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THE NONLINEAR EFFECTS OF AIR POLLUTION ON HEALTH: EVIDENCE FROM WILDFIRE SMOKE

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ABSTRACT

We estimate how acute air pollution exposure from wildfire smoke impacts human health in the U.S., allowing for nonlinear effects. Wildfire smoke is pervasive and produces air quality shocks of varying intensity, depending on wind patterns and plume thickness. Using administrative Medicare records for 2007–2019, we estimate that wildfire smoke accounts for 18% of ambient PM2.5 concentrations, 0.42% of deaths, and 0.69% of emergency room visits among adults aged 65 and over. Smaller pollution shocks have outsized health impacts, indicating significant health benefits from improving air quality, even in areas meeting current regulatory standards.

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1 Introduction

Many countries regulate air pollution to safeguard human health. The prevailing regulatory approach, including the U.S. Clean Air Act's air quality standards, targets areas where pollution levels exceed certain limits, while areas with lower exposure face little regulatory scrutiny. This approach can be rationalized if the relationship between ambient pollution concentrations and health is convex, with minimal health impact expected below a "safety threshold" near established limits and health damages increasing more rapidly as concentrations rise above these limits. While economic and regulatory models commonly assume a convex pollution–health relationship, evidence confirming this shape remains scarce, even though it frequently appears in regulatory discussions and revisions of existing standards (Landrigan et al., 2018).

We study how the health effects of acute air pollution scale with exposure magnitude, using wildfire smoke as a natural experiment to assess impacts on adult mortality and hospital use in the U.S. Wildfires provide an important and useful setting for this analysis for several reasons. First, they are a large and growing source of fine particulate matter ($PM_{2.5}$), tiny inhalable particles widely regarded as the component of wildfire smoke most harmful to public health (Burke et al., 2021; U.S. EPA, 2021, 2024). Second, wildfire smoke plumes can drift hundreds of miles from their source, and their daily locations and densities are directly measured via satellite remote sensing, even in areas lacking ground-level pollution monitors (Ruminski et al., 2006). Third, and importantly for our study, these plumes can significantly elevate ambient pollution concentrations, with magnitudes that vary greatly depending on wind patterns and plume density.

Our analysis relies on nationally comprehensive and spatially granular daily records of wildfire smoke plumes, ambient air pollution concentrations, and mortality and hospital use among U.S. adults aged 65 and over from 2007 to 2019. Our empirical strategy relates daily smoke exposure to $PM_{2.5}$ and health outcomes, leveraging cross-year variations in smoke in a given county on the same day of the year. This strategy isolates the causal pollution–health

relationship by flexibly controlling for potentially confounding cross-sectional and seasonal differences. We first estimate $PM_{2.5}$ concentration and health impacts separately for small to large smoke exposures ("smoke shocks"). We then match, for each smoke shock, the resulting effect on $PM_{2.5}$ concentrations to the corresponding health response, tracing out the concentration–response (C–R) relationship describing how health effects vary with the magnitude of a pollution shock.

We present three main sets of findings. First, ground-level $PM_{2.5}$ concentrations sharply rise on days a county is exposed to a smoke shock. These effects vary widely depending on smoke proximity and density and persist for about three days. Counties near but not directly beneath a smoke plume see three-day $PM_{2.5}$ levels rise modestly by 0.5–3.2 $\mu g/m^3$ (5%–35% of the smoke-free daily mean), depending on the distance to the smoke. Counties under a plume experience larger impacts of 5.2–14.4 $\mu g/m^3$ (58%–158% of the smoke-free daily mean), depending on smoke thickness. Accounting for the frequency of smoke shocks and applying population weights, we estimate an average daily smoke impact of 1.65 $\mu g/m^3$, indicating that smoke accounts for about 18% of ambient $PM_{2.5}$ concentrations affecting the U.S. population.

Second, smoke shocks coincide with substantially elevated deaths and emergency room (ER) visits, with impacts persisting for roughly three days from the exposure date, aligning with the period of elevated $PM_{2.5}$ levels. We find no evidence of reduced mortality or ER visits in the following weeks, indicating that short-run intertemporal substitution does not explain the health impacts. We estimate that the average daily impact of smoke exposure results in an additional 0.51 deaths and 9.7 ER visits per million adults, accounting for about 1 out of every 240 deaths (0.42%) and 1 out of every 145 ER visits (0.69%) in the sample. When scaled to the population of 54.1 million U.S. residents aged 65 and over in 2019, these effects translate to an annual impact of 10,070 premature deaths and 191,541 excess ER visits.

Third, comparing how health impacts vary with the magnitude of the smoke shock reveals

a striking, non-monotonic pattern: health impacts steeply increase when moving from small to medium smoke shocks but level off and slightly decrease from medium to large shocks. Plotting these health impacts against the corresponding $PM_{2.5}$ impact of each smoke shock reveals a strongly concave C–R relationship: small air pollution shocks have greater health impacts per unit of $PM_{2.5}$ than larger shocks. The C–R curve flattens at about $6\,\mu g/m^3$ of $PM_{2.5}$ from smoke, beyond which larger shocks do not cause additional damages. We use two-stage least squares (2SLS) instrumental variables (IV) estimation with smoke shocks as instruments to recover the slopes along different portions of the C–R curves. $PM_{2.5}$ from larger shocks has smaller effects than $PM_{2.5}$ from smaller shocks, corroborating the concavity of the C–R relationship. On average, our IV estimates of the mortality effects of $PM_{2.5}$ align with those from Deryugina et al. (2019) using wind direction instruments, suggesting that smoke's health impacts primarily stem from $PM_{2.5}$ and are comparable to those from other sources.

A key insight from our analysis is that the marginal health cost of pollution shocks is downward-sloping, contrasting with the upward-sloping marginal cost curves commonly seen in other economic contexts. This result has important policy implications. Figure 1 illustrates the textbook example of public goods provision, where the socially optimal air pollution level depends on the trade-off between the marginal private benefits (e.g., industrial savings) of allowing an additional unit of pollution and its marginal costs (e.g., health damages). Our finding that the pollution–health relationship is concave, rather than convex, suggests that the optimal pollution level—the point at which the cost of further reducing pollution exceeds the benefit—may be lower than previously thought.

This paper estimates C–R relationships for mortality and hospital use among U.S. adults aged 65 and over, using a unified natural experiment and IV framework. Most prior studies that derive C–R relationships are observational, comparing how outcomes relate to different pollution exposure levels across places or contexts, such as comparing the effects of ambient pollution exposure to those of cigarette smoking (Pope III et al., 2009; Crouse et al., 2012;

Burnett et al., 2014). However, these comparisons can be prone to omitted variables bias and sensitive to the choice of statistical model and estimation techniques (Dominici, Greenstone and Sunstein, 2014). Numerous studies in economics have used natural experiments and IV strategies to provide causal estimates of pollution impacts, but they generally estimate a single average impact and do not identify the shape of the C–R relationship (Currie and Neidell, 2005; Currie, Neidell and Schmieder, 2009; Currie, 2013; Schlenker and Walker, 2016; Deryugina et al., 2019). One exception is Heft-Neal et al. (2023), who find a concave C–R relationship between ER visits and wildfire pollution in California. While comparisons of estimates from observational and IV research designs usually emphasize identification and measurement error issues, our finding of a nonlinear C–R relationship shows that estimated impacts can also differ substantially due to analyzing pollution variation at different levels (Ishimaru, 2024).

This paper also adds to a growing multi-disciplinary literature on how wildfire smoke impacts various aspects of human health and well-being, including physical health (Jayachandran, 2009; Rangel and Vogl, 2019; Cullen, 2020; Heft-Neal et al., 2022; Qiu et al., 2024), mental health (Molitor, Mullins and White, 2023), worker health and earnings (Borgschulte, Molitor and Zou, 2022; Cabral and Dillender, 2024), health protection behaviors (Burke et al., 2022), learning outcomes (Graff Zivin et al., 2020; Wen and Burke, 2022), and crime (Burkhardt et al., 2020) (see Bayham et al. (2022) for a recent review). Our paper is the first to analyze how smoke affects both mortality and hospital use, using daily observations at a national scale. The comprehensive and granular data enable us to explore nonlinearities in smoke impacts, compare the C–R relationships for mortality and hospital use, and quantify the aggregate burden of smoke on each domain.

Our research highlights several areas for further work. First, our C–R relationship quantifies the short-run health impacts of acute air pollution shocks. While prior studies have estimated that the long-run impacts of pollution exposure may exceed the short-run impacts (Chay, Dobkin and Greenstone, 2003; Chen et al., 2013; Ebenstein et al., 2017;

Deryugina and Reif, 2023), little is known about the shape of the long-term C–R relationship or the long-term impacts of smoke pollution specifically. Second, lower marginal damages at higher concentrations may emerge due to adaptation, such as defensive and avoidance behaviors (Deschenes, Greenstone and Shapiro, 2017; Ito and Zhang, 2020; Graff Zivin and Neidell, 2009, 2013; Barwick et al., 2024; Chen, Oliva and Zhang, 2022). Understanding the behavioral basis of the C–R relationship could provide insights into designing effective policies and fueling innovative solutions to improve health and resilience to pollution shocks.

2 Data and Measurement

Our main analysis sample is a balanced panel of all counties in the contiguous U.S. for 2007–2019. The sample consists of daily observations (about 14.7 million in total) with measures of wildfire smoke exposure, ground-level ambient air pollution concentrations, and health outcomes among adults aged 65 and older. We describe here the primary data sources and measures used in the analysis.

2.1 Wildfire Smoke

We use wildfire smoke plume data from the National Oceanic and Atmospheric Administration's Hazard Mapping System (HMS) for 2007–2019. HMS produces daily, satellite-based smoke analyses for North America, using infrared and visible band images. Expert analysts evaluate these images to identify significant smoke plumes and manually outline their extent and density with georeferenced polygons, categorizing densities as light, medium, or thick based on appearance.

Figure 2 compares satellite-observed and HMS-recorded smoke plumes, featuring the 2013 Rim Fire's impact in California and Nevada. Adjacent panels show daily HMS smoke plumes for August 19–24, with shades indicating smoke density. Before August 22, smaller fires spread smoke across the western U.S., a common pattern during the study period. On

August 22, the Rim Fire intensified sharply, burning over 100,000 acres and generating thick plumes that traveled hundreds of miles north, affecting distant areas in the following days.

Our primary smoke exposure measure comprises nine mutually exclusive and exhaustive categories that we call "smoke shocks." These shocks specify a county's proximity to the nearest wildfire smoke plume in bins from 0–100 km to >1,000 km, and the thickest density (light, medium, or thick) if a plume covers the county. We consider a county to be covered by, or within, a certain distance of a plume if at least 20% of its land area satisfies the condition. Counties are assigned to the >1,000 km distance bin on days with no smoke reported.

Figure 3a reports the frequency of each smoke shock category in the sample, weighted by county Medicare population. These frequencies represent the percentage of days older Americans are exposed to each shock type. Smoke exposure is common: older Americans are within 1,000 km of a smoke plume on 71.4% of days (nearly 3 out of every 4 days) and are directly covered by a plume (of any thickness) on 7.7% of days, or about 28 days annually. Figure A.1 shows the distributions of daily $PM_{2.5}$ concentrations for each shock category, showing a progressive increase in pollution with greater smoke intensity.

Figure 3b maps the average annual number of days each county is covered by smoke of any thickness, while Figure 3c breaks down smoke days by year, highlighting substantial annual variation in their number and geographic distribution. Smoke is pervasive across the U.S., particularly in the Midwest, where downwind emissions from western U.S. and Canadian wildfires contribute to an average of 50 or more smoke days annually in Minnesota, North Dakota, and Iowa. Southern regions, including eastern Texas, Louisiana, and the Florida Panhandle, also frequently experience smoke from local and Central American fires.

2.2 Air Pollution

We use ambient air pollution data from the EPA's Air Quality System, sourced from ground monitors. We focus on $PM_{2.5}$, particles with a diameter less than 2.5 µm ($PM_{2.5}$) that form a major component of wildfire smoke and are considered particularly harmful to human

health (U.S. EPA, 2021). We analyze other criteria pollutants and $PM_{2.5}$ species (chemical components) in supplemental analyses. Daily $PM_{2.5}$ concentrations for each county are calculated as the weighted average of 24-hour readings from monitors within 20 miles of the county centroid, weighted by the inverse distance. $PM_{2.5}$ is observed for 30% of the sample, covering 56% of counties and 89% of the Medicare population.

2.3 Mortality and Hospital Use

We use Medicare administrative records for mortality and hospital use for beneficiaries aged 65 to 100, covering over 97% of the U.S. population in this age group (Heutel, Miller and Molitor, 2021). The data include death dates and county of residence for all beneficiaries, as well as all inpatient and outpatient ER visits for those in fee-for-service Medicare, representing 69% of our sample. We also observe total costs for inpatient stays. Our primary measure of hospital use is ER visits, including both outpatient visits and those leading to admissions. In supplemental analyses, we examine additional measures: ER admission spending, any hospital visit (outpatient or inpatient), and hospital admissions. All county-level health measures are based on county of residence.

3 Wildfire Smoke Event Study

To motivate and support our research design for estimating C–R relationships, we begin by estimating panel distributed lag and lead models relating air quality and health outcomes to wildfire smoke exposure. We refer to this analysis as an "event study," as it describes the dynamics of air quality and health outcomes in the days before and after a smoke exposure event. Our objectives are twofold: first, to validate our control strategy by checking for pre-exposure effects; and second, to ensure that increases in pollution and adverse health outcomes coincide with smoke arrival and to determine the appropriate post-exposure window for capturing any delayed or offset health effects (e.g., harvesting).

3.1 Event Study: Research Design

To conduct our event study, we consolidate the daily smoke shocks shown in Figure 3a into a single continuous exposure variable that we call the "smoke index." We then estimate how a one-unit change in the index relates to air quality and health outcomes in the days before and after the exposure.

We construct the index variable $SmokeIndex_{ct}$ as a scaled sum of the smoke shock variables in county c on date t. Each shock is scaled by an estimate of its effect on same-day $PM_{2.5}$, calculated by estimating equation (2) using one-day $PM_{2.5}$ as the dependent variable (see Table A.1, column (2)). Thus, by construction, each one-unit increase in the index corresponds to an increase of approximately 1 $\mu g/m^3$ in ground-level $PM_{2.5}$ on the event day.

Our empirical strategy relates year-over-year deviations in smoke exposure for a given county and day of the year (e.g., Orange County, CA, on July 1) to corresponding deviations in ground-level $PM_{2.5}$ and health outcomes. Specifically, using observations for each county c and date t, we estimate the following regression:

$$\begin{aligned} Y_{ct} = \sum_{d=-20}^{20} \beta_d \, SmokeIndex_{c(t-d)} + [\text{county} \times \text{day-of-year FEs}]_{ct} \\ + [\text{county} \times \text{day-of-week FEs}]_{ct} + [\text{state} \times \text{year} \times \text{month FEs}]_{ct} + \varepsilon_{ct}. \end{aligned} \tag{1}$$

We estimate this equation separately for three primary outcomes Y_{ct}: ground-level $PM_{2.5}$ ($\mu g/m^3$), mortality (deaths per million people), and ER visits (visits per million people). In supplemental analyses, we estimate this equation for additional outcomes, including other criteria air pollutants, $PM_{2.5}$ species, and health care metrics including ER admission spending, hospital visits (outpatient or inpatient), and hospital admissions. Observations are weighted by Medicare population and standard errors are two-way clustered at the county and date levels to allow for arbitrary serial correlation within a county and spatial correlation on a given date.

Equation (1) includes the smoke index variable for date t with 20 leads and lags. The

key parameters of interest are the corresponding coefficients: β_d captures how smoke affects the outcome d days after the smoke event. These coefficients map the effects of smoke from 20 days before to 20 days after the event.

The primary controls are county-by-day-of-year fixed effects, isolating variation in smoke exposure within a county and day of the year to address cross-sectional and seasonal confounds. Our baseline also includes two secondary sets of controls: county-by-day-of-week fixed effects account for county-specific day-of-week (Sunday, Monday, etc.) patterns in outcomes like hospital visits, and state-by-year-by-month fixed effects account for arbitrary regional trends and any policy or environmental influence common to all counties in a state and month, like a particularly hot July in California. Since smoke shocks should not exhibit strong day-of-week patterns and do vary significantly from year to year, these secondary controls should have little impact on our estimates. Supplemental analyses report results that omit or relax these secondary controls.

Our identifying assumption is that, with these controls, the residual variation in *SmokeIndex* is as-if randomly assigned. The event-study specification provides a falsification check: there should be no effects of smoke before its arrival, meaning $\hat{\beta}_d$ should equal zero for all $d < 0$. Because none of the leads or lags are normalized to zero, this check captures any pre-exposure differences in levels, not just trends.

3.2 Event Study: Results

Figure 4a reports smoke event-study coefficients β_d from equation (1). The left panel shows ground-level $PM_{2.5}$ outcomes, with a spike starting on the smoke event day (day 0) and lasting about three days. The day 0 "on impact" effect—approximately a 1-$\mu g/m^3$ increase in $PM_{2.5}$ per one-unit increase in the smoke index—reflects the index's normalization, with magnitudes on other days interpreted relative to this baseline. Importantly, $PM_{2.5}$ levels show no significant effects in the days leading up to the smoke event, except for a modest increase the day before, likely due to smoke arriving the evening prior and affecting the previous day's

24-hour $PM_{2.5}$ reading.[1] The lack of pre-exposure effects supports our research design and the assumption that $PM_{2.5}$ would have remained flat absent the smoke shock. Additionally, the smoke's initial impact on $PM_{2.5}$ fully persists into the next day, diminishes by about 50% on day two, and mostly subsides by day three, indicating that a three-day event window is appropriate for capturing the air quality impacts of smoke exposure.

The middle and right panels of Figure 4a extend the analysis to mortality and ER visit outcomes. Given the smoke index's normalization, the magnitude of the health responses can be interpreted as the change per 1-$\mu g/m^3$ on-impact increase in smoke-induced $PM_{2.5}$. The health effect dynamics generally mirror the air quality impacts, beginning on the smoke event day and subsiding within three days. Mortality rises gradually, peaking two days after the event, totaling about 0.5 additional deaths per million Medicare beneficiaries within three days. ER visits increase more sharply, with around 6 additional visits per million beneficiaries on the event day, and about 25% of this effect persisting into the next day. The absence of subsequent reductions in mortality and ER visits within the 20-day post-event window suggests that the health impacts are not merely accelerating outcomes that would have occurred soon after (i.e., harvesting). Additionally, the lack of significant effects on mortality and ER visits before the smoke event supports the validity of our research design for health outcomes.

Figure A.2 shows that smoke exposure increases several key air pollutants, including coarse particulate matter (PM_{10}), ozone (O_3), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO). $PM_{2.5}$ exhibits the largest standardized rise—17% of a standard deviation on the event day—highlighting it as a primary component of wildfire smoke. Figure A.3 breaks down this increase into twelve primary $PM_{2.5}$ species, including organic carbon, sulfate, and nitrate, identifying elements that may drive the health impacts. Figure A.4a shows that smoke exposure raises ER spending, hospital admissions, and visits,

[1]The HMS generates smoke files approximately twice daily, once shortly before sunrise and again shortly after sunset. While nighttime smoke exposure is not captured in the smoke data, its impact is recorded by pollution monitors.

indicating that the increase in ER visits is not offset by a reduction in other types of hospital care.

The event study findings validate our baseline control strategy and support using a three-day post-event window to capture the cumulative impacts of wildfire smoke on $PM_{2.5}$ and short-term health outcomes. We incorporate both into our estimation of C–R relationships.

4 Concentration–Response Relationships

The event-study analysis shows that wildfire smoke sharply impacts ground-level air quality and increases mortality and ER visit rates among older adults. In this section, we examine how these effects scale with the intensity of smoke events. We first estimate how pollution and health outcomes respond to smoke shocks of different magnitudes, then combine these estimates to graphically represent the causal C–R relationships.

4.1 Concentration–Response: Research Design

We estimate the relationship between air pollution from wildfire smoke and health, allowing for arbitrary nonlinearity in the effects of larger versus smaller smoke shocks, using the following regression equation:

$$\begin{aligned} Y_{ct}^{\text{3-day}} = \sum_{s \in S} \beta_s^Y \mathbf{1}(\mathit{SmokeShock}_{ct} = s) \; + \; & [\text{lead/lag of } \mathit{SmokeShock} \text{ FEs}]_{ct} \\ + \; & [\text{county} \times \text{day-of-year FEs}]_{ct} \; + \; [\text{county} \times \text{day-of-week FEs}]_{ct} \\ + \; & [\text{state} \times \text{year} \times \text{month FEs}]_{ct} \; + \; \varepsilon_{ct}. \end{aligned} \tag{2}$$

Our primary outcomes are ground-level $PM_{2.5}$, mortality, and ER visits. In supplemental analyses, we also examine ER admission spending, hospital visits (outpatient or inpatient), and hospital admissions. The event-study results showed that smoke impacts on these outcomes are concentrated within three days of exposure. To capture the cumulative three-

day effects, we define $Y_{ct}^{\text{3-day}}$ as the sum of the daily measures on the index day t and the following two days.

The right-hand side of equation (2) is identical to equation (1) except we have replaced the smoke index variable with a series of indicators $\mathbf{1}(SmokeShock_{ct} = s)$ for whether county c on date t was exposed to a smoke shock of intensity s, for all shocks S listed in Figure 3a. We also include fixed effects for one lead and lag of the shocks to address serial correlation in smoke exposure and isolate the date-t shock's impact. The omitted shock category is counties over 1,000 km from any smoke plume. Thus, the coefficients β_s^Y describe the effect on outcome Y of exposure to a shock of intensity s, relative to having been at least 1,000 km from a plume. Observations are weighted by Medicare population, and standard errors are clustered by county and date.

Using estimates from equation (2), we can trace out the pollution–health C–R relationship by plotting the points $\left(\hat{\beta}_s^H, \hat{\beta}_s^{\text{PM}_{2.5}}\right)$ that pair the change in health outcome H with the corresponding change in $\text{PM}_{2.5}$ for each smoke shock $s \in S$. The curve serves as a visual IV estimate of the health impact of $\text{PM}_{2.5}$ using smoke shocks as instruments. Its slope reflects the ratio of a smoke-induced change in health (the reduced form) to the corresponding change in $\text{PM}_{2.5}$ (the first stage), with changes in slope indicating a nonlinear relationship between air pollution and health. In Section 5, we directly estimate the slope of the C–R relationship using 2SLS estimation and discuss the exclusion restriction needed to interpret the slope as reflecting the causal effect of $\text{PM}_{2.5}$ on health.

We aim to attribute the effects estimated in equation (2) to wildfire smoke. A potential challenge is that the wind patterns transporting smoke to an area may also carry pollution from other sources. To disentangle the effects of smoke from generically polluted wind directions as in Deryugina et al. (2019), we examine the sensitivity of our estimates to including county-specific wind direction bins in 60-degree increments.

4.2 Concentration–Response: Results

The left panel of Figure 4b plots estimates of $\beta_s^{\text{PM2.5}}$ from equation (2), showing how each smoke shock s impacts three-day $PM_{2.5}$ concentrations. The effects increase monotonically with proximity to a smoke plume and coverage density. In counties near but not directly covered by a plume, $PM_{2.5}$ concentrations rise modestly by 0.5–3.2 $\mu g/m^3$ (5%–35% of the mean of 9.1 $\mu g/m^3$ on a smoke-free day, the reference category). Counties directly covered by a plume experience larger increases of 5.2–14.4 $\mu g/m^3$ (58%–158% of the smoke-free mean), depending on the smoke's thickness. These findings demonstrate that smoke significantly impacts ambient pollution concentrations, with effects varying substantially based on wind patterns and plume density, underscoring the value of smoke as a natural experiment to explore how pollution effects scale with the magnitude of exposure.

To quantify wildfire smoke's average contribution to ambient $PM_{2.5}$, we combine the $PM_{2.5}$ effects of each smoke shock type with their frequency over the sample period. Specifically, we calculate

$$\sum_{s \in S} \hat{\beta}_s^{\text{PM}_{2.5}} Pr(SmokeShock_{ct} = s), \tag{3}$$

where $\hat{\beta}_s^{\text{PM}_{2.5}}$ is the estimated $PM_{2.5}$ effect from smoke shock s, and $Pr(SmokeShock_{ct} = s)$ is the fraction of the sample in which this type of smoke shock occurs (Figure 3a). This sum equals 1.65 $\mu g/m^3$ (Table A.1). Compared to the average daily $PM_{2.5}$ concentration of 9.1 $\mu g/m^3$ on a smoke-free day (the reference category), these results imply that smoke accounts for 18% of ambient daily $PM_{2.5}$ in the sample. These findings complement National Emissions Inventory estimates that wildfires produced 12% of $PM_{2.5}$ emissions in 2007–2019 (U.S. EPA, 2024). Our finding that population-weighted ambient $PM_{2.5}$ concentrations attributable to smoke exceed wildfire emissions over the sample highlights wildfires as a significant contributor to pollution in populated areas.

The middle and right panels of Figure 4b present the estimated C–R relationships, illustrating how smoke exposure impacts mortality and ER visits at different exposure levels.

Each point on a curve corresponds to one of the nine smoke shocks from equation (2). The horizontal position reflects the change in $PM_{2.5}$ concentrations due to smoke ($\hat{\beta}_s^{PM_{2.5}}$), while the vertical position indicates the corresponding change in the health outcome, either mortality ($\hat{\beta}_s^{Mortality}$) or ER visits ($\hat{\beta}_s^{ER}$).

The resulting C–R relationships show that health impacts rise steeply with low to moderate smoke exposure and then plateau at around $6\,\mu g/m^3$, forming nonlinear, concave shapes. The steep initial slopes suggest substantial health benefits from additional air quality improvements, even on relatively clean days with little smoke exposure. The concavity of these curves indicates that smaller pollution shocks have a greater health impact per unit of $PM_{2.5}$ than larger shocks. In fact, the largest shocks exhibit marginally lower impacts than moderate ones; while these differences lack statistical significance, they point to potential non-monotonic effects of pollution exposure on health, which might occur as people reduce outdoor activities and avoid exposure on highly polluted days.

Figure A.4b shows similar concave and non-monotonic C–R relationships for ER spending, hospital visits, and admissions. Figure A.5 Panel (a) shows that C–R relationships for mortality and ER visits hold after controlling for county-daily wind direction and temperature, helping isolate smoke effects from generic pollution and weather influences. Panels (b) and (c) demonstrate robustness to relaxing secondary controls. Figure A.6 finds similar C–R patterns across counties in the lowest, middle, and highest terciles of average $PM_{2.5}$ levels, indicating that observed nonlinearities in the overall C–R relationships are not driven by cross-sectional treatment effect heterogeneity and underscoring that modest pollution increases can have steep health impacts, even in low-pollution areas.

We calculate wildfire smoke's aggregate contribution to elderly mortality and ER visits, applying equation (3) but replacing the $PM_{2.5}$ effects of each smoke shock with mortality and ER visit effects (Table A.1). We estimate that smoke causes an average of 0.51 deaths and 9.7 ER visits per million adults each day, implying that smoke exposure accounts for about 1 in every 240 (0.42%) deaths and 1 in every 145 (0.69%) ER visits in the sample.

When scaled to the U.S. population of 54.1 million adults aged 65 years and over in 2019, this corresponds to 10,070 premature elderly deaths and 191,541 excess ER visits annually.

Wildfire smoke's impact on mortality risk can be valued based on lives lost or life-years lost. EPA guidelines specify assigning a common value of statistical life of $9.4 million ($2019) to each excess death, resulting in an estimated annual cost of $95 billion (U.S. EPA, 2010). The EPA approach does not adjust for the potentially lower life expectancy of those who die from acute wildlife smoke exposure. We address this by valuing the life-years lost from smoke exposure. We use a value of $136,000 ($2019) per life-year (Cutler, 2004) and assume that those dying prematurely from wildfire smoke would have lived an additional 3.5 years (Deryugina et al. (2019)), yielding an annual mortality cost of $4.8 billion. By comparison, wildfires cause $617 million in annual damages to structures, with federal and state fire suppression and protection costs adding another $3.5 billion (National Institute of Standards and Technology, 2017). These costs, though significant, are less than the estimated annual mortality cost from smoke.

5 Two-Stage Least Squares Estimation

The C–R relationships presented above offer visual IV estimates of how $PM_{2.5}$ impacts mortality and ER visits. In this section, we directly estimate the slopes along different portions of these curves using two-stage least squares estimation and discuss the exclusion restriction needed to interpret the slopes as the causal effects of $PM_{2.5}$ on health.

5.1 Two-Stage Least Squares: Research Design

We are interested in the relationship between health outcomes and $\text{PM}_{2.5}$, as described by the following equation:

$$\begin{aligned} Y_{ct}^{\text{3-day}} = \beta \left[\text{PM}_{2.5}\right]_{ct} &+ \left[\text{lead/lag of } \text{PM}_{2.5}\right]_{ct} \\ &+ \left[\text{county} \times \text{day-of-year FEs}\right]_{ct} + \left[\text{county} \times \text{day-of-week FEs}\right]_{ct} \\ &+ \left[\text{state} \times \text{year} \times \text{month FEs}\right]_{ct} + \varepsilon_{ct}. \end{aligned} \tag{4}$$

Estimating this equation via ordinary least squares (OLS) could lead to biased estimates of β due to measurement error in $\text{PM}_{2.5}$ and omitted variables that affect health and are correlated with $\text{PM}_{2.5}$. To address these issues, we use smoke shocks as instruments for $\text{PM}_{2.5}$, applying a two-sample 2SLS estimator (Angrist and Krueger, 1992). In the first stage, we estimate equation (2) with three-day $\text{PM}_{2.5}$ as the outcome, using observations where $\text{PM}_{2.5}$ is observed. We then use the first-stage estimates to generate the predicted values, $\widehat{\text{PM}_{2.5}}$, for the full sample, as smoke exposure is measured for all observations. These predicted values serve as the key independent variable in the second-stage regression, specified as follows:

$$\begin{aligned} Y_{ct}^{\text{3-day}} = \beta_{IV}[\widehat{\text{PM}_{2.5}}]_{ct} &+ \sum_{s \in S_{included}} \beta_s^Y \mathbf{1}(SmokeShock_{ct} = s) \\ &+ \left[\text{lead/lag of } SmokeShock \text{ FEs}\right]_{ct} + \left[\text{county} \times \text{day-of-year FEs}\right]_{ct} \\ &+ \left[\text{county} \times \text{day-of-week FEs}\right]_{ct} + \left[\text{state} \times \text{year} \times \text{month FEs}\right]_{ct} + \varepsilon_{ct}. \end{aligned} \tag{5}$$

The coefficient β_{IV} captures a regression average of the C–R curve's slope, calculated over the portion of the curve determined by the smoke shocks excluded from the second-stage regression controls, i.e., shocks not in $S_{included}$. This is because residual variation $\widehat{\text{PM}_{2.5}}$ comes only from the excluded instruments. We consider three sets of excluded instruments: "all" shocks, where $S_{included}$ is the empty set; "small" shocks, including only those without direct smoke coverage; and "large" shocks, consisting of light, medium, or thick smoke coverage.

The omitted smoke shock category in $S_{included}$ is always the 0–100 km distance bin, marking the dividing point between small and large shocks.

We calculate standard errors for all two-sample IV estimates via bootstrap, clustering at both the county and date levels (Cameron and Miller, 2015). The bootstrap permits calculating standard errors for linear combinations of parameters, even if they derive from different equations. This allows us to formally test whether the slope of the C–R relationship differs at smaller versus larger concentrations of $PM_{2.5}$ by testing whether estimates of β_{IV} differ when based on different excluded instruments.

We also estimate equation (4) via OLS, which uses all residual variation in $PM_{2.5}$ not absorbed by the fixed effects in our regressions. By comparison, IV estimates rely only on variation from drifting smoke plumes. Comparing the OLS and IV estimates is useful for understanding whether and how conclusions about the pollution–health relationship depend on these different sources of variation.

For smoke to be a valid instrumental variable for $PM_{2.5}$, it must satisfy the relevance condition and the exclusion restriction. The relevance condition requires smoke to be strongly correlated with $PM_{2.5}$, which we demonstrate by reporting first-stage F-statistics for each IV regression.[2] The exclusion restriction requires that smoke not be correlated with unobserved determinants of health. Our control strategy addresses threats to this restriction arising from cross-sectional or seasonal patterns. However, wildfire smoke carries a mix of potentially harmful pollutants and may interact with weather conditions that directly affect health.

Supplemental analyses suggest that these potential violations of the exclusion restriction are limited. Regarding weather interactions, Figure A.5 Panel (a) demonstrates that the C–R relationships for mortality and ER visits hold after flexibly controlling for wind direction and temperature. For multiple pollutants, we align with the broader pollution-health literature by using $PM_{2.5}$ as the primary air quality indicator, recognizing that $PM_{2.5}$ inherently

[2]We report both the Cragg–Donald (1993) statistic, which assumes homoskedastic errors and can be compared to the well-known Stock and Yogo (2005) critical values that are valid only under homoskedasticity, and the Kleibergen–Paap (2006) statistic, which is robust to clustering at the county and date levels.

encompasses a complex mixture of secondary particles derived from gaseous pollutants like NO_2 and SO_2. Existing research has identified $PM_{2.5}$ as the main driver of adult health impacts from general pollution shocks, such as those resulting from changing wind direction (Deryugina et al., 2019). Specifically for wildfire smoke, $PM_{2.5}$ is the component widely considered to pose the greatest risk to public health (U.S. EPA, 2021). Figure A.2 further shows that, while smoke elevates several pollutants, its largest impact is on $PM_{2.5}$ when assessed on a standard-deviation scale.

To further validate interpreting smoke impacts as operating through $PM_{2.5}$, we directly compare our smoke IV estimate of mortality impacts with the wind direction IV estimate from Deryugina et al. (2019). Larger smoke IV estimates could indicate that smoke $PM_{2.5}$ is more harmful or that other pollutants in smoke contribute to health impacts, indicating a potential exclusion restriction violation. However, Table A.2 shows that both instruments yield comparable results. The wind direction estimate falls approximately midway between our IV estimates for small and large smoke shocks, suggesting that smoke effects operate through $PM_{2.5}$ similarly to other $PM_{2.5}$ sources.

5.2 Two-Stage Least Squares: Results

Table 1 reports estimates from equation (5). Panel A shows results for three-day mortality outcomes. Using all smoke shocks as excluded instruments, column (1) shows each unit of $PM_{2.5}$ corresponds to 0.20 additional deaths per million, on average. For small shocks, column (2) reports an increase of 0.37 deaths per million per unit of $PM_{2.5}$, while column (3) reports that large shocks have a small and statistically insignificant mortality impact relative to a distance of 0–100 km from smoke. Panel B shows similar patterns for three-day ER visits. In both panels, columns (4)–(6) show that estimates differ statistically across excluded instruments, corroborating the concave patterns seen in Figure 4b.

Column (7) reports OLS estimates of equation (4): each additional unit of $PM_{2.5}$ corresponds to 0.19 additional deaths and 1.40 additional ER visits per million over three

days. The OLS estimates lie between the IV estimates for large and small shocks and are similar to those using all shocks. This suggests that differences between IV and OLS estimates may stem more from varying regression weights in the presence of heterogeneous treatment effects (Ishimaru, 2024) than from bias, an explanation that has received little attention in prior comparisons IV and OLS pollution impact estimates.

6 Discussion and Conclusion

After over half a century of regulation, air quality in the U.S. and many other developed countries has improved substantially (Dominici, Greenstone and Sunstein, 2014). While the health risks from elevated $PM_{2.5}$ are widely acknowledged, some policymakers have questioned the health benefits of further air quality improvements amid relatively low ambient concentrations (U.S. EPA, 2020). Indeed, prevailing regulatory approaches assume the C–R relationship to be convex, with severe pollution episodes causing the greatest risks and pollution levels below a safety threshold causing little harm (McGartland et al., 2017; National Research Council, 2009).

Our findings challenge these views. We present new causal evidence that exploits the natural experiment of drifting wildfire smoke to show that the relationship between air pollution exposure and health is concave: small increases in pollution cause significant health impacts, while larger increases yield diminishing additional effects.

Evidence of a concave C–R relationship indicates significant health benefits of reducing $PM_{2.5}$ levels even in areas meeting regulatory standards. This evidence invites a reevaluation of regulatory strategies, particularly in countries like the U.S. with relatively low average pollution levels. By focusing on a broader range of pollution levels, especially smaller and more frequent pollution events, policies can better protect public health. Our findings indicate that further reductions in air pollution could significantly benefit health outcomes, even in regions that comply with current air quality standards.

Our findings also highlight the potential role of adaptation in the health effects of pollution exposure. First, the concave C–R relationship suggests that individuals exposed to large smoke shocks may adopt adaptive behaviors, such as avoidance, which reduces marginal harm and bends the C–R relationship downward. Second, we find evidence of a concave C-R relationship not only overall but also conditional on baseline pollution exposure. This is consistent with individuals adapting to baseline pollution levels over time, even as variability in pollution exposure above baseline significantly impacts health. Because air pollution sources can never be eliminated entirely, our results suggest value in policies that not only reduce population pollution exposure but also facilitate adaptation and resilience to fluctuating air quality conditions.

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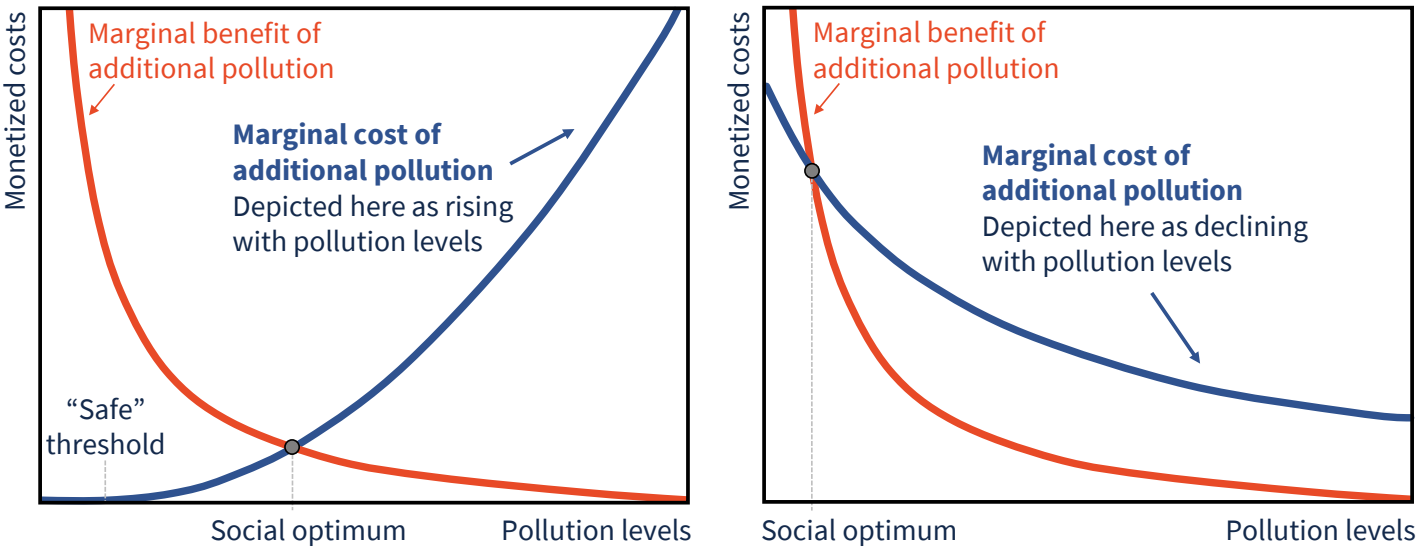
Figures and Tables

Figure 1: Role of the concentration–response function for optimal pollution abatement

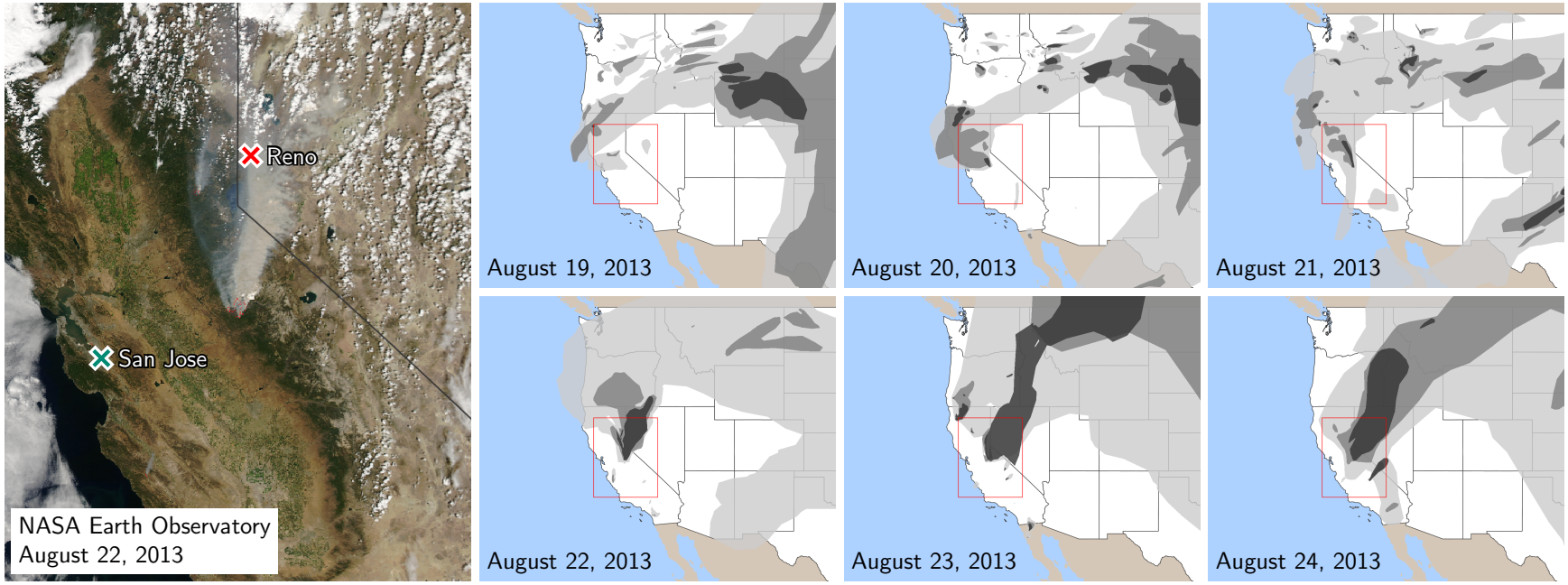


(a) Increasing marginal cost of pollution

(b) Decreasing marginal cost of pollution

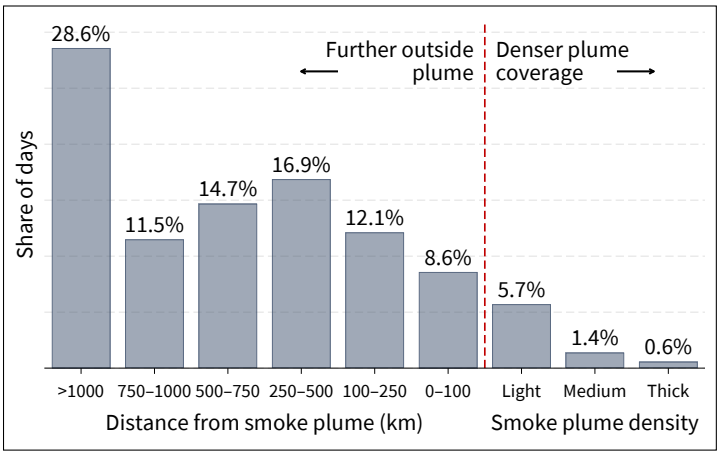
Notes: The figure shows the traditional conceptual framework for economic analysis of marginal cost versus marginal benefits of pollution abatement. Adapted from Figure 1 of Pope III et al. (2015).

Figure 2: Satellite-based smoke plumes (NOAA Hazard Mapping System)

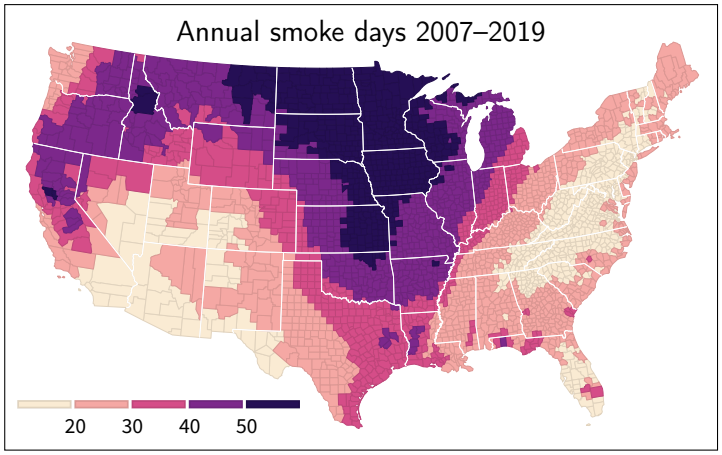


Notes: The left panel shows an image acquired by the Moderate Resolution Imaging Spectroradiometer (MODIS) on NASA's Aqua satellite of the Rim Fire burning in central California near Yosemite National Park, on August 22, 2013. The image reveals hot spots outlined in red and a thick smoke plume blowing northeast. The six panels to the right show a series of daily snapshots of the NOAA Hazard Mapping System (HMS) smoke plumes data for August 19–24, 2013. Polygons represent wildfire smoke plume boundaries, and shading indicates whether HMS categorized the smoke density as light, medium, or thick. The red rectangles show the extent of the NASA image in the left panel.

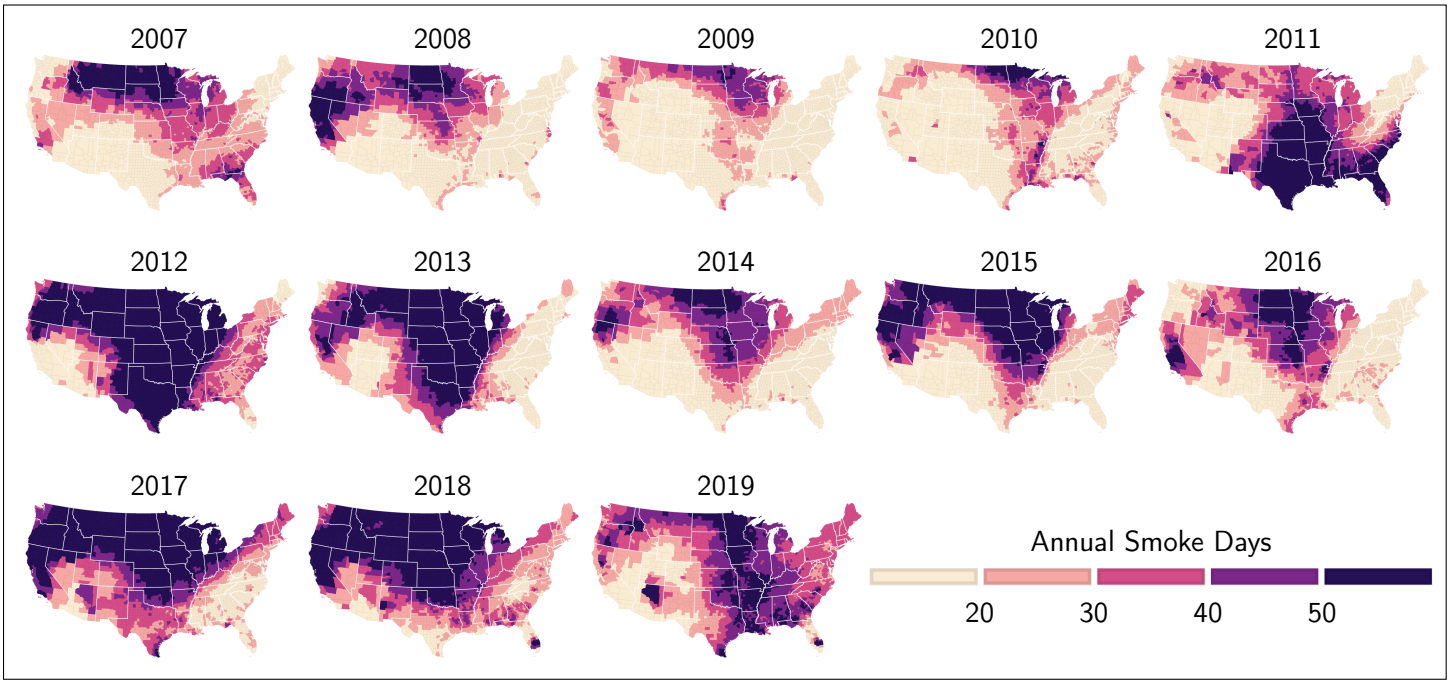
Figure 3: Wildfire smoke exposure in the U.S., 2007–2019



(a) Frequency of smoke exposure



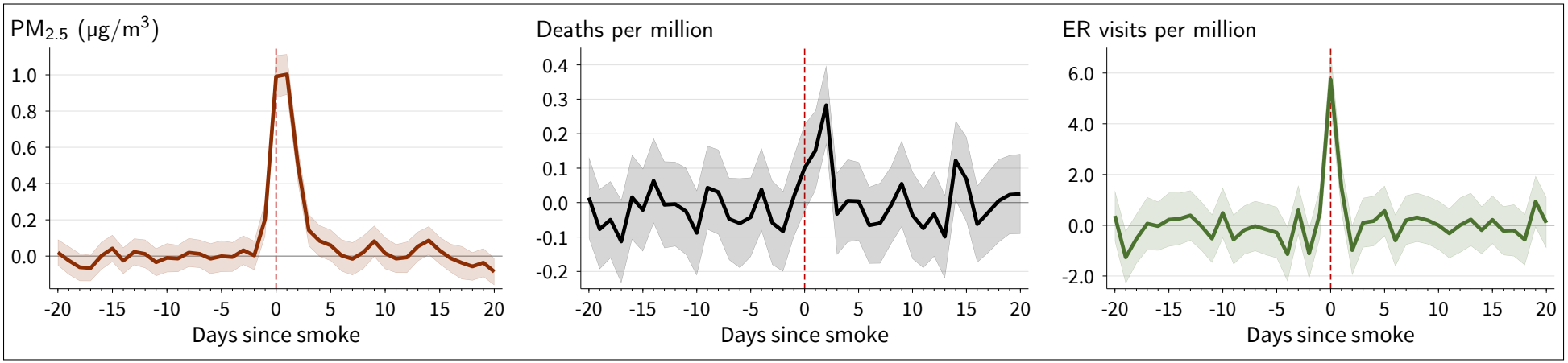
(b) Geography of smoke plume coverage



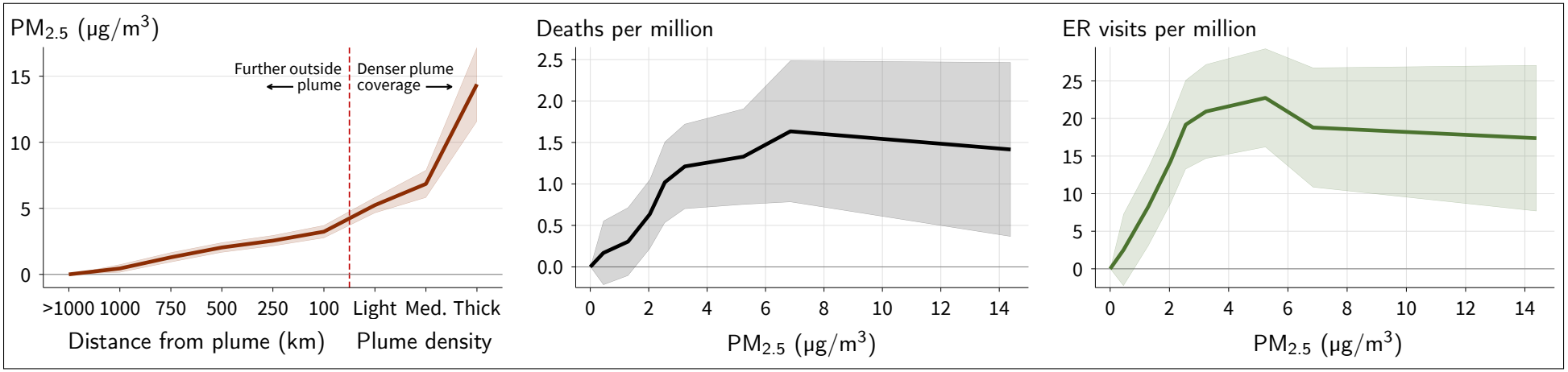
(c) Annual smoke plume coverage

Notes: Panel (a) reports Medicare population-weighted frequency distribution of the smoke shock categories at the county-daily level. Panel (b) shows annual average smoke days by county over the 2007–2019 period. Here we consider a county to be exposed to smoke if at least 20% of its land area is covered by smoke plumes. Panel (c) breaks down smoke days by year.

Figure 4: Effects of wildfire smoke on air pollution and health



(a) Event study



(b) Concentration–response

Notes: Panel (a) reports event-study estimates from equation (1). The estimates reflect the effect of a one-unit increase in the smoke index in county c on date t on ground-level $PM_{2.5}$ (left), mortality (middle), and ER visits (right) on the date of the event (day 0 on the graph) and up to 20 days before (negative day values) and after (positive day values) the event. Panel (b) reports estimated effects of exposure to smoke shocks of varying intensity using equation (2) (Table A.1, columns (3)–(5) report regression details). Each estimate reflects the effect of exposure to a smoke shock of intensity s, relative to having been at least 1,000 km away from a smoke plume. The effects of smoke exposure on ground-level $PM_{2.5}$ (left) are positioned on the horizontal axis according to the category of smoke intensity; effects on mortality (middle) and ER visits (right) are positioned on the horizontal axis according to the $PM_{2.5}$ effect of the associated category of smoke intensity. All regressions are weighted by the Medicare population in county c on date t. Shaded areas reflect 95% confidence intervals based on standard errors clustered at both the county and date levels.

Table 1: $PM_{2.5}$ impacts on mortality and ER visits

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	2SLS excluded smoke instruments:			IV differences			OLS
	All shocks	Small shocks	Large shocks	(2)–(3)	(2)–(1)	(1)–(3)	
A. 3-day deaths per million							
$PM_{2.5}$	0.20***	0.37***	0.033	0.34***	0.17***	0.17***	0.19***
	(0.041)	(0.085)	(0.034)	(0.098)	(0.059)	(0.046)	(0.019)
Excluded smoke instruments	All	Small	Large				
Included smoke instruments	None	Large	Small				
Cragg-Donald F-statistic	4485.5	2815.1	4699.5				
Kleibergen-Paap F-statistic	47.5	42.2	55.6				
Mean outcome	358.9	358.9	358.9	47.5	47.5	47.5	349.1
Sample size	14,594,910	14,594,910	14,594,910	358.9	358.9	358.9	3,320,622
B. 3-day ER visits per million							
$PM_{2.5}$	3.2***	7.0***	–0.23	7.2***	3.8***	3.4***	1.4***
	(0.53)	(1.1)	(0.38)	(1.2)	(0.77)	(0.59)	(0.19)
Excluded smoke instruments	All	Small	Large				
Included smoke instruments	None	Large	Small				
Cragg-Donald F-statistic	4484.8	2811.1	4705.5				
Kleibergen-Paap F-statistic	47.5	42.1	55.7				
Mean outcome	4,326.2	4,326.2	4,326.2	47.5	47.5	47.5	4,160.5
Sample size	14,591,808	14,591,808	14,591,808	4,326.2	4,326.2	4,326.2	3,319,490

Notes: The table reports IV and OLS estimates of the relationships between $PM_{2.5}$ ($\mu g/m^3$) and mortality (three-day deaths per million) and ER visits (three-day ER visits per million). Columns (1)–(3) present estimates of β_{IV} from equation (5), which relates health outcomes to variation in three-day $PM_{2.5}$ predicted by the excluded smoke instruments. The columns correspond to three sets of excluded instruments: All shocks include all smoke shock categories; small shocks are those without direct smoke coverage; and large shocks consist of light, medium, or thick smoke coverage. Columns (4)–(6) show simple differences between the IV estimates from columns (1)–(3), as specified by the column labels. Column (7) presents OLS estimates from equation (2), where smoke shock variables are replaced with $PM_{2.5}$. Standard errors are clustered at the county and date levels. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

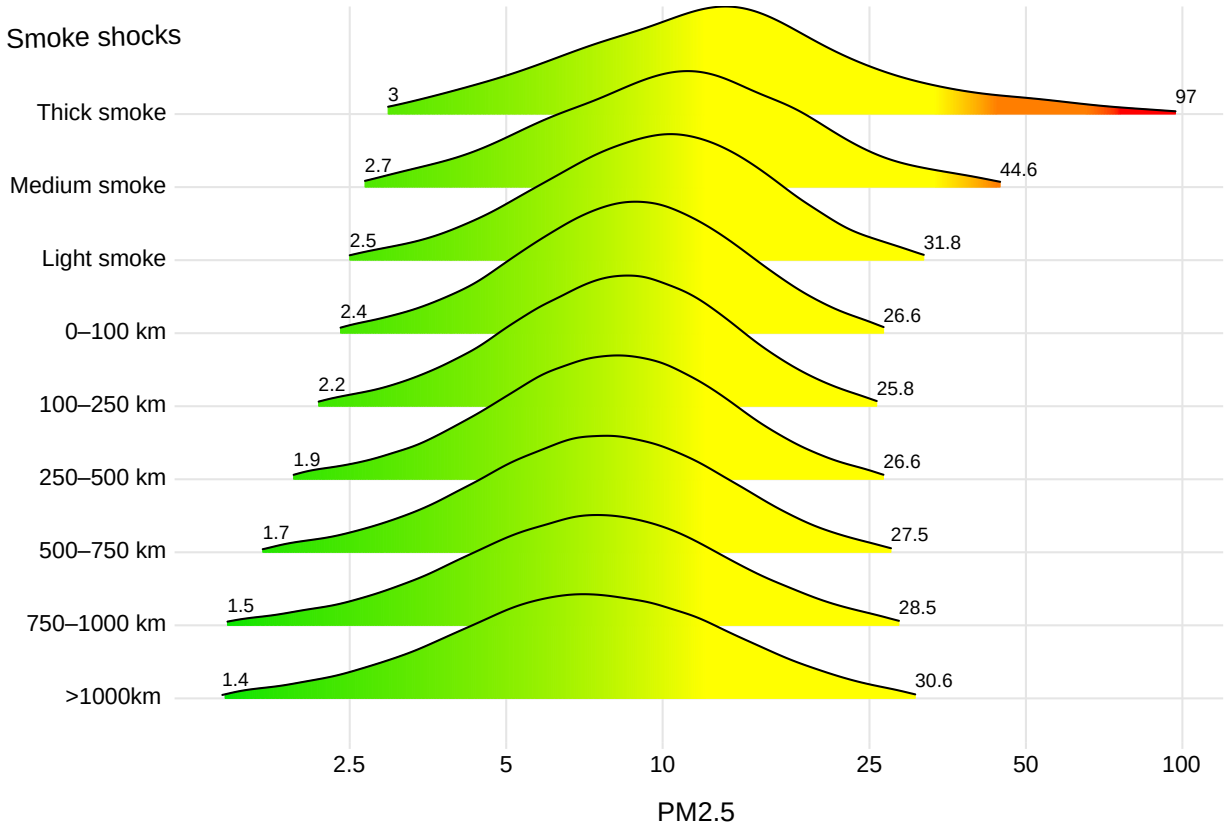
Online Appendix

The Nonlinear Effects of Air Pollution on Health: Evidence from Wildfire Smoke

Nolan Miller, University of Illinois and NBER
David Molitor, University of Illinois and NBER
Eric Zou, University of Michigan and NBER

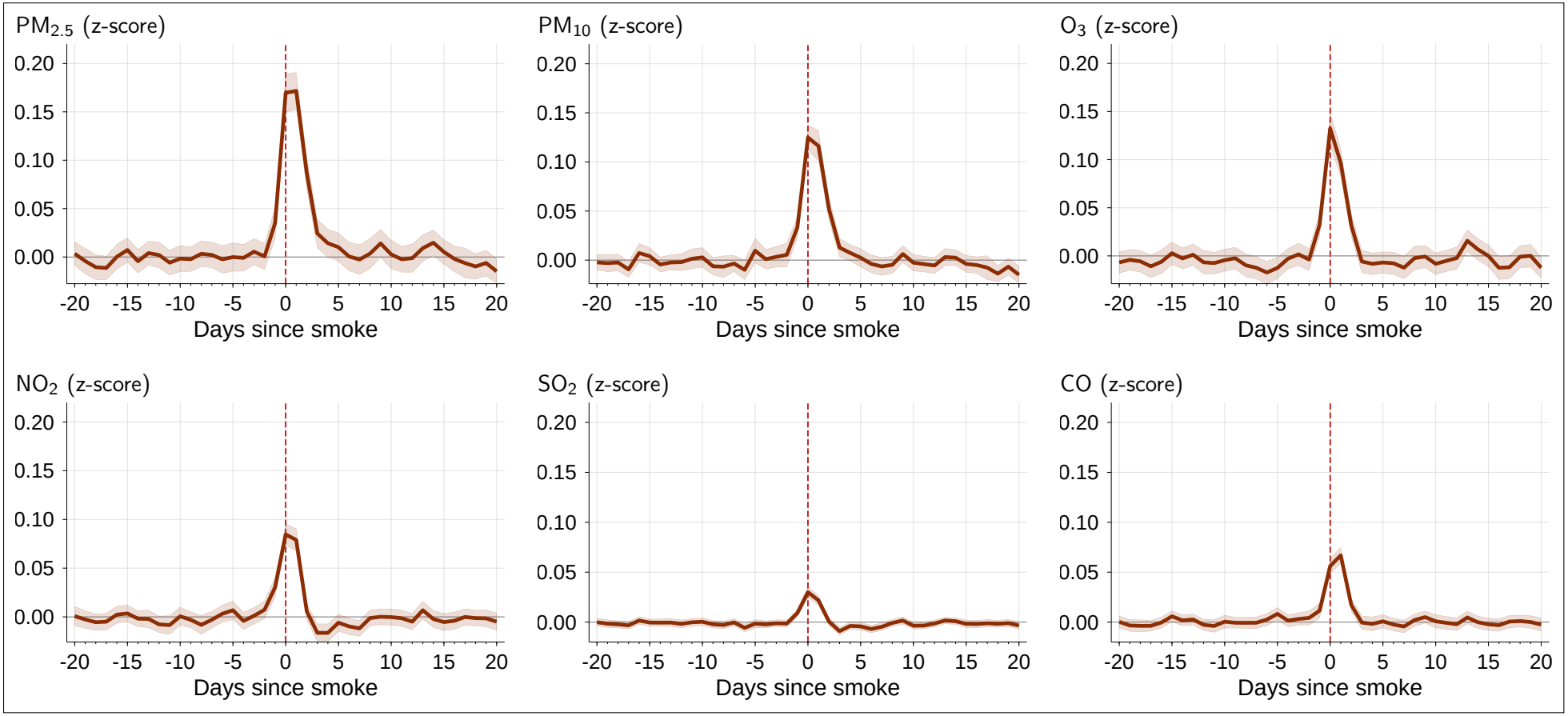
September 2024

Figure A.1: Distributions of $PM_{2.5}$ concentration by smoke shock categories



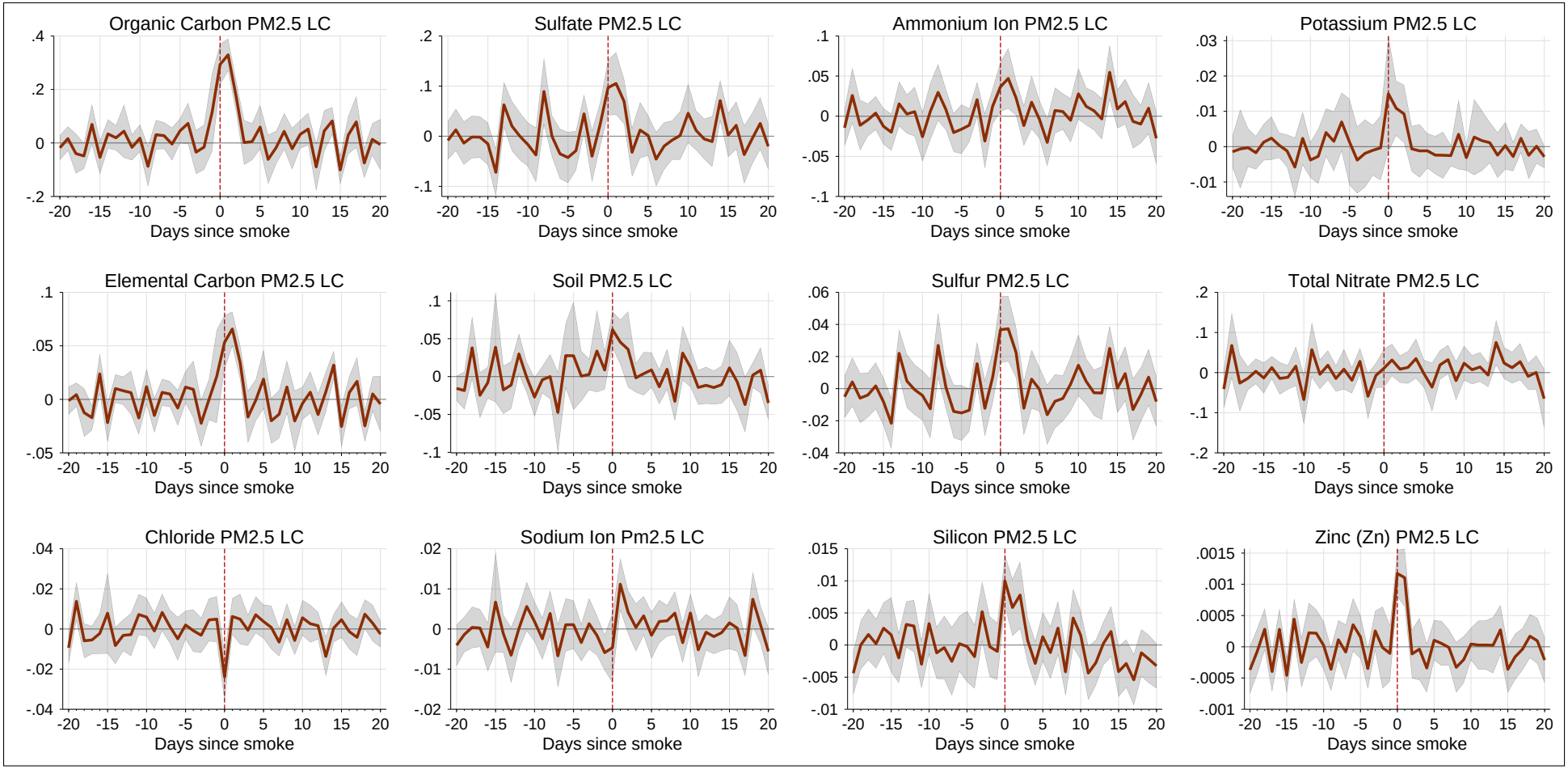
Notes: The plot shows the distribution of daily $PM_{2.5}$ concentrations across the categories of wildfire smoke shocks considered in the analysis. The distributions are color-coded according to the EPA's Air Quality Index, where orange and red zones represent air quality considered unhealthy for sensitive groups and the general population, respectively. The numbers at each end of the distributions represent the 1^{st} and 99^{th} percentiles.

Figure A.2: Effects of wildfire smoke on criteria air pollution: Event study



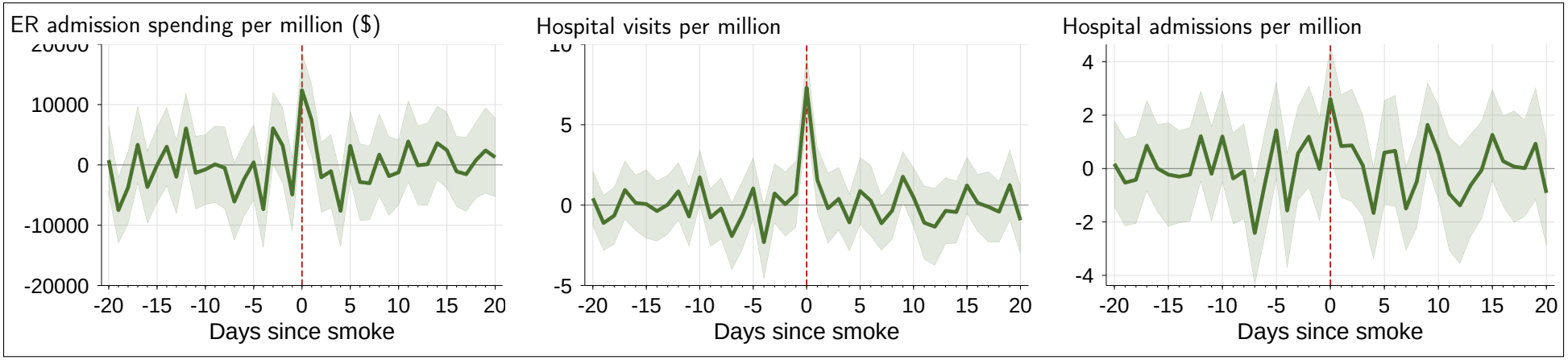
Notes: This figure reports event-study estimates from equation (1) for six criteria air pollutants. The outcome variables are standardized z-scores, with a mean of 0 and a standard deviation of 1. The estimates reflect the effect of a one-unit increase in the smoke index in county c on date t on ground-level pollutant concentration on the date of the event (day 0 on the graph) and up to 20 days before (negative day values) and after (positive day values) the event. All regressions are weighted by the Medicare population in county c on date t. Shaded areas reflect 95% confidence intervals based on standard errors clustered at both the county and date levels.

Figure A.3: Effects of wildfire smoke on $PM_{2.5}$ species: Event study

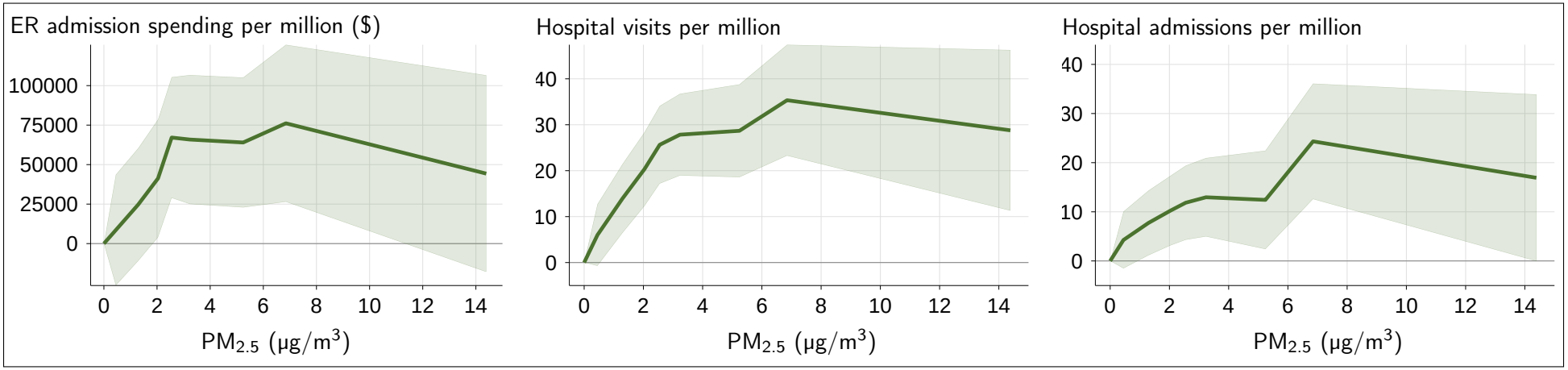


Notes: This figure reports event-study estimates from equation (1) for various $PM_{2.5}$ species (chemical components). The estimates reflect the effect of a one-unit increase in the smoke index in county c on date t on ground-level pollutant concentration on the date of the event (day 0 on the graph) and up to 20 days before (negative day values) and after (positive day values) the event. All regressions are weighted by the Medicare population in county c on date t. Shaded areas reflect 95% confidence intervals based on standard errors clustered at both the county and date levels.

Figure A.4: Effects of wildfire smoke on health: Other health care outcomes



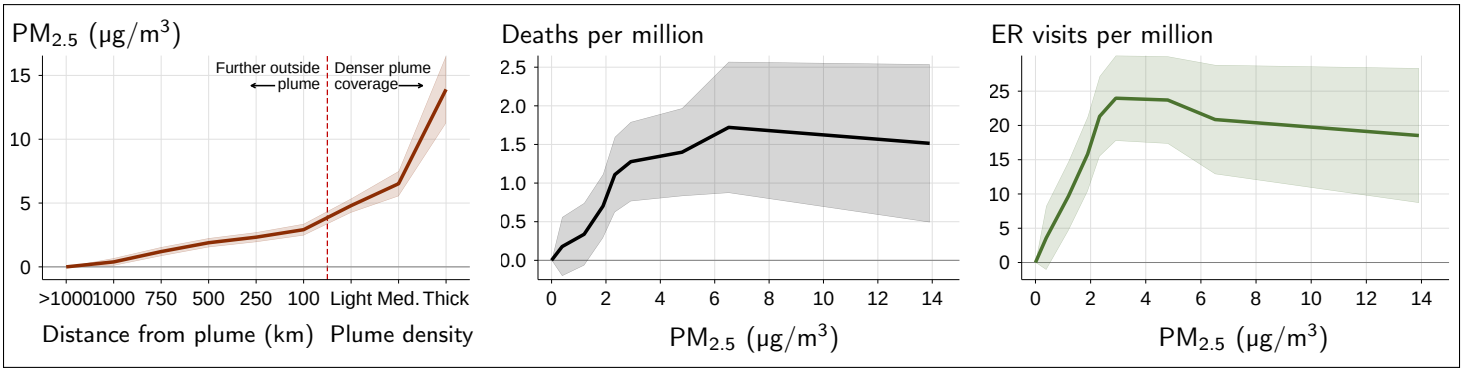
(a) Event study



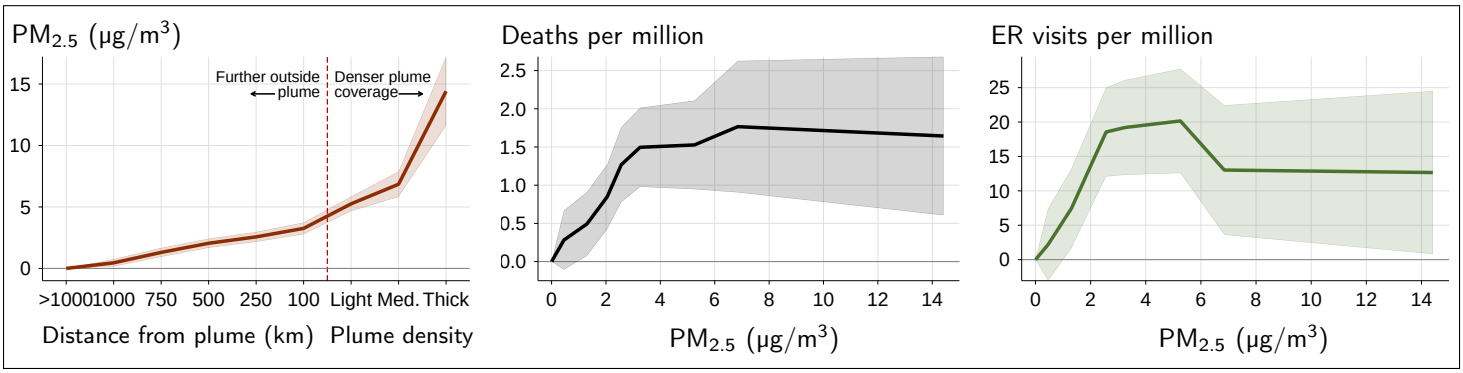
(b) Concentration–response

Notes: Panel (a) reports event-study estimates from equation (1). The estimates reflect the effect of a one-unit increase in the smoke index in county c on date t on total ER spending (left), hospital visits (middle), and hospital admissions (right) on the date of the event (day 0 on the graph) and up to 20 days before (negative day values) and after (positive day values) the event. Panel (b) reports the concentration–response relationships with respect to $PM_{2.5}$. All regressions are weighted by the Medicare population in county c on date t. Shaded areas reflect 95% confidence intervals based on standard errors clustered at both the county and date levels.

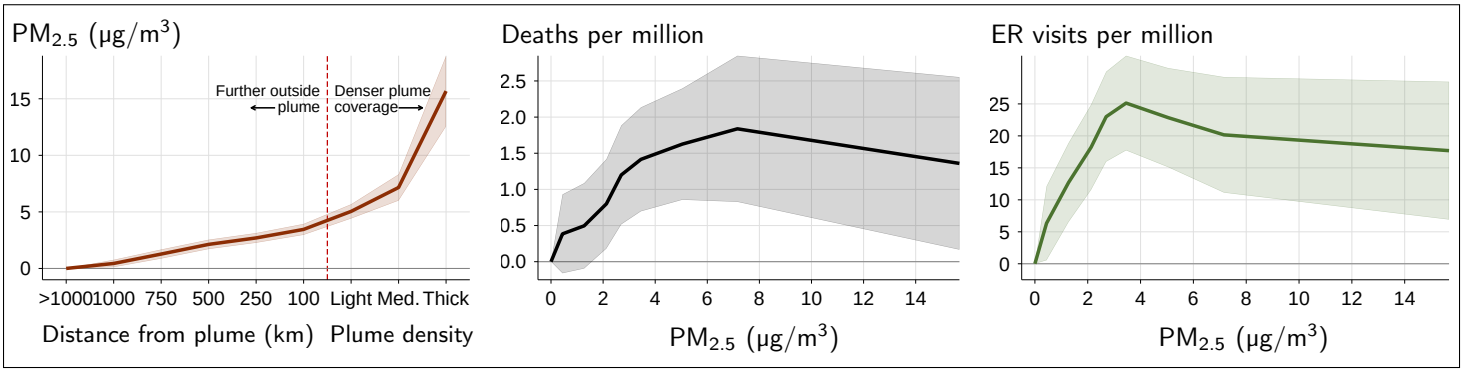
Figure A.5: Effects of wildfire smoke on air pollution and health: Robustness



(a) Add county × 60-degree wind direction bins, 10-degree temperature bins fixed effects



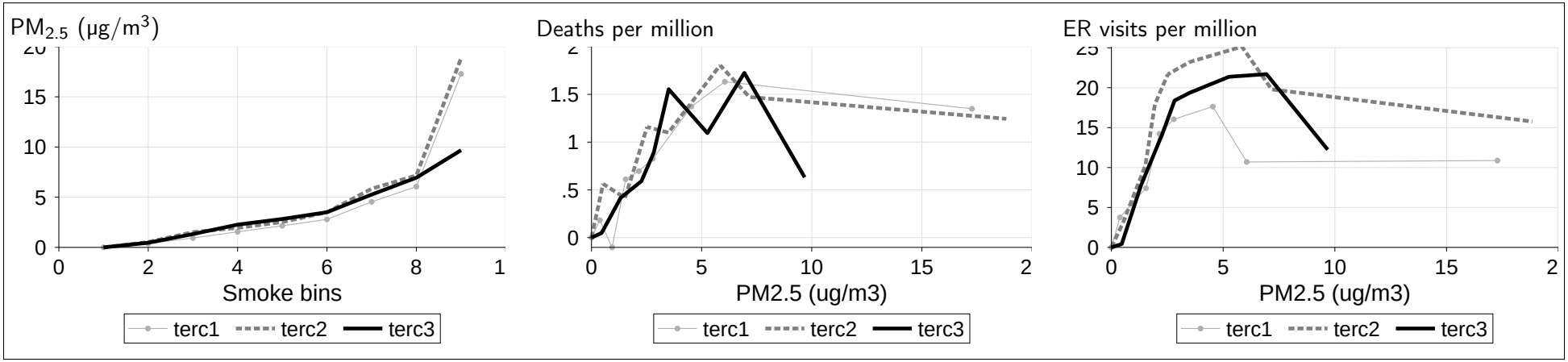
(b) Drop county × day-of-week fixed effects



(c) Replace state × year × month fixed effects with state × year

Notes: This figure reports various versions of the C–R estimates. Each panel corresponds to a robust specification, and each estimate reflects the effect of exposure to a smoke shock of intensity s, relative to having been at least 1,000 km away from a smoke plume. The effects of smoke exposure on ground-level $PM_{2.5}$ (left) are positioned on the horizontal axis according to the category of smoke intensity. The effects on mortality (middle) and ER visits (right) are positioned on the horizontal axis according to the $PM_{2.5}$ effect of the associated category of smoke intensity. All regressions are weighted by the Medicare population in county c on date t. Shaded areas reflect 95% confidence intervals based on standard errors clustered at both the county and date levels.

Figure A.6: Effects of wildfire smoke: Heterogeneity by county average PM2.5



Notes: This figure reports heterogeneity of the C–R estimates conducted separately by counties' average $PM_{2.5}$ levels. The groups "terc1/2/3" correspond to counties in the lower/medium/highest tercile of sample-average $PM_{2.5}$ concentration. Each panel corresponds to a robust specification, and each estimate reflects the effect of exposure to a smoke shock of intensity s, relative to having been at least 1,000 km away from a smoke plume. The effects of smoke exposure on ground-level $PM_{2.5}$ (left) are positioned on the horizontal axis according to the category of smoke intensity. The effects on mortality (middle) and ER visits (right) are positioned on the horizontal axis according to the $PM_{2.5}$ effect of the associated category of smoke intensity. All regressions are weighted by the Medicare population in county c on date t.

Table A.1: Effects of wildfire smoke on air pollution and health

	(1)	(2)	(3)	(4)	(5)
		First stage		Reduced form	
Smoke shock	Sample frequency (%)	1-day $PM_{2.5}$ ($\mu g/m^3$)	3-day $PM_{2.5}$ ($\mu g/m^3$)	3-day deaths per million	3-day ER visits per million
> 1,000 km from plume (ref. category)	28.58%	–	–	–	–
700–1,000 km from plume	11.50%	0.02	0.45***	0.17	2.50
		(0.07)	(0.15)	(0.20)	(2.44)
500–750 km from plume	14.68%	0.22**	1.29***	0.30	8.30***
		(0.08)	(0.19)	(0.21)	(2.67)
250–500 km from plume	16.87%	0.41***	2.04***	0.64***	14.28***
		(0.08)	(0.19)	(0.21)	(2.87)
100–250 km from plume	12.12%	0.53***	2.55***	1.02***	19.18***
		(0.08)	(0.21)	(0.25)	(3.02)
0–100 km from plume	8.59%	0.75***	3.24***	1.21***	20.93***
		(0.09)	(0.25)	(0.26)	(3.20)
Light smoke	5.71%	1.91***	5.24***	1.33***	22.74***
		(0.12)	(0.30)	(0.29)	(3.34)
Medium smoke	1.38%	2.72***	6.85***	1.63***	18.80***
		(0.22)	(0.53)	(0.43)	(4.06)
Thick smoke	0.57%	5.48***	14.39***	1.42***	17.38***
		(0.51)	(1.43)	(0.54)	(4.95)
Average daily effect of smoke		0.41***	1.65***	0.51***	9.70***
		(0.05)	(0.12)	(0.14)	(1.92)
Mean daily outcome (ref. category)		9.0	9.1	122.8	1,408.8
Sample size	14,705,570	4,410,645	3,321,357	14,594,910	14,591,808

Notes: The table reports the sample frequency and estimated effects of the smoke shocks examined in the paper. Column (1) shows the population-weighted sample frequency of each smoke shock category. Columns (2)–(5) represent separate regressions of equation (2). Columns (2)–(3) report the first-stage effects of smoke on one- and three-day $PM_{2.5}$, respectively. Columns (3)–(4) report the reduced-form effects of smoke on three-day mortality and ER visits, respectively. In all regressions, the ">1,000 km from plume" category is used as the reference (omitted) category in the regression. The average daily effect of smoke, reported below the shock-specific estimates, is calculated as the weighted sum of these estimates, each multiplied by its corresponding sample frequency. Observations are at the county-day level, and all regressions are weighted by the Medicare population in each county on each date. Standard errors are clustered at both the county and date levels. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Table A.2: $PM_{2.5}$ impacts on mortality: smoke versus wind direction IV

	(1)	(2)	(3)	(4)
	2SLS excluded instruments:			
	All smoke shocks	Small smoke shocks	Large smoke shocks	Wind direction
$PM_{2.5}$	0.43***	1.6***	0.085	0.69***
	(0.091)	(0.38)	(0.071)	(0.061)
Cragg-Donald F-statistic	4721.9	1104.4	7318.5	298.1
Mean outcome	358.9	358.9	358.9	384.6
Sample size	14,594,910	14,594,910	14,594,910	1,980,549

Notes: The table reports smoke and wind direction 2SLS IV estimates of the relationship between one-day $PM_{2.5}$ ($\mu g/m^3$) and mortality (three-day deaths per million). Columns differ in the instruments used to identify variation in $PM_{2.5}$. Columns (1)–(3) use smoke shocks as instruments and are estimated like columns (1)–(3) of Table 1 except that the endogenous variable is one-day $PM_{2.5}$. This is to align with the endogenous pollution measure used in the wind direction IV analysis of Deryugina et al. (2019), whose focal IV estimate (Table 2, Panel B, column (1)) is reproduced in column (4). Smoke IV estimates in this table are larger than the ones in Table 1 because the first-stage effects of smoke are smaller for one-day $PM_{2.5}$ than for three-day $PM_{2.5}$ (Table A.1), borrowing intuition from the just identified IV setting (i.e., a single instrument) where the IV estimate equals the ratio of the reduced form coefficient to the first stage coefficient. Observations in both the smoke and wind direction analyses are at the county and date level, but there are some differences in the sample periods and control strategies (see Deryugina et al. (2019) for details). For each IV regression, we report the Cragg–Donald (1993) first-stage F-statistic, the statistic reported in Deryugina et al. (2019). Standard errors for the smoke IV estimates in columns (1)–(3) are clustered at the county and date levels. The standard error for the wind direction IV estimate in column (4) is clustered at the county level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.