Air Quality, Mortality, and Perinatal Health: Causal Evidence from Wildfires[*†]

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Abstract

I demonstrate how scientific models of pollution processes can be leveraged in quasi-experimental econometric designs to credibly estimate the impacts of environmental quality on health or other outcomes while also improving precision and external validity over previous approaches. I simulate the geographic distribution of fine particulate matter (PM2.5) caused by wildfires for the entire continental United States during 2004-2010 using a set of scientific models of wildfire emissions and air pollution transport commonly used wildfire and air quality applications. Regressing observed concentrations of PM2.5 at pollution monitoring stations on simulated PM2.5 from wildfires, I find that wildfires can explain at least 15 percent of ambient ground-level PM2.5 and even larger fractions of toxic mercury and lead particulates. I then regress county-level health outcomes on station-measured PM2.5 using simualted wildfire PM2.5 as an instrumental variable. I find that a $10\mu gm^{-3}$ (approximately 2.3 standard deviation) increase in monthly PM2.5 concentration is associated with one additional premature death per 100,000 individuals. This effect is driven primarily by deaths from cardiovascular and respiratory diseases for individuals over age 65. With a control function approach, I find evidence that dose response is approximately linear below the U.S. ambient air quality standard for PM2.5. In addition, I find that in-utero exposure to PM2.5 is associated with higher rates of prematurity, lower birth weights, and changes in the sex ratio, which I interpret as evidence of fetal attrition. Finally, I show that the estimated health effects of PM2.5 are sensitive to the inclusion of controls for other pollutants. I present suggestive evidence that this sensitivity reflects a heterogeneous response to metallic particulates, which are known to be particularly detrimental to health. These findings contribute to a growing body of evidence on the health dangers of fine particulate matter.

1 Introduction

In the past 40 years, ambient air quality regulation has grown in response to the burgeoning evidence of the public health costs of air pollution. The Clean Air Act Amendments of 1970, the establishment of the Environmental Protection Agency (EPA), and subsequent refinements of air quality standards have all contributed to general downward trends in pollution levels. While the health benefits of air quality improvement are uncontroversial at the highest margins of pollutant levels, the important question remains whether additional reductions will also yield health benefits, and whether those health benefits exceed the increasing marginal costs of abatement. Because pollutants are not randomly assigned and may be correlated with other determinants of health outcomes, an important scientific challenge has been to develop research designs that provide precise, unbiased, and population-representative estimates of air pollution's effects.

In this paper, I exploit quasi-random shocks to ambient fine particulate matter (PM2.5) concentrations generated by large wildfires across the United States to estimate effects on mortality and infant health outcomes. Wildfires are uncontrolled fires primarily occurring in remote wildnerness areas, but cause significant variation in urban particulate levels through mechanisms that are plausibly unrelated to non-pollution determinants of health. First, I quantify the effect that wildfires have on air quality by applying a sequence of specialized emissions and dispersion models to historical fire data to generate measures of wildfire pollution for the continental U.S. over time. Then, I estimate the effects of short-term and *in utero* exposures on adult mortality and infant health outcomes, using modeled wildfire PM2.5 as an instrumental variable for station-observed PM2.5. I use extensive pollution monitoring data—spanning 60 PM2.5 subspecies and 18 criteria pollutant and organic gases—to decompose the shock to air quality represented by the wildfire PM2.5 instrument and present a methodology for assessing potential bias from omitted pollutants.

The U.S. Environmental Protection Agency (EPA) has identified six airborne "criteria" pollutants to be regulated under the Clean Air Act that are generally considered harmful to public health: particle pollution (PM2.5 and PM10), Carbon Monoxide (CO), Nitrogen Dioxide (NO2), Ozone (O3), Sulfur Dioxide (SO2), and Lead (Pb). This study estimates the effects of PM2.5 generated by wildfires and tests whether these estimates potentially reflect the effects of other criteria pollutants. Fine particulate matter, defined as particulate matter less than 2.5 micrometers in diameter (PM2.5), is considered most dangerous because of its ability to penetrate deep into the human lung and sometimes enter the bloodstream.

I make several contributions to the literature on both the health effects of air pollution and air quality effects of wildfires. First, I systematically assess the impact of large wildfires ($\geq$ 1000 acres) on ground-level air quality in the continental United States. I combine estimates of source emissions with an atmospheric model to retrospectively forecast the spatial distribution of wildfire-related pollutant concentrations in the period following a historical fire event, using the resulting data to predict pollutant concentrations at air pollution monitors. For 78 pollutants, I estimate a set of lower bounds for the percentage of each pollutant's average ambient concentration that is attributable to wildfires; notably, wildfires contribute at least 15% of ambient aggregate PM2.5, 5% of PM10, 5% of O3, and large fractions (15%-35%) of several dangerous metals bound to fine particulates, including arsenic, lead, mercury, nickel, and cadmium. In addition to the aggregate quantities of particulate pollutants they generate, wildfires cycle metallic and other highly toxic industrial emissions previously deposited into wildland vegetation and soils back into the atmosphere, resulting in new ground-level exposures in population centers. These findings underscore the potential significance of wildfires' contribution to public health and that socially optimal fire management policies must take wildfires' health costs from worsened air quality into account. Furthermore, I find that 75% of geographic exposure to wildfire PM2.5 occurs outside of the state of origin, raising the possibility wildfire management policy in the U.S. may be inefficient due to inter-state externalities.

Next, I estimate the effect of average monthly PM2.5 exposure on county-level mortality rates for 2004-2010 via

instrumental variables, controlling for weather variables and stringent sets of region-specific fixed effects. Short-term exposure to PM2.5 is associated with mortality with magnitudes consistent with prior literature, and the dose response is approximately linear below regulatory limits of PM2.5. A transitory $10\mu gm^{-3}$ increase in a county's average monthly PM2.5 (approximately a doubling of average ambient concentrations in the sample period) is associated with one additional death per 100,000 individuals. These effects are largely driven by cardiovascular and respiratory fatalities, but PM2.5 is also associated with general disease-related causes of death. Nearly all short-term PM2.5-related deaths are of individuals over age 65, and women are twice as susceptible as men. Because wildfires also emit large quantities of several gaseous pollutants, I attempt to control for potentially correlated pollutants by including a set of modeled controls for NO2, SO2, NH3, and VOC gases. I am unable to decisively rule out confounding effects from O3, even conditional on all controls, which are on the order of 35% of the estimated effect of PM2.5 based on an estimate from another study. Finally, I estimate the effects of prenatal exposure to PM2.5 on premature birth rates, birth weight, and sex ratios, finding small but statistically significant harmful effects. I also find marginal evidence for negative effects on the fraction of males in a birth cohort, and my estimates are consistent with higher susceptibility of male fetuses to death from pollution shocks estimated in Sanders and Stoecker (2011).

Controlling for non-PM2.5 emissions from wildfires results in a different composition of PM2.5 that more heavily favors toxic species, and I find larger effects in the presence of higher fractions of metals and lower fractions of non-metal particulates. While the intrusive quality of PM2.5 is the basis for the proposed dangers of PM2.5, there is wide heterogeneity in the chemical composition of PM2.5 and some evidence of heterogeneous effects, but relatively little understood about the relative toxicities of individual substances (Bell 2012). PM2.5 is composed of a wide range of substances, including elemental carbon (EC), organic carbon (OC), nitrates (NO3-), sulfates (SO42-), and metals bound to particulates (such as mercury and lead). Some of these are formed or released directly from an emission source (commonly EC, OC, and metals) and others are formed through chemical reactions in the atmosphere (e.g., nitrates and sulfates). Composition also varies widely by region and over time from cross-sectional differences and seasonal differences within regions (Franklin et al. 2008). This heterogeneity presents problems for the effective regulation of particulate levels, as small exposures of highly toxic species are potentially as dangerous as large exposures of EC, OC, or other species that account for most of PM2.5 mass. Relatedly, it presents statistical challenges for interpeting estimated effects for PM2.5. When controls for non-PM2.5 pollutants from wildfires are included, estimated effects on mortality increase by over two times. For infant health, effects approximately double for on prematurity, gestational age, and average birth weight. I interpret this pattern of estimates as evidence that the conditional mixture of PM2.5 identified has increased toxicity that exceeds any reduction in upward bias accomplished by adding controls.

This work attempts to make new methodological and evidentiary contributions to the already-large and diverse economic literature on the health effects of pollution. For short-term health outcomes, panel studies and regional natural experiment studies are two popular research designs. The widely-accepted truism motivating most of the contemporary air pollution literature is that pollution exposure is non-randomly assigned and systematically related to other determinants of health outcomes. Panel studies, such as Currie and Neidell (2005), attempt to address this non-random assignment through exploiting narrow variation through stringent fixed effects. Natural experiment studies try to provide a source of quasi-random assignment by isolating the variation they use to a particular type of pollution-generating (or reducing) event. Strategies have included exploiting the timing of the Clean Air Act of 1970 to predict relatively sudden decreases in particulate concentrations (Chay and Greenstone 2003); changes in daily airport traffic congestion in California caused by weather in other major airports (Schlenker and Walker 2011); weekly panel variation in automobile traffic to identify the effects of carbon monoxide, ozone, and particulate matter on infant mortality rates (Knittel, Miller, and Sanders 2011); and temperature inversions in Mexico City (Arceo-Gomez et al. 2011). Currie et al. (2013) provide an extensive survey of both types of papers exploring the effects of early-life exposure

to pollution, finding a general consensus that airborne pollutants are associated with infant mortality, premature birth, and low birth weight. Papers applying natural experiments to adult mortality have been more infrequent. Chay et al. (2003) uses the timing of the Clean Air Act of 1970, finding insignificant effects on adult and elderly mortality. Pope et al. (2007) use an 8-month national strike of copper smelter workers to estimate the effect of sulfate particulate reductions, finding a 2.5% reduction in mortality over the strike time period.

Several papers have attempted to estimate health effects of major wildfire events, implicitly taking their exposure measures as proxies for pollution shocks. Jayachandran (2009) examines sharp increases in particulate pollution from an intense wildfire season in Indonesia in 1997, tracking spatial and temporal variation in pollution from wildfires using satellite-based measures of particulate levels. She finds evidence that prenatal smoke exposures during that period caused a substantial increase in early-life mortality, on the order of a 20 percent increase in the under-age-three mortality rate. Breton, Park, and Wu (2011) estimate that prenatal exposure to high PM2.5 concentrations from a week-long wildfire event in California was associated with an 18g decrease in mean infant birth weight in comparison to counties unaffected by the fires.

Few studies have used modeled exposures from large emission events based on atmospheric transport models, and none have used exposures in tandem with monitoring data to predict health outcomes.[1] Rappold et al. (2012) use modeled wildfire exposures in North Carolina to assess increases in asthma and congestive heart failure risks with reduced-form Poisson regressions. I contribute to an open space in the literature by incorporating developments in emissions and atmospheric transport modeling and taking advantage of substantial increases in computational power made over the last decade. I unite quasi-random variation in pollution levels predicted from wildfire and atmospheric models with observed pollution levels in a panel econometrics framework to estimate health effects, providing a methodology that bridges some of the long-standing gaps between the atmospheric science, epidemiology, and economics literatures on air quality.

2 Data

2.1 Modeled Wildfire Air Pollution

Combining historical wildfire event data and meteorology with scientifically relevant fire and atmospheric transport models, I generate a high-resolution, gridded daily measure of wildfire pollution for the continental U.S. (CONUS) domain. The measure represents a retrospective forecast of where pollution from documented fire events would be likely to have traveled given what is known about atmospheric behavior during and after the fire. To this end, I use the BlueSky Framework software package, which integrates several existing models of emissions and transport processes into a unified process.

2.1.1 Modeling Tools

The interface between wildfire management and air quality standards has prompted extensive development of tools in the last two decades to appraise the downwind impacts of wildfires. Beginning in 2003, the National Oceanic and Atmospheric Administration (NOAA) developed and implemented the Smoke Forecasting System (SFS) to provide operational forecasts of wildfire PM2.5 (Rolph et al. 2008). A central tool in the NOAA SFS is the BlueSky Framework

[1] A class of study distinct from this one combines modeled exposures with pre-existing estimates of health risks to determine population-wide impacts. For example, Caiazzo et al. (2013) use the Community Multiscale Air Quality (CMAQ) model combined with the U.S. National Emissions Inventory for 2005 to create an annual predicted map of average pollution concentrations, and interpret this as a measure of long-term pollution exposure. Also, several studies use observed changes in particulate measurements and only employ "backward trajectory" calculations to indirectly verify that large changes are due to a specific event, such as wildfires or dust episodes.

(BSF), a modeling framework which connects independently developed models of fuel loading, fire consumption, fire emissions, and atmospheric transport (Larkin et al. 2009). The BSF has also been used in development of regional forecasting systems in the Pacific Northwest (O'Neill et al. 2009). The BSF readily accommodates several popular models of each component of the modeling process.

The Fuel Characteristic Classification System (FCCS) is a 1km-resolution spatial map of fuelbed types across the continental United States developed from a combination of fuel photo series, scientific literature, satellite imagery, and expert opinions (Ottmar et al. 2007). CONSUME 3.0 predicts how the amount of fuel consumption for a given fire event divides between flaming, smouldering, and residual phases, each of which have unique contributions to emissions due to differences in combustion efficiency (Prichard et al. 2005). The Fire Emissions Production Simulator (FEPS) is a software module that simulates emission production and plume buoyancy based on a provided consumption profile (Anderson et al. 2004).[2] These three modules have all been used, via the BSF, in the development of national fire emissions inventories since 2008. Lastly, the Hybrid Single-Particle Lagrangian Integrated Trajectory model (HYSPLIT) is a system which uses gridded meteorological data to simulate airmass trajectories, dispersion of concentrations from pollutant plumes, and deposition processes (Draxler and Hess 1997). In addition to being used in the NOAA SFS, HYPSLIT has been used in hundreds of applications, such as modeling fallout dispersion from the Fukushima Daichii nuclear disaster (Draxler et al. 2013), African dust transport to the Iberian peninsula (Escudero et al. 2006), and dispersion of particulate heavy metals from industrial emission sources in Spain (Chen et al. 2013).

2.1.2 Description of Wildfire Emissions and Air Pollution Modeling

Wildfires can be started by lightning strikes or direct sunlight when highly flammable fuels (e.g., forest underbrush) endure an extended dry period. Wildfires are also caused by human errors, such as escaped campfires, car accidents, or downed power lines. Occasionally, they are intentionally set by arsonists. Fires are also intentionally set by fire management agencies to preemptively burn fuels for naturally-occurring fires, among other functions. Wildfire incidence peaks in mid-to-late summer, but has varying seasonal peaks by region. The majority of large wildfire events (over 1,000 acres in size) occur in the Western and Northwestern United States.

There are several phenomena which contribute variation to the amount of wildfire-generated pollution at a given point in time in space. Broadly, these are the characteristics of the fire and the meteorological conditions at the time of and shortly after the fire event. The duration of the fire is a function of time till detection, containment efforts, and the containment difficulty of the fire. Besides its role in promoting the rate of spread and ultimate size of a fire, the fuel cover determines the volume and chemical composition of emissions from the fire per unit of area burned. Wildfires' dominant emissions by mass are PM10, PM2.5, CO, and NOx. In addition to PM2.5 generated by biomass burning, such as Organic Carbons (OC), wildfires release minerals and metals which accumulate in forest soils and vegetation from atmospheric deposition. Nearby historical industrial activity is strongly related to the amount of lead and mercury re-released by fires into the atmosphere, with these re-emissions representing a significant fraction of atmospheric concentrations.

Once generated, emissions travel upward at varying speeds depending on a variety of factors, resulting in a heterogeneous vertical distribution of pollutants in a fire. This vertical distribution then interacts with ambient pressure and wind conditions which result in airborne transport of emissions downwind. Emitted particles (and gases) interact with weather conditions heterogeneously, resulting in relative downwind changes in concentrations that vary by pollutant. Dry deposition is a set of processes by which pollutant concentrations decrease through contact with surfaces, which include gravitational settling and interception (collision with trees, buildings, etc.). Wet deposition is a set of processes by which atmospheric hydrometeors (e.g., precipitation) absorb particles.

[2]FEPS is capable of fuel consumption calculations, but this functionality is replaced by CONSUME 3.0 in this modeling process.

State and federal agencies responsible for wildfire management keep records on the location, size, and timing of wildfire events. Fire events larger than 1,000 acres are gathered from the Fire Protection Agency (FPA) Fire Occurrence Database (FOD), an interagency collection of fire event reports updated for accuracy and cleaned for duplicates using methods described in Short (2013). The fire event characteristics drawn from this database for modeling are the latitude and longitude point data of the fire, date and time the fire was detected, area of the fire burned in acres, and the date and time at which a fire agency declared it contained. For an available subset of federal fires, I draw the date and time at which a fire agency declared the fire extinguished from a U.S. Geological Survey database of fires reported by the six major federal agencies tasked with managing wildfires. If any of the values except for the containment or extinguish dates are missing, the fire is omitted. Where time of extinguishment data are missing, I empirically estimate the total burn time using a regression model with categorical dummies for the fire area and the duration from start to containment as predictors, adjusting for region-specific unobserved effects and seasonal effects; where both containment and extinguishment dates are missing, I use the same model without containment time to predict burn duration. The methodology, rationale, and results of the total burn time estimation procedure are described in Appendix 8.1. Figure 1 shows the total number of acres burned in fires larger than 1,000 acres mapped by state for 2000-2010, as calculated from the FPA FOD database. The majority of area burned is concentrated in the West, Northwest, and Southwestern United states, with a decreasing eastward and strongly decreasing Northeastward pattern. These large fires constitute over 85 percent of area burned in the United States over this period. Fires smaller than 1,000 acres are a significantly larger percentage of area burned for Northeastern and Central states, but are not included because of high computational costs of the modeling process relative to their small total emissions contributions compared to larger fires.

I utilize a sequence of wildfire models that exploit several facets of these wildfire emissions and pollution transport processes to predict the contribution to PM2.5 levels from wildfires and explicitly describe the computational workflow in Appendix Section 8.2.1; Figure 2 depicts the workflow visually. Historical fire events are input into the BlueSky Framework, where fuel loadings, fuel consumption, emissions, and vertical plume rise are estimated; these are fed as emission sources into HYSPLIT, which calculates the concentations' trajectory and dispersion from each source; hourly spatial concentration estimates are calculated at an approximately $1600 km^2$ resolution (approximately 5,000 unique points in CONUS); then, the HYSPLIT predicted concentrations are sampled at pollution monitoring station locations and averaged by county and month to create a monthly panel of county averages of wildfire pollution.

2.2 Birth and Mortality Data

Data on the population of births, linked infant deaths, and mortality events in the United States for 2004-2010 come from the U.S. Center for Disease Control's (CDC) National Center for Health Statistics' (NCHS) National Vital Statistics System (NVSS). Data sets contain all non-identifying information recorded on birth and death certificates. Each birth record contains the year and month of the birth event in addition to important perinatal health outcomes, such as birthweight, Apgar scores, estimated gestation, birth complications, and characteristics of the mother and father of the child. Table 3 summarizes these outcomes by gestational category (full-term and pre-term). The mortality data contain individual death records, which include the year and month, county, cause of death, and characteristics of the deceased individual (race, gender, and education). For 2005 and beyond, county identifiers are censored for all counties with fewer than 100,000 individuals.

Causes of death are coded into 39 groups, in accordance with the latest classifications of the International Statistical Classification of Diseases and Related Health Problems (ICD-10). County-by-month mortality rates for each cause are calculated by summing counts from the 34 categories causes of death, including cancers, heart failure, respiratory disease, and other diseases and dividing by a population measure. The population estimates used to calculate rates per

100,000 individuals are from the CDC NCHS Bridged-Race Population Estimates, a set of annual intercensal county population estimates with breakdowns by sex, age, and race. I generate an "all-cause" rate from all non-external, non-accidental causes of death for by the general population, gender, and infant, child, and 10-year age groups. Table 2 reports summary statistics for mortality rates in the sample.

2.3 Ambient Air Pollution and Weather Data

Daily average monitoring station observations of pollutant levels are gathered from the U.S. Environmental Protection Agency's Air Quality System (AQS), a centralized database of pollutant measurements from state and federal monitors. The geographic and temporal distribution of measurements varies widely by pollutant. The PM2.5 Chemical Speciation Network provides measurements of PM2.5 subspecies of interest, such as metals and nitrates. Some stations collect data at weekly rather than daily frequency. For county-months with missing station-days, I use the average of nonmissing observations by first averaging to monthly station observations, and then averaging station-month values to county-month values. County-months with no station observations are excluded from the sample. For birth and death outcomes, I define the mother's and decedent's county of residences, respectively, as the aggregate geographic units for calculating pollution exposure. For local weather measures, I use data from the North America Land Data Assimilation System on average monthly daily maximum and minimum air temperatures and monthly precipitation quantities for each U.S. county. These data were drawn from the CDC WONDER database. This data source is distinct from the meteorological reanalysis data used as inputs into the pollution transport model.

3 Econometric Approach

3.1 Statistical Model

I consider the following linear model of health outcome y_{it} with a $K \times 1$ vector of endogenous variables representing pollution levels, P_{it}, and a set of unobserved effects:

$$y_{it} = P_{it}\beta + R_{it}\psi + \alpha_i + g_{it}(t) + \varepsilon_{it} \tag{1}$$

$$P_{kit} = z_{it}\gamma_k + R_{it}\psi_k^f + \eta_{ki} + f_{ki}(t) + v_{kit} \tag{2}$$

$$g_{it}(t) = c_{i,y(t)} + s_{i,m(t)} + \tau_i\omega(t) \tag{3}$$

$$f_{ki}(t) = c_{ki,y(t)}^f + s_{ki,m(t)}^f + \tau_{ki}^f\omega(t) \tag{4}$$

Equation (1) shows the relationship between the health outcome (e.g., mean birthweight) y_{it} and pollutants P_{it} for county i in month t. R_{it} is a set of time-varying county characteristics, α_i represents a county fixed effect, and $g_i(t)$ generally represents time-varying unobserved heterogeneity. ε_{it} is an idiosyncratic error term which may generally be correlated with P_{it}. Equation (2) represents the first stage relationship between pollutant k and the vector of at least K excluded modeled wildfire pollution instruments, z_{it}, with a set of fixed effects η_{ki} and $f_{ki}(t)$ matching those in equation (1). Equation (3) defines $g_i(t)$ as the sum of sets of region-year fixed effects $c_{i,y(t)}$, region-month (seasonal) effects $s_{i,m(t)}$, and arbitrary regional time trends $\tau_i\omega(t)$. $y(t)$ and $m(t)$ are functions which convert the global time index t to the correct calendar year (e.g., 2004) and calendar month (e.g., July) indices. "Region" can generally refer to any

geographic unit which hierarchically nests counties, including counties, states, and NCDC climate regions. Equation (4) defines $f_{ki}(t)$ in parallel to (3) for P_{kit} (except naturally requiring that fixed effects vary by pollutant k).

Region-year fixed effects account for annual trends in the health outcome including those driven by changes in pollution from sources other than wildfires. This includes region-specific climatological changes and regulatory responses to wildfire incidence or pollution, which might simultaneously affect both wildfire incidence and health outcomes. Region-month fixed effects account for unobserved persistent seasonal differences between regions, such as weather patterns that drive seasonality in wildfire incidence and health outcomes. Including fixed effects increases the plausibility of the assumption that the instrument is exogenous in equation 1; namely, that $E[\varepsilon_{it}|z_{it}, R_{it}, \alpha_i, g_{it}(t)] = 0$.

3.2 Identification

The commonly shared movement of air parcels explains most of the spatiotemporal variation in all pollutants from wildfires simulated by HYSPLIT. However, fuel loadings, wet deposition, and dry deposition generate some independent variation among pollutant types that can separately identify their effects. The possibility of separate identification of pollutants breaks down as the pollutants become more similar in the ways that HYSPLIT is able to distinguish them. A corollary of this is that even a perfectly calibrated pollutant instrument will also proxy for the effects of its unmodeled close chemical neighbors. In this paper, independent variation in PM2.5 is uniquely identified primarily by differences in fuel combustion efficiency and the large difference in deposition rates between PM2.5 and gases. Interpretation of the estimated effects is complicated by heterogeneous effects, especially those driven by the chemical composition of the PM2.5 that is statistically identified. These problems hold true for nearly any attempt to identify the effects of PM2.5 by isolating exogenous changes.

The structural model of atmospheric transport represented by HYPSLIT seamlessly combines trajectory and dispersion calculations in space with pollutant removal from the atmosphere through deposition processes, which has several advantages over some commonly-used methods. Some studies have implicitly exploited atmospheric phenomena and pollutant characteristics through regression interactions. For example, Schlenker and Walker (2011) interact instrumented taxi time with wind speed to separately identify CO and NO2, which may be explained by differing dry deposition rates between CO and NO2. NO2 has a higher deposition velocity than CO. An alternative explanation they supply is that higher wind speeds change the composition of emissions from airplane engines to be more NO2-heavy. Assuming a fixed emission ratio of CO to NO2, higher wind speeds will carry parcels of both pollutants equally far but deposit more NO2 than CO, resulting in an increasing ratio of CO to NO2 in distance from the airport. This fact would be captured by HYSPLIT's deposition modeling process. Moreover, the wildfire modeling method explicitly models differences in emissions ratios using fuel types. Wet deposition processes, which are strong sources of variation in airborne particulate concentrations, enable Bharadwaj and Eberhard (2008) to identify significant reductions in PM10 using rainfall quantity as an instrument. HYSPLIT uses an approximation based on a dynamic washout process calibrated by user parameters, instead, providing a mechanistic interaction between the horizontal and vertical distributions of pollutants and the location and intensity of rainfall. While regression interaction-based identification methods are practical and easy to implement, they introduce complications in interpreting estimates beyond the concerns of omitted variables biases plaguing most methods. A purely regression-based approach makes diagnosing the role of heterogeneous effects more difficult, since an arbitrary set of instruments which are incidentally (or loosely scientifically) exploited for identification may identify a contextualized subset of the pollutant's variation and hence an unpredictable local average treatment effect (LATE) in the presence of heterogeneous effects. The modeling methodology used here helps illuminate the source of identifying variation by explicitly unpacking the "black box" of physical phenomena driving independent identification of pollutant levels (e.g., differential responses of pollutant concentrations to rainfall).

While moderating relationships and nonlinear effects may drive the detection of a LATE, the salient cause of heterogeneous effects for particulate matter is its heterogeneous chemical composition. Because of changes in the toxicological properties of the PM2.5 whose effects are being measured, the interpretation of changes in effect estimates across different identification strategies is potentially ambiguous, even when the regions and emissions sources being studied are identical across estimation methods. An intuitive confounding story for harmful pollutant A with a homogeneous health effect would be that its effect is biased upward by harmful pollutant B co-emitted from wildfires, implying that including controls for pollutant B would make the estimated effect of pollutant A smaller in expectation. This property does not always hold if there are heterogeneous effects. Specifically, heterogeneous chemical composition may result in some controls removing the statistical influence of some subspecies of pollutant A in favor of more harmful ones. A $1\,\mu gm^{-3}$ increase in ambient PM2.5 induced by general wildfire PM2.5 will have a smaller marginal health impact than a $1\,\mu gm^{-3}$ increase of wildfire-emitted PM2.5 subspecies with above-average toxicity. Indeed, in Section 4.2 I find that controlling for other wildfire pollution species more than doubles the estimated average effect on mortality. Controlling for another wildfire pollutant eliminates variation from emissions and atmospheric trajectory components common between the control pollutant and PM2.5 in the identification of the PM2.5 coefficient, resulting in greater weight on identification idiosyncratic to the fuel type at the fire and deposition behavior. For example, Organic Carbons (OC), common byproducts of primary combustion, are a large constituent of wildfire emissions by mass across all fuel types and have a large part of their influence removed by including any other wildfire pollutant controls due to their commonality to all fires. Furthermore, the deposition parameters chosen for PM2.5 place relatively more weight on PM2.5 species whose deposition characteristics mimic the chosen parameters most closely. This has ambiguous estimation consequences without further investigation of the distribution of emission deposition characteristics across PM2.5 subspecies. To address this ambiguity empirically, I examine how the identifying composition of PM2.5 varies by specification using monitoring data for over 60 subspecies of PM2.5 in Section 4.1.2.

3.3 Estimating, Testing, and Controlling for Effects from Multiple Pollutants

In the ideal scientific setting, one would have a large enough dataset with measurements of all species of interest with identifying instruments for each species and estimate the effects of multiple endogenous variables using 2SLS or otherwise appropriate IV estimator. In reality, station coverage is limited to fewer than 20% of county-month observations in the sample period, and further limited when overlapping species measurements are required. The generation of strong identifying instruments may be both scientifically constrained by the quality of models and practically constrained by computational power. In lieu of the ideal estimation of all pollutants' coefficients, it is feasible to consistently estimate a single structural parameter of interest (in this case PM2.5's effect) without any concern for the structural parameter values for other pollutants. Generally, evidence for a consistent estimate of the effect of PM2.5 can be established by exhausting potential confounding causal pathways through a combination of control variables and pre-testing for omitted variables.[3]

Under the assumption that estimates using the wildfire pollution instrument will reflect effects causally originating with wildfire events only, the primary risk of confounding comes from omitted pollutants which are correlated with the wildfire instrument. A measure of downwind PM2.5 from a wildfire will be correlated with other pollutants emitted concurrently in the same fire's combustion processes, which will also share at least some of its atmospheric trajectory. For example, wildfires simultaneously emit quantities of PM2.5 and NO2, and their atmospheric destinations are highly correlated. In this framework, the health-effect parameter for PM2.5, β_{PM25}, can be identified either through joint IV estimation of all pollutants, or through single-variable IV estimation of PM2.5 alone with controls for

[3]Causal pathways can also be credibly ruled out using evidence from rigorous studies that find no effects of an omitted explanator on the outcome of interest, but I do not do this here.

pollutants from the same source. This equivalence is motivated by writing the reduced form for equation 1 as follows, only substituting the first stage for endogenous PM2.5 using only wildfire PM2.5 as an instrument:

$$y_{it} = z_{pm25,it}\eta_{pm25} + P_{B,it}\eta_B + \Upsilon_{k,it} + \varepsilon^*_{it} \tag{5}$$

$z_{k,it}$ is defined as pollutant k originating from wildfires. $P_{B,it}$ is the vector of all pollutants excluding PM2.5 originating from all sources. For brevity, define $\Upsilon_{k,it}$ as the composite set of controls and effects and ε^*_{it} as a composite error term for equation 1. Partition each pollutant k into its concentration from wildfires and its concentration from all other sources, defining $P_{B,it} = z_{B,it} + \tilde{P}_{B,it}$. Then,

$$y_{it} = z_{pm25,it}\eta_{pm25} + z_{B,it}\eta^{wf}_B + \Upsilon_{k,it} + \tilde{P}_{B,it}\tilde{\eta}_B + \varepsilon^*_{it} \tag{6}$$

Because wildfire PM2.5 in part shares common emission and transport processes with other pollutants from fires, PM2.5 and other wildfire pollution are correlated: $E[z_{B,it}\,|\,z_{pm25,it}, \Upsilon_{k,it}] \neq 0$. Uncontroversially, $E[\tilde{P}_{B,it}\,|\,z_{pm25,it}, \Upsilon_{k,it}] = 0$ is a core assumption for the validity of the instrument; wildfire PM2.5 must be orthogonal to any pollutants in B from all non-wildfire sources. The reduced-form regression of y on z_{pm25} will be inconsistent for η_{pm25}. However, $z_{B,it}$ is observed by virtue of the same modeling process that generates z_{pm25}, and the reduced-form regression of y on z_{pm25} and z_B produces a consistent estimate for η_{pm25}. Correspondingly, provided the other key assumptions for the consistency of IV are met, IV estimation of y on P_{pm25} and z_B with (z_{pm25}, z_B) as instruments is consistent for β_{pm25}. While both the joint IV estimation and the single-variable IV procedures will be consistent for β_{PM25}, single-variable IV is far more feasible to implement; it only requires station observations of PM2.5, an instrument for PM2.5, and adequate controls for correlated pollutants. In some cases, modeled pollutants may be sufficient as controls but not sufficient as identifying instruments; joint IV estimation of PM2.5 and NO2 with a strong instrument for PM2.5 and weak instrument for NO2 may result in an inferior estimate for PM2.5 compared to the corresponding consistent one-variable IV estimate for PM2.5.

An alternative solution to creating an instrument or proxy is to use the endogenous measure of the omitted variable as a control, but in the pollution setting this is not always feasible. First, measurement coverage for each pollutant species incompletely overlaps both across stations and time. Second, while the quality of station measurements for a particular species might be sufficient for determining whether wildfires have an impact on concentrations of that species in a station-by-station analysis, they may not be appropriate measures of concentrations for aggregate geographic regions used to measure health outcomes (i.e., counties in this paper). Relatedly, to the extent that station measurements (whether due to direct station mismeasurement or spatial error) fail to capture variation from wildfires due to measurement error, the control would fail to account for the influence of the omitted variable. The estimates in this paper instead use the equivalent of a proxy for pollutants emitted from wildfires as controls, thereby reducing or removing their confounding role.

It is possible to meaningfully pre-test for potential omitted variables provided there are observations containing values of both the omitted variable and the instrument. Sufficient power in the test obviates the need to create develop instruments or controls for the omitted variable if the test is negative. The test is to run a pseudo-first-stage regression of the suspected omitted variable on the current set of instruments and controls and checking whether the current instruments are jointly significant predictors of the proposed omitted variable. In practice, a researcher may not be able to develop an adequate identifying instrument or proxy for the potential omitted variable, and she might not be able to directly control for measures of the omitted variable without losing sample size (or relying on imputation methods). The creation of new instruments or proxies for new pollutant species is constrained practically by computational requirements and development time for accurate emission factors and deposition parameters. Separate identification

of pollutants is also statistically limited by the mechanical richness of the modeling process. As separate identification of pollutants in the modeling process used here is driven by differential emissions and deposition behavior, pollutants with very similar emission and deposition properties will be weakly identifiable unless some part of the modeling process is upgraded to exploit other differences in characteristics not accounted for by HYSPLIT (e.g., buoyancy, aerodynamic, or photochemical properties).

To illustrate, consider a simple two-pollutant example with Pollutant A and Pollutant B and a wildfire-generated measure of Pollutant A as an identifying instrument. Assume we have a prior belief that Pollutant B causes mortality. If Pollutant B is positively correlated with wildfire-generated Pollutant A, then an instrumental variables regression of mortality on Pollutant A with wildfire-generated Pollutant A as an instrument and no control for Pollutant B will be biased upward due to the confounding effect of Pollutant B. Hence, a pseudo-first-stage regression of Pollutant B observations on wildfire-generated Pollutant A which produces a significantly positive coefficient on wildfire-generated Pollutant A is interpreted as evidence of this upward bias (in context of the prior belief that Pollutant B has an effect on mortality).

This test for omitted variables holds under one additional assumption: the direction, but not necessarily the magnitude, of the average partial effect of the instrument is the same between the samples used for testing and estimation. If the instrument is monotonically related to the endogenous variable of interest for the population, this assumption is satisfied. For the relationship between wildfire-generated pollution to observed pollution, these assumptions are likely to hold. While there may be first-stage heterogeneous effects of the modeled wildfire-generated pollution (either due to heterogeneous modeling error or because of true heterogeneity in the world due to chemistry or other processes), I assume that effects are bounded by zero. With the exception of a few highly reactive pollutants and/or pollutants with low atmospheric quantities, wildfire pollution can be generally expected to homogeneously weakly increase (or decrease) each pollutant type across geographic location and time. Let superscript A and superscript B denote that the variable is drawn from estimation sample's and testing sample's subpopulations, respectively. The assumption can be written as

$$\frac{\partial E(P_{it}^{A}|z_{it}^{A},R_{it}^{A},\eta_{ki}^{A},f_{ki}^{A}(t))}{\partial z_{it}^{A}}>0 \iff \frac{\partial E(P_{it}^{B}|z_{it}^{B},R_{it}^{B},\eta_{ki}^{B},f_{ki}^{B}(t))}{\partial z_{it}^{B}}>0. \tag{7}$$

In the linear case, this simply translates to the pseudo-first-stage coefficients having the same direction in each sample (i.e., $\gamma_k^A > 0 \iff \gamma_k^B > 0$). The corresponding hypothesis test is of $H_0 : \gamma_k^A = \gamma_k^B = 0$, $H_A : \gamma_k^A \neq 0$ using the t-test of $H_0 : \gamma_k^B = 0$ from the regression using the testing sample B. Because of sampling error, failure to reject the null does not rule out omitted pollutants, but the estimate's confidence interval can be informative of the largest effect that is statistically supported by the given estimate. The true coefficient in the testing sample could be substantively smaller than the coefficient in the estimation sample, in which case the confidence interval bound may be misleadingly low. A more stringent assumption, which would imply (7), is similar to the necessary assumption for the consistency of two-sample IV estimators: $\gamma_k^A = \gamma_k^B$. This assumption permits a more literal interpretation of the coefficients and confidence intervals when the omitted variables test is conducted with a set of observations that is not identical to that being used to estimate the equation of interest. I perform and interpret this test for criteria and organic gases in Section 4.1.1.

4 Results

4.1 Wildfires' Effect on Ambient Air Quality

4.1.1 First Stage: Wildfires' Effect on Ambient Concentrations of Pollutants

Wildfires have a considerable impact on urban air quality, and noticeably and dangerously so for larger wildfires close to urban centers. The wildfire PM2.5 instrument is a strong predictor of PM2.5, but also captures some of the relationship between wildfires and other criteria pollutants. For each pollutant, I regress the county-monthly average of its station values on the county-monthly average of the wildfire PM2.5 instrument (sampled at the station sites), and I control for county, state-year, and state-month fixed effects, and quadratics of average minimum temperature, maximum temperature, and precipitation. I measure the average contribution by wildfires for each pollutant's concentration in the estimation sample by calculating its partial fitted value $z_{it}\hat{\gamma}^B$, and calculate the percentage of all concentrations of that pollutant attributable to the instrument by dividing by the average measured concentration. These percentages can be interpreted as lower bounds of the amount of each pollutant attributable to wildfires in the CONUS. I repeat this procedure controlling for estimates of NO2, SO2, NH3, and organic (VOC) gases from wildfires and assess how a unit increase in the wildfire instrument predicts downwind concentrations of criteria gases, organic gases, and PM2.5 subspecies. In another specification, I control for only wildfire NO2 and SO2.

Panel A in Tables 4, 6, and 5 shows the estimated regression coefficients and percentage of average ambient concentrations contributed by wildfires for criteria pollutants, non-metallic PM2.5, and five of the most toxic PM2.5 species. Appendix Tables 16 through 20 repeat this exercise for all other metallic PM2.5 species. Under the assumption that the estimated coefficients reflect purely causal relationships, the maximum of the wildfire percentage of ambient concentration across different control pollutant specifications can be interpreted as an estimated lower bound on the true percentage of ambient concentrations caused by wildfires. Assuming the station sets are representative of the U.S., the instrument predicts nearly 15% of PM2.5 levels and 5% of PM10 levels. Controlling for non-PM2.5 species alters the distribution of pollutants predicted by the instrument, which has significant implications for health effects estimates. Panel B of the pollution regression tables report the estimated coefficients for the regression with controls for other pollutants. The wildfire instrument ceases to be a statistically (and chemically) significant predictor of PM10, while still predicting 15% of PM2.5 mass.

Interpreting hypothesis tests for these estimates as the omitted variables test described in Section 3.3 for IV estimates with no controls for other pollutants, we expect the effect identified by the wildfire PM2.5 instrument to be biased upward by any health effects of non-PM2.5 pollutants that are significantly associated with the PM2.5 instrument. Hence, PM10 and two criteria gases, O3 and NO2, are possible confounders, though the contributions predicted by the instrument for these pollutants are only 4.7%, 3%, and 5.7% of ambient levels. Organic gases are insignificantly predicted, both statistically and in magnitude. Because of sampling error, this test does not rule out that other pollutants with statistically insignificant coefficients may still confound estimates, especially if their 95% confidence interval upper bound is a quantity that could have meaningful health effects. For example, benzene is insignificantly predicted at 6% of total concentrations, but its confidence interval upper bound is 16.2% of benzene, which is arguably a quantity that could have a marginal health impact. Benzene concentrations may only be poorly detected statistically; short-lived organic gases, such as m-xylene and toluene (8 to 48 hours, Prinn et al. 1987) show neither statistically nor substantively significant effects, while benzene has a comparatively long atmospheric lifetime (2 weeks to 2 months).

Wildfires have the unique property of inducing changes in PM2.5 almost uniformly across both highly and lightly polluted areas. This property is favorable to estimating population-representative effects, since an area's non-wildfire pollution levels drives nonlinear dose response and might also be correlated with effect heterogeneity due to other

factors (e.g., highly-polluted areas also have low-income individuals who are more vulnerable to pollution shocks). Figure 4a shows a quantile-quantile plot of all PM2.5 against the estimated implied counterfactual PM2.5 (a world with no wildfire PM2.5), with each point representing the numerical values at which the same quantile occurs in each distribution. The quantile relationships are approximately parallel to the line of distributional equivalence and shifted upward, suggesting that wildfire PM2.5 largely preserves the shape of the distribution of PM2.5 and only shifts the mean. For comparison, Figure 4b shows a comparable quantile-quantile plot when the counterfactual is estimated using the same set of fixed effects and station observations (i.e., a pure panel data approach) instead of fixed effects-IV, revealing a considerably different distribution of margins of change for PM2.5 driven mostly by left- and right-tail behavior.

4.1.2 PM2.5 Chemical Composition Identified by Wildfire Instrument

The types and quantities of PM2.5 predicted by the instrument significantly change when non-PM2.5 controls are included. Section 3.2 outlined an argument for how this changes the interpretation of the effect estimate because of changes in the level of toxicity per unit PM2.5. While the total mass of PM2.5 predicted by the instrument only decreases by 10%, the fractions of subspecies groups change significantly. In the non-metallic category, Organic Carbons decrease in concentration by 60-75% per unit wildfire PM2.5, Elemental Carbons by 40-50%, and hydrogen PM2.5 by 70-85%. Bromine PM2.5 increases by 100%, and nitrates by 50%, while the influence of sulfates stays approximately the same. Several metallic PM2.5 species become more strongly represented per unit of wildfire PM2.5 by at least 50%: Arsenic, Lead, Nickel, Mercury, Cadmium, Barium, Cesium, Cobalt, Gallium, Lanthanum, Selenium, Niobium, and Rubidium. The estimated fraction of atmospheric mercury PM2.5 attributable to wildfires becomes approximately 30 percent, parallel to the fraction established in an inventory of mercury wildfire emissions in the U.S. (Wiedenmyer and Friedli 2007). Predicted arsenic increases by a factor of nearly 30, now accounting for 19 percent of ambient arsenic concentrations. Lead, Nickel, and Cadmium are also all significantly enhanced per unit wildfire PM2.5.

The speciated PM2.5 data present a fairly complete picture of PM2.5 in the U.S. Over 81% of average PM2.5 concentration is accounted for by the subspecies I model. The remaining unexplained PM2.5 concentration may be due to known PM2.5 species which I measure imperfectly or not at all (such as sea salt and dust) and differences in mean concentrations between the PM2.5 Speciation network and general PM2.5 station samples. Moreover, heterogeneous coefficients between testing and estimation samples are not likely to drive most of the results. The number of observations measuring total PM2.5 exceeds the number of observations measuring individual species by 20,000 to 30,000, driven mostly by spatial variation in station coverage. Despite the disparity in spatial sampling, the instrument's estimated effect on total PM2.5 concentration is closely matched by the sum of coefficients for individual PM2.5 species (in the no-controls case, a less than 1% difference). This suggests that any between-sample differences in the relationship between the wildfire instrument and pollutants are mean-zero across PM2.5 subspecies.

Some coefficients for metallic species are negative. The causal interpretation for negative coefficients is that something in the pollutant plume causes a chemical reaction that removes quantities of another species or its precursors (e.g., through oxidation or binding). Many metal PM2.5 species, including mercury, are defined as the metal bound to other airborne particles, such as black carbon (soot). Chemical reactions with wildfire emissions may change such metals back to their gaseous phases, or additional substances may bind to and change the particle to a larger size class. Another possibility is that the relationship is not causal. The PM2.5 instrument is generated using a set of emissions factors for all PM2.5. If there is geographic heterogeneity of subspecies emissions (e.g., aluminum, silicon, and other metals) that is negatively correlated with the total amount of PM2.5 emitted, high downwind PM2.5 values will also be negatively correlated with those metals. The final possibility is that stations' measurement methods may have some

systematic measurement error for subspecies measurements that varies with the amount of other substances in the air.

4.2 Short-term Effects on Mortality

4.2.1 Short-term Effects of PM2.5 on All-Cause Mortality

Panel A of Table 8 reports the 2SLS estimates of the effect of average monthly PM2.5 on monthly all-cause mortality rates using wildfire PM2.5 as an instrument, each column reporting a specification with a different set of fixed effects. Estimates range from 0.67 to 1.05 additional deaths per 100,000 people per monthly $10\mu gm^{-3}$ increase in PM2.5. Panel D reports OLS estimates with the same fixed effects and weather controls as the 2SLS estimates; they are insignificant and sharply estimated close to zero, reflecting the important role of exposure measurement error and omitted variables causing downward bias. Estimated effects using 2SLS increase with the inclusion of more stringent region-specific fixed effects, providing some evidence of region-specific confounders to wildfire PM2.5 such as unobserved seasonal weather factors or endogenous annual policy responses to poor air quality or high wildfire activity. It is also partially explainable by changes in the finite-sample bias of the 2SLS estimator across specifications because of relative changes in the ratio of endogeneity in PM2.5 to the strength of the first-stage relationship (see Appendix 8.5); however, confidence intervals based on the inverted Anderson-Rubin test statistic (Anderson and Rubin 1949; Finlay and Magnusson 2009) are very close to the conventional asymptotic confidence intervals, which is evidence against any meaningful bias from weak instruments. Finally, these changes can be attributable to changes in the PM2.5 composition identified by wildfire PM2.5, since different fixed effects may remove certain correspondingly invariant characteristics of wildfire PM2.5. The effect size in column 4 translates to approximately 39,230 premature deaths per year in the U.S. due to monthly exposure to PM2.5, based on the 2010 U.S. population and assuming the sample average PM2.5 of $10.6\mu gm^{-3}$ is representative of the entire U.S. I find evidence that many of these deaths are driven by forward displacement of mortality within six months in Section 4.2.3.

OLS estimates may be downward-biased because of some combination of correlated unobservables not removed by fixed effects or measurement errors (potentially worsened by fixed effects). The traditional culprits for bias, such as residential sorting, seasonality, and coincidental trends presumably have their influence removed by the stringent fixed effects imposed in each specification. The identifying variation for the OLS estimates remaining is based on within-region, within-year, within-season comparisons, with variation likely to be driven by the totality of incidental variations in PM2.5 emissions and weather patterns. Co-emission would bias estimates upward, as changes in PM2.5 emissions would likely be accompanied by changes in other pollutants. On the other hand, the activities underlying emissions of PM2.5 are likely correlated with several time-varying economic and health behavior processes, including changes in traffic, smoking and drug use, short-term health inputs, physical activity, and stressful events.

More likely is that measurement error plays a significant role in shrinking both OLS and 2SLS estimates toward zero, though the 2SLS estimate corrects this measurement error to the extent that both PM2.5 and the wildfire PM2.5 are characterized by classical measurement error. County-level averages of PM2.5 and the wildfire PM2.5 instrument are calculated from raw averages of measurements at the sites of pollution monitors, which are not always spatially representative. In the traditional errors-in-variables setup, nonzero correlation between the true value of the regressor and the measurement error (i.e., non-classical error) has different implications for bias (expression in Appendix 8.4). In the case of negative correlation large enough relative to the signal value of the mismeasured regressor, the coefficient estimate can also reverse sign. Stations tend to be located in more densely populated and plausibly more polluted areas. More densely populated areas have higher pollution but would have their aggregate exposures well-measured by local station observations. Less-densely populated and less-polluted areas will use information on PM2.5 from more highly-populated areas, resulting in overestimation of PM2.5 levels. The combination of these two factors may

result in a negative correlation between the measurement error and PM2.5 levels.

Table 11 reports estimates by age group, revealing that the observed aggregate effects are primarily driven by the three age groups over age 65. Elderly individuals are more likely to be living at vulnerable health margins, and thus are more susceptible to a relatively short-term shock to pollution cause a life-threatening health complication. Also (not reported in tables), the estimated effect is twice as large for women as it is for men. Similarly, Chen et al. (2005) find a higher increased relative risk for females for fatal heart disease and Kunzli et al. (2005) for atherosclerosis from PM2.5 exposure.

The inclusion of any of the controls for other pollutants results in a sharp increase in the estimated effect of PM2.5 on all-cause mortality by about two and a half times. Because including controls changes the breakdown of PM2.5 that is identifying the effect to favor relatively more mass from highly toxic species (as demonstrated in Section 4.1.2), we can not unequivocally expect including controls to have a net downward effect on the magnitude of health effects estimates. Hence, these estimates are prima facie evidence of large compositional effects in PM2.5. As shown in Section 4.1.2, the specifications in Panel B and Panel C reflect changes in PM2.5 composed of greater proportions of metals and nitrates than the no-control specifications in Panel A. A common finding in the epidemiological and medical literature is that PM2.5 effects are higher in the presence of metallic PM2.5 subspecies. Bell (2012) finds 15% larger PM2.5 effect estimates for cardiovascular and respiratory morbidity when ambient Nickel (N) is elevated. In a study of rats, Pozzi et al. (2003) find evidence that inflammatory response from particulates is driven by contaminants adsorpted onto particles by comparing inflammatory responses between exposures to urban-sampled particulate matter and pure black carbon.

There are also some shifts in predictions of criteria and organic gases depending on the set of pollutant controls, suggesting a potential role of changing correlations with omitted pollutants driving the increase in mortality effects. However, the loss of PM2.5 mass from carbons and gain from metals is roughly stable across estimates using different pollutant control groups; the changes in estimated mass contributions by the instrument to these gaseous species varies widely with control groups; and estimates are relatively stable across control group sets after the first control pollutant is included. While this analysis is not a substitute for joint IV estimation of all pollutants, this is evidence that most of the increases in effects are driven by a set of PM2.5 species and not from confounding by simultaneous wildfire emissions of criteria and organic gases. Despite attempts to control for O3 production by modeling its key precursor NO2, the instrument predicts approximately 1ppb of O3 per $0.1\mu gm^{-3}$ of PM2.5 predicted by the instrument across specifications. Bell et al. (2004) find a 0.52% increase in daily mortality per 10ppb increase in the previous week's O3; if this effect were true and the base mortality rate is 67.6 deaths per 100,000, then 0.35 of the 1.04 deaths per $10\mu gm^{-3}$ of PM2.5 estimated with the wildfire PM2.5 instrument and no controls are attributable to bias from O3. The estimate for O3-related bias is comparable for the effect with all non-PM2.5 controls, but relatively smaller (0.35 of 2.68 deaths).

Using a control function approach to estimate nonlinear dose response of short-term mortality, I find that the marginal effect of PM2.5 slightly declines at low concentrations (less than $5\mu gm^{-3}$) and becomes approximately linear. Previous studies using multi-city time series analyses examining short-term PM2.5 dose response have also found a roughly linear relationship below the NAAQS concentration level of $25\mu gm^{-3}$ for all-cause mortality (Schwartz, Laden, and Zanobetti 2002; Stieb et al. 2008); Daniels et al. (2000) additionally finds approximate linearity in PM10 for all-cause mortality. Piecewise regressions for an endogenous variable can be easily estimated via control function methods without the need to develop additional identifying instruments. In the control function procedure, the first-stage regression is identical to the conventional IV first stage, but the residuals from that regression are generated and used as a control variable in a regression of the outcome on the endogenous variable. Results can be made further robust to endogeneity by controlling for corresponding nonlinear functions of the control function residual, accounting

for changing correlation with the error term across the support of the endogenous variable. The only other required assumptions are mean independence of the instrument from the structural error and that the distribution of the first stage is correctly specified; in the case of wildfire pollution, the endogenous explanatory variable is continuous and its relationship to the instrument is conceptually linear. Figure 5 is a graph of the fitted values and 95% confidence interval of a spline regression dividing average monthly PM2.5 concentrations into splines by decile (denoted by vertical bars), controlling for the linear control function residual.

4.2.2 Short-Term Effects of PM2.5 by Cause of Death

I estimate effects on mortality rates by broad cause-of-death categories using specification (4) from Table 8, and report the results in Table 9. Unsurprisingly, the fatal effects of PM2.5 manifest most strongly through cardiovascular and respiratory causes, consistent with prior literature. A $10 \mu g m^{-3}$ increase in average monthly PM2.5 is associated with additional deaths from ischemic heart disease (0.26 additional deaths per 100,000), cerebrovascular disorders (0.17), influenza and pneumonia (0.15), and chronic lower respiratory disease (0.19). PM2.5 also has an impact (0.16) on deaths in the ICD-10's broad "Other Diseases" category, suggesting that PM2.5 exposures either lead to complications for already-vulnerable individuals or also cause cardiovascular and respiratory-related deaths for individuals whose cause of death is coded in accordance with the presence of another major health condition.

These wide-ranging effects are supported by the medical literature, which generally finds various undesirable immune system and other bodily responses to fine particulates. Proposed pathophysiological pathways for short-term effects to exposure of PM reviewed in Brook et al. (2010) and Pope et al. (2003) include the production of proinflammatory cytokines that create a systemic inflammatory response affecting bodily areas outside the lungs (also in van Eeden et al. 2001), systemic oxidative stress, changes in coagulation, changes in blood pressure, impaired vascular function, and increased heart rate variability. Brook et al. (2010) cite some conflicting evidence on the effects of particulates on biomarkers for these pathways, likely due to heterogeneity in chemical composition and exposure duration and intensity, but nevertheless reveal a common association between PM2.5 and important biomarkers related elevated risks of cardiovascular and respiratory morbidity. Specific studies have also specifically tied certain types of morbidity to particulate pollution, such as pneumonia (Zeikloff et al. 2002; Zeikloff et al. 2003) and chronic obstructive pulmonary disease (MacNee and Donaldson 2003). There are also research findings which associate subspecies with certain respiratory and cardiovascular health effects. Dye et al. (2001) find pulmonary injury in rats after exposure to PM2.5 subcomponents, with suggestive evidence of the high pulmonary toxicity of metal particulates, while Huang and Ghio (2006) implicate arsenic, mercury, and nickel exposure as causes for anemia, tachycardia, and increased blood pressure.

The inclusion of the wildfire non-PM2.5 pollution controls show the corresponding increases of toxicity of implied changes in PM2.5 across these dominant causes of death. Effects per unit mass PM2.5 on increase by factors of approximately 1.8 for ischemic heart disease and cerebrovascular deaths and 2.3 for chronic lower respiratory deaths, while increasing by a factor of 3 for influenza/pneumonia and other disease-related deaths (though individually remain within sampling error of the no-control effect sizes). Assuming these accurately represent the comparative magnitudes of true effects and that changes in identified PM2.5 composition explain most of the estimated increase in mortality per unit mass, this implies greater toxicity of PM2.5 metals for respiratory and general illnesses relative to cardiovascular illnesses. One explanation is that metals interfere with antimicrobial processes in the lungs, thereby raising the risk and severity of infection. Systemic inflammatory response may also inhibit the body's ability to fight infections outside the lungs.

As a sensitivity check, I estimate whether wildfire-instrumented PM2.5 has an impact on external causes of death (Table 10), with rationale comparable to Heutel and Ruhm (2013): if effect estimates are driven by confounding

variation from seasonal or trending factors related to both wildfires and mortality, then external causes of death physiologically unrelated to wildfires might show an effect. I consider 5 outcome groups as classified by the ICD-10: deaths from motor vehicle accidents, accidents, suicides, assaults/homicides, and from "all other external and unspecified causes." Motor vehicle accidents may regardless be affected by wildfires in extreme cases, as wildfires near major roadways can rapidly impede visibility causing massive, multi-vehicle accidents (Collins et al. 2009). ICD-10's "all other external and unspecified" category contains deaths due to fire exposure and acute smoke inhalation, which would reflect the deaths of firefighters, rural residents, campers, hikers, and other individuals who may be trapped in the vicinity of a wildfire. However, neither of these show any relationship to wildfire smoke, which is some evidence that wildfire pollution exposure is driven by fires distant enough to not have potential direct effects of fire events themselves (e.g., stress caused by imminent danger or property damage). I also find no relationship to suicides and homicides. With no wildfire pollution controls, I find a moderate, marginally statistically significant positive effect on the deaths under the "other unspecified accidents or adverse effects" category, which includes all deaths due to complications related to surgery or medication. This result may be explained by expected increase in the frequency of medical care being administered for increased rates of morbidity due to pollution.

4.2.3 Lagged and Lead Short-Term Associations with PM2.5

Estimating causal associations of air pollution with health outcomes is complicated by a wide range of potential intertemporal relationships between outcome and regressor, both causal and non-causal. There are three reasons to expect lagged pollution values to have negative effects: forward displacement of deaths, depletion of wildfire fuel stocks combined with contemporaneous measurement error, and denominator error in population rates due to annual population measures. In Table 12, I report reduced form estimates of lead, lagged, and both lead and lagged effects of the instrument on all-cause mortality, as well as the joint F-statistic of lead/lagged coefficients. I find evidence of forward displacement and generally violations of the strict exogeneity assumption for fixed effects estimators.

Pollution exposure causes forward displacement of an event if it causes the relocation of an event that otherwise would have occurred to an earlier time period. Schlenker and Walker (2011) argue that welfare impacts of air pollution through morbidity would be be overestimated if forward displacement occurs and is not taken into account (but they test for and find no evidence of forward displacement of hospitalizations). Unless there is a value on postponing a particular outcome, the only negative impact pollution exposure would have on welfare is through events that counterfactually would not have existed if not for the exposure. Since everybody dies[4], welfare effects of pollution-induced mortality can only be measured through the average change in life expectancy. Short-term pollution exposures may primarily only affect those who would otherwise die within a few months, but forward displacement of mortality in this sense is still economically meaningful as long as individuals place positive value on an additional month of life, though one might expect that such value is lower than that of a healthy working individual. The estimates in the second column reveal significant forward displacement.

If wildfire smoke is measured with substantial error, part of the error term of observed pollution is a function of the true level of wildfire pollution, which may in turn be predicted by past (or future) wildfire pollution due to fuel stock dynamics. A large wildfire may burn fuels accumulated over long periods that are not immediately replaced. Wildfires in the near future in the same area are well-situated to affect the same downwind areas as the past large wildfire, but likely to have smaller sizes and shorter durations. In turn, high concentrations in the past predict low concentrations in the present, which would result in lower present mortality.

Lastly, error in the population measure used to calculate mortality rates may cause a lagged negative relationship between mortality and pollution to appear. I measure mortality rates using annual intercensal estimates of population,

[4] I was unable to find a citation for this.

but measure mortality effects with monthly frequency. Holding changes due to births and migrations fixed, if contemporaneous pollution causes deaths in one month, then the following month's population count is too high, resulting in a measured mortality rate lower than the true rate. The measured rate is hence negatively correlated with the previous month's pollution, generating downward bias in estimates of lagged effects.

Jointly significant lead and lagged effects are interpreted as evidence of violation of the strict exogeneity assumption needed for large-N (number of cross-sectional observations) consistency of fixed effects estimators with a small number of time periods. The inconsistency has bounds shrinking at a rate proportional to the number of time periods (Wooldridge 2010), which in this case is 84 months. In all three specifications I find evidence that strict exogeneity is violated. Lagged and lead effects may also be indicative of shocks correlated with the regressor that affect multiple time periods and the outcome variable. In the wildfire setting, this may be weather or climatological variables not adequately captured by temperature, precipitation, and annual and seasonal regional fixed effects.

4.3 Effects on Infant Health

While infants at the most vulnerable health margins may be more likely to die from pollution shocks, the larger population of surviving infants may have their health after birth and subsequent quality of life impacted by *in utero* pollution exposure. Table 13 reports IV estimates for average exposures over the 9 months preceding birth and 4 months preceding birth. Prenatal exposure to PM2.5 has a strong effect on premature births, with effects concentrated in the 4 months leading up to birth. A $10\mu gm^{-3}$ increase in PM2.5 over the gestational period is associated with a 2.6 percentage point increase in the number of premature births and an average decrease in gestational age of 0.23 weeks. There are also negative, but not statistically significant effects on average birth weight, amounting to a $19g$ decrease per $10\mu gm^{-3}$ increase in PM2.5.

As with the mortality outcomes, controlling for NO2 and SO2 strengthens effects, testament to the increased relative toxicity of a unit change in PM2.5; in the final 4 months before birth, a $10\mu gm^{-3}$ increase in PM2.5 lowers average birth weights by $31g$, but there is no significant increase in the likelihood of low birth weight. If the increased toxicity also would result in increased fetal attrition (weakly suggested by the increase in the effect on percentage of female births), then this effect is likely to be occurring for healthier neonates. Alternatively, the effect could be driven by additional growth losses for neonates who regardless of exposure would have been low birth weight. There are at least four classes of physiological mechanisms which may explain the observed negative associations with birth weight: intrauterine growth restriction, fetal genetic or epigenetic changes, pollutant-DNA adducts, and premature birth (Slama et al. 2008). Prematurity may be highly correlated with any of the other mechanisms, or the increased rates of prematurity alone could be driving most of the effect.

The complex interaction of birth timing, overlapping exposures between birth cohorts, and strict exogeneity requirements for fixed effects estimators are possible hazards to identifying meaningful effects of in utero exposure. Because these exposure estimates are framed relative to the birth month, and not the month of conception, substantial harmful effects may be attributable to displacement of unhealthy births from future cohorts into current ones via decreases in gestational age. In the same vein, I expect that displacement due to premature births (and fetal deaths) caused by PM2.5 exposure will cause bias in the opposite direction due to cohort composition effects, as infants with worse health outcomes are deselected from a birth cohort and displaced into earlier cohorts (or completely removed the sample due to fetal death). Exposure timing varies even for births within the same month (by as much as 30 days), resulting in a mixture of true exposure effects estimated in each exposure window. More complicatedly, if the error is not strictly exogenous conditional on the exposure measures and controls, then exposure windows with a mixture of true exposure period and non-exposure periods will reflect a mixture of exposure effects and strict exogeneity violations (i.e., feedback between the dependent variable and lead/lagged values of the regressor).

Attrition from fetal deaths is likely to cause downward bias in the magnitude of these estimates. One key piece of evidence for fetal attrition is the large, albeit imprecisely estimated, effect of average exposure on the sex ratio: each $1\mu gm^{-3}$ increase in average PM2.5 exposure over nine months before birth raises the percentage of female births by 0.2 percentage points with no non-PM2.5 controls and 0.4 percentage points with NO2 and SO2 controls. This magnitude is comparable to the findings of Sanders and Stoecker (2011) for Total Suspended Particulates (TSPs), which are all particles less than $100\mu m$. Limited monitor coverage at the time of Clean Air Act makes it impossible to ascertain the effects TSP reductions had on fine particulates. Using rough conversion factors (based on ratios of means in the AQS data) for TSPs to PM10 of 0.55, and PM10 to PM2.5 of 0.6, a one-unit change in TSPs corresponds to a 0.33 unit change in PM2.5, translating the estimate to 0.067 percentage points per unit change in TSP compared to Sanders and Stoecker's (2011) 0.088.

This pattern of results is comparable to Bharadwaj and Eberhard's (2008) estimates of the effects of PM10 in Santiago, Chile on birth outcomes, but with smaller magnitudes. They estimate a $125g$ effect on birth weight per $17.57\mu gm^{-3}$ (one standard deviation) increase in PM10 pollution 1-16 weeks before birth, whereas I estimate a substantially smaller effect of $32g$ for a comparable change in PM2.5 (again using a conversion factor of $PM10 = 0.6 \times PM2.5$). Besides differences in toxicity between PM10 and PM2.5 (which we regardless might expect to make the difference smaller), this large difference is likely to be driven by some combination of nonlinear effects due to the substantially higher pollution levels in their sample period and the effects of omitted pollutants that also significantly decrease with rainfall. Average PM10 in the U.S. sample period is $18\mu gm^{-3}$ compared to $76\mu gm^{-3}$ in the Santiago sample, and any increasing dose response would be reflected. The rainfall instrument is likely to be strongly associated with decreases in non-PM10 pollutants relative to its association with PM10. While the wildfire instrument does predict some non-PM2.5 pollution levels, this contribution (and thus potential upward bias in estimates' magnitudes) is constrained by the wildfire instrument's dependence on wildfire-specific PM2.5 emissions and PM2.5-specific deposition parameters, compared to the broad and relatively less PM-heavy distribution of pollutants from all industrial sources in or near Santiago. Lastly, because they identify their pollution changes through rainfall, they also identify effects on health outcomes through the associated changes in water quality generated thru deposition; deposited pollutants run off into water and food supplies and are exposed to individuals through consumption and skin contact. This can bias their estimates either way, depending on whether the pollutants are more harmful after deposition or in the air. In contrast, I control for local rainfall, which will generally account for the aggregate effect of deposited pollutants that could affect health outcomes through the water supply. If deposition occurs in watersheds outside of the area that affect the area's water supply and precipitation differs significantly between the two areas, then the airborne pollutant estimated effects may still pick up effects from associated changes in water supply quality.

5 Wildfire Externalities and Current Management Policy

Wildfires induce significant changes in PM2.5 concentrations over long distances, with polluted air parcels crossing intranational and international boundaries. Assuming that monitoring stations are representative of a state's overall exposure to wildfire pollution, I calculate the fraction of wildfire PM2.5-months that occur outside the state of the wildfire, find that over 75% of geographic exposure to PM2.5 from large wildfire events in the CONUS occurs in states other than the state of origin. Table 7 reports the percentage of modeled wildfire PM2.5 exposure that occurs outside of each state of the wildfire occurrence as an approximation of the intensity of inter-state pollution externalities from wildfires. Because of the implied externalities, wildfire management is subject to the classic tradeoff between inefficient local management behavior and potentially inefficient centralized, uniform policies for environmental goods. To the extent that local jurisdictions in charge of wildfires (e.g., state fire agencies) are individual actors and ignore

inter-state pollution spillovers in making fire management decisions, then they will tend to under-suppress wildfire activity or engage in more aggressive prescribed burning for other local benefits. The structure of wildfire management in the U.S. is a complicated mixture of many agencies acting individually and collaborating at multiple levels of government, while the Clean Air Act does not penalize states for pollution from naturally-occurring wildfires. Hence, it is unclear whether current wildfire management efforts properly account for the welfare effects from poor air quality.

While air quality externalities largely make wildfire abatement a national environmental good, it is uncertain whether fire policy would strongly improve with greater centralization. Banzhaf and Chupp (2011) show for the U.S. electricity sector that a uniform federal pollution abatement policy has better welfare implications than decentralized state policies because the inter-state spillovers addressed by a uniform policy are relatively more important than the between-state heterogeneity of benefits addressed by decentralized policies. They argue that relatively inelastic marginal cost of abatement in the relevant region of the uniform policy results in smaller distortions from ignoring between-state heterogeneity of marginal benefits. Wildfires are characterized by large inter-state spillovers, but the concavity or convexity properties of the marginal costs of abatement are unclear, as are their true marginal damages. Wildfire management has two dimensions of abatement: pre-fire measures, such as prescribed burning and fuel clearing, and suppression efforts. Marginal costs of suppression efforts are relatively easy to measure; for example, Donovan (2006) finds a convex marginal cost function for the number of contract-based firefighting crews hired in a season. Regardless, all abatement measures may have strong heterogeneity and uncertainty in marginal benefits and costs associated with them. Prescribed wildfires themselves generate pollution and some ecological hazards because of their artificial timing (Knapp et al. 2009). Naturally-occurring wildfires have ecological benefits, such as biodiversity and better disease regulation, which may potentially counterbalance the marginal benefits of improved air quality (e.g., Keane and Karau 2010). Even suppression's benefits cannot be well-accounted for, as aggressive suppression can lead to higher likelihood and intensity of future fires by altering the nature of fuel accumulation (Yoder 2004).

Despite federal guidelines governing fire suppression attempts in the interest of protecting public health (Fire Executive Council 2009), the incentives facing the agencies making fire management choices are vague relative to the regulation of agents generating industrial air pollution. The Clean Air Act distinguishes between "unplanned" and "planned" fire, only penalizing states for the pollution generated by planned fire (i.e., prescribed burns), resulting in the adverse health effects of natural wildfires not being inherently taken into account by air quality regulations (Engel and Reeves 2011). There are federal directives and funding for wildfire management, with $3.9 billion allocated for FY2014 (Bracmort 2013). Decision-making regarding suppression and prescribed burning is not federally-determined, however. Currently, fire management in the U.S. predominantly falls upon five federal agencies for fires over 1,000 acres[5] and individual state, county, and local agencies, with frequent interagency collaboration. For the fires in the sample period, 36% were reported by state, county, and local agencies, while the remainder were federally-reported. The Forest Service and and Bureau of Land Management reported the majority of the remaining fires. There are ambiguities regarding which agency is responsible for suppression decisions; for example, the agency making the report does not always commit all of the resources tasked with managing the fire, and multiple agencies may report the same fire but only one record is retained in the FPA fire database.

6 Conclusion

This study uses new tools to measure the health externality costs of both industrial and natural sources of air pollution and provides estimates for the effects of fine particulate matter on mortality and infant health. To my knowledge, it is

[5]These are the the Bureau of Land Management (BLM), Bureau of Indian Affairs (BIA), the U.S. Forest Service (USFS), Fish and Wildfire Service (FWS), and National Park Service (NPS) for 99% of federally-reported fires.

the first to synthesize historical emissions, atmospheric transport models, and ground-level monitoring data at a large scale to estimate the distribution of environmental pollutants and their health effects in the United States. Its design provides spatially and temporally smooth measures of pollution shocks, and the ability to construct a full emissions-to-destination modeling process provides a large degree of customizability and control over the variation used to identify changes in air quality. The choice of wildfires as emissions source results in geographically wide-reaching variation in particulate levels, inducing both small and large shocks to highly polluted and relatively unpolluted areas. The findings of effects on short-term mortality and infant health contribute to the body of evidence supporting that PM2.5, and generally air quality, has important impacts on human health. They also highlight the importance of fire management as an important public health issue.

As might be expected with a new source of data, there are several statistical issues which must be addressed order to fully realize the potential of wildfires to identify useful, policy-relevant health effects parameters. Incorrect exposure measurements in both space and time create potentially serious measurement error problems which are only partially alleviated by instrumental variables techniques. Imperfect monitoring coverage results in measurement error of exposures both within and between geographic units. Spatial measurement error can be alleviated through more comprehensive measures of ambient pollution, generated through a combination of interpolation of data points, remote sensing data, and two-sample instrumental variables estimation techniques (Khawand 2014). Two-sample IV techniques can also be used to include geographic regions with no monitoring coverage in estimating health effects, resulting in estimated average effects more representative of the U.S. population. In the short run, this study can be improved upon through developing richer model inputs from higher-quality data products that require substantially greater computational input to implement. Satellite products for fire detection allow wildfire burn dynamics to be better parsed out in space and time. Higher-resolution meteorological products can be used to better capture short-range dispersion patterns, which in turn require more intensive geographic sampling schemes to properly translate to aggregate concentrations.

The modeling of wildfires' air quality impact itself also stands to be significantly improved. The relationship between the wildfire pollution forecasts and actual pollution levels, while intuitively seeming to be relatively uncomplicated, is subject to situation-specific measurement errors due to the complex interaction among fire, fuel, and meteorological data inputs and modeling assumptions. Modeling errors may occur due to unmodeled heterogeneity at the source or between the source and the destination. A richer exploration of heterogeneous source-receptor relationships is needed to understand where modeling errors may result in putting undue weight on health effects in certain areas or discarding useful variation in others. Extensive further work, particularly in collaboration with scientists in the wildfire community, is required to improve the realism and predictive power of the wildfire pollution simulation.

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7 Tables and Figures

Figure 1: Number of Acres Burned (Thousands) for All Fires Greater than 1,000 Acres, 2000-2010

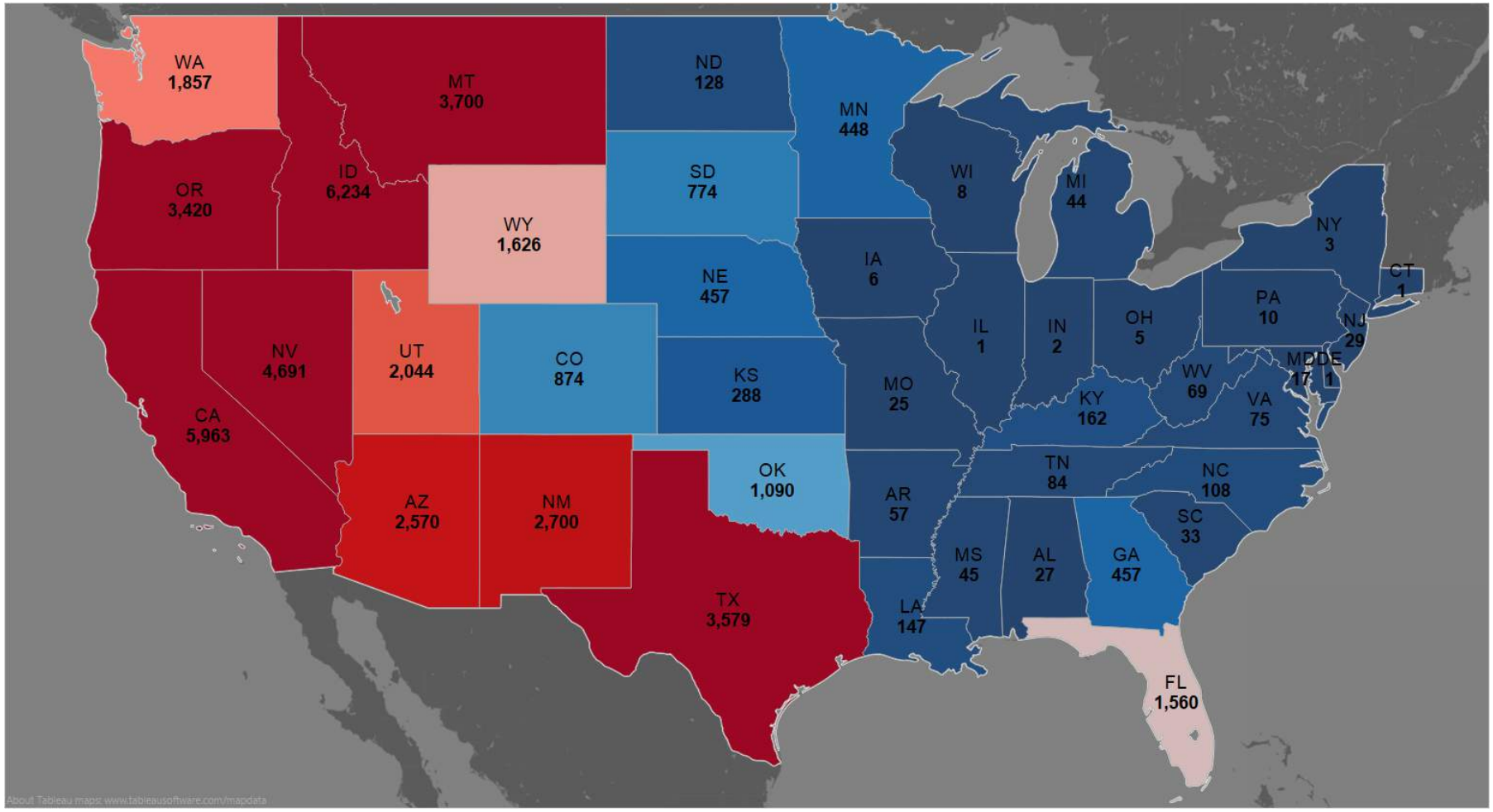


Figure 2: Wildfire Air Pollution Modeling - BlueSky Framework Workflow

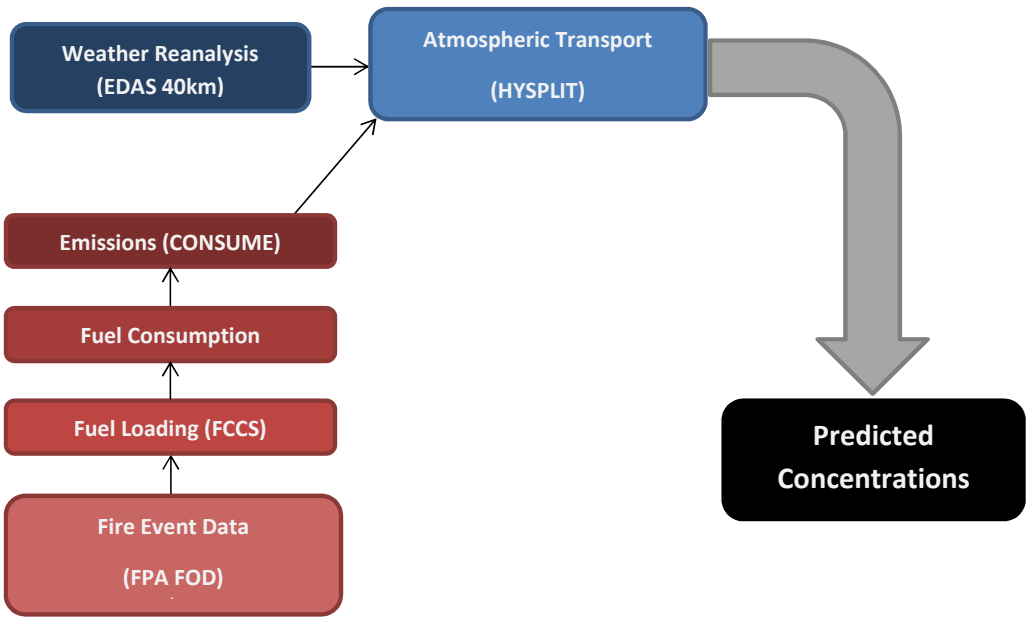
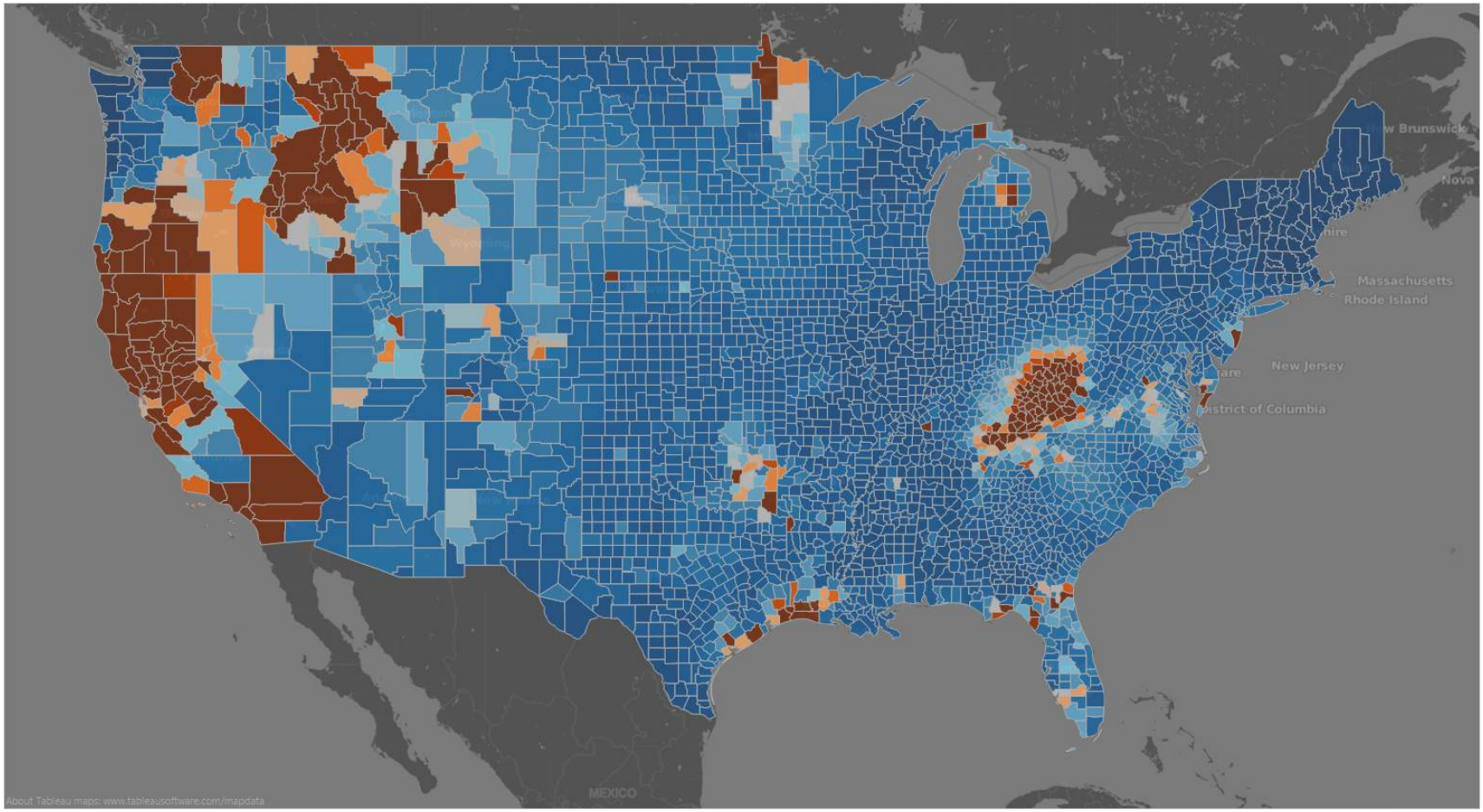


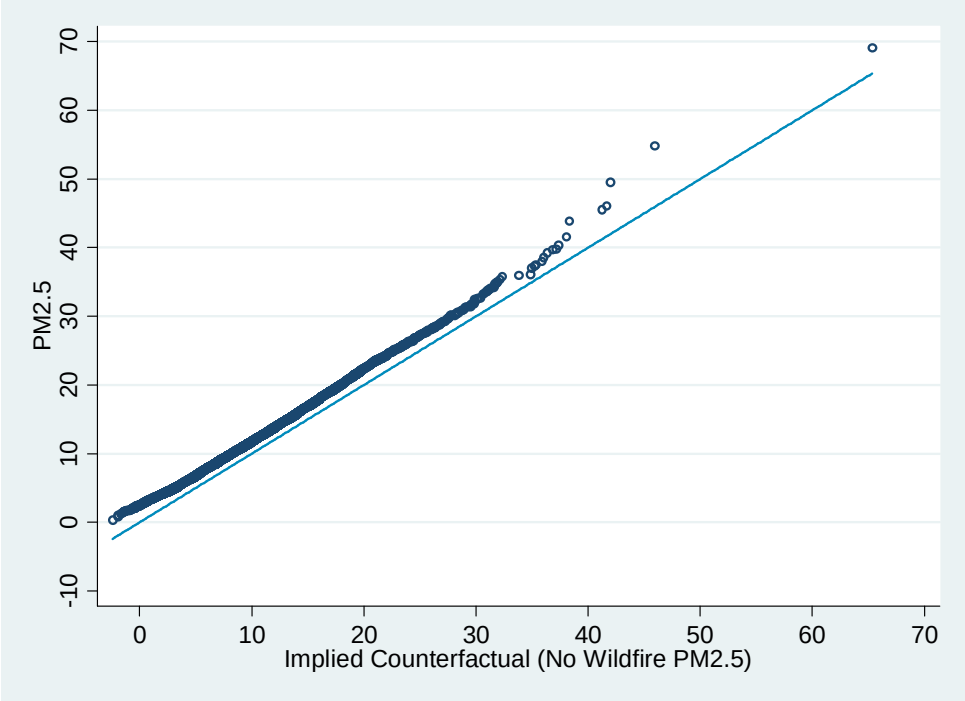
Figure 3: Average Raw Wildfire PM2.5 Output by County, CONUS, 2004-2010



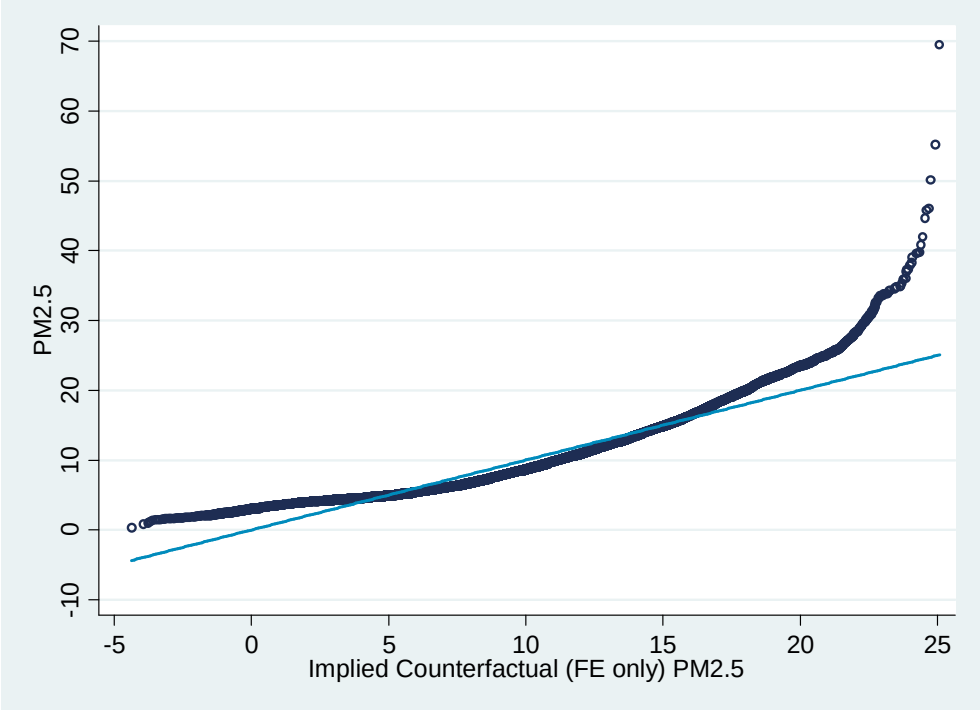
*Dark blue values represent low concentrations and brown values high concentrations (e.g, California has high concentrations, while Maine has low concentrations)

Figure 4: Quantile-Quantile Plots of PM2.5 versus Counterfactuals

(a) PM2.5 (with Wildfire PM2.5) versus Estimated Counterfactual PM2.5 (No Wildfire PM2.5)

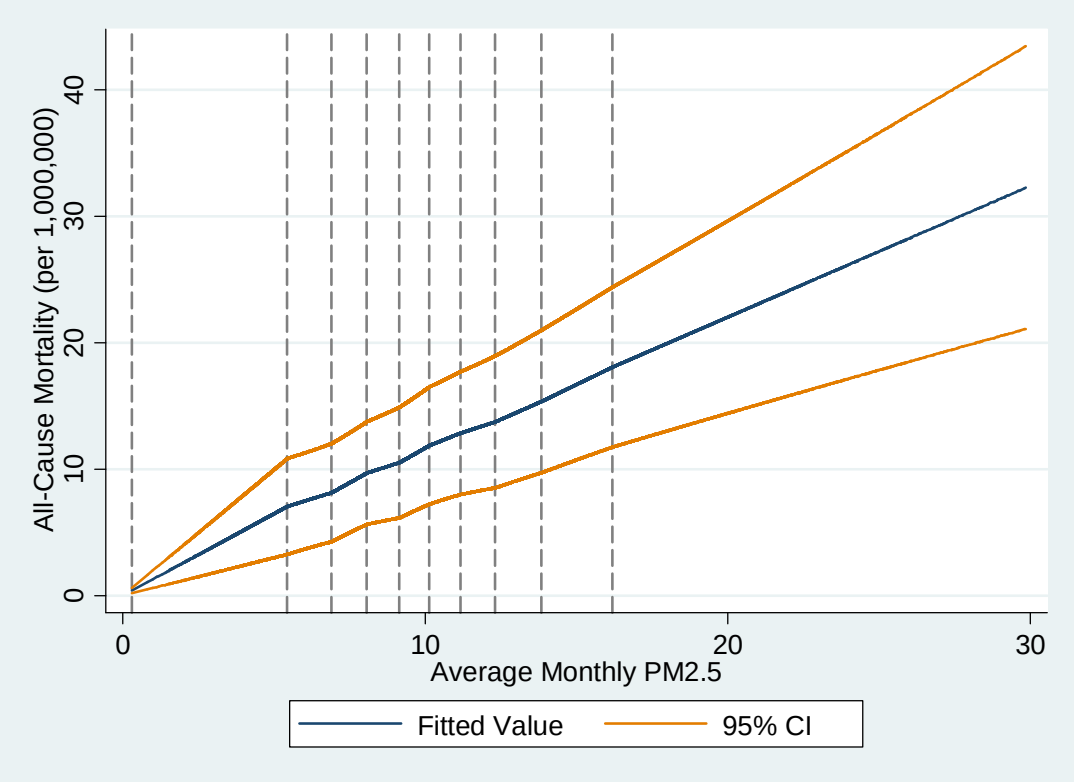


(b) PM2.5 versus Counterfactual PM2.5 Estimated with Fixed Effects



Sub-figure A plots PM2.5 against the counterfactual estimated using the wildfire PM2.5 instrument; sub-figure B plots it against the counterfactual as estimated using state-year, state-month, and county fixed effects. Each point on the plot represents the values in each distribution at which the quantiles are equivalent.

Figure 5: Spline Control Function Regression of All-Cause Mortality on PM2.5, by Decile



This is a plot of the estimated effect of average monthly PM2.5 estimated by splines in deciles of average monthly PM2.5 conditional on a linear control function residual using wildfire PM2.5 as the excluded instrument.

Table 1: AQS PM2.5 and NLDAS Weather Descriptive Statistics

	Mean	Std. Dev	5th Pctile.	Median	95th Pctile.	N
Average Daily Pollution and Weather Measures						
PM2.5 Concentration (ug/m3)	10.60	4.37	4.50	10.13	18.47	37,259
Precipitation (mm)	2.72	2.04	0.25	2.35	6.45	171,014
Maximum Air Temperature (F)	63.82	19.43	29.13	65.96	91.02	171,014
Minimum Air Temperature (F)	46.09	17.23	16.88	46.49	72.48	171,014

Table 2: Monthly, County-Level Mortality Rate (per 100,000) by Subgroup from U.S. Death Certificates, 2004-2010

	Mean	Std. Dev	5th Pctile.	Median	95th Pctile.	N
All individuals	78.58	33.62	36.38	73.73	136.38	171,182
< 1 year	54.67	222.98	0.00	0.00	278.55	170,782
01 to 04	1.56	18.77	0.00	0.00	2.15	170,787
05 to 14	0.69	6.50	0.00	0.00	1.52	170,796
15 to 24	1.71	10.65	0.00	0.00	7.56	170,855
25 to 34	4.18	18.92	0.00	0.00	22.82	170,836
35 to 44	11.52	27.14	0.00	0.00	50.48	170,837
45 to 54	31.70	41.41	0.00	24.25	98.14	170,847
55 to 64	73.93	70.76	0.00	64.44	188.95	170,834
65 to 74	173.19	128.88	0.00	159.49	386.10	170,820
75 to 84	431.85	255.33	0.00	410.98	833.33	170,831
85+	1247.40	747.52	0.00	1173.70	2395.20	170,823
All Ages, Male	76.67	42.35	24.85	70.17	147.49	171,075
All Ages, Female	79.28	43.25	26.26	72.74	152.65	170,917
County Population	110,000	350,000	3,394	28,494	490,000	171,182

Table 3: Monthly, County-Level Mean Birth Outcomes and Rates for Birth Cohorts from U.S. Birth Certificates, 2004-2010

	Mean	Std. Dev	5th Pctile.	Median	95th Pctile.
All Births					
# Births	113	391	3	28	476
Avg. Birth Weight (g)	3268	197	2966	3274	3556
% Male	48.8%	15.1%	25.0%	49.1%	71.4%
% Low Birth Weight	8.1%	8.3%	0.0%	7.1%	21.4%
APGAR 0-3	0.5%	2.3%	0.0%	0.0%	2.9%
APGAR 4-6	1.4%	3.9%	0.0%	0.0%	6.7%
APGAR 7-8	13.0%	14.4%	0.0%	9.4%	40.0%
APGAR 9-10	82.7%	19.2%	46.0%	88.0%	100.0%
APGAR Unknown	2.4%	13.7%	0.0%	0.0%	3.8%
Preterm Birth	12.6%	10.4%	0.0%	11.7%	29.2%
Full-Term Birth	87.3%	10.5%	70.5%	88.1%	100.0%
Gest. Age Unknown	0.2%	1.3%	0.0%	0.0%	0.4%
N = 259,471					
Full-Term Births Only (< 37 wks.)					
# Births	98	339	2	24	417
Avg. Birth Weight (g)	3368	167	3108	3371	3619
% Male	49.1%	15.9%	25.0%	49.8%	75.0%
% Low Birth Weight	3.2%	5.5%	0.0%	1.8%	11.9%
APGAR 0-3	0.2%	1.6%	0.0%	0.0%	0.9%
APGAR 4-6	1.0%	3.5%	0.0%	0.0%	5.0%
APGAR 7-8	11.5%	14.5%	0.0%	7.7%	40.0%
APGAR 9-10	85.0%	19.4%	50.0%	90.6%	100.0%
APGAR Unknown	2.3%	13.7%	0.0%	0.0%	3.1%
N = 258,748					
Pre-Term Births Only (≥ 37 wks.)					
# Births	14	48	0	3	60
Avg. Birth Weight (g)	2570	481	1778	2574	3323
% Male	46.4%	29.2%	0.0%	48.5%	100.0%
% Low Birth Weight	41.3%	29.4%	0.0%	42.3%	100.0%
APGAR 0-3	2.8%	9.8%	0.0%	0.0%	16.7%
APGAR 4-6	4.2%	12.1%	0.0%	0.0%	25.0%
APGAR 7-8	22.3%	25.8%	0.0%	16.7%	100.0%
APGAR 9-10	68.1%	30.0%	0.0%	74.1%	100.0%
APGAR Unknown	2.6%	14.5%	0.0%	0.0%	4.7%
N = 216,452					

Table 4: First Stage Regression of PM2.5 and Regressions of Criteria Pollutants on Wildfire Instrument

	Fine Particulate (PM2.5)	**Coarse Particulate (PM10)**	**Carbon Monoxide (CO)**	**Sulfur Dioxide (SO2)**	**Nitric Oxide (NO)**	**Nitrogen Dioxide (NO2)**	**Ozone (O3)**
				Panel A: 2SLS			
Coefficient	**1.1e-01*****	**5.5e-02****	-1.2e-04	2.9e-03	-2.2e-02	**2.2e-02*****	**1.1e-04*****
	(1.1e-02)	(2.4e-02)	(3.2e-04)	(3.4e-03)	(1.5e-02)	(5.8e-03)	(1.1e-05)
% Wildfire	15.3%	4.7%	-0.4%	1.5%	-4.8%	3.0%	5.7%
95% CI Upper	*18.5%*	*8.7%*	*1.6%*	*4.8%*	*1.6%*	*4.6%*	*6.9%*
95% CI Lower	*12.2%*	*0.7%*	*-2.4%*	*-1.9%*	*-11.2%*	*1.4%*	*4.6%*
				Panel B: 2SLS with Wildfire NO2, SO2 Controls			
Coefficient	**1.1e-01*****	4.7e-02	6.1e-04	4.0e-03	-1.4e-02	5.5e-03	**9.2e-05*****
	(1.2e-02)	(2.9e-02)	(3.7e-04)	(4.9e-03)	(2.1e-02)	(8.0e-03)	(1.8e-05)
% Wildfire	16.1%	4.0%	2.0%	2.0%	-3.1%	0.8%	4.8%
95% CI Upper	*19.5%*	*9.0%*	*4.3%*	*6.8%*	*5.8%*	*2.9%*	*6.6%*
95% CI Lower	*12.8%*	*-0.9%*	*-0.4%*	*-2.8%*	*-12.1%*	*-1.4%*	*3.0%*
				Panel C: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls			
Coefficient	**1.0e-01*****	3.3e-02	5.6e-04	4.6e-03	8.5e-03	3.8e-03	**9.7e-05*****
	(1.1e-02)	(2.7e-02)	(3.7e-04)	(4.8e-03)	(1.8e-02)	(8.6e-03)	(1.9e-05)
% Wildfire	14.5%	2.8%	1.8%	2.3%	1.8%	0.5%	5.1%
95% CI Upper	*17.5%*	*7.3%*	*4.1%*	*7.1%*	*9.6%*	*2.9%*	*7.0%*
95% CI Lower	*11.5%*	*-1.7%*	*-0.5%*	*-2.4%*	*-6.0%*	*-1.8%*	*3.1%*
Mean Conc.	1.10E+01	1.80E+01	4.60E-01	2.90E+00	7.00E+00	1.10E+01	3.00E-02
N	36752	14706	11955	14719	10665	13119	26063

Coefficients are for a 1µgm-3 change in PM2.5. Units are ppb for SO2, NO, and NO2 and ppm for O3 and CO. "% Wildfire" is calculated as the overall quantity of pollutant predicted by the instrument divided by the mean concentration times 100%. Standard errors clustered at state-year level are in parentheses. Significance stars represent $p < 0.1$ (*), $p < .05$ (**), $p < .01$ (***).

Table 5: Regressions of Highly Toxic PM2.5 Subspecies on Wildfire PM2.5

	Arsenic	**Mercury**	**Lead**	**Nickel**	**Cadmium**
			Panel A: 2SLS		
Coefficient	2.1e-07	**-1.6e-05*** **	**2.0e-05** **	4.7e-06	9.5e-06
	(1.5e-06)	(4.1e-06)	(8.6e-06)	(3.2e-06)	(6.5e-06)
% Wildfire	0.4%	-24.0%	10.6%	6.7%	8.4%
95% CI Upper	*6.8%*	*-12.1%*	*19.7%*	*15.5%*	*19.8%*
95% CI Lower	*-5.9%*	*-35.9%*	*1.5%*	*-2.2%*	*-3.0%*
			Panel B: 2SLS with Wildfire NO2, SO2 Controls		
Coefficient	**8.7e-06*** **	**2.4e-05*** **	**2.6e-05***	8.5e-06	**3.8e-05*** **
	(2.3e-06)	(6.3e-06)	(1.4e-05)	(6.3e-06)	(9.4e-06)
% Wildfire	18.6%	35.5%	14.1%	12.1%	33.9%
95% CI Upper	*28.0%*	*53.6%*	*28.6%*	*29.8%*	*50.2%*
95% CI Lower	*9.1%*	*17.4%*	*-0.5%*	*-5.6%*	*17.6%*
			Panel C: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls		
Coefficient	**9.1e-06*** **	**2.2e-05*** **	**3.3e-05** **	**1.1e-05***	**3.5e-05*** **
	(2.4e-06)	(6.2e-06)	(1.3e-05)	(6.3e-06)	(9.1e-06)
% Wildfire	19.4%	31.7%	17.9%	16.3%	30.7%
95% CI Upper	*29.3%*	*49.6%*	*32.2%*	*33.9%*	*46.5%*
95% CI Lower	*9.6%*	*13.9%*	*3.6%*	*-1.2%*	*14.9%*
Mean Concentration	7.00E-04	1.10E-03	2.80E-03	1.00E-03	1.70E-03
N	15,566	8,300	15,624	15,624	10,439

Coefficients are for a 1-unit change in the wildfire PM2.5 instrument. Units are in µgm-3 for all PM2.5. "% Wildfire" is calculated as the overall quantity of pollutant predicted by the instrument divided by the mean concentration times 100%. Standard errors clustered at state-year level are in parentheses. Significance stars represent $p < 0.1$ (*), $p < .05$ (**), $p < .01$ (***).

Table 6: Regressions of Non-Metallic PM2.5 Subspecies on Wildfire PM2.5

	Organic Carbon (OC)	**Elemental Carbon (EC)**	**Hydrogen**	**Chloride**	**Bromine**	**Sulfur**	**Nitrite**	**Soil**	**Sulfate**	**Nitrate**
					Panel A: 2SLS					
Coefficient	**2.3e-02*** **	**3.0e-03*** **	**2.9e-03*** **	-2.6e-04	**1.9e-05*** **	**9.7e-03*** **	**1.1e-04** **	-2.3e-04	**2.7e-02*** **	**2.1e-02*** **
	(4.8e-03)	(8.4e-04)	(4.9e-04)	(4.8e-04)	(3.7e-06)	(1.3e-03)	(4.5e-05)	(1.8e-03)	(3.8e-03)	(3.1e-03)
% Wildfire	25.3%	13.7%	15.9%	-4.9%	10.4%	17.9%	10.8%	-0.5%	17.4%	28.2%
95% CI Upper	*35.9%*	*21.2%*	*21.2%*	*12.9%*	*14.4%*	*22.5%*	*19.2%*	*6.7%*	*22.1%*	*36.4%*
95% CI Lower	*14.8%*	*6.3%*	*10.6%*	*-22.8%*	*6.4%*	*13.3%*	*2.4%*	*-7.7%*	*12.6%*	*20.1%*
					Panel B: 2SLS with Wildfire NO2, SO2 Controls					
Coefficient	5.6e-03	1.5e-03	6.7e-04	-1.4e-03	**3.8e-05*** **	**1.1e-02*** **	4.4e-05	8.7e-04	**3.2e-02*** **	**3.2e-02*** **
	(4.7e-03)	(1.7e-03)	(6.7e-04)	(9.8e-04)	(6.1e-06)	(1.5e-03)	(4.4e-05)	(2.3e-03)	(4.6e-03)	(4.5e-03)
% Wildfire	6.3%	6.8%	3.7%	-26.8%	20.8%	20.0%	4.2%	1.8%	20.6%	42.4%
95% CI Upper	*16.7%*	*21.8%*	*10.9%*	*9.7%*	*27.4%*	*25.6%*	*12.5%*	*11.2%*	*26.2%*	*54.2%*
95% CI Lower	*-4.1%*	*-8.2%*	*-3.6%*	*-63.2%*	*14.2%*	*14.3%*	*-4.1%*	*-7.7%*	*14.9%*	*30.6%*
					Panel C: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls					
Coefficient	**9.6e-03***	1.7e-03	4.1e-04	-9.9e-04	**3.6e-05*** **	**8.6e-03*** **	5.7e-05	2.1e-03	**2.5e-02*** **	**3.2e-02*** **
	(5.3e-03)	(1.5e-03)	(6.7e-04)	(9.7e-04)	(5.4e-06)	(1.7e-03)	(4.6e-05)	(2.5e-03)	(5.0e-03)	(4.1e-03)
% Wildfire	10.7%	7.7%	2.3%	-18.8%	19.9%	15.9%	5.4%	4.3%	15.9%	42.6%
95% CI Upper	*22.3%*	*20.6%*	*9.5%*	*17.5%*	*25.7%*	*22.1%*	*14.1%*	*14.2%*	*22.2%*	*53.4%*
95% CI Lower	*-0.8%*	*-5.3%*	*-5.0%*	*-55.0%*	*14.1%*	*9.7%*	*-3.3%*	*-5.6%*	*9.7%*	*31.7%*
Mean Concentration	1.30E+00	3.20E-01	2.60E-01	7.60E-02	2.70E-03	8.00E-01	1.50E-02	7.10E-01	2.30E+00	1.10E+00
N	6,477	6,469	6,281	6,359	15,481	15,561	6,299	6,281	15,628	15,378

Coefficients are for a 1-unit change in the wildfire PM2.5 instrument. Units are in µgm-3 for all PM2.5. "% Wildfire" is calculated as the overall quantity of pollutant predicted by the instrument divided by the mean concentration times 100%. Standard errors clustered at state-year level are in parentheses. Significance stars represent p < 0.1 (*), p < .05 (**), p < .01 (***).

Table 7: Percentage of Wildfire PM2.5 Exposure Outside of the State of Origin

AL	AR	AZ	CA	CO	CT	DE	FL	GA	IA	ID	IL	IN	KS	KY	LA
72.2%	72.1%	77.9%	80.3%	56.9%	7.3%	--	83.4%	86.7%	--	74.1%	83.6%	74.5%	92.5%	82.0%	73.7%
MA	**MD**	**ME**	**MI**	**MN**	**MO**	**MS**	**MT**	**NC**	**ND**	**NE**	**NH**	**NJ**	**NM**	**NV**	**NY**
--	96.5%	--	84.8%	94.3%	69.8%	58.5%	75.7%	86.1%	75.1%	90.6%	--	65.8%	69.8%	73.7%	68.0%
OH	**OK**	**OR**	**PA**	**RI**	**SC**	**SD**	**TN**	**TX**	**UT**	**VA**	**VT**	**WA**	**WI**	**WV**	**WY**
80.4%	61.7%	72.0%	78.4%	--	58.0%	50.2%	75.2%	92.3%	65.2%	78.6%	--	76.2%	--	--	54.5%

Each cell represents the fraction of raw average wildfire PM2.5 unit-months that occurs within the wildfire's state of origin. Empty cells indicate states with no wildfires larger than 1,000 acres in the sample period.

Table 8: IV Estimates: PM2.5 Effects on All-Cause Mortality (by Fixed-Effects Specification)

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: 2SLS						
Avg. PM2.5 ($10\mu gm^{-3}$)	**0.671*** **	**0.806*** **	**0.881*** **	**1.041*** **	**1.049*** **	**0.926*** **
	(0.121)	(0.126)	(0.123)	(0.199)	(0.125)	(0.129)
First-Stage F-Statistic	67.791	80.118	79.523	79.078	82.994	76.378
First-Stage Partial R^2	0.03	0.025	0.025	0.024	0.025	0.028
Panel B: 2SLS - Wildfire NO2, SO2 Controls						
Avg. PM2.5 ($10\mu gm^{-3}$)	**1.590** **	**1.965*** **	**2.067*** **	**2.350*** **	**2.419*** **	**2.875*** **
	-0.701	(0.670)	(0.695)	(0.737)	(0.736)	(1.015)
First-Stage F-Statistic	10.102	17.334	16.737	19.645	20.799	13.569
First-Stage Partial R^2	0.003	0.004	0.004	0.004	0.004	0.003
Panel C: 2SLS -Wildfire NO2, SO2, NH3, VOC Controls						
Avg. PM2.5 ($10\mu gm^{-3}$)	**1.791** **	**2.269*** **	**2.365*** **	**2.680*** **	**2.779*** **	**3.175*** **
	(0.780)	(0.802)	(0.823)	(0.870)	(0.874)	(1.145)
First-Stage F-Statistic	9.123	14.695	14.556	17.441	18.459	12.713
First-Stage Partial R^2	0.003	0.003	0.003	0.003	0.003	0.003
Panel D: OLS						
Avg. PM2.5 ($10\mu gm^{-3}$)	***-0.043****	-0.017	-0.022	-0.013	-0.009	0.001
	(0.024)	(0.025)	(0.025)	(0.024)	(0.025)	(0.026)
Fixed Effects						
Year	Y	Y	--	--	--	--
Month	Y	--	--	--	--	--
County	Y	Y	Y	Y	--	--
County-Month	N	N	N	N	N	Y
County-Year	N	N	N	N	Y	N
Climate Region-Month	N	Y	Y	--	--	--
State-Year	N	N	Y	Y	--	Y
State-Month	N	N	N	Y	Y	--

N = 36,752. Coefficients are effects for mortality rate per 100,000 population for a 10µgm-3 change in PM2.5. Standard errors clustered at state-year level are in parentheses. Significance stars represent $p < 0.1$ (*), $p < .05$ (**), $p < .01$ (***).

Table 9: IV Estimates: PM2.5 Effects on Mortality (by Cause)

All-Cause	All-Cause (ln(rate))	Ischemic Heart Disease	Other Heart Disease	Cerebrovascular	Influenza & Pneumonia	Chronic Lower Respiratory	ICD-10 "All Other (Residual)"
Panel A: 2SLS							
1.041* **	**0.013*** **	**0.258*** **	0.066	**0.166*** **	**0.146*** **	**0.194*** **	**0.163** **
(0.233)	(0.003)	(0.073)	(0.041)	(0.043)	(0.044)	(0.048)	(0.070)
Panel B: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls							
2.680* **	**0.033*** **	**0.472** **	**0.257***	**0.292** **	**0.458*** **	**0.454*** **	**0.529** **
(0.885)	(0.012)	(0.225)	(0.133)	(0.134)	(0.167)	(0.169)	(0.230)
Panel C: OLS							
-0.013	0.000	-0.010	0.007	***-0.015*** **	***-0.009*** **	-0.003	-0.003
(0.024)	(0.000)	(0.011)	(0.007)	(0.006)	(0.004)	(0.006)	(0.009)
Outcome Means (monthly, per 100,000)							
67.64	4.16	11.97	5.53	4.20	1.67	4.20	11.92

N = 36,752

Coefficients are effects for mortality rate per 100,000 population for a 10µgm-3 change in PM2.5. Standard errors clustered at state-year level are in parentheses. Significance stars represent p < 0.1 (*), p < .05 (**), p < .01 (***).

Table 10: IV Estimates: PM2.5 (Non-)Effects on Mortality from External Causes

Motor Vehicle Accidents	**Other Unspecified & Adverse Effects**	**Homicides**	**Suicides**	**Other External Causes**
		Panel A: 2SLS		
0.020	**0.083****	-0.031	0.001	0.008
(0.026)	(0.040)	(0.021)	(0.009)	(0.007)
		Panel B: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls		
0.067	0.060	-0.063	-0.007	0.006
(0.076)	(0.111)	(0.060)	(0.024)	(0.020)
		Panel C: OLS		
-0.001	-0.003	0.007**	-0.000	-0.000
(0.004)	(0.005)	(0.003)	(0.002)	(0.001)
		Outcome Means (monthly, per 100,000)		
1.35	2.53	1.10	0.42	0.18

N = 36,752

Coefficients are effects for mortality rate per 100,000 population for a 10μgm^{-3} change in PM2.5. Standard errors clustered at state-year level are in parentheses. Significance stars represent $p < 0.1$ (*), $p < .05$ (**), $p < .01$ (***).

Table 11: IV Estimates: PM2.5 Effect on All-Cause Mortality by Age Group

01 to 04	**05 to 14**	**15 to 24**	**25 to 34**	**35 to 44**
		Panel A: 2SLS		
-0.028	-0.043	0.011	***-0.230****	0.032
(0.088)	(0.037)	(0.072)	(0.124)	(0.148)
		Panel C: 2SLS -Wildfire NO2, SO2, NH3, VOC Controls		
-0.014	-0.057	0.069	-0.395	0.042
(0.241)	(0.102)	(0.199)	(0.344)	(0.408)
45 to 54	**55 to 64**	**65 to 74**	**75 to 84**	**85+**
		Panel A: 2SLS		
0.103	0.584	**3.140*** **	**7.009*** **	**27.027*** **
(0.227)	(0.419)	(0.907)	(1.956)	(6.978)
		Panel C: 2SLS -Wildfire NO2, SO2, NH3, VOC Controls		
0.275	1.615	**6.042** **	**14.892** **	**78.988*** **
(0.626)	(1.223)	(2.795)	(6.181)	(26.583)

Coefficients are effects for mortality rate per 100,000 population for a 10µgm-3 change in PM2.5. Standard errors clustered at state-year level are in parentheses. Significance stars represent $p < 0.1$ (*), $p < .05$ (**), $p < .01$ (***).

Table 12: Reduced Form Lead and Lagged Wildfire PM2.5 Effect on All-Cause Mortality

	(1)	(2)	(3)
6 Month Lead		-0.007	0.011
		(0.01)	(0.02)
5 Month Lead		0.009	**-0.035****
		(0.019)	(0.016)
4 Month Lead		**0.046****	**0.046****
		(0.019)	(0.023)
3 Month Lead		0.016	0.008
		(0.018)	(0.022)
2 Month Lead		**-0.035***	-0.009
		(0.019)	(0.019)
1 Month Lead		0.015	-0.018
		(0.019)	(0.023)
Contemp.	**0.077*****	**0.046*****	**0.036***
	(0.018)	(0.018)	(0.022)
1 Month Lag	-0.022		**-0.043****
	(0.016)		(0.018)
2 Month Lag	-0.008		0.016
	(0.016)		(0.019)
3 Month Lag	**-0.049*****		-0.03
	(0.016)		(0.019)
4 Month Lag	-0.021		-0.031
	(0.019)		(0.021)
5 Month Lag	**-0.052*****		**-0.062*****
	(0.019)		(0.022)
6 Month Lag	0.009		0.036
	(0.018)		(0.023)
Joint F-test Leads/Lags (p-value)	0.00015	0.00055	0.00028
N	30,355	30,394	24,096

Table 13: IV Estimates: Effect of PM2.5 Exposure for Full Gestation and 16 Weeks Before Birth on Birth Outcomes

	% Female	Gestational Age (Weeks)	% Premature (< 37 Weeks GA)	Avg. Birth Weight (g)	Low Birth Weight (< 2500g)	Low Apgar (< 5)
			Panel A: 2SLS			
Avg. PM2.5 (9 mo. Before Birth)	**0.00226***	**-0.0228*****	**0.00257*****	-1.9235	0.0090	-0.0003
	(0.00116)	(0.0077)	(0.00083)	(1.7574)	(0.0070)	(0.0005)
1-16 Weeks Before Birth	0.00117	**-0.0238*****	**0.00195*****	-1.7355	0.0031	0.0000
	(0.00086)	(0.0055)	(0.00064)	(1.1570)	(0.0046)	(0.0003)
FS F-stat F = 156.45, Partial R2 = 0.0977, N = 43,585						
			Panel B: 2SLS - Wildfire NO2, SO2 Controls			
Avg. PM2.5 (9 mo. Before Birth)	0.00397	**-0.0464*****	**0.00388****	-4.4164	0.0006	0.0001
	(0.00244)	(0.0164)	(0.00177)	(3.6309)	(0.0015)	(0.0010)
1-16 Weeks Before Birth	0.00107	**-0.039*****	**0.00297*****	**-3.1059****	0.00001	0.0002
	(0.00112)	(0.0078)	(0.00088)	(1.4327)	(0.0006)	(0.0004)

FS F-stat = 37.68, Partial R2 = 0.023, N = 43,585. PM2.5 is denominated in 1ugm-3 and averaged over the specified period. The instrument is wildfire PM2.5 averaged over the same period, and all controls are averaged over the same period.

8 Appendix

8.1 Estimating Fire Burn Durations

Wildfires can last for a period of hours to hundreds of days (for large, remote complex fires). The best measure in the FPA database of a fire's start time is the discovery time by the reporting agency, which is almost always reported. The time of the fire's containment, which indicates a judgment by the fire managing agency that the fire perimeter is secured from spreading further, is reported with similar frequency. Only some of the FPA database sources also have reports of their fires' extinguishment dates. Substantial emissions may still occur during the period between containment and extinguishment, especially for large fires. For fires greater than 300 acres, approximately 43% of burn time is post-containment. To better calibrate the time profile of emissions from fires, I use these fire events to fit a model and predict the burn duration for all fires in the absence of an explicitly-reported extinguishment or "put-out" time.

I merge fire extinguishment dates from the DOI-USGS database of fire reports from six major federal agencies. Then, estimate a linear model of a fire's burn duration:

$$D_i = c_i\xi + s(i)\theta_s + m(i)\theta_m + y(i)\theta_y + r_i$$

A fire's burn duration is a function of its time to containment D_i; its final land-area size, measured by a categorical "size class" c_i; some unobserved seasonal-, year-, and state-specific factors; and idiosyncratic factors r_i. I estimate this relationship using all fires from 2000-2010 larger than 300 acres. The containment time is naturally a strong predictor, as it is the earliest a fire can be extinguished. Its coefficient is sharply estimated close to 1, suggesting that time-to-containment is at least conditionally unrelated to unobservable characteristics of the fire that affect its total duration.

Where both containment and put-out dates are unavailable, a fire is assigned its duration based on the same model, estimated without including containment date as a covariate. All predictions less than 1 day due to the linear fit of the model are assigned a value of 1 day of burning. All fires with reported and predicted durations exceeding 160 days are assigned 160 days of burning to lower computational overhead. This is based on an assumption that fires reported to burn in excess of 160 days have reporting error in their records or are long-burning smoldering fires, which do not have comparable emissions to flaming fires. This truncation procedure removes approximately 10 percent of fire emission days, and less than 4 percent of emissions when weighted by the total land area of the fire.

The purpose of these duration estimates is to improve the predictive power of modeled concentrations. Errors in the prediction from reporting errors or misspecification of the model for fire burn duration will result in emissions profiles of incorrect length. These errors will not affect the validity of the modeled pollutant concentrations as instruments for observed pollutant concentrations, provided they are statistically unrelated to the determinants of the observed pollutant concentrations I do not include in my first stage estimation.

8.2 Wildfire Modeling Details

8.2.1 Modeling Workflow

The fire events from the FPA FOD database are each input as individual events into the BSF. The CONSUME module reads the coordinate data of the event and determines the likely fuel type using the FCCS fuel map. CONSUME then divides the fuel consumption into flaming, smouldering, and residual emission phase, each of which has a distinct contribution to emissions volume for the same fuel (as a model of fuel combustion efficiency). Combining the fuel

consumption profile with empirically-derived emissions factors, FEPS then estimates the quantities of heat and the pollutant of interest released by the fire. Using an empirically-derived diurnal (i.e., daily recurring) time profile embedded in FEPS, I generate a 24-hour emissions pattern that repeats for each day a fire burns and terminates at the estimated date of the fire's extinguishment. The pattern distributes the total emissions calculated by CONSUME among hours of the day. This modeling step is designed to improve downwind concentration estimates by accounting for fire burn cycles that vary with meteorological parameters that systematically vary with time of day, with lower emissions during nighttime hours.

The FEPS Plume Rise module estimates the buoyancy of the emitted pollutant due to the heat calculated by CONSUME and assigns 20 heights into which fractions of the hourly emissions are injected. This step reflects that quantities of a pollutant will be lofted higher from a fire location the more heat the fire releases, and that larger fires will also tend to have higher plumes that will result in longer-range transport. The result is a set of hourly point-source emissions for each fire event, with 20 emissions quantities in each hour released at the FEPS-calculated altitudes.

The point-source emissions generated by the CONSUME and FEPS models from the fire event data are then inputted into HYSPLIT, which calculates the trajectory and dispersion of the emitted pollutants and outputs a spatial field of concentrations over time. To calculate concentrations, HYSPLIT requires continuous meteorological data spanning the time period of the fire event and its corresponding downwind impacts of interest. Meteorological reanalysis data sets or archived forecasts are typically used for retrospective applications. Here, I use the Eta Data Assimilation System 40km (EDAS40), an archived 3-hourly forecast spanning 2004 to the present with a spatial resolution of 40km. This forecast system was developed and maintained by National Weather Service's National Centers for Environmental Prediction.

HYSPLIT represents the distribution of pollutants from a source through the behavior of a large number of individual "particles" (which are computational representations of pollutant masses, not to be confused with particulate pollutants in themselves). These particles are released over the duration of an emission and HYSPLIT models their advective motion using three-dimensional velocity vectors from the meteorological data. In addition, the particle approach adds a random component to their advective motion that approximates a random walk process calibrated by local atmospheric turbulence. HYSPLIT particles are assigned a proportional fraction of pollutant mass at the time of emission and shed mass through atmospheric removal processes (dry and wet deposition). Concentrations for a grid cell are calculated through the sum of masses of particles within the grid cell divided by the size of the grid cell. All HYSPLIT calculation methods are described in detail in Draxler and Hess (1997). I describe deposition processes and my choice of calibration parameters in the next section.

Each HYSPLIT run uses a 5-day set of hourly burning emissions at 21 vertical levels for a single fire location. I set HYSPLIT to release 300,000 particles per emissions hour, which are evenly divided among the vertical emission levels. I allow HYSPLIT to calculate the travel of particles for 920 hours (approximately five and a half weeks) from the hour of the first emission. From these calculations, HYSPLIT creates an hourly concentration grid for the CONUS model domain roughly matching the resolution of the meteorological data, with each grid square encompassing approximately 1,600 km sq. for 2004-2010. I sample concentrations from each fire event's grid at 10 meters above ground level at pollution monitoring sites and census tract centroids, sum concentrations across all fire events, and average the resulting hourly concentrations to daily average concentrations by each sampling site.

While the raw output is constructed from emissions measures and conversions that would denominate it in μgm^{-3} if it were to be taken literally, I remain agnostic about the units of the output and allow first-stage regressions to implicitly rescale the wildfire PM2.5 measure. In Appendix Section 8.3, I establish that the output has a strongly logarithmic fit to observed pollution data and take a logarithmic transformation of the raw concentrations shifted by a small constant. This will be the wildfire PM2.5 instrument used for the remainder of the paper.

8.2.2 Deposition Processes

HYSPLIT's modeling of deposition, or the removal of pollutants from the atmosphere by precipitation and settling or impaction upon terrain, plays an important role in generating independent variation among pollutants to allow the separate identification of their health effects. HYSPLIT dynamically accounts for the amount of air pollution lost to precipitation by modeling the interaction of traveling parcels of air pollution from origin to destination with temporally and spatially smooth representations of precipitation events. HYSPLIT models particle pollutant wet deposition (also referred to as "wet removal" and "wet scavenging") via two processes described as in-cloud removal ("washout") and below-cloud removal ("rainout").[6] For gaseous pollutants, it uses a calculation method based on gas solubility. HYSPLIT has one common process for both particles and gases for modeling dry deposition which assumes a rate of removal driven by wind speed. One pollutant-specific constant calibrates the intensity of each process: the washout ratio, representing an average ratio of pollutant concentration in air to concentration in water at the ground; the rainout rate, or a fixed rate of pollutant removal while pollutant concentrations are in a meteorological layer with precipitation (s^{-1}); the Henry's Law Constant for wet removal of soluble gases ($mol\,atm^{-1}$); and the dry deposition velocity (ms^{-1}). The constants I choose for each pollutant type, along with corresponding citations, are reported in Appendix Table 14. For reference, I also report constants for related pollutants that I do not model.

Wet deposition of particulate pollutants is characterized by HYSPLIT through one process in which polluted air is ingested over time into proximal atmospheric moisture (washout), and another in which rain falls through polluted air (rainout). Wet deposition processes play a relatively larger role in mass removal of fine particulate pollutants than they do for gaseous pollutants, up to an order of magnitude higher, though this relation varies by species. While there is substantial heterogeneity in the efficiency with which PM2.5 pollutants are removed by rain because of the many component subspecies and variation in the particle size distribution, a washout ratio of 1×10^5 is broadly used as an estimate for the washout ratio of general PM2.5. In the absence of well-established parameters for rainout rates, I use HYSPLIT's suggested particle rainout rate of $5 \times 10^{-5} s^{-1}$ which has been used in other HYSPLIT particulate modeling applications (Chand et al. 2008; Wen et al. 2013). I expect that empirically-derived washout ratios will capture most deposition since they are often measured without HYSPLIT's deposition process distinction in mind, and at least one study finds that below-cloud deposition is insignificant for fine particles except in extreme precipitation events (Andronache 2003).

Instead of explicit washout and rainout parameters, gaseous pollutants' wet deposition is calibrated by the appropriate Henry's Law constant for the water-soluble gas. Henry's Law holds that at a constant temperature, the solubility of gas in a liquid is proportional to the pressure of the gas surrounding the liquid. An intuitive example of Henry's Law at work is a carbonated soda: while sealed, a soda bottle contains liquid with dissolved CO2 and a space above the liquid with CO2 gas. The opening of the bottle lowers the resulting pressure above the liquid, and over time the CO2 escapes from the liquid and into the open air through the bottle opening. The reverse process occurs if there is liquid in the same bottle with no CO2, and CO2 is injected into the empty space of the sealed bottle: the higher the pressure of the resulting air space in the bottle (and the greater the concentration of CO2), the greater the equilibrium concentration of CO2 in the liquid will be. Henry's Law constants are chosen from an extensive collection of estimates from academic papers (Sander 1999). Estimates are typically calculated in one of three ways: by theoretical calculations, extrapolations from other measured constants, or by field measurements and experiments. For each gas, I choose the most recent estimate from a literature review where available. If a literature review-based estimate is not available, I choose the modal Henry's Law constant reported in Sander (1999).

Dry deposition is modeled through gravitational settling and impaction at ground level which intensifies with wind velocity. In the absence of precipitation and chemical reactions, dry deposition is the primary determinant of a

[6] There is some inconsistent usage of the terms "washout" and "rainout" in across some papers, their meanings occasionally swapped.

pollutant's lifetime in the atmosphere following emissions. I conduct a literature search for dry deposition velocities, using the compound name and "deposition velocity" as search terms. For deposition velocities for gases drawn from field observations, urban-setting deposition velocities are preferred. Many gases, such as NO and HCHO, do not have significant dry deposition fluxes over land. I use a deposition velocity of $0 ms^{-1}$ for such gases with trivial land deposition rates, and also for any gases for which I am unable to find any direct reference to deposition fluxes or velocities. The deposition velocities I choose are reported in Appendix Table 14.

8.3 Nonlinear First Stage Transformation

The relationship between measured concentrations and modeled concentrations is extremely nonlinear, requiring a monotonic transformation to maximize the predictive power of the wildfire pollution instrument. Figure 6a shows the estimated coefficients of a piecewise linear regression of daily station PM2.5 on wildfire PM2.5 interacted with vigintile (5-percentile-block) indicator, representing an approximation of the first derivative of the true dose response function between measured and raw modeled PM2.5 across the raw modeled PM2.5 distribution. This regression controls for year, month, and county fixed effects, with standard errors clustered at the state level. The pattern is highly nonlinear, scaling multiple orders of magnitude, with the estimated slope monotonically decreasing in concentration. A function of the form $f(x) = \frac{a}{x+c}$ (with $a > 0, c \geq 0$) follows a comparable pattern, suggesting that a linear approximation better predicts station PM2.5 using as a regressor the natural logarithm of modeled wildfire PM2.5 plus some constant. This nonlinear pattern implies that some combination of the emissions calculations and HYSPLIT is resulting in systematic overestimation of large concentrations and underestimation of small concentrations. The monotonically decreasing slope across the domain of concentrations implicates the dispersion calculation of HYSPLIT, which relies on calibration from atmospheric parameters to determine turbulent velocities and a Gaussian random component that determine the random-walk-like dispersive behavior of the particle. One explanation for the subsequent logarithmic fit is that the calibration of the Gaussian component's variance does not account for how the true variance is itself positively related to concentration level, resulting in systematic underestimation of dispersion for large concentrations and overestimation for small concentrations (causing overestimated and underestimated concentrations, respectively).

A logarithmic transformation of the wildfire pollution measure in the first stage accounts for the implicit overdispersion of concentrations along trajectories by compressing the distribution of magnitudes. To accomplish this transformation without discarding zero values, I take the natural logarithm of daily average wildfire PM2.5 plus a constant. The choice of constant by which to shift the raw concentration before taking the logarithm, the "shift parameter" has two important impacts: it determines the position of zeroes on the log function, and relatedly, it changes the relative curvature of the fit of logged concentrations to observed concentrations. Shift parameters that are too small will result in the log transformation overestimating the contrast between the effect of positive wildfire concentrations relative to zero wildfire concentrations, while shift parameters that are too large will cause an underestimated contrast. Large shift parameters may also distort the marginal effects for larger values in the distribution. One sensible choice of shift parameter is a point at which positive concentrations could be considered effectively zero for the dependent variable of interest. HYSPLIT's concentration outputs near zero can be reasonably framed as a sensitivity problem: there is a computational threshold below which it will never give a positive value, and the distribution of values approaching zero is continuous until the trivial minimum value at $4.92 \times 10^{-34} \mu g m^{-3}$. I choose a value corresponding to the 10th percentile of positive values ($7.21 \times 10^{-14} \mu g m^{-3}$), add it to the raw concentration value, and take the logarithm. For ease of interpretation later, I also shift all transformed values by the minimum of the transformed values to make all values nonnegative. Figure 6b shows the same regression as in Figure 6b, but now with logged daily wildfire PM2.5 interacted with vigintile indicator. The slopes now fall within the same order of magnitude, slightly increasing in vigintile (implying a gradual shift to underestimation of marginal changes in concentrations relative to smaller

vigintiles).

Additionally, there are several numerically large outliers which may affect the fit, but station data provides a way of trimming outlier values sensibly. I account for these right-tail outliers by assigning the instrument the station PM2.5 value if the raw modeled wildfire PM2.5 exceeds the station-observed PM2.5 value, and both values are greater than $65\mu gm^{-3}$. Empirically, the latter condition implies the former in 100 percent of cases, which motivated the selection of this cutoff. Less than 0.1 percent of station-days have measured PM2.5 exceeding $65\mu gm^{-3}$. All other wildfire PM2.5 values exceeding $65\mu gm^{-3}$ (approximately 0.6 percent of all values) are set to $65\mu gm^{-3}$. This adjustment compresses the right tail of the distribution, enhancing the performance of the logarithmic transformation I take to improve the fit of the instrument (in exchange for losing some variation in extreme values). Because the observed nonlinear relationship and outliers are ostensibly due to HYSPLIT's dispersion calculation methods, which are not unique to any pollutant, I assume that concentrations for other pollutant species follow a comparable relationship with their observed values (in the absence of daily station data to do a pollutant-specific adjustment). For all control species, I reduce all right-tail values for other pollutant species to their 98th percentile of positive values before taking the logarithmic transformation, since that is the approximate point at which the PM2.5 values always exceed station values. Then, I take the logarithm of the outlier-adjusted modeled concentration outputs plus the 10th percentile of their positive values added.

8.4 Coefficient Estimates under Non-Classical Measurement Error

Consider a simplified cross-sectional setting, with health outcome y as a function of true exposure x^*,

$$y_i = x_i^* \beta + \varepsilon_i$$

Assume x_i^* is uncorrelated with ε_i, and that the researcher only observes an imperfect measure x of x^*such that $x = x^* + e$. Define $var(x^*) = \sigma^2_{x^*}$, $var(e) = \sigma^2_e$, and $cov(x^*, e) = \sigma_{x^*e}$. Then, the probability limit of the ordinary least squares estimator of y on x can be written as

$$plim\hat{\beta} \quad = \quad \beta \frac{(\sigma^2_{x^*} + \sigma_{x^*e})}{\sigma^2_{x^*} + \sigma^2_e + 2\sigma_{x^*e}}$$

By the Cauchy-Schwarz inequality, the denominator is always positive: it represents the variance of the error-prone regressor x. $\sigma_{x^*e} = 0$ corresponds to the classical errors-in-variables assumption, which results in attenuation bias. The probability limit of the OLS estimate $\hat{\beta}$ is both attenuated and incorrectly-signed if $\sigma_{x^*e} < 0$ and $\sigma^2_{x^*} < |\sigma_{x^*e}|$. Negative correlation between the true regressor value and the size of the measurement error is plausible in the pollution setting if population density increases both pollution levels and reduces exposure measurement error asymmetrically across polluted areas.

8.5 Change in Finite-Sample Bias of IV when Fixed Effects are Included

Another possibility for the increase in estimates across different fixed effects specifications is that the inclusion of fixed effects potentially changes the finite-sample bias of the 2SLS estimate, even with equally "strong" instruments in the Staiger and Stock (1997) nomenclature. This is because both the strength of the first-stage relationship and the amount of correlation between the endogenous variable and structural equation error term both determine finite-sample bias of IV estimators. In this interpretation, the inclusion of fixed effects chooses variation in observed PM2.5

that is less correlated with unobserved determinants of health than the total variation in PM2.5, while simultaneously not weakening the relationship between wildfire PM2.5 and station PM2.5 sufficiently to counterbalance the change. The corresponding OLS estimates for PM2.5 are close to zero and relatively precise, implying that IV estimates would be biased toward zero. One might be inclined to believe that between-county variation in pollution is more strongly associated with unobserved determinants of health than within-county variation is, both based on the mechanisms proposed for either correlation (e.g., residential and industrial sorting versus micro-level changes in economic activity) and more pragmatically through the revealed preference of researchers for multiple time-series and panel studies over cross-sectional studies. Murray (2006) provides a simplified approximation of the finite-sample bias of 2SLS based on Hahn and Hausman (2001) (where the structural and first-stage equation error terms have varianced normalized to one) as follows:

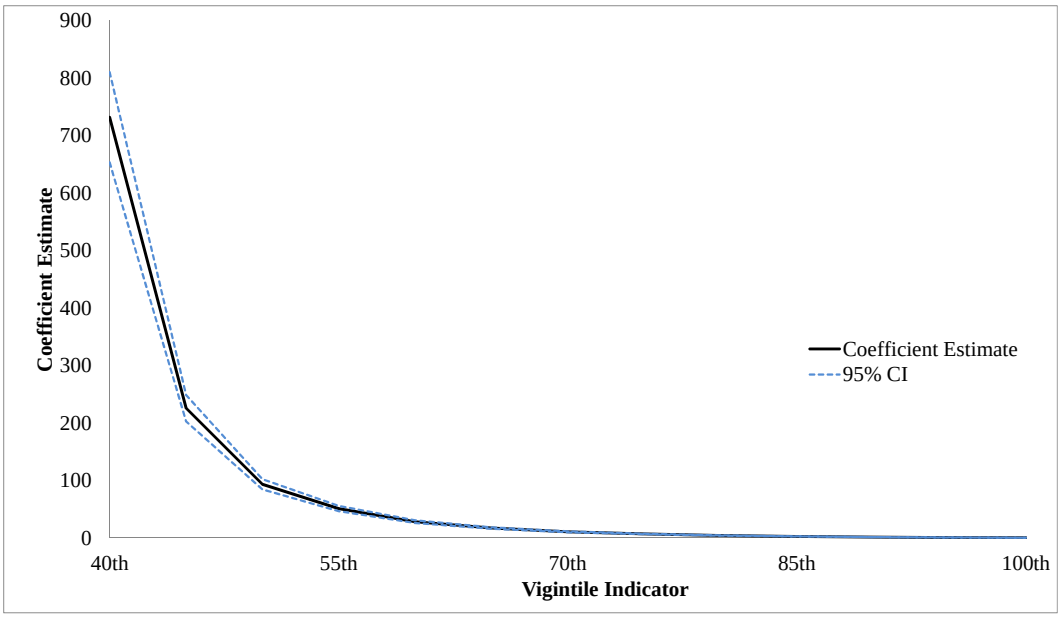
$$E(\hat{\beta}_{,2SLS}) - \beta \approx \frac{l\rho(1-\tilde{R}^2)}{N\tilde{R}^2}.$$

Here, β is the effect of PM2.5 on mortality, $l = 1$ is the number of instruments, ρ is the correlation between the structural and first-stage equation error terms (a measure of the level of endogeneity), $\tilde{R}^2$ is the partial R-squared of the first stage regression, and N is the sample size. If the inclusion of fixed effects decreases ρ to ρ_{fe}, but decreases $\tilde{R}^2$ to $\tilde{R}^2_{fe}$, then the approximate bias decreases as long as $\frac{\rho}{\rho_{fe}} > \left(\frac{(1-\tilde{R}^2)}{\tilde{R}^2}\right)\left(\frac{(1-\tilde{R}^2_{fe})}{\tilde{R}^2_{fe}}\right)^{-1}$.

8.6 Appendix Tables and Figures

Figure 6: Piecewise Regression Coefficient Estimates of Daily Station PM2.5 on Raw and Log-transformed Wildfire PM2.5 Model Output, by Vigintile

(a) Raw Wildfire PM2.5 Output



(b) Log-Transformed Wildfire PM2.5 Output

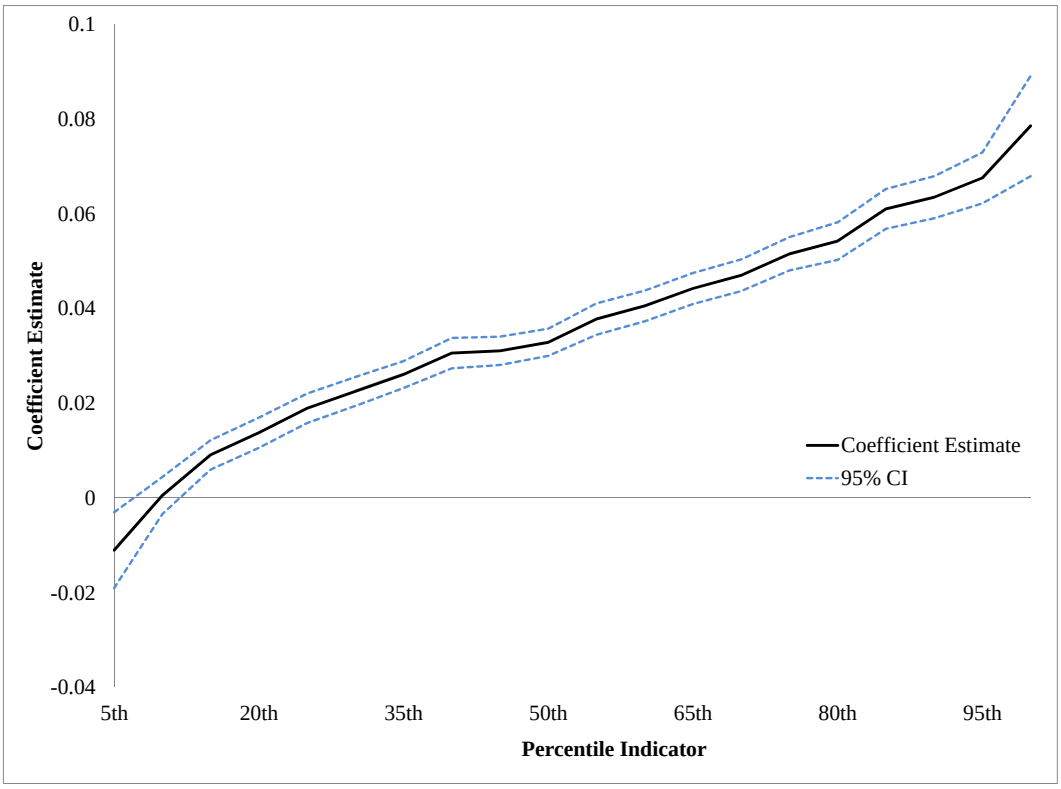


Table 14: Henry's Law Constants and Dry Deposition Velocities for Gaseous Pollutants

Pollutant	Henry's Law Constant	Citation	Dry Deposition Velocity	Citation
Ozone (O3)	9.40E-03		1.25E-02	
Sulfur Dioxide (SO2)	**1.23E+01**		1.50E-02	
Nitrogen Dioxide (NO2	**1.20E-02**		3.60E-03	
Nitric Oxide (NO)	**1.90E-03**		0.00E+00	
Carbon Monoxide (CO)	9.40E-04		3.00E-04	
Methane (CH4)	1.40E-03			
Carbon Dioxide (CO2)	3.40E-02			
Ammonia (NH3)	**6.10E+01**		6.50E-03	
Formaldehyde (HCHO)	3.20E+03		5.00E-03	
Mercury Elemental (Gas	9.30E-02		1.00E-04	
Mercury Reactive Gaseo	1.40E+06		1.00E-03	
Toluene (C6H5CH3)	**1.50E-01**		**0.00E+00**	
Benzene	1.60E-01			
O-Xylene	1.30E-01			
M-Xylene	1.90E-01			
P-Xylene	1.30E-01			

Table 15: Regression of Organic Gases on Wildfire PM2.5

	M/P Xylene	**Benzene**	**Toluene**	**Ethylbenzene**	**O-Xylene**	**Styrene**
			Panel A: 2SLS			
Coefficient	-2.4e-04	8.7e-03	1.5e-02	8.5e-04	1.6e-03	-5.5e-03
	(5.1e-03)	(6.5e-03)	(1.7e-02)	(2.6e-03)	(2.8e-03)	(3.8e-03)
% Wildfire	-0.2%	6.6%	4.6%	1.8%	2.9%	-18.8%
95% CI Upper	7.1%	16.2%	15.2%	12.5%	13.0%	7.2%
95% CI Lower	-7.5%	-3.0%	-6.0%	-9.0%	-7.3%	-44.9%
			Panel B: 2SLS with Wildfire NO2, SO2 Controls			
Coefficient	1.1e-02	1.3e-02	6.0e-03	-2.0e-03	-4.3e-03	-2.0e-03
	(7.4e-03)	(8.1e-03)	(4.0e-02)	(6.4e-03)	(7.3e-03)	(4.3e-03)
% Wildfire	8.1%	9.6%	1.9%	-4.3%	-7.9%	-7.0%
95% CI Upper	18.7%	21.5%	26.9%	22.1%	18.6%	22.1%
95% CI Lower	-2.5%	-2.4%	-23.1%	-30.7%	-34.5%	-36.1%
			Panel C: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls			
Coefficient	1.0e-02	**1.7e-02****	1.9e-02	1.7e-03	-6.3e-05	-2.3e-03
	(7.7e-03)	(7.7e-03)	(3.1e-02)	(4.8e-03)	(5.2e-03)	(5.1e-03)
% Wildfire	7.6%	12.9%	6.0%	3.6%	-0.1%	-7.8%
95% CI Upper	18.6%	24.4%	25.3%	23.4%	18.9%	26.8%
95% CI Lower	-3.5%	1.5%	-13.3%	-16.2%	-19.1%	-42.4%
Mean Concentration	2.00E+00	1.90E+00	4.60E+00	6.90E-01	7.90E-01	4.30E-01
N	8044	8924	8653	8580	8364	7761
	Chloroform	**Carbon Tetrachloride**	**Methyl Chloroform**	**Tetrachloro-ethylene**	**Trichloro-ethylene**	**Dichloro-Trifluoroethane**
			Panel A: 2SLS			
Coefficient	-8.6e-05	-1.8e-04	-2.6e-04	-2.4e-04	-5.6e-05	-5.6e-05
	(7.6e-05)	(1.3e-04)	(5.1e-04)	(2.8e-04)	(4.9e-04)	(4.9e-04)
% Wildfire	-4.9%	-2.9%	-6.4%	-4.5%	-2.2%	-2.2%
95% CI Upper	3.6%	1.3%	17.8%	5.8%	36.5%	36.5%
95% CI Lower	-13.3%	-7.1%	-30.6%	-14.7%	-41.0%	-41.0%
			Panel B: 2SLS with Wildfire NO2, SO2 Controls			
Coefficient	-1.8e-04	-9.6e-05	-4.2e-04	**-1.0e-03***	8.5e-04	8.5e-04
	(1.5e-04)	(1.4e-04)	(8.7e-04)	(5.9e-04)	(6.4e-04)	(6.4e-04)
% Wildfire	-10.5%	-1.5%	-10.1%	-18.7%	34.2%	34.2%
95% CI Upper	6.1%	2.9%	31.2%	3.1%	84.9%	84.9%
95% CI Lower	-27.0%	-6.0%	-51.4%	-40.6%	-16.4%	-16.4%
			Panel C: 2SLS with Wildfire NO2, SO2, NH3, VOC Controls			
Coefficient	-1.9e-04	**-2.4e-04***	-7.6e-04	-6.3e-04	9.5e-04	9.5e-04
	(1.6e-04)	(1.5e-04)	(1.1e-03)	(6.0e-04)	(7.6e-04)	(7.6e-04)
% Wildfire	-11.0%	-3.8%	-18.3%	-11.9%	38.2%	38.2%
95% CI Upper	6.6%	0.7%	34.1%	10.3%	98.9%	98.9%
95% CI Lower	-28.6%	-8.4%	-70.7%	-34.2%	-22.5%	-22.5%
Mean Concentration	2.50E-02	8.80E-02	6.00E-02	7.60E-02	3.50E-02	3.50E-02
N	8086	7687	7569	8131	8090	8090

Coefficients are for a one-unit change in the wildfire PM2.5 instrument. Units for organic gases are ppbC (parts per billion carbon). "% Wildfire" is calculated as the overall quantity of pollutant predicted by the instrument divided by the mean concentration times 100%. Standard errors clustered at state-year level are in parentheses. Significance stars represent p < 0.1 (*), p < .05 (**), p < .01 (***).

Table 16: Regression of PM2.5 Metal Subspecies on Wildfire PM2.5, Set I

	Aluminum	Ammonium Ion	Ammonium Nitrate	Ammonium Sulfate	Antimony	Barium	Calcium	Cerium	Cesium	Chlorine
Coefficient	**-4.6e-04*** **	**2.1e-02*** **	**9.8e-03*** **	**1.8e-02*** **	-2.4e-05	**7.7e-05*** **	4.2e-05	**6.4e-05** **	**6.6e-05*** **	-1.1e-04
	(1.2e-04)	(2.6e-03)	(2.7e-03)	(3.7e-03)	(1.9e-05)	(2.5e-05)	(8.8e-05)	(2.7e-05)	(1.7e-05)	(1.9e-04)
% Wildfire	-15.9%	24.0%	23.1%	12.3%	-2.3%	9.0%	1.2%	7.0%	9.0%	-5.0%
95% CI Upper	*-7.5%*	*29.9%*	*35.7%*	*17.3%*	*1.3%*	*14.8%*	*6.1%*	*12.7%*	*13.7%*	*11.1%*
95% CI Lower	*-24.3%*	*18.2%*	*10.6%*	*7.3%*	*-5.9%*	*3.2%*	*-3.7%*	*1.2%*	*4.3%*	*-21.1%*
Coefficient	**-6.0e-04*** **	**2.8e-02*** **	**2.0e-02*** **	**2.1e-02*** **	**-5.3e-05***	**1.2e-04*** **	1.1e-04	**2.2e-04*** **	**5.5e-05** **	-5.0e-04
	(1.5e-04)	(3.5e-03)	(5.1e-03)	(6.3e-03)	(2.8e-05)	(4.1e-05)	(1.2e-04)	(3.9e-05)	(2.6e-05)	(3.7e-04)
% Wildfire	-20.8%	32.2%	0.4806919	14.3%	-5.1%	13.6%	3.2%	23.8%	7.4%	-21.8%
95% CI Upper	*-10.5%*	*40.2%*	*0.7209205*	*22.8%*	*0.3%*	*22.9%*	*10.2%*	*32.2%*	*14.4%*	*9.7%*
95% CI Lower	*-31.1%*	*24.2%*	*0.2404632*	*5.8%*	*-10.6%*	*4.3%*	*-3.8%*	*15.4%*	*0.4%*	*-53.4%*
Coefficient	**-5.6e-04*** **	**2.4e-02*** **	**1.8e-02*** **	**1.5e-02** **	-4.0e-05	**1.2e-04*** **	1.2e-04	**2.3e-04*** **	**6.5e-05** **	-3.8e-04
	(1.6e-04)	(3.2e-03)	(4.6e-03)	(6.4e-03)	(3.0e-05)	(4.0e-05)	(1.2e-04)	(4.0e-05)	(3.0e-05)	(3.4e-04)
% Wildfire	-19.3%	27.5%	43.4%	10.2%	-3.9%	14.5%	3.4%	25.4%	8.8%	-16.5%
95% CI Upper	*-8.6%*	*34.7%*	*64.7%*	*18.9%*	*1.8%*	*23.6%*	*10.4%*	*34.0%*	*16.8%*	*12.9%*
95% CI Lower	*-30.1%*	*20.2%*	*22.0%*	*1.5%*	*-9.5%*	*5.5%*	*-3.6%*	*16.8%*	*0.8%*	*-45.9%*
Mean Concentration	4.30E-02	1.30E+00	6.10E-01	2.10E+00	1.60E-02	1.30E-02	5.20E-02	1.40E-02	1.10E-02	3.40E-02
N	15529	10899	6299	6295	10859	10719	15516	10273	10432	15561

Table 17: Regression of PM2.5 Metal Subspecies on Wildfire PM2.5, Set II

	Chromium	**Chromium Vi**	**Cobalt**	**Copper**	**Europium**	**Gallium**	**Gold**	**Hafnium**	**Indium**
Coefficient	-6.1e-06	**6.6e-05*** **	**1.6e-06*** **	5.8e-06	-1.1e-05	2.6e-06	2.0e-06	**4.2e-05*** **	**-1.3e-05** **
	(1.0e-05)	(1.7e-05)	(5.7e-07)	(8.3e-06)	(1.5e-05)	(1.6e-06)	(2.6e-06)	(1.1e-05)	(6.0e-06)
% Wildfire	-5.5%	9.0%	3.2%	2.5%	-3.6%	2.7%	1.3%	8.4%	-2.5%
95% CI Upper	*12.6%*	*13.7%*	*5.3%*	*9.5%*	*5.5%*	*6.1%*	*4.6%*	*12.6%*	*-0.2%*
95% CI Lower	*-23.6%*	*4.3%*	*1.0%*	*-4.5%*	*-12.7%*	*-0.7%*	*-2.0%*	*4.2%*	*-4.7%*
Coefficient	2.5e-06	**5.5e-05** **	**2.8e-06** **	5.4e-06	9.1e-06	-6.4e-07	**-7.3e-06** **	-1.6e-05	-8.6e-06
	(1.8e-05)	(2.6e-05)	(1.2e-06)	(1.6e-05)	(2.6e-05)	(2.3e-06)	(3.4e-06)	(1.2e-05)	(7.8e-06)
% Wildfire	2.2%	7.4%	5.5%	2.3%	2.9%	-0.7%	-4.7%	-3.2%	-1.6%
95% CI Upper	*34.3%*	*14.4%*	*9.9%*	*16.1%*	*19.2%*	*4.0%*	*-0.4%*	*1.6%*	*1.3%*
95% CI Lower	*-29.8%*	*0.4%*	*1.0%*	*-11.5%*	*-13.4%*	*-5.3%*	*-9.1%*	*-7.9%*	*-4.5%*
Coefficient	7.5e-06	**6.5e-05** **	**3.7e-06*** **	5.0e-06	-6.2e-06	**4.4e-06***	-8.3e-07	7.2e-06	-6.9e-06
	(1.8e-05)	(3.0e-05)	(1.2e-06)	(2.0e-05)	(2.9e-05)	(2.6e-06)	(3.9e-06)	(1.2e-05)	(8.0e-06)
% Wildfire	6.7%	8.8%	7.1%	2.1%	-2.0%	4.6%	-0.5%	1.4%	-1.3%
95% CI Upper	*37.9%*	*16.8%*	*11.6%*	*18.7%*	*15.9%*	*9.9%*	*4.3%*	*6.3%*	*1.7%*
95% CI Lower	*-24.4%*	*0.8%*	*2.5%*	*-14.5%*	*-19.9%*	*-0.8%*	*-5.4%*	*-3.4%*	*-4.3%*
Mean Concentration	1.70E-03	1.10E-02	7.80E-04	3.50E-03	4.80E-03	1.50E-03	2.40E-03	7.70E-03	7.90E-03
N	15579	10432	10769	15561	7850	7896	7896	7850	10319

Table 18: Regression of PM2.5 Metal Subspecies on Wildfire PM2.5, Set III

	Iridium	**Iron**	***Lanthanum***	**Magnesium**	**Manganese**	**Molybdenum**	**Niobium**	**Potassium**	**Potassium Ion**
Coefficient	-9.1e-07	-2.0e-04	**1.0e-04*****	**-1.4e-04*****	5.2e-06	4.3e-06	**2.5e-06***	**6.4e-04*****	**5.7e-04*****
	(3.0e-06)	(1.3e-04)	*(2.5e-05)*	(3.4e-05)	(1.7e-05)	(3.9e-06)	(1.4e-06)	(1.4e-04)	(2.0e-04)
% Wildfire	-0.5%	-4.2%	*11.8%*	-13.4%	2.8%	2.1%	2.0%	15.1%	13.9%
95% CI Upper	*2.7%*	*1.1%*	*17.6%*	*-7.0%*	*20.8%*	*5.8%*	*4.2%*	*21.7%*	*23.6%*
95% CI Lower	*-3.7%*	*-9.4%*	*5.9%*	*-19.8%*	*-15.3%*	*-1.6%*	*-0.2%*	*8.4%*	*4.2%*
Coefficient	**-1.5e-05*****	-2.8e-05	**2.2e-04*****	-7.2e-05	6.9e-06	**-2.1e-05*****	1.9e-06	**5.5e-04****	5.0e-04
	(4.1e-06)	(2.0e-04)	*(3.6e-05)*	(5.3e-05)	(3.4e-05)	(6.7e-06)	(1.9e-06)	(2.2e-04)	(3.3e-04)
% Wildfire	-8.3%	-0.6%	*26.2%*	-6.9%	3.7%	-10.4%	1.5%	12.9%	12.1%
95% CI Upper	*-3.9%*	*7.6%*	*34.7%*	*3.1%*	*39.3%*	*-4.0%*	*4.5%*	*23.1%*	*27.6%*
95% CI Lower	*-12.8%*	*-8.7%*	*17.8%*	*-17.0%*	*-32.0%*	*-16.8%*	*-1.6%*	*2.7%*	*-3.4%*
Coefficient	-3.4e-06	4.9e-05	**2.4e-04*****	**-9.0e-05***	3.1e-05	-1.0e-05	**5.1e-06****	**7.0e-04*****	**7.2e-04****
	(4.7e-06)	(2.2e-04)	*(3.9e-05)*	(5.3e-05)	(3.7e-05)	(7.8e-06)	(2.1e-06)	(2.4e-04)	(3.4e-04)
% Wildfire	-1.9%	1.0%	*28.2%*	-8.6%	16.8%	-5.0%	4.0%	16.5%	17.5%
95% CI Upper	*3.2%*	*10.0%*	*37.3%*	*1.4%*	*56.0%*	*2.4%*	*7.3%*	*27.4%*	*33.9%*
95% CI Lower	*-7.0%*	*-7.9%*	*19.1%*	*-18.7%*	*-22.4%*	*-12.5%*	*0.7%*	*5.5%*	*1.2%*
Mean Concentration	2.80E-03	7.20E-02	*1.30E-02*	1.50E-02	2.80E-03	3.20E-03	1.90E-03	6.40E-02	6.20E-02
N	7850	15574	*7896*	15081	15611	8378	7850	15592	10628

Table 19: Regression of PM2.5 Metal Subspecies on Wildfire PM2.5, Set IV

	Rubidium	**Samarium**	**Scandium**	**Selenium**	**Silicon**	**Silver**	**Sodium**	**Sodium Ion**	**Strontium**
Coefficient	**1.2e-06****	-1.1e-05	**-5.7e-05*****	**4.6e-06*****	**-6.7e-04****	**-1.5e-05*****	-7.2e-05	**6.4e-04*****	1.4e-07
	(5.7e-07)	(1.1e-05)	(9.5e-06)	(1.6e-06)	(2.7e-04)	(5.1e-06)	(1.5e-04)	(2.1e-04)	(2.3e-06)
% Wildfire	2.6%	-4.3%	-14.1%	6.4%	-8.2%	-3.7%	-1.4%	7.8%	0.2%
95% CI Upper	*4.9%*	*4.8%*	*-9.5%*	*10.7%*	*-1.8%*	*-1.3%*	*4.1%*	*12.8%*	*5.7%*
95% CI Lower	*0.2%*	*-13.5%*	*-18.7%*	*2.1%*	*-14.7%*	*-6.1%*	*-6.9%*	*2.7%*	*-5.3%*
Coefficient	**4.2e-06*****	-5.1e-06	**-1.3e-04*****	**1.1e-05*****	**-7.6e-04****	9.8e-06	1.4e-04	3.4e-04	**-6.7e-06***
	(7.3e-07)	(2.0e-05)	(1.5e-05)	(2.6e-06)	(3.1e-04)	(6.3e-06)	(2.7e-04)	(2.9e-04)	(3.6e-06)
% Wildfire	8.8%	-2.1%	-31.2%	14.8%	-9.3%	2.4%	2.6%	4.1%	-8.2%
95% CI Upper	*11.8%*	*14.2%*	*-23.9%*	*21.8%*	*-1.8%*	*5.4%*	*12.9%*	*11.1%*	*0.3%*
95% CI Lower	*5.8%*	*-18.3%*	*-38.5%*	*7.8%*	*-16.8%*	*-0.6%*	*-7.7%*	*-2.9%*	*-16.8%*
Coefficient	**4.7e-06*****	-9.0e-06	**-1.1e-04*****	**1.1e-05*****	**-6.8e-04****	9.3e-06	2.3e-04	4.3e-04	-5.2e-06
	(7.9e-07)	(2.2e-05)	(1.6e-05)	(2.7e-06)	(3.3e-04)	(6.8e-06)	(3.0e-04)	(3.1e-04)	(3.9e-06)
% Wildfire	9.8%	-3.7%	-26.3%	15.6%	-8.4%	2.2%	4.4%	5.3%	-6.4%
95% CI Upper	*13.1%*	*14.1%*	*-18.6%*	*23.1%*	*-0.4%*	*5.5%*	*15.6%*	*12.8%*	*2.9%*
95% CI Lower	*6.6%*	*-21.5%*	*-34.1%*	*8.0%*	*-16.3%*	*-1.0%*	*-6.8%*	*-2.2%*	*-15.7%*
Mean Concentration	7.10E-04	3.80E-03	6.30E-03	1.10E-03	1.20E-01	6.20E-03	7.80E-02	1.20E-01	1.20E-03
N	15561	7850	7863	15611	15561	10319	15112	10697	15561

Table 20: Regression of PM2.5 Metal Subspecies on Wildfire PM2.5, Set VI

	Tantalum	**Terbium**	**Tin**	**Titanium**	**Tungsten**	**Vanadium**	**Yttrium**	**Zinc**	**Zirconium**
Coefficient	**1.6e-05****	-8.7e-06	3.8e-06	**-2.3e-05****	**1.2e-05****	2.3e-06	**2.5e-06***	-1.6e-06	**-4.2e-06****
	(6.3e-06)	(1.8e-05)	(6.5e-06)	(9.1e-06)	(5.6e-06)	(2.6e-06)	(1.4e-06)	(3.2e-05)	(2.1e-06)
% Wildfire	4.7%	-2.2%	0.5%	-8.2%	4.5%	1.9%	2.8%	-0.2%	-3.9%
95% CI Upper	*8.5%*	*6.9%*	*2.3%*	*-1.8%*	*8.8%*	*6.4%*	*5.9%*	*9.0%*	*-0.2%*
95% CI Lower	*1.0%*	*-11.4%*	*-1.3%*	*-14.6%*	*0.3%*	*-2.5%*	*-0.2%*	*-9.5%*	*-7.6%*
Coefficient	8.9e-06	6.7e-06	-1.7e-05	-1.1e-05	-1.1e-07	3.0e-06	**4.6e-06*****	5.4e-05	**-8.6e-06****
	(7.5e-06)	(3.1e-05)	(1.2e-05)	(1.2e-05)	(7.1e-06)	(4.8e-06)	(1.7e-06)	(4.0e-05)	(3.4e-06)
% Wildfire	2.7%	1.7%	-2.4%	-3.8%	0.0%	2.6%	5.2%	8.1%	-7.9%
95% CI Upper	*7.2%*	*17.4%*	*1.1%*	*4.5%*	*5.3%*	*10.7%*	*9.0%*	*19.8%*	*-1.7%*
95% CI Lower	*-1.8%*	*-14.0%*	*-5.8%*	*-12.1%*	*-5.4%*	*-5.5%*	*1.3%*	*-3.7%*	*-14.0%*
Coefficient	**2.7e-05*****	4.4e-06	-1.4e-05	-5.7e-06	1.3e-05	8.6e-07	**6.5e-06*****	6.4e-05	**-6.6e-06***
	(8.8e-06)	(3.3e-05)	(1.3e-05)	(1.2e-05)	(7.7e-06)	(4.6e-06)	(2.0e-06)	(4.1e-05)	(3.5e-06)
% Wildfire	8.1%	1.1%	-1.9%	-2.0%	4.9%	0.7%	7.3%	9.5%	-6.0%
95% CI Upper	*13.3%*	*17.9%*	*1.7%*	*6.3%*	*10.8%*	*8.4%*	*11.6%*	*21.6%*	*0.3%*
95% CI Lower	*2.8%*	*-15.6%*	*-5.5%*	*-10.4%*	*-1.0%*	*-6.9%*	*2.9%*	*-2.5%*	*-12.3%*
Mean Concentration	5.10E-03	6.00E-03	1.10E-02	4.20E-03	4.00E-03	1.70E-03	1.40E-03	1.00E-02	1.60E-03
N	7850	7850	10809	15561	7850	15574	8470	15574	15161