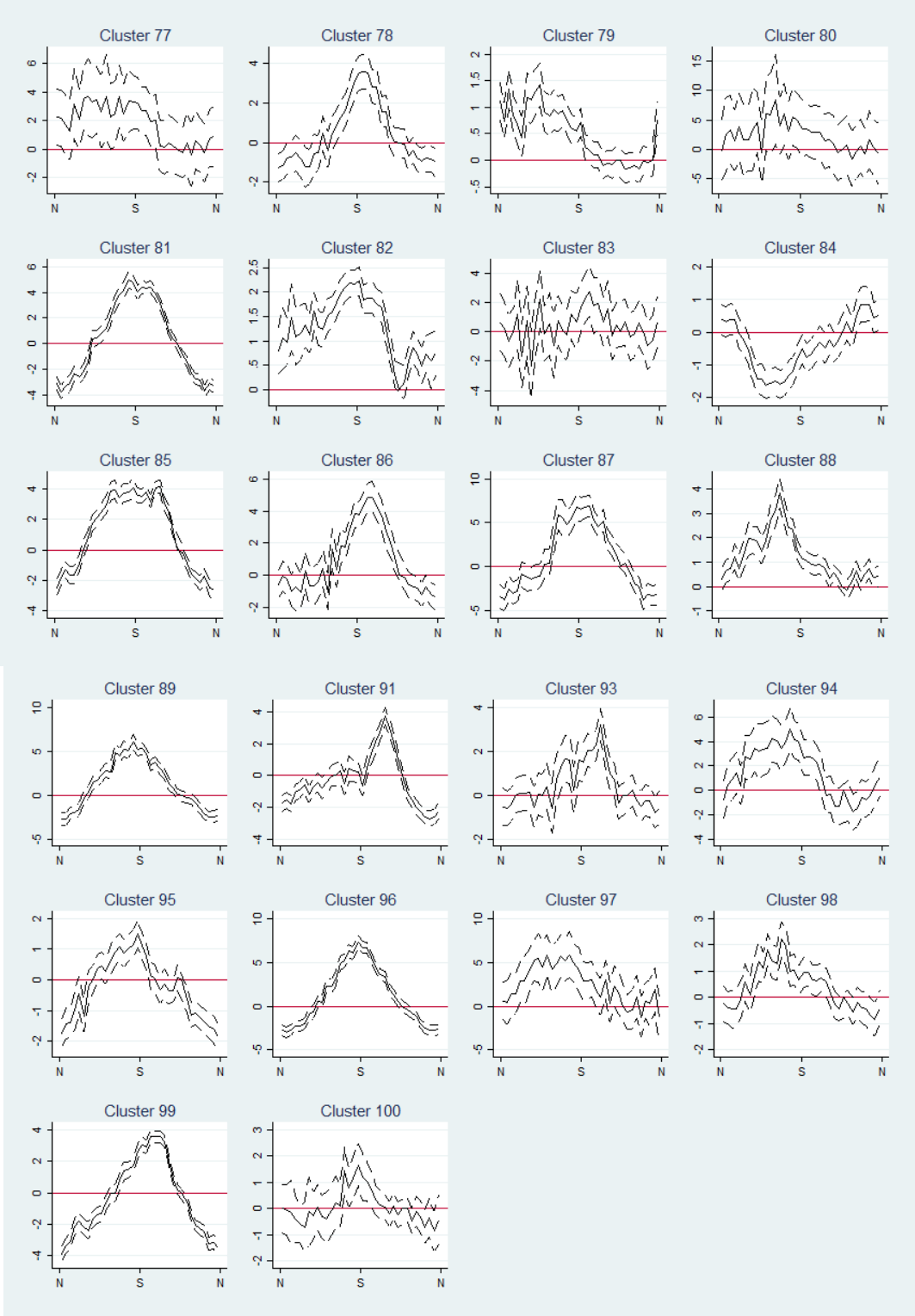
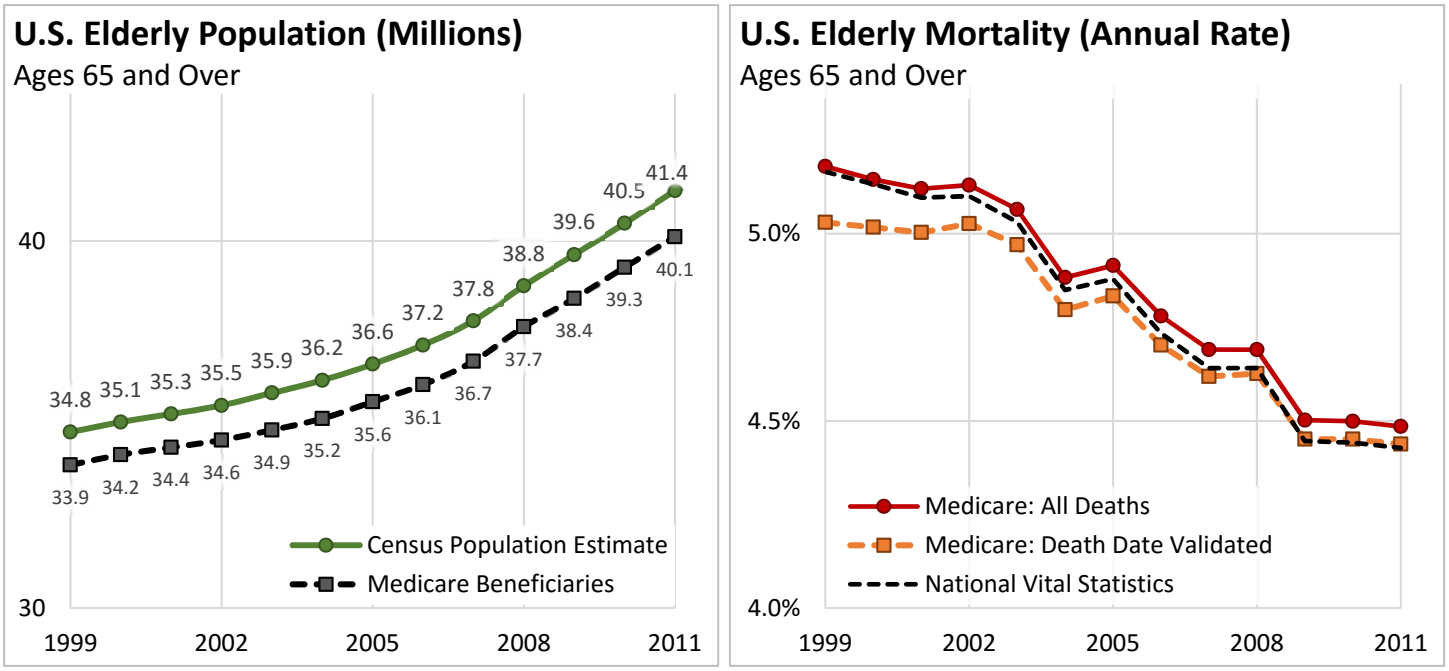


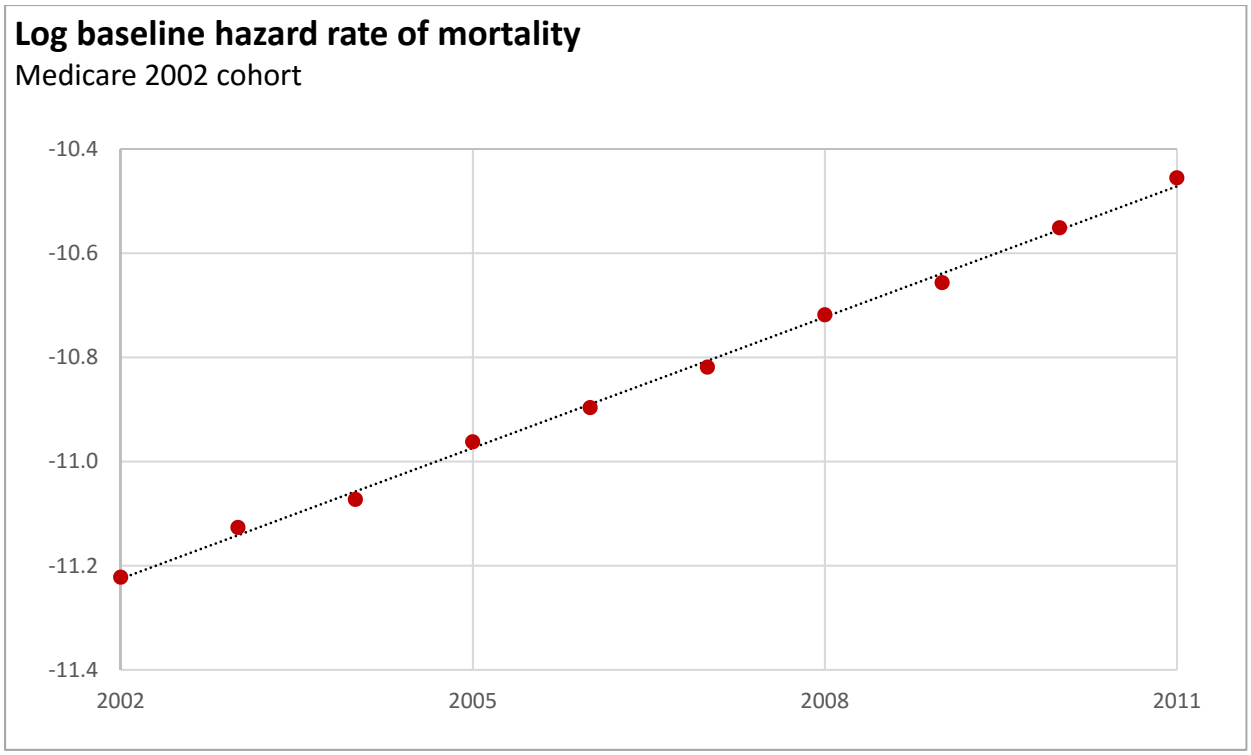
Appendix Figure 1. Relationship between wind direction and PM 2.5 concentrations for each monitor group. Pollution monitor groups with fewer than 1,000 PM 2.5 readings are not shown.



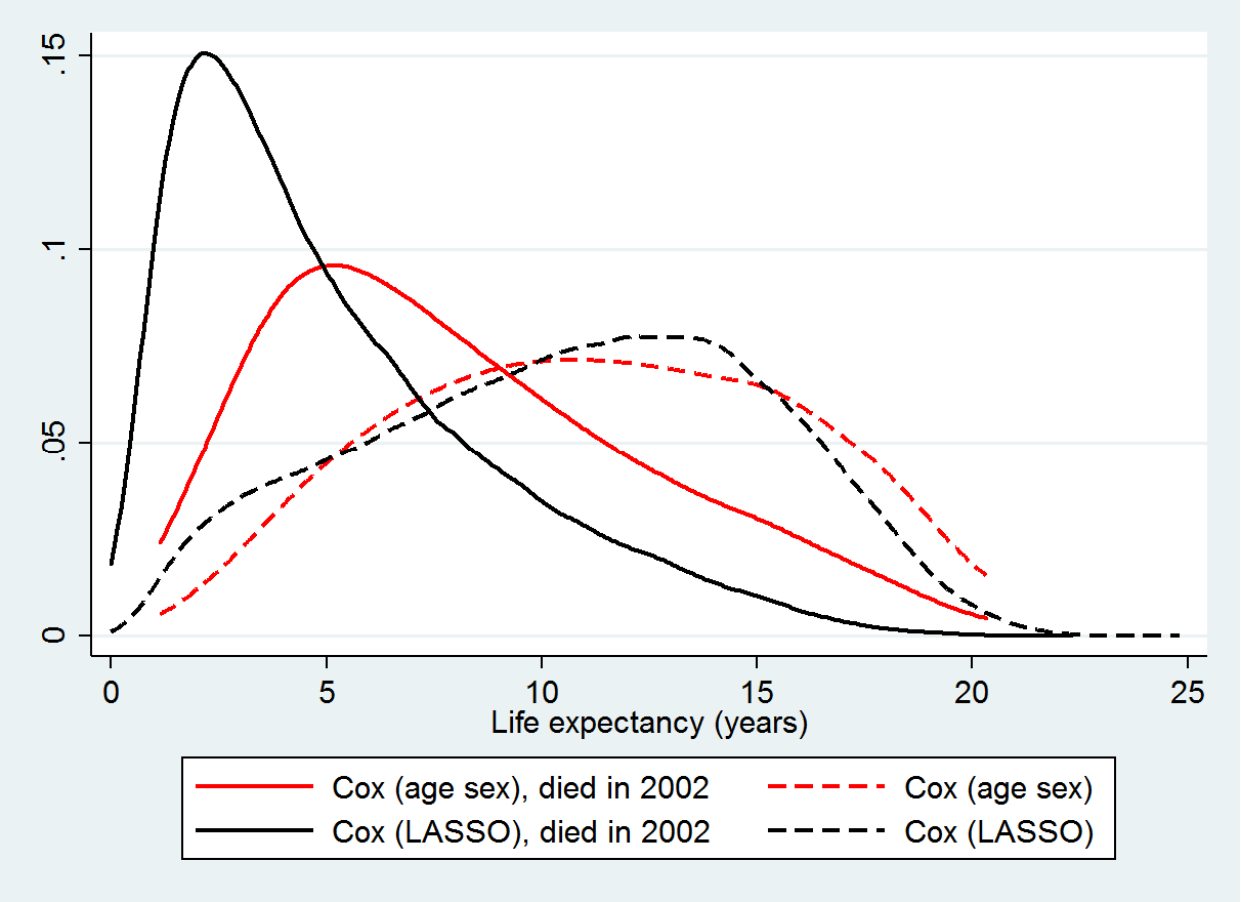
Appendix Figure 2. Population and Mortality Among U.S. Elderly, 1999-2011.

Left Panel: Census population estimates are derived from U.S. Census Bureau files. Estimates for 1999-2009 are intercensal estimates of the July 1 resident population age 65 and over; estimates for 2010-2011 are postcensal estimates of the July 1 resident population age 65 and over. Medicare beneficiaries for a given calendar year include all individuals age 65-100 in the corresponding annual Medicare enrollment file, limited to those who turned 65 before July 1 of the year and have a ZIP code of residence located in the 50 states or the District of Columbia.

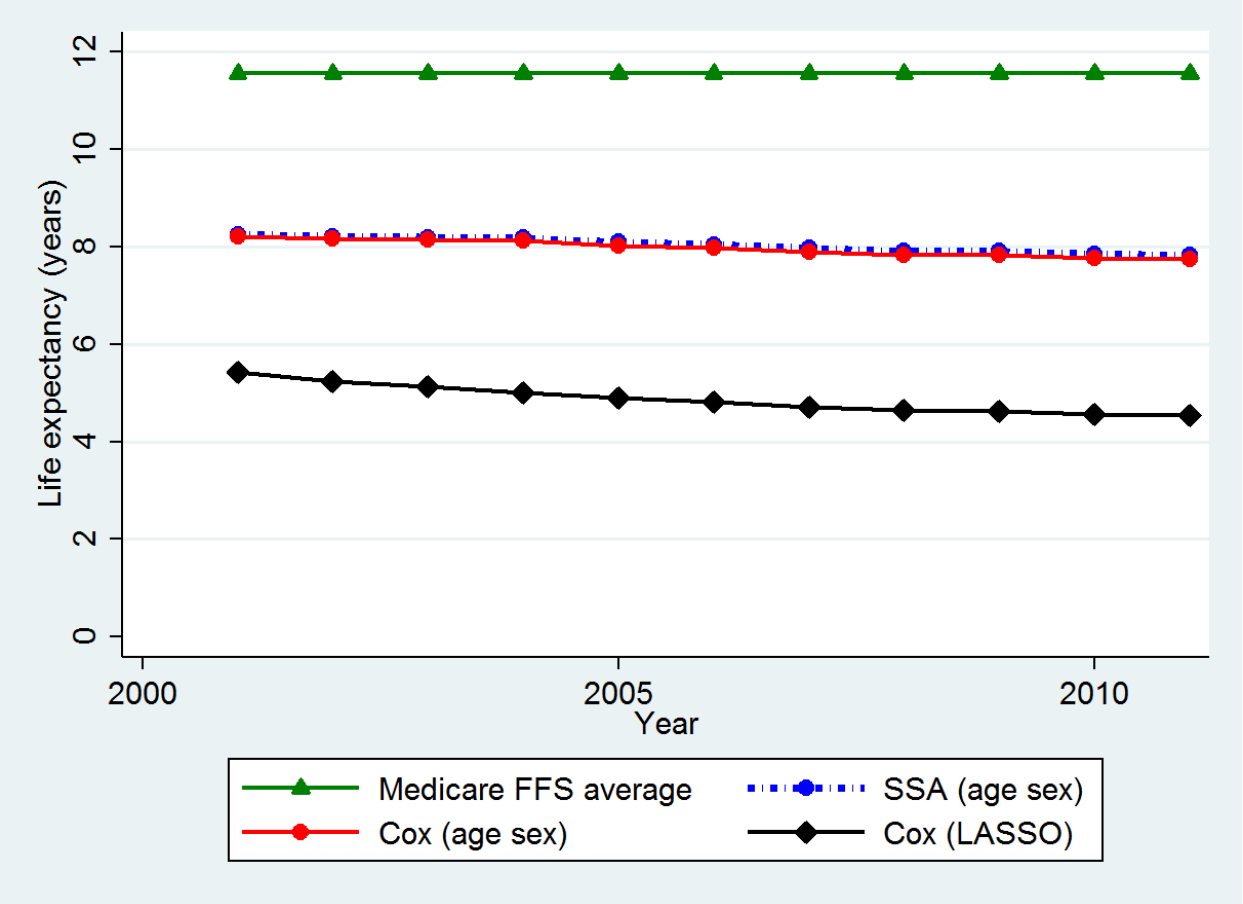
Right Panel: National vital statistics mortality data come from the Compressed Mortality File (CMF), which is produced by the National Center for Health Statistics and is based on death certificates filed in the 50 states and the District of Columbia. To obtain vital statistics mortality rates, we divide total CMF deaths among the 65 and over population in a given year by the Census population estimates shown in the *Left Panel*. The dashed lines report annual mortality rates based on death dates recorded in the Medicare annual enrollment files. The figure reports both the total mortality rate in the Medicare sample, as well as the mortality rate among the analytical sample used in the paper which excludes individuals who have a validated death that year but do not have a validated death *date* flag.



Appendix Figure 3. Log of the baseline hazard rate for the Medicare 2002 cohort. The red points in the figure correspond to the log of the baseline hazard rate of mortality for the Medicare 2002 cohort, as estimated by equation (A3) when using age and gender as controls. The coefficients on age and gender were estimated using the Cox proportional hazards model (A2). The figure also displays a dotted line of best fit.



Appendix Figure 4. Kernel density plot of life expectancy estimates for Medicare beneficiaries alive on January 1, 2002. The dashed lines display the distributions of life expectancy for all Medicare beneficiaries alive on January 1, 2002. The solid lines limit the distribution to the subset of those beneficiaries who later died during the 2002 calendar year. The red lines display estimates from a Cox proportional hazards model that includes only age and gender as regressors. The black lines display estimates generated by estimating model (A3) using LASSO with 1,062 regressors.



Appendix Figure 5. Average ex ante life expectancy for Medicare fee-for-service beneficiaries who later die within one year, by year. Estimates for "Medicare FFS average" are produced by estimating (A1) with no covariates. Estimates for "Cox (age sex)" are produced by estimating (A1) using only age and gender as predictors. Estimates for "Cox (LASSO)" are produced by estimating (A3) with 1,062 included regressors. Estimates for "SSA (age sex)" are obtained from the 2011 period life table for the Social Security area population (source: https://www.ssa.gov/oact/STATS/table4c6.html, accessed August 7, 2015).