# The Wind's Fatal Blow: the Effect of Transported Air Pollution on Mortality\*

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#### Abstract

We estimate the nationwide mortality effects of acute pollution exposure in the US over the period 1972–1988. Using wind direction as an instrument for changes in daily pollution levels, we estimate that a one-day increase in sulfur dioxide (SO<sub>2</sub>) exposure of one part per billion increases all-age mortality by 0.15 deaths per million over three days. Our estimated effects are largest for the elderly, but are also significant for infants and non-elderly adults. We also find that the marginal mortality effect of SO<sub>2</sub> is larger in counties that are richer and less polluted. Our results suggest that a uniform reduction in pollution levels would benefit high-income individuals more than low-income individuals and lead to higher mortality reductions in areas with lower pollution levels, all else equal.

JEL Classification: I18, Q52, Q53 Keywords: Air pollution, mortality, distributional effects

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## 1 Introduction

The World Health Organization (WHO) estimates that ambient air pollution caused 3 million premature deaths in 2012 (WHO, 2018). While such assessments are grounded in a large number of epidemiological studies, the studies themselves generally lack an exogenous source of pollution variation, casting doubts on the validity of their estimates.<sup>1</sup> Although quasi-experimental studies can overcome this identification challenge, they are typically based on a small subset of the population of interest. This makes it difficult to draw conclusions for the general population, especially if the marginal effects of air pollution depend on variables such as income, access to health-care, or existing pollution levels, which vary throughout the population and over time. Such variation also matters for understanding the distributional consequences of pollution regulation.

We use a novel source of exogenous variation-changes in the local wind directionto estimate the causal impact of acute sulfur dioxide (SO<sub>2</sub>) exposure on county-level mortality in the US, using national data from a 17-year period (1972–1988). To our knowledge, ours is the largest-scale study of acute pollution exposure on mortality, encompassing nearly 18 million deaths. We show that daily changes in the local wind direction are strongly correlated with changes in daily SO<sub>2</sub> concentrations, even after flexibly controlling for temperature, wind speed, and precipitation. Although other recent studies have combined a local pollution source – such as a highway, factory, or airport – with information on whether an area is upwind or downwind,<sup>2</sup> Deryugina et al. (2018) and this paper are the first to exploit such variation at the national scale. In addition, our large sample size enables us to separately estimate the effects

<sup>&</sup>lt;sup>1</sup>See Englert (2004) for a review, and Dominici, Greenstone and Sunstein (2014) for additional discussion.

<sup>&</sup>lt;sup>2</sup>See, for example, Anderson (2015); Hanna and Oliva (2015); Schlenker and Walker (2016); Knittel, Miller and Sanders (2016).

of multiple pollutants.

Our comprehensive data also allow us to investigate the distributional burden of air pollution, which depends not just on the relationship between pollution *levels* and income, but also on how the *marginal* effects of pollution are related to income. Specifically, we estimate how the marginal effect of  $SO_2$  varies as a function of average county-level income and pollution. Future versions of this paper will also include data from the years 1989–2015, enabling us to estimate contemporary relationships between marginal pollution damages, pollution levels, and income, and to investigate whether these relationships have changed over time.<sup>3</sup>

We find that a one-part-per-billion daily increase in  $SO_2$  (about 10 percent of the mean) raises overall mortality by 0.15 deaths per million over three days (0.20 percent of the mean). The largest absolute and relative increase occurs among those aged 65 and older: their mortality rate increases by 1.02 deaths per million (0.23 percent). Those aged between 1 and 64 also experience significant mortality increases of 0.035 deaths per million (0.14 percent). Infant mortality increases by a marginally significant 0.16 deaths per million. Our results are robust to including rich sets of control variables and fixed effects, and are not driven by other pollutants. Importantly, our IV results are up to ten times larger than the corresponding OLS estimates, suggesting that observational estimates from prior studies may significantly understate the health effects of pollution.

Surprisingly, we find that the marginal effect of  $SO_2$  rises with income and with the percentage of residents who are above the poverty line, implying that poorer individuals are less affected by acute pollution fluctuations. We also find that the mortality impacts of increases in  $SO_2$  fall with average  $SO_2$  levels, implying a con-

<sup>&</sup>lt;sup>3</sup>County-level daily mortality data during this time period are not publicly available, necessitating the use of a Census Research Data Center to extend the analysis to encompass this time period.

cave pollution-mortality relationship. To be clear, these results are descriptive, not causal: counties' pollution levels, incomes, and poverty rates are correlated with numerous other characteristics that could plausibly mediate or exacerbate the impact of pollution. Nevertheless, these results matter for understanding the distributional benefits of a given reduction in air pollution and imply that a uniform reduction would have relatively larger mortality benefits in areas that are richer and less polluted.

The Environmental Protection Agency (EPA) estimates that sulfur concentrations have fallen by about 87 percent between 1980 and 2016 (EPA, 2016). If we conservatively assume that each additional death results in just one year of life lost, then our estimates imply that the reduction in  $SO_2$  saved 780,000 life-years in 1972–1988. Adopting a conventional value of a statistical life year of \$100,000 (Cutler, 2004), the economic value of these mortality reductions is \$78 billion.

While several prior studies have investigated the effect of pollution on infant mortality (e.g., Chay and Greenstone, 2003; Currie and Neidell, 2005; Currie and Walker, 2011; Knittel, Miller and Sanders, 2016), the existing evidence for adult mortality is either limited geographically or lacks credible exogenous variation in pollution (e.g., Borja-Aburto et al., 1997; Laden et al., 2000; Chay, Dobkin and Greenstone, 2003; Moretti and Neidell, 2011). Important exceptions include Chen et al. (2013), who study the long-term mortality effects of particulate matter in China; Deschênes, Greenstone and Shapiro (2017), who estimate the mortality impacts of the US  $NO_x$ budget program; and Barreca, Neidell and Sanders (2017), who consider the long-run mortality effects of sulfur dioxide in the US. However, none of these studies considers heterogeneity in the pollution-mortality relationship, other than by age. We show that the heterogeneity along other dimensions is substantial, underscoring the need for nationally representative samples. Finally, researchers and policymakers are often interested in understanding whether low-income individuals are more vulnerable to pollution and whether the marginal impact of pollution falls or rises with increasing ambient concentrations (i.e., whether the pollution-mortality gradient is convex or concave). Our study provides these answers for sulfur dioxide and develops a general methodology that can be applied to other pollutants.

The rest of the paper is organized as follows. Section 2 provides a brief background on air pollution, describes the data we employ, and provides the intuition underlying our identification strategy. Section 3 discusses our empirical strategy in more detail. Section 4 presents and discusses the results, and Section 5 concludes.

### 2 Background and data

#### 2.1 Air pollution

Sulfur dioxide harms human health through two main channels. First, direct exposure to  $SO_2$  has a negative short-run effect on respiratory function, especially in people with asthma (Agency for Toxic Substances and Disease Registry, 1998). Second, sulfur dioxide frequently transforms into sulfate,  $SO_4(2^-)$ , which is a primary component of fine particulate matter (PM 2.5). Due to its own harmful health effects, PM 2.5 has increasingly become the focus of EPA air quality regulations. Unfortunately, fine particulate matter itself was not widely monitored until the late 1990's, so we cannot include it in our analysis.

Our air pollution data come from the EPA's Air Quality System database, which provides hourly data at the pollution monitor level for the six different criteria pollutants regulated by the EPA. The amount of spatial and temporal coverage depends on the pollutant. Our analysis focuses on the mortality impacts of sulfur dioxide (SO<sub>2</sub>), but we also consider three other air pollutants that have been widely monitored over the past several decades due to health concerns: nitrogen dioxide  $(NO_2)$ , ozone  $(O_3)$ , and carbon monoxide (CO).

Figure 1 displays the population-weighted concentrations and the number of monitored counties over time for each pollutant. Except for ozone, the population-weighted mean for all pollutants declines substantially during our sample period. CO data are readily available since the mid-1970's and maintain consistent coverage of approximately 225 counties per year, while ozone data are unavailable prior to 1980. Data on SO<sub>2</sub> and NO<sub>2</sub> are available for a larger number of counties than CO, although this spatial coverage declines beginning in 1976. Each year during our sample period, at least 400 counties monitor sulfur dioxide concentrations, and about 50 percent of US individuals live in a county that monitors SO<sub>2</sub>.

Panel A of Table 1 shows summary statistics for daily ambient pollution concentrations during 1972–1988. The average sulfur dioxide concentration during our sample period is 9.31 parts per billion, with a standard deviation of 12.6. The average levels of nitrogen dioxide and ozone are higher, at about 22 and 25.5 parts per billion, respectively. The most prevalent pollutant is carbon monoxide, with an average concentration of 1.64 parts *per million* (1,640 parts per billion). We are at least twice as likely to observe sulfur dioxide levels as any of the other three pollutants, which is one of the reasons we focus on  $SO_2$ .

We do not include data on total suspended particulates (TSP's) in our analysis for two reasons. First, TSP's include particles up to 100 micrometers in diameter, but the current scientific consensus is that the negative health effects are predominantly caused by small particles with diameters of less than 10 micrometers (PM 10), and particularly by those with less than 2.5 micrometers (PM 2.5). Indeed, although EPA regulated TSP's in the 1970's, current regulations apply only to those smaller particles. Second, both SO<sub>2</sub> and NO<sub>2</sub> are precursors to TSP's; adding TSP to the analysis would therefore complicate the interpretation of results.

#### 2.2 Wind and weather conditions

Our identification strategy is motivated by the fact that a lot of the air pollution in the United States is transported over long distances by wind currents. Figure 2 shows the fraction of PM 2.5 and sulfates-for which  $SO_2$  is a precursor-that can be attributed to local versus regional sources for thirteen large U.S. cities. In the vast majority of these examples, regional contributions to air pollution far exceed the local ones.

It is possible to explicitly model the transport of pollutants by wind currents and then use the predicted pollution as an instrument for changes in local pollution levels. But doing so is both computationally intensive, especially at the daily level, and requires fairly comprehensive data on emissions, which are largely not available during our study period. However, a valid instrument needs only to meet two criteria: a strong first stage and exogeneity with respect to the variable of interest. Thus, we take a much simpler approach and use changes in local wind direction as instruments for changes in  $SO_2$ . Our key identifying assumption is that, conditional on other climatic variables, wind direction is plausibly exogenous with respect to mortality. In this section, we provide evidence that wind direction is also a good predictor of changes in local pollution. (This will also be confirmed formally in the next section.)

We obtain wind speed and wind direction data from a 6-hour reanalysis dataset published by the Japan Meteorological Agency (JMA).<sup>4</sup> These data are available going back as far as 1958. They consist of vector pairs, one for the East-West wind direction (u-component) and one for the North-South wind direction (v-component), reported on a grid with a resolution of 1.25 degrees (about 86 miles). We first interpolate be-

<sup>&</sup>lt;sup>4</sup>Available from http://rda.ucar.edu/datasets/ds628.0/.

tween grid points in the original dataset to calculate the 6-hour u- and v-components at the location of each pollution monitor in the EPA data. We then calculate the average daily wind direction and wind speed to match the frequency of our mortality data. Specifically, we average the u- and v- components within a station-day and use trigonometry to convert the average component into wind direction and speed.

A potential threat to our identification strategy is that changes in wind direction may be correlated with certain other atmospheric conditions, which could themselves affect mortality. For this reason, we include flexible controls for daily temperature and precipitation in our estimation and probe the sensitivity of our results to their inclusion. We obtain daily temperature and precipitation from Schlenker and Roberts (2009). Combining monthly PRISM with daily data from weather stations, which are unevenly distributed throughout the US, Schlenker and Roberts (2009) derive a similarly spatially detailed weather map at the daily level. The final dataset spans the years 1950–2015 and includes total daily precipitation and daily maximum and daily minimum temperatures for each point on a 2.5 by 2.5 mile grid covering the contiguous United States.<sup>5</sup> To aggregate the gridded data to the county level, we simply average the daily measures across all grid points located in a particular county.

Figure 3 illustrates the variation we use to estimate the causal impacts of acute pollution exposure. The graphs show the relationships between daily average wind direction (in 10-degree bins) and  $SO_2$  in two states, Illinois and Massachusetts, after controlling for other temperatures, precipitation, wind speed, as well as county and month-by-year fixed effects in the same way we will control for them in our main specification.<sup>6</sup> The maps next to each graph show the locations of the included

<sup>&</sup>lt;sup>5</sup>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.

<sup>&</sup>lt;sup>6</sup>Appendix Figure A1 shows corresponding graphs for each mainland state.

monitors.

It is immediately apparent that there is a very strong relationship between wind direction and pollution in both places. Across all the monitors in Illinois, relative pollution levels are highest when the wind is blowing from the South and Southeast, where many coal power plants are located, and lowest when the wind is blowing from the West, Northwest, or North, a much cleaner area. In Massachusetts, the pattern is slightly different:  $SO_2$  levels are highest when the wind is blowing from the Southwest, a relatively industrial area.  $SO_2$  levels are substantially lower for many other directions, such as when the wind is blowing from the Northwest, North, Northeast, and East, areas that include the Atlantic Ocean and more sparsely populated states. Over long distances, it is common for wind patterns to be non-linear, so these estimates are not necessarily informative of the ultimate emission sources. However, they are generally consistent with patterns of industrial activity, lending credence to our identification strategy.

#### 2.3 Mortality and county-level characteristics

We obtain daily mortality data from the National Vital Statistics System of the National Center for Health Statistics. These data include counts of death, by county of occurrence, along with the cause of death and basic demographic information about the decedent such as age, gender, and race. We focus our analysis on the years 1972–1988 because those years contain data on the exact date of death.<sup>7</sup> We classify causes of death into four categories: cardiovascular, cancer, external, and "other". Cardiovascular disease is the leading cause of death, accounting for almost half of the deaths in our sample. Cancer deaths make up slightly over twenty percent of

 $<sup>^7\</sup>mathrm{The}$  exact date of death is unavailable prior to 1972, and is suppressed after 1988 for confidentiality reasons.

overall mortality. External causes of deaths are responsible for about eight percent of all deaths and include car accidents, poisonings, suicides, and other causes not originating in the body. The remaining twenty or so percent of deaths are grouped into the "other" category because none is large enough to warrant its own. The two largest components of the "other" category are chronic lower respiratory illnesses and diabetes.

To calculate death *rates*, we obtain annual intercensal population estimates from the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute. We calculate the death rate by dividing the count of deaths by the size of the relevant population. Figure 4 displays death rates by age group and by cause of death during the time period 1972–1988. Death rates are highest for the elderly. The infant mortality rate steadily declines over time, and nearly equals the average death rate of the population by the end of our sample period.

Panel B of Table 1 shows the average 3-day mortality rates for different subgroups over this time period. Across all ages and all causes, the death rate is 74 per million over any given 3-day period. The rate is higher for those aged 1 and under (99 per million) and substantially higher for those aged 65 and over (446 per million). Individuals between the ages of 1 and 65 die at much lower rates (about 25 per million).

The panel also shows mortality rates by major causes of death. Over a typical 3-day period, about 36 deaths per million were from cardiovascular causes. Cancer and "other" causes each accounted for 16 deaths per million, and the remaining 6 deaths per million are attributable to external causes.

We obtain county-level poverty rates from the U.S. Census Bureau for the years 1970, 1980, 1990, and 2000. Data on county-level median incomes come from the Regional Economic Information System (REIS), published by the Bureau of Economic Analysis. Finally, data on the share of the population that is black come from SEER. To simplify interpretation, we use 1970 values of these variables in our current analysis.

# 3 Using wind direction to estimate the effect of acute pollution exposure on mortality

We expect the effect of wind direction on pollution concentration to differ across space. For example, a westerly wind may be associated with high pollution levels in Pennsylvania, a state located just to the east of the manufacturing-heavy state of Ohio. A westerly wind in California, however, blows in from the Pacific Ocean and thus may be associated with relatively low pollution levels for that area.

Estimating the effect of wind direction on local pollution separately for each pollution monitor, however, may capture pollution transport *within* a county. Intuitively, pollution transport within a county may increase or decrease the measured pollution concentrations at the monitor while leaving the average person's pollution exposure unchanged. The first stage would then suffer from measurement error that leads to attenuation bias in the second stage. To avoid this problem, we allow the wind-pollution relationship to vary by state, using local measures of wind direction but restricting the coefficients on wind direction to be the same across all monitors within the same state. Specifically, we estimate the following first stage equation:

$$SO2_{cdmy} = \sum_{s=1}^{50} \sum_{b=0}^{2} \beta_{bs} 1[S = s] \times 1[WD_{cdmy} \subseteq [90b, 90b + 90)] + \sum_{l=0}^{2} f_{l}(Temp_{c(d+l)my}, Prcp_{c(d+l)my}, WS_{c(d+l)my}) + \sum_{l=-2, l \neq 0}^{2} \sum_{s=1}^{50} \sum_{b=0}^{2} \gamma_{bsl} 1[S = s] \times 1[WD_{c(d+l)my} \subseteq [90b, 90b + 90)] + \alpha_{c} + \alpha_{sm} + \alpha_{my} + \varepsilon_{cdmy},$$
(1)

where c indexes counties, s indexes states, and d, m, y are day, month, and year, respectively. The dependent variable,  $SO2_{cdmy}$ , is the daily average sulfur dioxide concentration, in parts per billion. The key independent variables are the 150 indicators formed by the interaction of state indicators, 1[S = s], and wind direction indicators,  $1[WD_{cdmy} \subseteq [90b, 90b + 90)]$  for b = 0, 1, and 2. The wind direction indicators are equal to 1 if the daily average wind direction falls in the 90-degree bin given by 90b and 90b + 90, and 0 otherwise. The choice of relatively coarse wind angle bins is driven by computational considerations, as increasing the number of bins increases computational time drastically. As we demonstrate in Section 4.2, however, our results are robust to increasing the number of wind direction bins.

We control for temperature,  $Temp_{cdmy}$ , using a set of indicators for the minimum or maximum temperature falling into a particular 3-degree Celsius range. The minimum and maximum bins capture temperatures below -15 degrees or over 30 degrees. We control for precipitation  $Prcp_{cdmy}$  and wind speed  $WS_{cdmy}$  with two sets of indicators for whether these climatic variables fall into one of ten deciles. The function f() then forms a set of all possible interactions of these atmospheric controls, allowing for tens of thousands of weather indicators. Again, our results are very stable when we vary the weather controls, supporting the validity of our instrumental variables strategy.

As we discuss below, our main outcome of interest is the death rate over a threeday period that includes the day of the pollution shock and the two subsequent days. It is thus important to control for all variables that could be correlated with our instruments and affect mortality over this time period. We control for two weather leads with  $f_l()$ , constructed in the same way as contemporaneous weather controls. We also control for two leads and lags of the instruments, which will capture any autocorrelation in wind direction. Finally, we also control for county, state-by-month, and month-by-year fixed effects ( $\alpha_c$ ,  $\alpha_{sm}$ , and  $\alpha_{my}$ , respectively). Similar to our other modeling choices, omitting the instrument lags and varying fixed effects does not change our results much.

Our second-stage specification follows naturally from the first-stage specification:

$$DeathRate3day_{cdmy} = \rho \widehat{SO2_{cdmy}} + \sum_{l=0}^{2} g_l(Temp_{c(d+l)my}, Prcp_{c(d+l)my}, WS_{c(d+l)my})$$
$$+ \sum_{l=-2, l \neq 0}^{2} \sum_{s=1}^{50} \sum_{b=0}^{2} \theta_{bsl} \mathbb{1}[S = s] \times \mathbb{1}[WD_{c(d+l)my} \subseteq [90b, 90b + 90)]$$
$$+ \alpha_c + \alpha_{sm} + \alpha_{my} + \epsilon_{cdmy}.$$
$$(2)$$

The variable  $DeathRate3day_{cdmy}$  is the 3-day mortality rate, calculated as the number of deaths on days d, d + 1, and d + 2 per million people. We also consider death rates of infants (<1 years old), those between the ages of 1 and 64, those who are 65 and older, and deaths due to different causes. The key coefficient of interest is  $\rho$ , which measures the effect of daily average sulfur dioxide concentration,  $\widehat{SO2}_{cdmy}$ , on the mortality rate. The other control variables are the same as those shown in

equation (1). Standard errors are clustered by county, and the regression is weighted by county-year population.

While we focus our attention on  $SO_2$ , our empirical approach does allow us to instrument for multiple pollutants simultaneously. If there is sufficient variation in the pollutant-wind relationship across pollutants, we can identify the acute mortality impacts of each one. The cost of doing so, however, is a greatly reduced sample size because many counties do not monitor all criteria pollutants at the same time. The smaller sample size, in turn, reduces the power of our instruments and, more importantly, the generalizability of our estimates. Thus, we do not include multiple pollutants in our primary specification. Instead, we later perform a robustness check to ensure that our conclusions about  $SO_2$  are not driven by other pollutants.

We also analyze how the marginal effects of pollution vary by income and pollution levels. To capture variation in the marginal effects of  $SO_2$  along these dimensions, we augment equations (1) and (2) with an interaction of the  $SO_2$  variable and each county's 1970 poverty rate, per-capita income, or average  $SO_2$  levels. To calculate each county's average pollution level, we average residuals from a regression of daily  $SO_2$  levels in 1972–1988 on month and year fixed effects. Because each interaction includes daily  $SO_2$ , the resulting variables are also endogenous. We thus instrument for them using our state-specific wind direction instruments.

While our heterogeneity analysis will correctly estimate how the causal marginal effects of  $SO_2$  varies with each heterogeneity dimension of interest, it would be improper to attribute causality to the latter variable. For example, while we can estimate how the marginal effects of  $SO_2$  vary with income, we cannot conclude that the variation is *caused* by income differences as opposed to merely correlated with them. This is because we have quasi-experimental variation in daily  $SO_2$  changes, but not in the dimensions of heterogeneity that we study. Other unobservables that

are correlated with average pollution levels or with income, such as the general health of the population or the quality of hospitals and other local infrastructure, may ultimately be responsible for producing any observed heterogeneity. Nonetheless, such heterogeneity is informative about the incidence of various pollution control policies, at least in partial equilibrium.

### 4 Results

#### 4.1 Effects of changes in short-run SO<sub>2</sub> exposure on mortality

Table 2 reports OLS and IV estimates of the relationship between sulfur dioxide  $(SO_2)$ and 3-day mortality for different age groups. While most of the OLS estimates are statistically significant (Panel A), they are on average about ten times smaller in magnitude than the corresponding IV estimates (Panel B). The IV estimates indicate that a one part-per-billion increase in  $SO_2$  increases 3-day all-age mortality by 0.14 per million (0.20 percent of the mean). Infants and the elderly are most susceptible, both in absolute and relative terms, although our estimates for infants are only marginally significant. The mortality of those under 1 year of age increases by 0.16 per million (0.16 percent of the mean), while the mortality rate of those 65 and older increases by 1.02 per million (0.22 percent of the mean). Those between the ages of 1 and 64 see a much smaller, but still significant, increase of 0.04 per million. However, because their average mortality rate is low, the relative pollution risk (0.16 percent of the mean) is exactly the same as it is for infants. In our preliminary estimates for the time period 1972–2015, 3-day all-age mortality increases by 0.21 deaths per million. Infant mortality increases by 0.24 deaths per million, mortality for 1–64 year olds increases by 0.06 per million, while mortality for those aged 65 and older increases by 1.3 per million. These infant mortality results are significant at the 5% level. All other estimates are significant at the 1% level.

Table 3 shows the OLS and IV estimates by cause of death. Again, the IV estimates are nearly an order of magnitude larger than OLS. The IV estimates in Panel B suggest that about half of the overall increase in mortality in Table 2 is attributable to cardiovascular causes (0.075 deaths per million). The next largest increase is among categories classified as "other" (0.036 deaths per million), followed by cancer (0.026 deaths per million). Because it is impossible to develop and die from cancer in such a short time period, this result suggests that SO<sub>2</sub> is more likely to kill individuals whose bodies are already weakened by other conditions. Finally, we find a small but significant increase in external deaths (0.008 per million). While we lack detailed data to probe this result further, it could be due to behavioral responses to pollution (e.g., Currie et al., 2009; Neidell, 2009; Moretti and Neidell, 2011) or due to negative effects of pollution on cognitive function (e.g., Crüts et al., 2008; Fonken et al., 2011; Bishop, Ketcham and Kuminoff, 2017), which could lead to a small increase in the likelihood of death.

Next, we gauge the extent to which our findings could be driven by other pollutants by sequentially instrumenting for additional ones. To ensure comparability, we limit our sample to observations that have pollution readings for  $SO_2$ , nitrogen dioxide (NO<sub>2</sub>), ozone (O<sub>3</sub>), and carbon monoxide (CO). The results for all-age allcause mortality are shown in Table 4. Note that because the pollutants have different average concentrations and standard deviations, and CO is expressed in parts per million, the different coefficients are not directly comparable to each other. However, the emerging pattern is that the effects of  $SO_2$  are consistently significant and large.

A natural question is how long the decedents in our sample would have survived if they were not killed by acute pollution exposure. The answer has first-order policy implications: society may value saving someone who otherwise would live for only five more days differently than saving someone who would live for ten years. The idea that acute pollution exposure may primarily affect individuals who would have died soon anyway is called "mortality displacement" or "harvesting". To see whether short-run mortality displacement is a concern in our setting, we extend the time window over which we consider mortality, up to 21 days after the change in pollution exposure. To ensure that we are still capturing the impact of a one-time increase in  $SO_2$ , we control for the appropriate number of weather and instrument leads: four leads plus contemporaneous controls for 5-day mortality, six leads plus contemporaneous controls for 7-day mortality, and so on.

The results are shown in Figure 5. If short-term mortality displacement were an issue in our setting, we would expect the estimated mortality effects to decline over time, potentially all the way to zero. Instead, they are monotonically *increasing* from 0.15 deaths per million over three days to 0.26 deaths per million over ten days. The estimates appear to converge at this point, with minimal changes in the estimated mortality effects at 14 or 21 days. This convergence suggests that 10-day estimates are sufficient to capture the long-run effects of acute pollution exposure.

#### 4.2 Robustness

We now demonstrate the robustness of our results to various modifications of our main estimating equation. A key identifying assumption in our analysis is that changes in wind direction are unrelated to mortality except through their effects on pollution levels. This identifying assumption is violated if wind direction is correlated with certain weather patterns that affected mortality (e.g., high-temperature days) or with seasonal phenomena that are also correlated with mortality (e.g., summer months). While this identifying assumption is impossible to assess directly, we can probe it indirectly by testing the sensitivity of our estimates to different ways of controlling for temperature, precipitation, and wind speed, and to different fixed effects.

Table 5 shows how the estimated effect of  $SO_2$  on all-age all-cause mortality as we vary the fixed effects and the weather controls. In column (1), we do not generate interactions between temperature, precipitation, and wind speed indicators, instead including them separately. In column (2), we drop the weather controls entirely, and in column (3) we omit minimum temperature and wind speed. Regardless of the specification, our estimates are very similar. In columns (4)-(6), we vary the fixed effects. Replacing our county, month-by-year, and state-by-month fixed effects with county-by-year and state-by-month (column (4)), county and state-by-month-by-year (column (5)), or county-by-month-by-year fixed effects (column (6)) does not have a meaningful impact on our results.<sup>8</sup> Thus, we are not concerned that omitted variables along these dimensions are biasing our IV estimates.

We have demonstrated that wind direction is a strong predictor of pollution concentrations and that our first-stage F-statistic is large. For the sake of completeness, we also re-estimate the IV specifications in Tables 2 and 3 using Limited Information Maximum Likelihood (LIML), which is median unbiased even in the presence of weak instruments. Our results, shown in Table 6, are nearly identical to our main results, suggesting that our results do not suffer from weak instrument bias.

Relatedly, we generate random wind directions to see if the way we construct our instruments is susceptible to spurious correlation. We leave the other controls as in Equation (2) and use these placebo wind directions as instruments for  $SO_2$ . The results, shown in Table 7, are completely insignificant. Importantly, the first stage

<sup>&</sup>lt;sup>8</sup>We have also estimated specifications that include only county and year fixed effects and a few other variations. These produce very similar results and are not shown for the sake of brevity.

F-statistics are very low, demonstrating that our strong first stage is not spurious.

In Online Appendix Table A1, we show that are results are likewise insensitive to controlling for only one instrument lag and to excluding the instrument lags altogether. Finally, we also demonstrate the robustness of our mortality estimates to varying the number of wind direction bins, increasing them from four to six, nine, or twenty-four (Online Appendix Table A2).

#### 4.3 Heterogeneity analysis

Heterogeneity analysis is frequently used to understand underlying mechanisms and to identify groups that are most affected. For example, low-income individuals are less likely to have access to high quality healthcare than high-income individuals and may thus be more susceptible to pollution, all else equal. Table 8 shows the relationship between the marginal effect of  $SO_2$  and an area's wealth, as measured by the average income in 1970 (panel A) or by the percentage of residents who are below the poverty line in 1970 (Panel B). The all-age mortality results in column (1) are striking: areas with lower per capita incomes or a higher proportion of residents below the poverty line are *less* susceptible to increases in sulfur dioxide. Specifically, a one-percent increase in per capita income increases the marginal effect of one part of  $SO_2$  per billion by 0.32 deaths per million people, which is more than twice of the average effect in Table 2. A one percentage point increase in the percent of people above the poverty line raises the marginal effect of  $SO_2$  by 0.009 deaths per million, which corresponds to 0.101 deaths per million per standard deviation in the percent of people above the poverty line.

Both of these results are driven by the oldest age group (column (4) of Table 8). A one-percent increase in per capita income is associated with a 2.88 deaths-per-

million increase in the marginal effect of  $SO_2$  for those 65 and older. Similarly, a one percentage point (standard deviation) increase in the share above the poverty line raises the marginal effect of  $SO_2$  by 0.13 (1.46) deaths per million elderly. Thus, the average estimates in Table 2 mask substantial income heterogeneity.

It is often assumed that the pollution-health relationship is convex, with marginal damages from pollution increasing with pollution levels. One way in which heterogeneity analysis can shed light on that is by looking at the marginal effects of  $SO_2$  in places with relatively high versus relatively low pollution levels. Table 9 shows how the marginal effects of  $SO_2$  change with higher pollution levels. Here, we again obtain counterintuitive results: the marginal effects of  $SO_2$  on mortality are falling with  $SO_2$  (column (1)). Specifically, a one part-per-billion (standard deviation) increase in *average*  $SO_2$  concentrations is associated with a decline in the *marginal* effect of  $SO_2$  of 0.013 deaths per million (0.075 deaths per million).

We see similar patterns in the two oldest age groups where the interaction terms are significant at above the ten percent level (columns (3) and (4)). The magnitude is especially large for individuals aged 65 and older, with a one-standard-deviation increase in average  $SO_2$  levels being associated with a 0.60 deaths per million decline in the marginal effect of  $SO_2$ . If interpreted causally, these estimates imply that the pollution-mortality relationship is concave.

For infants (column (2)), higher average  $SO_2$  levels are associated with an *increase* in the marginal effect of  $SO_2$  of about 0.045 deaths per million (0.26 in terms of the standard deviation of mean  $SO_2$ ). This estimate is only marginally significant and the future addition of newer data should help establish whether it is spurious or not.

Next, we consider heterogeneity by  $SO_2$  and by income jointly (Table 10). Our estimates for how the marginal effects of  $SO_2$  change with income levels largely cease to be significant (an exception is the gradient with respect to percent above the poverty line for ages 65 and older). However, the point estimates are positive, and we are hopeful that with the addition of more data, we will obtain smaller standard errors. The gradient of the marginal effects of  $SO_2$  with respect to average  $SO_2$  levels looks very similar to when we do not control for income.

Of course, it is only appropriate to interpret these results as being caused by poverty rates or  $SO_2$  levels if there is no omitted variable that is correlated with (a) poverty rates, average incomes, or average pollution levels and (b) the marginal effect of  $SO_2$  on mortality. In Online Appendix Tables A3 and A4, we regress poverty rates and  $SO_2$  on various county characteristics and show that this is unlikely to be the case. For example, poorer counties also have *lower* average  $SO_2$  levels, a lower population density, a higher share of black, young (<1 year), and old (>64 years) residents in 1970 and, unsurprisingly, a lower per-capita income. Counties with higher  $SO_2$ levels have lower poverty rates but also lower per capita income. They have a higher population density, higher shares of young and old residents, but a lower share of black residents. Each of these variables is significantly and independently correlated with the heterogeneity dimension of interest and it is likely that many of them are also correlated with the marginal damages caused by  $SO_2$ .

Despite the fact that the correlations between the marginal effects of  $SO_2$ , income, and average pollution levels may not reflect a causal relationship, they still have important implications for how the mortality benefits of pollution reduction efforts are distributed. In particular, they show that there may be a tradeoff between efficiency and equity, as targeting richer and less polluted areas would produce the largest mortality reductions but also exacerbate existing inequality.

#### 4.4 Implications of the results

Between 1972 and 1988, population-weighted  $SO_2$  concentrations fell by about 10 parts per billion (Figure 1). We can use our results to estimate the mortality reduction benefits of this decrease in air pollution. In our calculation, we conservatively assume that each life lost to sulfur dioxide corresponds to one life-year lost, i.e., that individuals killed by  $SO_2$  would have only lived for one additional year in the absence of this shock.<sup>9</sup> This assumption also makes it easy for interested readers to re-scale our estimates by their preferred quantity of life-years lost. We focus on the sample of counties with  $SO_2$  monitors, as this is the population for which our estimates are likely to be a lower bound.

Figure 6 shows the estimated decreases in the annual number of deaths as a result of these pollution reductions, as well as the corresponding economic benefits, assuming that one life-year is worth \$100,000 and that one life-year is lost per death. Because no mortality reduction is truly permanent, these estimates should be interpreted as the number of deaths *delayed* by at least one year (by exactly one year if each death corresponds to one life-year lost). As pollution concentrations are falling, the benefits are growing: our estimates imply that if SO<sub>2</sub> levels had remained at their 1972 levels, there would have been about 66,000 more deaths per year in 1987–1988, producing economic losses of \$6.6 billion per year. While 66,000 additional deaths may seem like a large number, it should be compared to the overall number of deaths per year in the sample of counties covered by SO<sub>2</sub> monitors.<sup>10</sup> Adding up the annual estimates, we conclude

<sup>&</sup>lt;sup>9</sup>Deryugina et al. (2018) estimate that the typical elderly individual killed by PM 2.5 has a counterfactual life expectancy of 3.6 years. Because our sample is on average much younger, assuming a counterfactual life expectancy of one year is thus very likely to be conservative.

 $<sup>^{10}</sup>$ If we assume that counties not covered by SO<sub>2</sub> monitors experience the same average reduction

that the 1972–1988 reductions in  $SO_2$  reduced the total number of deaths over this time period by 780,000, corresponding to economic gains of \$78 billion.

### 5 Conclusion

Accurate estimates of the effect of pollution exposure on health and mortality are vital for making informed policy decisions. Yet, reliable causal estimates remain scarce, especially for the non-infant population. We use daily average wind direction as an instrument for short-run changes in sulfur dioxide to estimate its causal effect on nationwide mortality over two decades. Our results show that naive OLS estimates are significantly biased, and that mortality among all age groups rises significantly over the three days following a one-day increase in  $SO_2$ .

We exploit our large sample size to estimate how the causal effect of sulfur dioxide varies by income and by a county's average pollution levels. Surprisingly, we find that the marginal impacts of  $SO_2$  are larger in richer and less polluted counties. This finding suggests a tradeoff between efficiency and equity, as targeting richer and less polluted areas would produce the largest mortality reductions yet also make the burdens of pollution less equal.

An important caveat is that the correlation between the marginal effects of pollution, income, and average pollution levels may have changed over time. We have begun analysis that will allow us to incorporate data from 1989–2015 in the next revision of this paper. We also plan to investigate whether these relationships have changed in recent years.

in  $SO_2$  concentrations, we would conclude that by 1988 there were almost 100,000 fewer deaths per year (out of 1.5 million deaths total), with annual benefits of almost \$10 billion.

### References

- Agency for Toxic Substances and Disease Registry. 1998. "Public Health Statement for Sulfur Dioxide."
- Anderson, Michael L. 2015. "As the Wind Blows: The Effects of Long-Term Exposure to Air Pollution on Mortality." NBER Working Paper No. 21578.
- Barreca, Alan I, Matthew Neidell, and Nicholas J Sanders. 2017. "Long-Run Pollution Exposure and Adult Mortality: Evidence from the Acid Rain Program." National Bureau of Economic Research.
- Bishop, Kelly C, Jonathan D Ketcham, and Nicolai V Kuminoff. 2017. "Hazed and Confused: Air Pollution, Dementia, and Financial Decision Making." mimeo.
- Borja-Aburto, Victor H., Dana P. Loomis, Shnkant I. Bangdiwala, Carl M. Shy, and Ramon A. Rascon-Pacheco. 1997. "Ozone, Suspended Particulates, and Daily Mortality in Mexico City." *American Journal of Epidemiology*, 145(3).
- Chay, Kenneth, and Michael Greenstone. 2003. "Air Quality, Infant Mortality, and the Clean Air Act of 1970."
- Chay, Kenneth, Carlos Dobkin, and Michael Greenstone. 2003. "The Clean Air Act of 1970 and Adult Mortality." *Journal of Risk and Uncertainty*, 27(3): 279–300.
- Chen, Yuyu, Avraham Ebenstein, Michael Greenstone, and Hongbin Li. 2013. "Evidence on the impact of sustained exposure to air pollution on life expectancy from China's Huai River policy." *Proceedings of the National Academy of Sciences*, 110(32): 12936–12941.
- Crüts, Björn, Ludo van Etten, Håkan Törnqvist, Anders Blomberg, Thomas Sandström, Nicholas L Mills, and Paul JA Borm. 2008. "Exposure to diesel exhaust induces changes in EEG in human volunteers." *Particle and fibre toxicology*, 5(1): 4.
- Currie, Janet, and Matthew Neidell. 2005. "Air Pollution and Infant Health: What Can We Learn from California's Recent Experience?" *Quarterly journal of economics*, , (3): 1003–1030.
- Currie, Janet, and Reed Walker. 2011. "Traffic Congestion and Infant Health: Evidence from E-ZPass." *American Economic Journal: Applied Economics*, 3(1): 65–90.

- Currie, Janet, Eric A Hanushek, E Megan Kahn, Matthew Neidell, and Steven G Rivkin. 2009. "Does pollution increase school absences?" The Review of Economics and Statistics, 91(4): 682–694.
- Cutler, David M. 2004. Your money or your life: Strong medicine for America's health care system. Oxford University Press.
- Deryugina, Tatyana, Garth Heutel, Nolan Miller, David Molitor, and Julian Reif. 2018. "The Mortality and Medical Costs of Air Pollution: Evidence from Changes in Wind Direction." NBER Working Paper 22796.
- **Deschênes, Olivier, Michael Greenstone, and Joseph S Shapiro.** 2017. "Defensive investments and the demand for air quality: Evidence from the NOx budget program." *American Economic Review*, 107(10): 2958–89.
- Dominici, Francesca, Michael Greenstone, and Cass R Sunstein. 2014. "Particulate matter matters." *Science*, 344(6181): 257–259.
- Englert, Norbert. 2004. "Fine particles and human health a review of epidemiological studies." *Toxicology Letters*, 149: 235–242.
- EPA. 2016. "Sulfur Dioxide Trends." Accessed February 6, 2018.
- Fonken, Laura K, Xiaohua Xu, Zachary M Weil, Guohua Chen, Qinghua Sun, Sanjay Rajagopalan, and Randy J Nelson. 2011. "Air pollution impairs cognition, provokes depressive-like behaviors and alters hippocampal cytokine expression and morphology." *Molecular psychiatry*, 16(10): 987.
- Hanna, Rema, and Paulina Oliva. 2015. "The effect of pollution on labor supply: Evidence from a natural experiment in Mexico City." *Journal of Public Economics*, 122: 68–79.
- Knittel, Christopher R, Douglas L Miller, and Nicholas J Sanders. 2016. "Caution, drivers! Children present: Traffic, pollution, and infant health." *Review of Economics and Statistics*, 98(2): 350–366.
- Laden, Francine, Lucas M. Neas, Douglas W. Dockery, and Joel Schwartz. 2000. "Association of Fine Particulate Matter from Different Sources with Daily Mortality in Six U.S. Cities." *Environmental Health Perspectives*, 108(10): 941–947.
- Moretti, Enrico, and Matthew Neidell. 2011. "Pollution, Health, and Avoidance Behavior: Evidence from the Ports of Los Angeles." *Journal of Human Resources*, 46(1): 154–175.
- Neidell, Matthew. 2009. "Information, avoidance behavior, and health the effect of ozone on asthma hospitalizations." *Journal of Human resources*, 44(2): 450–478.

- Schlenker, Wolfram, and Michael J. Roberts. 2009. "Nonlinear Temperature Effects indicate Severe Damages to U.S. Crop Yields under Climate Change." Proceedings of the National Academy of Sciences, 106(37): 15594–15598.
- Schlenker, Wolfram, and W Reed Walker. 2016. "Airports, air pollution, and contemporaneous health." *Review of Economic Studies*, 83(2): 768–809.
- WHO. 2018. "Ambient (outdoor) air quality and health."

# Figures



Figure 1: Air pollution and number of monitored counties, by year

Notes: This figure displays average daily pollution levels from 1972–2015 for all US counties with operational pollution monitors, along with the fraction of the US population covered by those counties. Data are obtained from the the EPA Air Quality database. Pollution data for CO,  $NO_2$ , and  $O_3$  end in 2014.



Figure 2: A lot of air pollution originates from distant sources, especially in the East

Notes: This figure, reproduced from EPA (2004), shows that a large fraction of measured PM 2.5 and sulfates does not originate from local sources. Sulfates, which are an important component of PM 2.5, are formed from the atmospheric transformation of sulfur dioxide.



Figure 3: The relationship between wind direction and  $SO_2$  concentrations

Notes: The left graphs plot coefficients from two regressions of sulfur dioxide on wind direction in 10-degree angle bins, using the samples of monitors displayed by the two maps on the right. The regressions include flexible controls for weather conditions, described in equation (1), county, stateby-month, and month-by-year fixed effects. The x-axis shows the direction from which the wind is blowing ("N" = North, "NE" = Northeast, "E" = East, and so on), while the y-axis shows the corresponding changes in SO<sub>2</sub>, in parts per billion, and the 95 percent confidence interval. Standard errors are robust to heteroskedasticity.



Figure 4: Trends in United States mortality rates, 1972-1988

Notes: These two figures report annual mortality rates from 1972-1988. These rates are calculated using mortality data from the National Vital Statistics and population data from SEER.



Figure 5: The effect of acute  $SO_2$  exposure on mortality over different time periods

Notes: Each point represents an estimate and 95% confidence intervals from an IV regression of the mortality rate over the given number of days on a 1-day change in SO<sub>2</sub>. Controls include county, month-by-year, and state-by-month fixed effects, two lags of the instruments, contemporaneous temperature and precipitation, as well as instrument, temperature, and precipitation leads. The number of leads in each regression is equal to the number of days minus one.



Figure 6: Annual mortality reduction benefits of 1972-1988 decreases in SO<sub>2</sub>

Notes: Estimates are constructed using the all-age IV mortality estimate from Table 2 and annual population-weighted  $SO_2$  concentrations. To construct our estimate of economic gains, we assume that each death results in a loss of one life-year and that each life-year is worth \$100,000. Both the mortality reductions and economic value of these reductions are per year. Only counties with  $SO_2$  monitors are included in this calculation.

# Tables

	(1)	(2)	(3)
	Mean	Standard deviation	Observations
	Panel A: Pollutio	n	
Sulfur dioxide, parts per billion	9.31	12.64	1,861,504
Nitrogen dioxide, parts per billion	22.06	15.79	706,968
Carbon monoxide, parts per million	1.64	1.36	842,841
Ozone, parts per billion	25.53	13.69	$666,\!801$
	Panel B: Mortalit	У	
All-cause, all-age	74.14	50.61	1,861,504
Age 1 and under	98.65	275.69	1,861,504
Age 1-64	25	26.97	1,861,504
Age 65 and older	446.22	301.66	1,861,504
Cardiovascular	36.44	30.61	$1,\!861,\!504$
Cancer	15.66	17.04	$1,\!861,\!504$
Other	16.45	18.59	$1,\!861,\!504$
External	5.59	13.47	$1,\!861,\!504$

### Table 1: Summary statistics

Sources: Environmental Protection Agency, National Center for Health Statistics, and National Cancer Institute. Sample restricted to observations where both mortality and sulfur dioxide are non-missing.

	(1) All ages	(2) <1 y.o.	(3) 1-64 y.o.	(4) $> 64$ y.o.
Pan	el A: OLS est	timates		
SO2, parts per billion	$0.015^{***}$ (0.006)	$0.013 \\ (0.019)$	$0.005^{***}$ (0.001)	$\begin{array}{c} 0.113^{***} \\ (0.034) \end{array}$
Dep. var. mean Effect relative to daily mean, percent Observations	$74.034 \\ 0.020 \\ 1,786,321$	$97.608 \\ 0.013 \\ 1,786,321$	$24.887 \\ 0.018 \\ 1,786,321$	$\begin{array}{c} 445.161 \\ 0.025 \\ 1,786,321 \end{array}$
Pa	nel B: IV esti	mates		
SO2, parts per billion	$0.144^{***} \\ (0.026)$	$0.164^{*}$ (0.099)	$0.035^{***}$ (0.009)	$\begin{array}{c} 1.017^{***} \\ (0.177) \end{array}$
F-statistic Dep. var. mean Effect relative to daily mean, percent Observations	$264.933 \\74.122 \\0.195 \\1,837,094$	$\begin{array}{c} 238.841 \\ 98.559 \\ 0.166 \\ 1,837,094 \end{array}$	$261.909 \\ 24.983 \\ 0.140 \\ 1,837,094$	295.755445.971 $0.2281,837,094$

Table 2: Daily	SO2	concentrations	and	mortality	across	age	group	$\mathbf{s}$
						()	· · · · · ·	

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people in the age group specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; and two leads of the weather controls. OLS estimates also include two leads and two lags of sulfur dioxide; IV estimates also include two leads and two lags of the instruments. Estimates are weighted by the number of people in the relevant age group.

	(1) Cardiovascular	(2) Cancer	(3) Other	(4) External
Par	nel A: OLS estin	nates		
SO2, parts per billion	$0.008^{**}$ (0.004)	$0.004^{***}$ (0.001)	$0.002 \\ (0.001)$	$0.001^{***}$ (0.001)
Dep. var. mean Effect relative to daily mean, percent Observations	$36.370 \\ 0.022 \\ 1,786,321$	$15.654 \\ 0.024 \\ 1,786,321$	$16.437 \\ 0.010 \\ 1,786,321$	$5.573 \\ 0.025 \\ 1,786,321$
Ра	anel B: IV estim	ates		
SO2, parts per billion	$0.075^{***}$ (0.015)	$0.026^{***}$ (0.005)	$0.036^{***}$ (0.006)	$0.008^{*}$ (0.004)
F-statistic Dep. var. mean Effect relative to daily mean, percent Observations	$264.933 \\ 36.401 \\ 0.207 \\ 1,837,094$	$264.933 \\ 15.673 \\ 0.165 \\ 1,837,094$	$\begin{array}{r} 264.933 \\ 16.458 \\ 0.217 \\ 1,837,094 \end{array}$	$264.933 \\ 5.590 \\ 0.135 \\ 1,837,094$

Table 3: Daily	I SO2	concentrations	and	mortality	across	causes	of	deat	h
•/				•/					

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people from the cause specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; and two leads of the weather controls. OLS estimates also include two leads and two lags of sulfur dioxide; IV estimates also include two leads and two lags of the instruments. Estimates are weighted by the number of people.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
SO2, parts per billion	0.160***	0.102***	0.151***	0.136***	0.099***	0.104***	0.099***
	(0.021)	(0.030)	(0.024)	(0.024)	(0.031)	(0.030)	(0.031)
NO2, parts per billion		$0.069^{***}$			$0.066^{***}$	$0.055^{*}$	0.045
Ozone parts per hillion		(0.022)	0.031		(0.021) 0.016	(0.029)	(0.028) 0.029
Ozone, parts per billon			(0.031)		(0.027)		(0.025)
CO, parts per million			· /	$0.819^{**}$	· /	0.338	0.491
				(0.357)		(0.439)	(0.467)
F-statistic	103.416	30.926	22.767	33.298	21.420	22.207	14.965
Observations	$274,\!269$	$274,\!269$	$274,\!269$	$274,\!269$	$274,\!269$	$274,\!269$	$274,\!269$
Dep. var. mean	26.190	78.573	78.573	78.573	78.573	78.573	78.573

Table 4: Other pollutants and mortality

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two leads of the weather controls. Estimates are weighted by the county population.

	(1)	(2)	(3)	(4)	(5)	(6)
SO2, parts per billion	$\begin{array}{c} 0.150^{***} \\ (0.024) \end{array}$	$0.120^{***}$ (0.019)	$\begin{array}{c} 0.152^{***} \\ (0.021) \end{array}$	$\begin{array}{c} 0.143^{***} \\ (0.024) \end{array}$	$\begin{array}{c} 0.141^{***} \\ (0.025) \end{array}$	$\begin{array}{c} 0.140^{***} \\ (0.025) \end{array}$
Weather controls	separate	none	no min. temp., no wind speed	full	full	full
Fixed effects	county, month- by-year, state-by- month	county, month- by-year, state-by- month	county, month- by-year, state-by- month	county- by-year, state-by- month	county, state-by- month- by-year	county- by- month- by-year
	200.000	241.000	200.000	017 515	000 570	004.007
F-statistic Dep var mean	289.223 74 126	$341.382 \\ 74.126$	$299.662 \\ 74.126$	$317.515 \\74.122$	$298.572 \\ 74.121$	$384.307 \\74 122$
Observations	1,838,847	1,838,847	1,838,847	1,837,075	1,837,062	1,836,566

Table 5: Robustness of estimates to different fixed effects and weather controls

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of 3-day deaths per million people. Controls and fixed effects are as specified in each column. All specifications include two leads and two lags of the instruments. Estimates are weighted by the total population.

	(1) All ages	(2) <1 y.o.	(3) 1-64 y.o.	(4) $>64$ y.o.
SO2, parts per billion	$0.145^{***}$ (0.026)	$0.165^{*}$ (0.100)	$0.035^{***}$ (0.009)	$\begin{array}{c} 1.023^{***} \\ (0.179) \end{array}$
F-statistic Dep. var. mean Effect relative to daily mean, percent Observations	$264.933 \\74.122 \\0.196 \\1,837,094$	$238.841 \\98.559 \\0.167 \\1,837,094$	$261.909 \\ 24.983 \\ 0.140 \\ 1,837,094$	$295.755 \\ 445.971 \\ 0.229 \\ 1,837,094$
	Cardiovascular	Cancer	Other	External
SO2, parts per billion	$0.076^{***}$ (0.015)	$0.026^{***}$ (0.005)	$0.036^{***}$ (0.006)	$0.008^{*}$ (0.004)
F-statistic Dep. var. mean Effect relative to daily mean, percent Observations	$264.933 \\ 36.401 \\ 0.208 \\ 1,837,094$	264.933 15.673 0.165 1,837,094	$264.933 \\ 16.458 \\ 0.218 \\ 1,837,094$	$264.933 \\ 5.590 \\ 0.136 \\ 1,837,094$

Table 6: Daily SO2 concentrations and mortality, LIML estimation

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people in the age group or from the cause of death specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the instruments; and two leads of the weather controls. Estimates are weighted by the number of people in the relevant age group.

	(1) All ages	(2) <1 y.o.	(3) 1-64 y.o.	(4) $> 64$ y.o.
SO2, parts per billion	-0.004 (0.179)	-1.569 (1.487)	$0.037 \\ (0.078)$	-0.284 (1.277)
F-statistic Dep. var. mean Effect relative to daily mean, percent Observations	2.103 74.034 -0.005 1,786,321	2.230 97.608 -1.608 1,786,321	2.132 24.887 0.147 1,786,321	$1.898 \\ 445.161 \\ -0.064 \\ 1,786,321$
	Cardiovascular	Cancer	Other	External
SO2, parts per billion	-0.019 (0.090)	$0.043 \\ (0.054)$	-0.067 (0.088)	$0.039 \\ (0.034)$
F-statistic Dep. var. mean Effect relative to daily mean, percent Observations	2.103 36.370 -0.052 1,786,321	2.103 15.654 0.275 1,786,321	$2.103 \\ 16.437 \\ -0.406 \\ 1,786,321$	2.103 5.573 0.695 1,786,321

#### Table 7: Placebo daily SO2 concentrations and mortality

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people in the age group of from the cause specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the placebo instruments; and two leads of the weather controls. Estimates are weighted by the number of people in the relevant age group.

	(1)	(2)	(3)	(4)					
	All ages	<1 y.o.	1-64 y.o.	>64 y.o.					
Panel A: using inc	Panel A: using income per capita as income measure								
SO2, parts per billion	-2.575***	-1.468	-0.576	-23.309***					
	(0.987)	(8.461)	(0.410)	(8.663)					
$SO2 \ge \log(\text{per capita income in } 1970)$	0.321***	0.189	0.072	2.877***					
	(0.117)	(1.005)	(0.049)	(1.030)					
F-statistic	85.601	79.212	84.999	92.344					
Dep. var. mean	72.519	97.029	24.303	437.659					
Observations	1,797,521	1,797,521	1,797,521	1,797,521					
Panel B: using percent	t above pover	ty line as incom	ne measure						
SO2, parts per billion	-0.714**	-3.463	0.178	-10.778***					
	(0.348)	(3.060)	(0.168)	(3.064)					
SO2 x % above poverty line in 1970	$0.009^{**}$	0.040	-0.002	$0.130^{***}$					
	(0.004)	(0.034)	(0.002)	(0.034)					
F-statistic	81.771	80.170	81.886	84.712					
Dep. var. mean	72.519	97.029	24.303	437.659					
Observations	1,797,521	1,797,521	1,797,521	1,797,521					

Table 8:	Heterogen	neity in	the effect	of SO2	by	income

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people in the age group specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the instruments; and two leads of the weather controls. Estimates are weighted by the number of people in the relevant age group.

	(1) All ages	(2) <1 y.o.	(3) 1-64 y.o.	(4) $> 64$ y.o.
SO2, parts per billion	0.209***	-0.049	0.058***	1.585***
$SO2 \ge average SO2$	(0.034) -0.013***	(0.138) $0.045^*$	(0.011) -0.005***	(0.237) -0.105***
	(0.004)	(0.025)	(0.002)	(0.026)
F-statistic	85.749	67.285	84.391	99.027
Dep. var. mean	74.122	98.559	24.983	445.971
Observations	$1,\!837,\!094$	$1,\!837,\!094$	$1,\!837,\!094$	$1,\!837,\!094$

Table 9: Heterogeneity in the effect of SO2 by average SO2 levels

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people in the age group specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the instruments; and two leads of the weather controls. Estimates are weighted by the number of people in the relevant age group.

	(1)	(2)	(3)	(4)		
	All ages	<1 y.o.	1-64 y.o.	>64 y.o.		
Panel A: using income per capita as income measure						
SO2, parts per billion	-1.378	-5.948	-0.115	-11.804		
	(1.036)	(8.030)	(0.435)	(8.307)		
$SO2 \ge \log(\text{per capita income in } 1970)$	0.184	0.698	0.019	1.560		
	(0.123)	(0.952)	(0.052)	(0.983)		
SO2 x average SO2	-0.010***	$0.045^{*}$	-0.004**	-0.081***		
	(0.003)	(0.026)	(0.002)	(0.023)		
F-statistic	68.652	63.681	68.744	68.398		
Dep. var. mean	72.519	97.029	24.303	437.659		
Observations	1,797,521	$1,\!797,\!521$	1,797,521	1,797,521		
Panel B: using percent above poverty line as income measure						
SO2, parts per billion	-0.434	-4.225	0.288*	-7.880**		
	(0.389)	(2.966)	(0.172)	(3.100)		
SO2 x % above poverty line in 1970	0.007	0.046	-0.003	0.102***		
	(0.004)	(0.033)	(0.002)	(0.034)		
SO2 x average SO2	-0.011***	0.044	-0.004**	-0.080***		
	(0.003)	(0.029)	(0.002)	(0.021)		
F-statistic	65.918	60.427	66.033	66.539		
Dep. var. mean	72.519	97.029	24.303	437.659		
Observations	1,797,521	1,797,521	$1,\!797,\!521$	1,797,521		

Table 10: Heterogeneity in the effect of SO2 by average SO2 levels

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of deaths over 3 days per million people in the age group specified by the column. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the instruments; and two leads of the weather controls. Estimates are weighted by the number of people in the relevant age group.

# **Online Appendix**

# **Appendix Figures**

Figure A1: The relationship between wind direction and pollution concentrations by state



















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Graphs show coefficients from state-specific regressions of sulfur dioxide on wind direction in 10degree angle bins. Regressions include flexible controls for weather conditions, described in equation (1), county, state-by-month, and month-by-year fixed effects. The x-axis shows the direction from which the wind is blowing ("N" = North, "NE" = Northeast, "E" = East, and so on), while the y-axis shows the corresponding changes in SO<sub>2</sub>, in parts per billion, and the 95 percent confidence interval. Standard errors are robust to heteroskedasticity.

## **Appendix Tables**

	(1)	(2)	(3)
	2 lags	$1 \log$	No lags
	Panel A: All-cau	use all-age mortality	
SO2, parts per billion	0.144***	0.151***	0.149***
	(0.016)	(0.016)	(0.018)
F-statistic	211.833	218.521	237.429
Dep. var. mean	73.897	73.906	73.915
Observations	1,776,618	1,788,048	1,799,476
	Panel B: Cardie	ovascular mortality	
SO2, parts per billion	0.074***	0.076***	0.076***
	(0.010)	(0.010)	(0.011)
F-statistic	211.833	218.521	237.429
Dep. var. mean	36.246	36.263	36.281
Observations	1,776,618	1,788,048	1,799,476

Table A1: Robustness of estimates to different numbers of instrument lags

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Robust standard errors in parentheses. Dependent variable is number of 3-day deaths per million people. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the instruments; and two leads of the weather controls. Estimates are weighted by the total population.

	(1)	(2)	(3)	(4)
SO2, parts per billion	$0.144^{***}$ (0.026)	$0.137^{***}$ (0.024)	$0.133^{***}$ (0.023)	$0.131^{***}$ (0.021)
Number of wind direction bins	4	6	9	24
F-statistic Dep. var. mean Observations	$264.933 \\ 74.122 \\ 1,837,094$	$186.126 \\ 74.034 \\ 1,786,321$	$\begin{array}{c} 128.197 \\ 74.034 \\ 1,786,321 \end{array}$	$\begin{array}{c} 49.594 \\ 74.034 \\ 1,786,321 \end{array}$

Table A2: Robustness of estimates to different wind direction bins

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Standard errors (in parentheses) clustered by county. Dependent variable is number of 3-day deaths per million people. All regressions include county, state-by-month and month-by-year fixed effects, as well as flexible controls for minimum and maximum temperature, precipitation, and wind speed; two leads and two lags of the placebo instruments; and two leads of the weather controls. Estimates are weighted by the total population.

	(1)	(2)	(3)	(4)
Mean SO2 levels, 1972-1988	0.35***	0.13***	0.14***	0.15***
	(0.04)	(0.04)	(0.04)	(0.02)
Population density (log)	$1.48^{***}$	2.49***	$2.18^{***}$	0.34**
	(0.16)	(0.15)	(0.15)	(0.16)
% black		-0.38***	-0.38***	-0.25***
		(0.02)	(0.02)	(0.02)
% aged below 1			-4.49***	-3.09**
			(1.58)	(1.24)
% aged 65 and older			-0.44***	-0.27***
			(0.07)	(0.05)
Per capita income (log)			· · ·	25.24***
. (0)				(1.32)
Observations	1,003	986	986	986
Adjusted R-squared	0.18	0.46	0.49	0.73

Table A3: Correlations between poverty levels and other characteristics

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Robust standard errors in parentheses. Dependent variable is the percentage of a county's 1970 population that lived above the poverty line. All independent variables are as of 1970 unless otherwise indicated.

	(1)	(2)	(3)	(4)	(5)	(6)
% above poverty line	0.23***	0.17***	0.10***	0.20***	0.11***	0.21***
	(0.02)	(0.02)	(0.03)	(0.04)	(0.04)	(0.04)
Population density (log)	× /	0.80***	1.07***	1.23***	1.14***	1.30***
		(0.15)	(0.21)	(0.22)	(0.19)	(0.21)
% black			-0.09***	-0.07***	-0.08***	-0.07***
			(0.02)	(0.02)	(0.02)	(0.02)
Per capita income (log)				-5.45***	( )	-5.56***
1 (0)				(1.31)		(1.31)
% aged below 1					$2.28^{*}$	$2.40^{*}$
6					(1.22)	(1.23)
% aged 65 and older					0.11**	0.11**
					(0.05)	(0.05)
					(	(1.00)
Observations	1,003	1,003	986	986	986	986
Adjusted R-squared	0.11	0.15	0.17	0.18	0.17	0.19

Table A4: Correlations between average SO2 concentrations and 1970 characteristics

Significance levels: \* 10 percent, \*\* 5 percent, \*\*\* 1 percent. Robust standard errors in parentheses. Dependent variable is county's average SO2 concentration between 1972-1988, adjusted for seasonal and temporal variation. All independent variables are as of 1970.