

**Special Section:**

Fire in the Earth System

**Key Points:**

- While the majority of large fires occur in the United States (US) West, a majority of smoke-attributable US mortality and morbidity occur east of ~100 degW
- A higher percent of mortality and morbidity is attributable to smoke in high fire-impacted northwestern states, relative to other US states
- Disability-adjusted life years attributable to fine particles in smoke are much higher than that from gas-phase hazardous air pollutants

**Supporting Information:**

Supporting Information may be found in the online version of this article.

**Correspondence to:**

K. O'Dell,  
[katelyn.odell@colostate.edu](mailto:katelyn.odell@colostate.edu)

**Citation:**






O'Dell, K., Bilsback, K., Ford, B., Martenies, S. E., Magzamen, S., Fischer, E. V., & Pierce, J. R. (2021). Estimated mortality and morbidity attributable to smoke plumes in the United States: Not just a western US problem. *GeoHealth*, 5, e2021GH000457. <https://doi.org/10.1029/2021GH000457>

Received 18 MAY 2021

Accepted 11 AUG 2021

© 2021. The Authors. GeoHealth published by Wiley Periodicals LLC on behalf of American Geophysical Union. This is an open access article under the terms of the [Creative Commons Attribution-NonCommercial-NoDerivs License](#), which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

## Estimated Mortality and Morbidity Attributable to Smoke Plumes in the United States: Not Just a Western US Problem

Katelyn O'Dell<sup>1</sup> , Kelsey Bilsback<sup>1</sup>, Bonne Ford<sup>1</sup> , Sheena E. Martenies<sup>2</sup>, Sheryl Magzamen<sup>3</sup> , Emily V. Fischer<sup>1</sup> , and Jeffrey R. Pierce<sup>1</sup> 

<sup>1</sup>Department of Atmospheric Science, Colorado State University, Fort Collins, CO, USA, <sup>2</sup>Department of Kinesiology and Community Health, University of Illinois at Urbana-Champaign, Urbana, IL, USA, <sup>3</sup>Department of Environmental and Radiological Health Sciences, Colorado State University, Fort Collins, CO, USA

**Abstract** As anthropogenic emissions continue to decline and emissions from landscape (wild, prescribed, and agricultural) fires increase across the coming century, the relative importance of landscape-fire smoke on air quality and health in the United States (US) will increase. Landscape fires are a large source of fine particulate matter (PM<sub>2.5</sub>), which has known negative impacts on human health. The seasonal and spatial distribution, particle composition, and co-emitted species in landscape-fire emissions are different from anthropogenic sources of PM<sub>2.5</sub>. The implications of landscape-fire emissions on the sub-national temporal and spatial distribution of health events and the relative health importance of specific pollutants within smoke are not well understood. We use a health impact assessment with observation-based smoke PM<sub>2.5</sub> to determine the sub-national distribution of mortality and the sub-national and sub-annual distribution of asthma morbidity attributable to US smoke PM<sub>2.5</sub> from 2006 to 2018. We estimate disability-adjusted life years (DALYs) for PM<sub>2.5</sub> and 18 gas-phase hazardous air pollutants (HAPs) in smoke. Although the majority of large landscape fires occur in the western US, we find the majority of mortality (74%) and asthma morbidity (on average 75% across 2006–2018) attributable to smoke PM<sub>2.5</sub> occurs outside the West, due to higher population density in the East. Across the US, smoke-attributable asthma morbidity predominantly occurs in spring and summer. The number of DALYs associated with smoke PM<sub>2.5</sub> is approximately three orders of magnitude higher than DALYs associated with gas-phase smoke HAPs. Our results indicate awareness and mitigation of landscape-fire smoke exposure is important across the US.

**Plain Language Summary** The pollutants from landscape (wild, prescribed, and agricultural) fires are expected to have an increasing impact on air quality and health in the United States (US) across the current century. The implications of landscape-fire smoke on the regional and seasonal distribution of health events and the relative health importance of specific pollutants within smoke are not well understood. In the present study, we assess the seasonal and regional distribution of the health impacts from US smoke exposure from 2006 to 2018. We also estimate the long-term health impacts for both fine particles (PM<sub>2.5</sub>) and gas-phase hazardous air pollutants (HAPs) in smoke. Although the majority of large landscape fires occur in the western US, we find the majority of deaths (74%) and asthma emergency department visits and hospital admissions (on average 75% across 2006–2018) attributable to smoke occur outside the West. Across the US, smoke-attributable asthma emergency department visits predominantly occur in spring and summer. The long-term health impacts associated with smoke PM<sub>2.5</sub> are much higher than the estimated long-term health impacts of gas-phase smoke HAPs. Our results indicate awareness and mitigation of landscape-fire smoke exposure is important across the US, not just in regions in proximity to large wildfires.

### 1. Introduction and Background

Smoke from landscape (wild, prescribed, and agricultural) fires significantly degrades air quality across the United States (US) (Brey, Barnes, et al., 2018; Brey & Fischer, 2016; Buysse et al., 2019; Ford et al., 2017; Kaulfus et al., 2017; Val Martin et al., 2015). Landscape-fire smoke, hereafter simply “smoke,” contributes over 40% of primary emissions of particulate matter with diameters smaller than 2.5 microns (PM<sub>2.5</sub>) in

the US (US EPA, 2017) and is responsible for a majority of non-anthropogenic exceedances in National Ambient Air Quality Standards (NAAQS) for  $PM_{2.5}$  (David et al., 2021). In heavily fire-impacted parts of the western US, fires dominate interannual variability in PM (Spracklen et al., 2007), have led to observed increases in the intensity of extreme  $PM_{2.5}$  events (McClure & Jaffe, 2018), and possible increases in summer mean  $PM_{2.5}$ , despite decreasing anthropogenic emissions (O'Dell et al., 2019). As anthropogenic emissions of  $PM_{2.5}$  continue to decline (Lam et al., 2011; Leibensperger et al., 2012; Tagaris et al., 2007; Val Martin et al., 2015) and smoke  $PM_{2.5}$  increases (Ford et al., 2018; Li et al., 2020; Liu et al., 2016; Neumann et al., 2021; Yue et al., 2013), the relative importance of smoke  $PM_{2.5}$  for US air quality will likely increase.

Acute exposure to smoke has negative impacts on human health (Cascio, 2018; Liu et al., 2015; Reid, Brauer, et al., 2016, and references within), which may differ from the health effects of anthropogenic  $PM_{2.5}$  due to differences in composition and exposure. Many epidemiological studies of acute exposure to smoke  $PM_{2.5}$  have observed impacts on respiratory morbidity (e.g., Aguilera et al., 2021; DeFlorio-Barker et al., 2019; Gan et al., 2020; Hutchinson et al., 2018; Magzamen et al., 2021; Rappold et al., 2012; Reid, Jerrett, et al., 2016). Impacts on mortality and cardiovascular morbidity are less certain (e.g., Reid, Brauer, et al., 2016), but evidence for these outcomes of acute smoke exposure is growing (e.g., Doubleday et al., 2020; Magzamen et al., 2021; Wettstein et al., 2018). Several recent works have investigated differences in asthma-related and respiratory hospital admissions on smoke-impacted days compared to non-smoke-impacted days and found larger concentration response functions for  $PM_{2.5}$  on smoke-impacted days (Aguilera et al., 2021; DeFlorio-Barker et al., 2019; Kiser et al., 2020). A potentially different impact of smoke  $PM_{2.5}$  versus anthropogenic  $PM_{2.5}$  on health is also supported by evidence from toxicological studies that suggest that smoke-sourced  $PM_{2.5}$  may be more harmful than other sources of  $PM_{2.5}$  due to compositional differences (Wegesser et al., 2009). In addition to differences in particle composition, differences in exposure may also differentially impact health. While there are seasonal differences in  $PM_{2.5}$  abundance and composition driven by modest variability in anthropogenic sources and atmospheric chemistry (Bell et al., 2007), emissions of  $PM_{2.5}$  from landscape fires are highly episodic and have distinct seasonal cycles. The seasonality of fires and smoke events varies by US region due to both climate and human factors (Balch et al., 2017; Brey, Barnes, et al., 2018; McCarty et al., 2009; Westerling et al., 2003). The implications of the unique composition and exposure timing of smoke-specific  $PM_{2.5}$  on the US healthcare system are not well understood.

Repeated acute smoke events from landscape fires contribute to the overall long-term exposure to multiple health-relevant pollutants. Health effects of chronic exposure to smoke-specific  $PM_{2.5}$  have yet to be quantified. However, chronic exposure to anthropogenic  $PM_{2.5}$  has been associated with all-cause mortality, cardiopulmonary mortality, and lung cancer (Crouse et al., 2019; Krewski et al., 2009; Pope et al., 2009). In addition to  $PM_{2.5}$ , wildfire smoke also contains many hazardous air pollutants (HAPs; Andreae, 2019; O'Dell et al., 2020; US EPA, 2015) which are compounds known or suspected to lead to serious health impacts (US EPA, 2015). The relative contribution of these different pollutants to potential health impacts of chronic smoke exposure is currently understudied.

This work leverages a growing knowledge of smoke concentrations and health responses to use a health impact assessment (HIA) to quantify: (a) the seasonal and spatial distribution of US asthma hospital admissions and emergency department (ED) visits attributable to acute smoke  $PM_{2.5}$  exposure, (b) the mortality from chronic smoke  $PM_{2.5}$  exposure by state, and (c) the relative contribution of HAPs to health impacts of chronic smoke exposure. We build upon previous US smoke HIAs and leverage new knowledge of smoke in several ways. In this HIA, we use observation-based smoke  $PM_{2.5}$  estimates (O'Dell et al., 2019), as opposed to previous model-based estimates (Fann et al., 2018; Ford et al., 2018; Neumann et al., 2021). In addition, we apply a recent meta-analysis of the impacts of smoke  $PM_{2.5}$  exposure on asthma morbidity (Borchers Arriagada et al., 2019) to estimate the asthma hospital admissions and asthma ED visits attributable to acute smoke  $PM_{2.5}$  exposure. Finally, we incorporate observation-based estimates of HAPs in smoke (O'Dell et al., 2020) into our HIA. To our knowledge, this is the first time HAPs have been included in a smoke HIA. The results of this HIA will be beneficial for individual, state, and regional awareness and preparedness for the health burdens posed by smoke exposure.

## 2. Materials and Methods

### 2.1. Smoke PM<sub>2.5</sub> and HAPs Concentration Estimates

To conduct this HIA on smoke in the US, we estimated observation-based daily smoke PM<sub>2.5</sub> concentrations by combining surface observations and satellite-based smoke plume estimates. This method was developed by O'Dell et al. (2019), and the data are available from 2006 to 2018. Here, we provide a brief description of the data. For a full description, please refer to O'Dell et al. (2019) or the metadata available in the data repository linked in the data availability statement. Daily average PM<sub>2.5</sub> observations from the surface monitors in the US EPA Air Quality System (AQS) were interpolated to a 15 × 15 km grid using ordinary kriging. Daily smoke plume information was obtained from NOAA Hazard Mapping System (HMS) smoke plume polygons (Brey, Ruminski, et al., 2018; Ruminski et al., 2006). These polygons indicate where smoke is likely present somewhere in the daytime atmospheric column according to visible satellite imagery. Combining the daily HMS smoke plume polygons with the gridded daily average PM<sub>2.5</sub> concentrations, we estimated a non-smoke background PM<sub>2.5</sub> as the seasonal median of the gridded PM<sub>2.5</sub> on days without an overlapping HMS smoke plume. We also conduct our analysis using the seasonal mean of the gridded PM<sub>2.5</sub> on days without an overlapping HMS smoke plume (results provided in Figures S1 and S2 and Table S1) and find this choice does not impact our main conclusions. The smoke PM<sub>2.5</sub> was then calculated as the difference between the kriged PM<sub>2.5</sub> and the non-smoke background PM<sub>2.5</sub> on smoke-impacted days. On non-smoke-impacted days, smoke PM<sub>2.5</sub> was set to zero. These data have been previously used in atmospheric science, epidemiological, and economic studies of US smoke PM<sub>2.5</sub> (Abdo et al., 2019; Burkhardt et al., 2019, 2020; Gan et al., 2020; Lipner et al., 2019; Magzamen et al., 2021; O'Dell et al., 2019, 2020).

We estimated gas-phase HAPs enhancements in smoke (hereafter “smoke HAPs”) using a previously published method from O'Dell et al. (2020). Briefly, ratios of smoke HAPs to PM<sub>1</sub> (particulate matter with aerodynamic diameters smaller than 1 μm) were developed using observations from the Western Wild-fire Experiment on Cloud Chemistry, Aerosol Absorption, and Nitrogen (WE-CAN). WE-CAN was an aircraft-based field campaign which sampled lofted smoke plumes from large western US wildfires in summer 2018. Ratios of smoke HAPs to smoke PM<sub>1</sub> were developed for “young,” “medium,” and “old” smoke with approximate chemical ages of <1 day, 1–3 days, and >3 days, respectively. Here, we used the “young” ratios for an upper-estimate of smoke HAPs concentrations. We multiplied these ratios by 2006–2018 mean kriged smoke PM<sub>2.5</sub> by grid cell for a gridded estimate of chronic smoke HAPs exposures. To perform this calculation, we made several assumptions. First, the WE-CAN ratios of HAPs to PM relied on smoke PM<sub>1</sub> mass concentrations, however, our kriged estimates were of smoke PM<sub>2.5</sub> mass concentrations. Thus, in order to use these HAP to PM<sub>1</sub> ratios with our kriged smoke PM<sub>2.5</sub> estimates, we assumed the mass concentration of particles with diameters between 1 and 2.5 μm was negligible. Volume size distributions of smoke aerosol from Bian et al. (2020) indicate that <5% of PM<sub>2.5</sub> volume (and hence mass) exists in the diameter range of 1–2.5 μm, thus errors due to this assumption are <5%, smaller than the relative uncertainty from the concentration response function. Further, we assumed that the WE-CAN HAPs to PM<sub>1</sub> ratios are representative of all US smoke plumes, but smoke HAPs concentrations may vary by fuel type (e.g., Gilman et al., 2015), burn conditions (Sekimoto et al., 2018), and smoke age (O'Dell et al., 2020). However, as we show in the results, the estimated health impacts of smoke HAPs are much smaller than that of smoke PM<sub>2.5</sub>, such that the overall health estimates of smoke are not greatly influenced by our assumptions in the HAPs calculation.

### 2.2. HIA of Acute Smoke Exposure

We focused the present HIA of acute smoke exposure on asthma hospitalizations and asthma ED visits as these outcomes are consistently associated with smoke exposure (e.g., Reid, Brauer, et al., 2016) and have been included in a meta-analysis of acute smoke PM<sub>2.5</sub> exposure (Borchers Arriagada et al., 2019). We estimated asthma hospitalizations and ED visits attributable to acute smoke PM<sub>2.5</sub> exposure with the following health impact function,

$$\Delta \text{Events} = \text{Population} \times \left( Y_0 / 365 \right) \times \left( 1 - e^{-\beta \times \Delta \text{PM}_{2.5}} \right), \quad (1)$$

from Anenberg et al. (2014, 2010) for chronic PM<sub>2.5</sub> exposure. We assumed the acute smoke health impact function follows the same functional form (e.g., Pratt et al., 2019). In Equation 1,  $Y_0$  is the annual baseline

asthma hospital admission rate or asthma ED visit rate,  $\Delta PM_{2.5}$  is the daily smoke  $PM_{2.5}$  concentration, and  $\beta$  is defined,

$$\beta = \ln(RR)/\Delta X \quad (2)$$

where RR is the relative risk per  $\Delta X$  increase in smoke  $PM_{2.5}$ . We used smoke-specific RRs for asthma hospital admissions and asthma ED visits from a meta-analysis of smoke  $PM_{2.5}$  exposure in the US (Borchers Arriagada et al., 2019). The meta-analysis RRs for the US are provided in the supplement of Borchers Arriagada et al. (2019) and incorporate RRs from several different time lags (i.e., admissions/visits at different numbers of days after smoke  $PM_{2.5}$  exposure) but is similar in magnitude to the meta-analysis of lag-0-specific RRs for both asthma hospital admissions and asthma ED visits. We also calculated a pooled smoke-specific RR using the US studies from Borchers Arriagada et al. (2019) (Alman et al., 2016; Delfino et al., 2009; Gan et al., 2017; Hutchinson et al., 2018; Le et al., 2014; Reid, Jerrett, et al., 2016; Resnick et al., 2015) and additional RRs from eastern US fires (Rappold et al., 2012; Tinling et al., 2016) as well as two recently published RRs based on smoke  $PM_{2.5}$  from western US fires (Gan et al., 2020; Magzamen et al., 2021). RRs from these individual studies, the pooled RR, and meta-analysis RR are plotted in Figure S3. As shown in Figure S3, we found the meta-analysis central estimate and 95<sup>th</sup> percent confidence interval (CI) lies within the much wider 95<sup>th</sup> percent CI of our pooled RRs estimate. Thus, despite our addition of several RRs from both eastern and western US fires, our pooled RR and the US-specific meta-analysis RR from Borchers Arriagada et al. (2019) are not statistically different. We used the US-specific meta-analysis RR in our calculations due to its tighter CI.

Values of the smoke-specific RRs and baseline rates used in Equations 1 and 2 are provided in Table S2. National annual baseline rates for the year 2010 for asthma (ICD9-493) hospital admissions and ED visits were obtained from the Healthcare Cost and Utilization Project (HCUP). We used national estimates of baseline rates from the National Emergency Department Sample (NEDS) and National Inpatient Sample (NIS), which are weighted national estimates based on state-provided data (AHRQ, 2006). Although asthma prevalence varies by state (BRFSS/CDC, 2019) and asthma hospitalization and ED visit rates vary by season (Verdier et al., 2017), to our knowledge, a complete national database of sub-national or sub-annual asthma ED/hospitalization rates is currently unavailable. Gridded population estimates for 2010 were obtained from the National Space Administration's Socioeconomic Data and Applications Center (NASA SEDAC, 2018), which we regridded from the original 2.5 arc-minute grid resolution to our  $15 \times 15$  km kriged  $PM_{2.5}$  grid. Daily, kriged smoke  $PM_{2.5}$  estimates, described previously, are used as the smoke  $PM_{2.5}$  input in Equation 1. These data were applied in Equation 1 to estimate daily, gridded asthma hospital admissions and asthma ED visits attributable to smoke  $PM_{2.5}$  at lag day 0. The largest contributors of uncertainty in US smoke  $PM_{2.5}$  HIAs are uncertainty in the shape of the concentration response function and smoke-specific  $PM_{2.5}$  concentrations (Cleland et al., 2021). We represent uncertainty in the asthma morbidity attributable to smoke  $PM_{2.5}$  as the range of asthma ED visits and asthma hospitalizations estimated by calculating asthma ED visits and asthma hospitalizations using the lower and upper bounds of the 95% CI in the smoke-specific RRs.

We defined nine US regions following Brey, Ruminski, et al. (2018). The regions are roughly the 10 EPA regions; however, only contiguous US states are included and several regions are combined/altered to follow likely fire and smoke patterns. The list of states in each region are provided in Table S3. Seasons were defined as follows: Winter: January, February, March; Spring: April, May June; Summer: July, August, September; Fall: October, November, December. We chose this less conventional seasonal categorization so that we better group regional US wildfire activity into a single season category where possible (results using a more standard seasonal categorization are provided in Figures S4 and S5). Gridded asthma ED visits and hospital admissions attributable to smoke  $PM_{2.5}$  were summed by each region and season. We sum by region for asthma morbidity, as opposed to by state as was done for mortality (described in the next section), because we found seasonal, by-state totals for each year to be too cumbersome for the main text. We present the seasonal fraction of asthma ED visits attributable to smoke  $PM_{2.5}$  by state in Figures S6–S9.

### 2.3. HIA for Chronic Exposure to Smoke $PM_{2.5}$

As no concentration response function for mortality specific to chronic exposure to smoke  $PM_{2.5}$  currently exists, we used the Global Exposure Mortality Model (GEMM, Burnett et al., 2018) to estimate premature



mortality and disability-adjusted life years (DALYs) attributable to chronic exposure to both all-source and smoke  $PM_{2.5}$ . We note excess risk of mortality from chronic exposure to smoke  $PM_{2.5}$  may differ from all-source  $PM_{2.5}$  due to differences in  $PM_{2.5}$  composition, toxicity, and exposure type (e.g., episodic vs. consistent). However, at present, there are no studies of increased mortality risk from chronic exposure to smoke  $PM_{2.5}$ , thus we assumed the GEMM is applicable to smoke  $PM_{2.5}$ . The GEMM was developed from 41 cohort studies in 16 different countries on the increased mortality risk from chronic exposure to all-source ambient  $PM_{2.5}$ . We estimated mortality and DALYs attributable to all-source  $PM_{2.5}$  from the GEMM following,

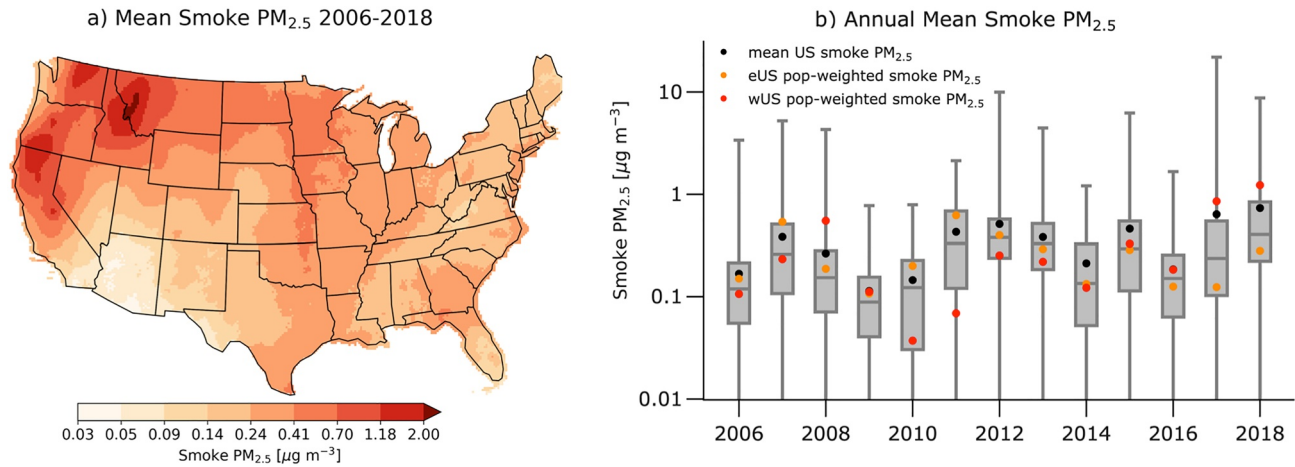
$$\Delta \text{Events} = \text{Population} \times Y_0 \times (1 - 1/HR), \quad (3)$$

where *Events* is mortalities or DALYs attributable to  $PM_{2.5}$ , *Population* is the regridded 2010 population from SEDAC described in Section 2.2,  $Y_0$  is the sum of baseline mortality or DALY rates for non-communicable diseases and lower respiratory infections, and *HR* is the hazard ratio from Burnett et al. (2018). Although the HR from Burnett et al. (2018) was developed specifically for mortality, we assume it can be applied to estimate DALYs (the sum of years of life lost and years of living with disability), as with prior  $PM_{2.5}$  mortality HRs (Burnett et al., 2014; Cohen et al., 2017). We used the all-cause mortality HR function with all countries included, which includes all non-communicable diseases and lower respiratory infections, with a threshold concentration of  $2.4 \mu\text{g m}^{-3}$ , the lowest observed concentration in the cohort studies used to develop the GEMM. Baeline all-cause (sum of non-communicable diseases and lower respiratory infections) mortality and DALY rates for 2010 were obtained from the GBD (GBD, 2019) and are provided in Table S2. In Table S1, we provide estimated mortalities and DALYs for smoke  $PM_{2.5}$  using the five leading causes of death HRs from the GEMM.

The mortality and DALYs attributable to smoke  $PM_{2.5}$  were estimated by multiplying the mortality (or DALYs) attributable to all-source  $PM_{2.5}$  from Equation 3 by the smoke  $PM_{2.5}$  fraction of 2006–2018 mean  $PM_{2.5}$  at each grid cell. We follow this approach, as opposed to applying the GEMM directly to smoke  $PM_{2.5}$  concentrations, due to the non-linearity of the concentration response function (the “attribution method” from Bilsback et al., 2020 and Kodros et al., 2016). With this method, we estimate excess mortalities and DALYs attributable to all-source and smoke  $PM_{2.5}$  for each grid cell and summed the excess mortality across each US state. We represent an uncertainty range in mortality and DALYs attributable to all-source  $PM_{2.5}$  and smoke  $PM_{2.5}$  as the range of deaths (or DALYs) estimated by calculating mortality using the upper and lower bounds of the uncertainty range ( $\pm 2$  standard error) in the GEMM concentration response function coefficients.

#### 2.4. HIA for Chronic Exposure to Smoke HAPs

To estimate DALYs attributable to smoke HAPs, we took a different approach than that used to estimate DALYs attributable to smoke  $PM_{2.5}$ . The method used to estimate DALYs from smoke  $PM_{2.5}$ , described previously in Section 2.3, relies on epidemiological concentration response functions relating exposure with specific diseases (e.g., Burnett et al., 2018), which are subsequently associated with an estimated number of DALYs (GBD, 2019; see Table S2). There are currently no concentration response functions associating the speciated smoke HAPs studied in this work with incidence of certain diseases in humans. Therefore, to estimate the DALYs attributable to smoke HAPs, we used estimates of human damage factors, expressed as DALYs, per annual intake of HAPs from Huijbregts et al. (2005). A full description of the calculation of DALYs per pollutant intake can be found in Huijbregts et al. (2005). Briefly, the DALY per intake factors were estimated through extrapolation of animal toxicity literature to estimate pollutant toxicity and subsequent disease incidence in humans. Disease incidence per pollutant exposure estimates were then combined with an estimated number of DALYs per disease. The disease per intake and DALY per disease factors were then combined to determine a final DALYs per pollutant intake factor. With this method, Huijbregts et al. (2005) estimated DALYs per year due to cancer and noncancer effects per mass intake of 1,192 pollutants. These DALY factors have been previously applied to estimate DALYs from HAPs in third-hand cigarette smoke exposure (Sleiman et al., 2014) and indoor exposure to HAPs (Logue et al., 2012). While these DALY factors allow us to compare health impacts of smoke  $PM_{2.5}$  and HAPs with the same metric (DALYs), we note the two methods used to estimate DALYs are very different. Although the approach based on concentration response functions in humans is the more precise method, it is not possible to apply such an approach to estimate DALYs from speciated HAPs, thus we rely on the DALY factor method.



**Figure 1.** 2006–2018 mean smoke  $PM_{2.5}$  on a  $15 \times 15$  km grid (panel a) and the distribution of annual average smoke  $PM_{2.5}$  across all grid cells for each year (panel b). Boxes in panel (b) extend from the 25th to 75th percentile, with a bar across the box indicating the median value. Box whiskers extend from the minimum to maximum value. Black points represent the area-weighted mean smoke  $PM_{2.5}$  and orange and red points indicate the population-weighted mean  $PM_{2.5}$  in the eastern and western US states, respectively.

We applied the DALY factors to estimate DALYs per person attributable to chronic exposure to smoke HAPs by,

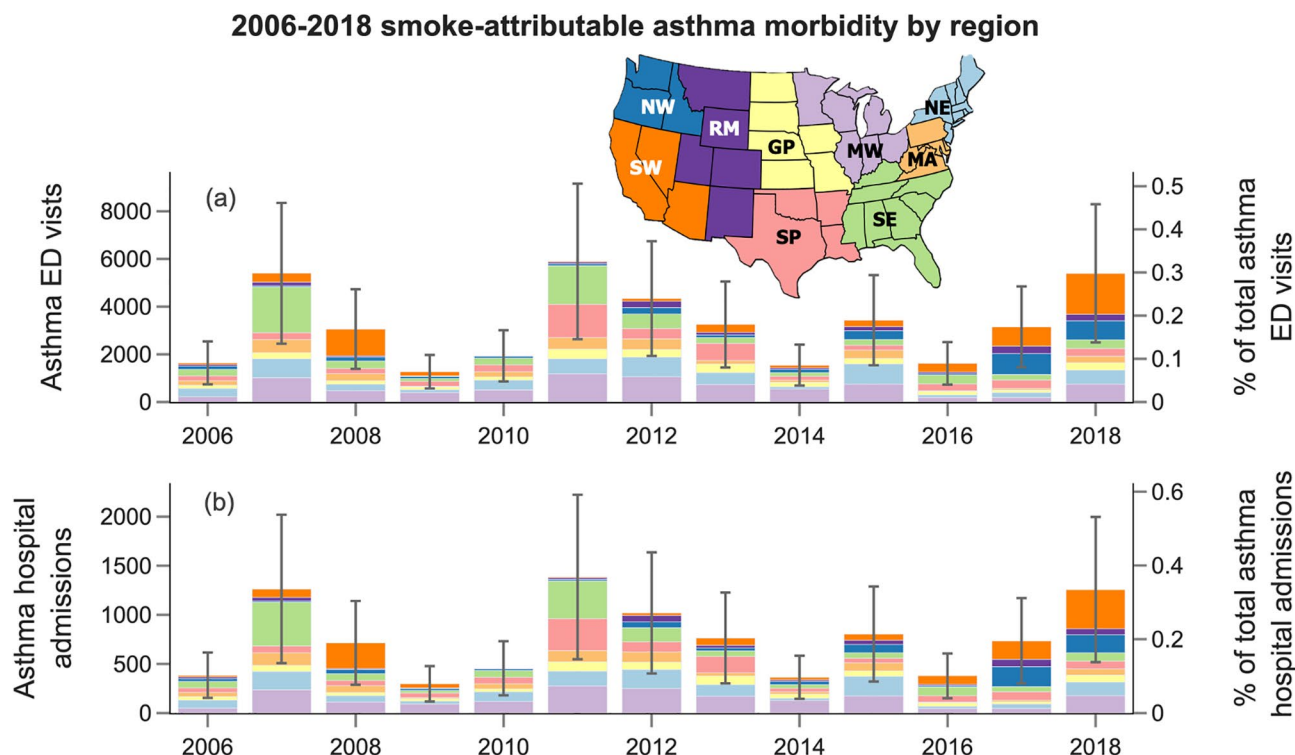
$$DALYs = C_i \times 365 \times V \times \left[ \left( \partial DALY_{cancer} / \partial intake \right)_i + \left( \partial DALY_{noncancer} / \partial intake \right)_i \right], \quad (4)$$

similar to Logue et al., (2012). In Equation 4,  $C_i$  is the concentration of smoke HAP  $i$  described in Section 2.1,  $V$  is  $14.4 \text{ m}^3 \text{ day}^{-1}$ , an estimated population-mean volume of air inhaled per day from Logue et al. (2012), and  $(\partial DALY_{cancer} / \partial intake)_i$  is the estimated DALYs due to cancer effects, and  $(\partial DALY_{noncancer} / \partial intake)_i$  is the estimated DALYs due to noncancer effects, per intake of pollutant  $i$  from Huijbregts et al. (2005). Unlike the smoke  $PM_{2.5}$  DALYs calculation, there is no threshold concentration applied for smoke HAPs. Implications of this are discussed in the results. Of the 32 smoke HAPs with estimated concentrations from Section 2.1, 25 have DALY factors for cancer and/or noncancer effects reported in Huijbregts et al. (2005). Huijbregts et al. (2005) reports the median estimate of DALY factors and provides an uncertainty estimate,  $k_i$ , expressed as the square root of the ratio of the 97.5<sup>th</sup> and 2.5<sup>th</sup> percentiles. This value is defined such that 95% of the distribution of DALY factors lie within a factor of  $k_i$  of the reported median estimate. We thus represent the 95% CI around the cancer and noncancer DALY factors as  $(\partial DALY / \partial intake)_i k_i^{-1}$  to  $(\partial DALY / \partial intake)_i k_i$ . The 95% CI around the DALY factors is large, spanning several orders of magnitude, driven by large uncertainties in extrapolating animal toxicity studies to humans and uncertainty in noncancer disease incidence and human impact (Huijbregts et al., 2005). We apply these upper and lower bounds on the 95% CI into Equation 4 to estimate the uncertainty bounds for our DALY estimates.

### 3. Results

#### 3.1. Landscape-Fire Smoke $PM_{2.5}$

Observation-based smoke  $PM_{2.5}$  estimates across the study period are presented in Figures 1a and 1b. Mean total  $PM_{2.5}$  from 2006 to 2018 and the long-term smoke  $PM_{2.5}$  fraction are shown in Figure S10. The 2006–2018 mean smoke  $PM_{2.5}$ , Figure 1a, reaches over  $2 \mu\text{g m}^{-3}$  in heavily fire-impacted regions of the western US. The box plots in Figure 1b show the distribution of annual average smoke  $PM_{2.5}$  across all  $15 \times 15$  km grid cells in the US for each year in our study period. In 2017, in several grid cells in Montana, the annual average smoke  $PM_{2.5}$  exceeded  $10 \mu\text{g m}^{-3}$ . Across all US grid cells, the area-weighted mean annual smoke  $PM_{2.5}$  (black points in Figure 1b) is much lower ranging from  $0.11 \mu\text{g m}^{-3}$  in 2009 to  $0.73 \mu\text{g m}^{-3}$  in 2018. In Figure 1b, we also provide population-weighted mean smoke  $PM_{2.5}$  estimates for the eastern and western US (orange and red points, respectively). These values were calculated as the sum of each western (eastern) US grid-cell annual mean  $PM_{2.5}$  multiplied by the fraction of the total western (eastern) US population in that grid cell. We find the area-weighted mean smoke  $PM_{2.5}$  across the US is often similar to



**Figure 2.** Asthma emergency department (ED, panel a) and asthma hospital admissions (panel b) attributable to smoke  $PM_{2.5}$  across the contiguous US for each year from 2006 to 2018. Colors represent different US regions, as indicated by the map, where darker colors represent regions in the western US and lighter colors represent regions outside the western US. The left y-axis on each panel represents the total number of events attributable to smoke  $PM_{2.5}$  and the right y-axis represents the percent of all asthma ED visits (panel a) or asthma hospital admissions (panel b) in the US that are attributable to smoke  $PM_{2.5}$ . Errors bars represent the range of morbidities estimated using the upper and lower 95% CI bounds on smoke-specific relative risk.

the population-weighted mean smoke  $PM_{2.5}$  in the eastern and western US. The mean population-weighted smoke  $PM_{2.5}$  across the full time period is higher in the western US ( $0.33 \mu g m^{-3}$ ) than the eastern US ( $0.26 \mu g m^{-3}$ ), however there is high inter-annual variability in both the western and eastern US population-weighted mean smoke  $PM_{2.5}$ . Our US-wide annual mean observation-based smoke  $PM_{2.5}$  is a factor of 2–6 lower than model-based estimates used in Fann et al. (2018), depending on the year. In addition, our long-term average smoke  $PM_{2.5}$  is lower than Ford et al. (2018) but of similar magnitude to Neumann et al. (2021), both model-based estimates. A direct, quantitative national or regional comparison between our chronic  $PM_{2.5}$  exposure estimates and these previous works is not possible due to a difference in time periods and regional definitions (or a lack of regional-level estimates). There are limitations to both model-based estimates (e.g., smoke dispersion in complex topography (Gan et al., 2017), determining plume injection height (Paugam et al., 2016), estimating fuel burned) and our observation-based estimates (e.g., lack of information on vertical smoke distribution (Brey, Ruminski, et al., 2018), sparse surface monitoring) that lead to uncertainties in total smoke  $PM_{2.5}$  concentrations.

### 3.2. Spatial Distribution of Asthma Morbidity Attributable to Smoke $PM_{2.5}$

In Figure 2, we show the contribution of each region to the total number and percent of asthma ED visits (Figure 2a) and asthma hospital admissions (Figure 2b) attributable to smoke  $PM_{2.5}$  in the US by year from 2006 to 2018. There is high inter-annual variability in the total amount of asthma morbidity attributable to smoke  $PM_{2.5}$  in the US over this time period. There is similarly high inter-annual variability in smoke  $PM_{2.5}$  concentrations over this same time period (O'Dell et al., 2019). Asthma ED visits attributable to smoke  $PM_{2.5}$  in the US range from approximately 1,300 to 5,900 visits per year, or 0.07%–0.33% of all asthma ED visits. The asthma hospital admissions attributable to smoke  $PM_{2.5}$  contribute a similar percent (0.08%–0.37%) of total annual asthma hospital admissions, compared to the asthma ED visits. We find a lower total number

**Table 1**

*Total Asthma Hospital Admissions, Asthma Emergency Department (ED) Visits, and Mortality Attributable to Smoke  $PM_{2.5}$  Exposure and Mortality Attributable to Total  $PM_{2.5}$  Exposure Across the Contiguous US From This Work Compared Results From Three Previous US Smoke Health Impact Assessments*

Outcome	This work	Ford et al. (2018) <sup>a</sup>	Fann et al. (2018) <sup>b</sup>	Neumann et al. (2021) <sup>c</sup>
All respiratory hospital admissions	-	-	3,900–8,500	350
Asthma hospital admissions	300–1,400 <sup>d</sup>	-	-	68
Asthma ED visits	1,300–5,900 <sup>d</sup>	-	-	400
Annual mortality attributable to chronic smoke $PM_{2.5}$ exposure	6,300 (CI: 4,800–7,800)	7,000–28,000	8,700–32,000	720–1,600
Annual mortality attributable to chronic $PM_{2.5}$ exposure	216,000 (CI: 163,000–266,000)	69,000–222,000	-	-

<sup>a</sup>Ford et al.(2018) results for the decade centered around 2000. Range given represents the range across multiple concentration response functions used in the study. <sup>b</sup>Fann et al.(2018) results were presented annually with two concentration response functions for mortality and two odds ratios for respiratory hospital admissions. The given range is the full range across all years and both concentration response functions. <sup>c</sup>Neumann et al. (2021) results for health impacts of western wildfires on the full US (i.e., non-western wildfires, prescribed burning, and agricultural burning were not included). The given range for mortality is the range across both concentration response functions used. <sup>d</sup>Range across all years in this analysis (2006–2018).

US, United States.

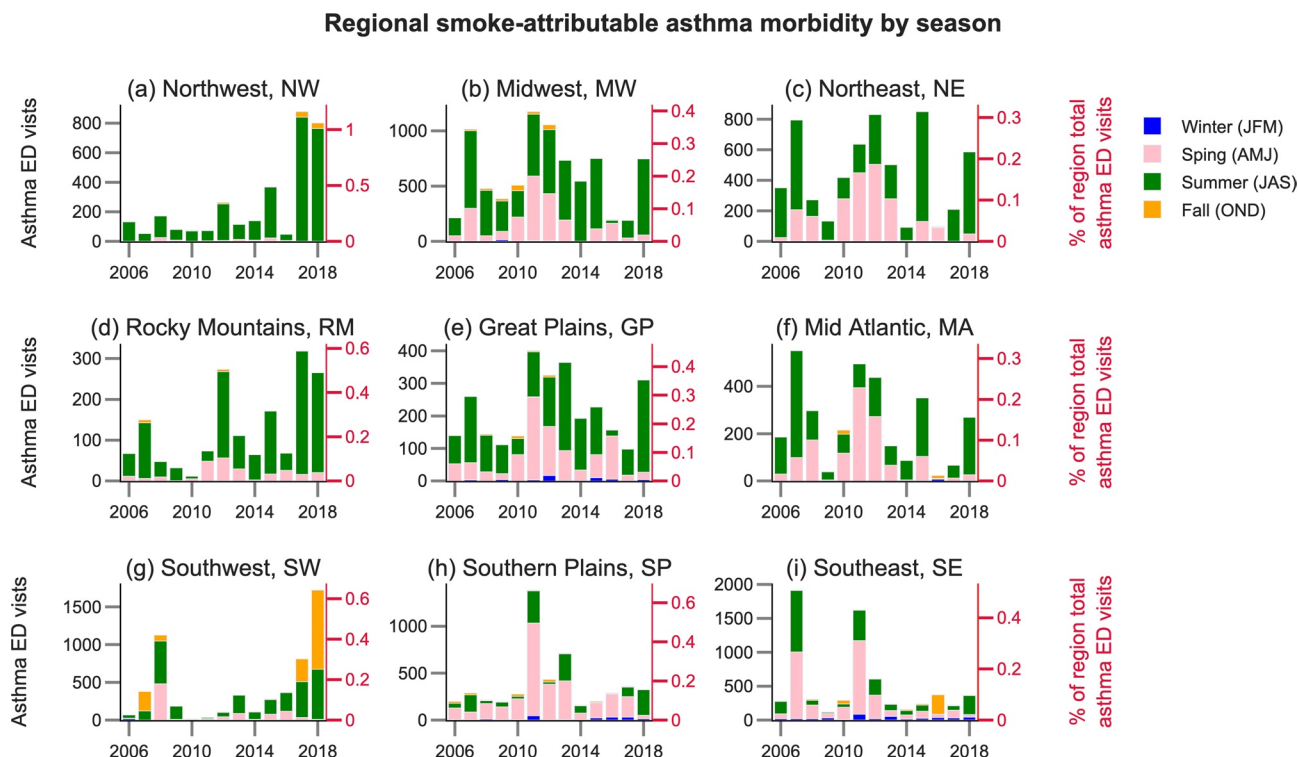
(300–1,400) of smoke  $PM_{2.5}$  attributable asthma hospital admissions than ED visits, due to a lower baseline rate in the former.

Total numbers and percent of asthma ED visits and hospital admissions attributable to smoke  $PM_{2.5}$  by year are given in Table 1 alongside previous estimates of respiratory morbidity attributable to smoke  $PM_{2.5}$  in the US from Fann et al. (2018) and Neumann et al. (2021). Our estimated asthma ED visits and hospital admissions are considerably higher than those from Neumann et al. (2021) of 400 and 68 per year, respectively. However, the estimates from Neumann et al. (2021) only account for smoke originating from fires in the western US and rely on different smoke-estimation methods and health impact functions than this work. In contrast, our estimates of asthma hospital admissions attributable to smoke  $PM_{2.5}$  are a factor of 6–8 lower than the all respiratory hospital admissions attributable to smoke  $PM_{2.5}$  in Fann et al. (2018). A lower number of asthma hospital admissions compared to all respiratory hospital admissions attributable to smoke  $PM_{2.5}$  is expected, due to a lower baseline rate in the former (AHRQ, 2006).

In most years, the majority of asthma ED visits and asthma hospital admissions attributable to smoke  $PM_{2.5}$  occur in non-western states (lighter colors in Figure 2, including the Midwest (MW), Great Plains (GP), Southern Plains (SP), Northeast (NE), Mid Atlantic (MA), and Southeast (SE) regions). There are only 2 years during our 13-year study period when over 50% of asthma morbidity attributable to smoke  $PM_{2.5}$  occurs in the western US (darker colors in Figure 2, including the Northwest (NW), Southwest (SW), and Rocky Mountain (RM) regions). In 2017 and 2018, 64%, and 52%, respectively, of all US asthma morbidity attributable to smoke  $PM_{2.5}$  occurred in the western states. In all other years during our study period, the western regions contributed on average 19% of US asthma morbidity attributable to smoke  $PM_{2.5}$ . The high inter-annual variability in the total amount of asthma morbidity attributable to smoke  $PM_{2.5}$  is also not exclusively driven by the western states. In fact, in the year with the most asthma morbidity attributable to smoke  $PM_{2.5}$ , 2011, less than 5% occurred in all the western states combined. This is largely driven by higher population densities in the East. As mentioned previously, we find the 2006–2018 population-weighted mean smoke  $PM_{2.5}$  concentration is higher in the western states ( $0.33 \mu\text{g m}^{-3}$ ) than the eastern states ( $0.26 \mu\text{g m}^{-3}$ ). However, the population is much higher in the East (around 226 million people) than the West (around 64 million people) overall. Thus, locations typically not considered to be heavily smoke impacted due to lower average concentrations of smoke  $PM_{2.5}$ , but with large population densities, can still experience a significant population health impact from smoke  $PM_{2.5}$ . In addition to population, variability in asthma prevalence by state (BRFSS/CDC, 2019) may influence the spatial distribution of smoke-attributable asthma morbidity, however, we are unable to account for this uncertainty in the present work.

In Figure 3, we show the number and percent of asthma ED visits attributable to smoke  $PM_{2.5}$  within each region. In general, the total number of asthma ED visits attributable to smoke  $PM_{2.5}$  in the worst wildfire years for the western US is similar to the total number of asthma ED visits attributable to smoke  $PM_{2.5}$  in several





**Figure 3.** Asthma emergency department (ED) visits attributable to smoke  $PM_{2.5}$  by each US region colored by season. The left y-axis represents the total number of asthma ED visits attributable to smoke  $PM_{2.5}$  in each region and the right y-axis represents the percent of region total asthma ED visits attributable to smoke  $PM_{2.5}$ . Region names and abbreviations follow the same regions from the map in Figure 2.

non-western regions such as the Northeast, Southeast, and Midwest. However, in the western regions, the percent of all asthma ED visits that are attributable to smoke  $PM_{2.5}$  is much higher than most other regions during heavy smoke years (e.g., 2017 and 2018). In the northwest region (Figure 3a), the percent of asthma ED visits attributable to smoke reaches over 1% in 2017 and 2018. This highlights that smoke  $PM_{2.5}$  has important, yet different, impacts on asthma morbidity across the US. In the western regions, where smoke concentrations are generally higher, but population density is lower, smoke  $PM_{2.5}$  contributes a higher fraction of regional asthma morbidity. In contrast, many eastern regions, which generally have higher population density but lower smoke  $PM_{2.5}$  concentrations see a larger total number of asthma morbidities attributable to smoke  $PM_{2.5}$ , yet these constitute a smaller fraction of all regional asthma morbidities.

### 3.3. Seasonality of Asthma Morbidity Attributable to Smoke $PM_{2.5}$

In Figure 3, we also show the seasonality of asthma morbidity attributable to smoke  $PM_{2.5}$  by US region. We present the seasonal fraction of asthma ED visits attributable to smoke  $PM_{2.5}$  by state in Figures S6–S9. In Figure S4, we show the percent contribution of each season to regional asthma morbidity attributable to smoke  $PM_{2.5}$  summed across all years. Across the US, most smoke-attributable asthma ED visits occur in spring and summer when 35% and 57%, respectively, of all asthma ED visits attributable to smoke  $PM_{2.5}$  from 2006 to 2018 occur. We note our seasonal attribution of smoke-specific asthma morbidity is based on annual asthma ED visit and hospitalization baseline rates. However, there are additional, non-smoke related, seasonal trends in asthma ED visits and hospitalizations, leading to an annual nadir in the total number of asthma ED visits and hospitalizations over the summer (Pendergraft et al., 2005; Silverman et al., 2003).

The seasonality of asthma ED visits attributable to smoke  $PM_{2.5}$  varies by US region. In the western regions shown in Figures 3a, 3d and 3g, most asthma ED visits attributable to smoke  $PM_{2.5}$  occur in the summer (see also Figure S4). This is largely driven by the timing of large wildfires (e.g., Brey, Barnes, et al., 2018; Jin et al., 2015; Westerling et al., 2003). In the Southeast and Southern Plains, 50% and 64%, respectively, of all asthma ED visits attributable to smoke  $PM_{2.5}$  from 2006 to 2018 occur in the spring (see Figures 3i

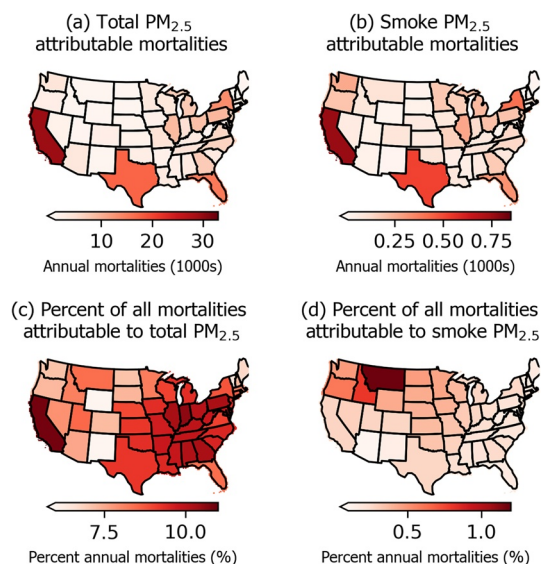
and 3b). This partially aligns with observed timing of local landscape fires (Brenner, 1991; Brey, Ruminski, et al., 2018; Dennis et al., 2002; McCarty et al., 2009). However, there is additional prescribed and agricultural burning in these regions in other seasons (Brey, Ruminski, et al., 2018; Dennis et al., 2002; McCarty et al., 2009), which is generally missed in our smoke  $PM_{2.5}$  method and hence is not reflected in the asthma morbidity attributable to smoke  $PM_{2.5}$ . The remainder of the regions (Midwest, Northeast, Great Plains, and Mid Atlantic) show a more even distribution of asthma ED visits attributable to smoke  $PM_{2.5}$  between spring and summer, with a majority of these visits occurring in summer (see Figures 3b, 3c, 3e, and 3f). Smoke in these regions is likely a combination of local landscape fires and transported smoke from other US regions and Canada (Brey, Ruminski, et al., 2018; DeBell et al., 2004; Le et al., 2014; Rogers et al., 2020; Wu et al., 2018). Overall, the timing of smoke-attributable asthma morbidity in the West follows the typical western wildfire season. However, the seasonal distribution of smoke-attributable asthma morbidity in the East is more mixed and varies by region, likely due to different combinations of smoke from both local and distant fires that follow different seasonal patterns (Brey, Ruminski, et al., 2018).

The timing and intensity of asthma morbidity attributable to smoke  $PM_{2.5}$  across the US presented here from 2006 to 2018 is likely to change in the future due to changes in human-fire interactions (Balch et al., 2017; Kupfer et al., 2020), population, land-management strategies (Ford et al., 2018), and climate-driven changes in fire regimes (Abatzoglou & Williams, 2016; Barbero et al., 2015; Goss et al., 2020; Spracklen et al., 2009; Williams et al., 2019). Balch et al. (2017) showed that human impacts on landscape fires have expanded fire seasons in the US. Climate impacts may also alter fire weather and seasonality in the future, altering timing of extreme wildfire conditions (Goss et al., 2020; Williams et al., 2019) and seasonal availability of suitable prescribed burning days (Kupfer et al., 2020). In addition, intensity and frequency of large fires is projected to increase in the western US (Barbero et al., 2015; Spracklen et al., 2009). Notably, in 2018 and more recently in the 2020 fire season (not included in our data set), large fires had extended, dramatic impacts on air quality in multiple large cities. For example, in Figure 3, we show regional asthma ED visits attributable to smoke  $PM_{2.5}$  in 2018 (and 2017 for the Northwest and Rocky Mountains) were well-above most other years in our time period for the western regions. Based on projected changes in wildfire intensity mentioned previously, 2018 may be more representative of western wildfire seasons in the future.

### 3.4. Spatial Distribution of Chronic Total $PM_{2.5}$ and Smoke $PM_{2.5}$ Mortalities

We estimate long-term exposure to smoke  $PM_{2.5}$  leads to 6,300 (CI: 4,800–7,800) additional deaths per year, 3% of all  $PM_{2.5}$  mortality in the contiguous US. We present our estimates of smoke  $PM_{2.5}$  and total  $PM_{2.5}$  mortality totals alongside previously published estimates of US mortalities attributable to smoke  $PM_{2.5}$  in Table 1. Our estimates of mortality attributable to smoke  $PM_{2.5}$  is generally lower than previous estimates, but our uncertainty range overlaps with the range of estimated mortalities presented in Ford et al. (2018), but not with Fann et al. (2018) nor Neumann et al. (2021). We note there are meaningful differences in methodology. Each of these previous HIAs focused on a different time period and used a different health impact function from this work. Ford et al. (2018) estimated mortality over the 1995–2004 decade using a range of relative risks and threshold concentrations, Fann et al. (2018) estimated annual mortality attributable to smoke  $PM_{2.5}$  based on annual-average concentrations for each year from 2008 to 2012, and Neumann et al. (2021) estimated health impacts of western wildfire smoke on the full US over the 1995–2004 decade. The earlier time periods studied in Ford et al. (2018) and Neumann et al. (2021) do not overlap with our time period. In Table 1, we present the full range of mortality attributable to smoke  $PM_{2.5}$  from Ford et al. (2018) and Neumann et al. (2021) over all relative risks and threshold concentrations used and the range across all years from Fann et al. (2018). There are additional factors that may also contribute to differences observed between our estimates and these previous works including different smoke  $PM_{2.5}$  estimates, population estimates, and mortality rates.

Total and smoke  $PM_{2.5}$  mortalities differ significantly by US state. In Figure 4, we show the total annual number and percent of all-cause mortality attributable to total  $PM_{2.5}$  (Figures 4a and 4c) and smoke  $PM_{2.5}$  (Figures 4b and 4d) by US state. The fraction of all mortality attributable to total  $PM_{2.5}$  ranges from approximately 5%–10% in each state and the fraction of mortality attributable to smoke  $PM_{2.5}$  ranges from approximately 0.1%–1.2%. In general, the fraction of mortality attributable to total  $PM_{2.5}$  is higher in the eastern states, where total  $PM_{2.5}$  concentrations are often higher (Figure S10a). California has the highest



**Figure 4.** Total annual mortalities (in 1000s) attributable to total  $PM_{2.5}$  (panel a) and smoke  $PM_{2.5}$  (panel b) in each state. Panels c and d show the percent of all mortalities in each state attributable to total  $PM_{2.5}$  and smoke  $PM_{2.5}$ , respectively.

percentage of mortalities attributable to total  $PM_{2.5}$ , but not smoke  $PM_{2.5}$ . The fraction of mortality attributable to smoke  $PM_{2.5}$  is higher in several northwestern states with the highest fraction occurring in Montana. There, we estimate 1.2% (CI: 0.9%–1.5%) of all annual mortalities from 2006 to 2018 are attributable to smoke  $PM_{2.5}$  exposure. These northwestern states may have additional deaths attributable to biomass burning due to emissions from winter wood burning. This  $PM_{2.5}$  source is not included in the deaths attributable to smoke  $PM_{2.5}$  here, but is included in the deaths attributable to total  $PM_{2.5}$ . Overall, 0.32% (CI: 0.25%–0.40%) of all mortalities in the western states are attributable to smoke  $PM_{2.5}$ , while 0.26% (CI: 0.20%–0.32%) of all mortalities in the eastern states are attributable to smoke  $PM_{2.5}$ . In terms of the total number of deaths attributable to total and smoke  $PM_{2.5}$ , we again see a heavy influence of population over concentration. The total number of deaths attributable to both smoke  $PM_{2.5}$  and total  $PM_{2.5}$  are highest in high-population states. We find a lower number of deaths attributable to smoke  $PM_{2.5}$  across all western states with 1,700 (CI: 1,300–2,000) deaths across the Northwest, Rocky Mountain, and Southwest regions, compared to 4,700 (CI: 3,500–7,800) deaths across all eastern states. This again highlights the important, yet different, impacts of smoke  $PM_{2.5}$  across the US where smoke contributes a higher percentage of mortality in heavily smoke-impacted western states and a higher total number of deaths in eastern states with high population density, but lower long-term population-average smoke  $PM_{2.5}$  exposure.

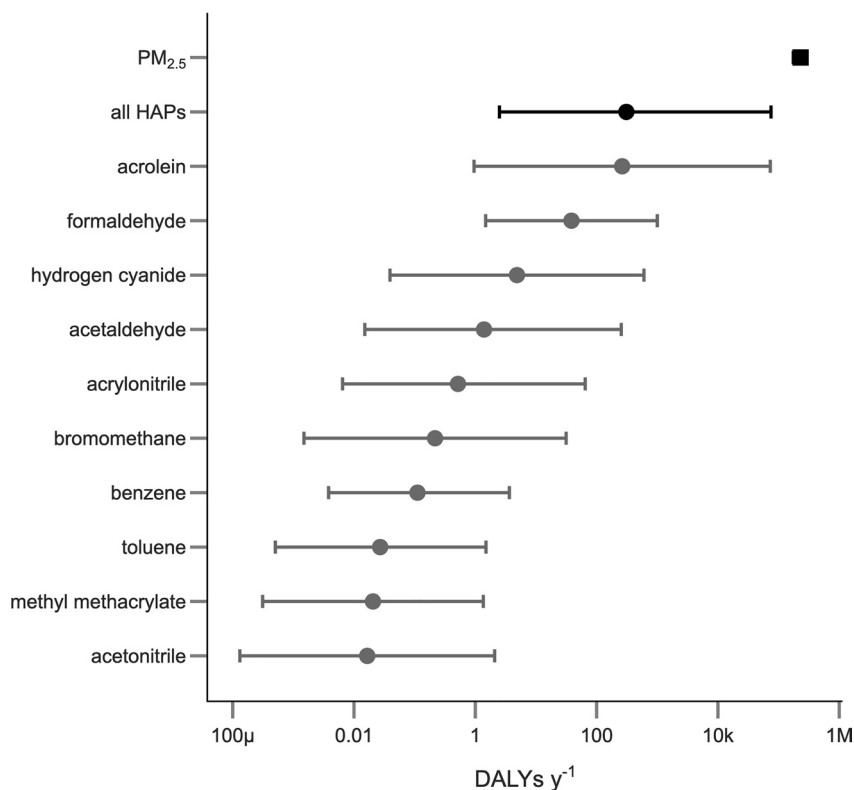
### 3.5. Smoke-Attributable DALYs Due to Hazardous Air Pollutants and $PM_{2.5}$

Figure 5 shows DALYs attributable to smoke  $PM_{2.5}$  and speciated gas-phase smoke HAPs in the US. We find the DALYs due to smoke  $PM_{2.5}$ , 231,000 (CI: 175,000–285,000) per year, is approximately three orders of magnitude higher than DALYs attributable to all gas-phase smoke HAPs included in our study, 309 (CI: 3–75,000) per year. However, there is a large amount of uncertainty in our estimates of DALYs from HAPs, the upper bound of which estimates DALYs from HAPs are within an order of magnitude of the DALYs attributable to smoke  $PM_{2.5}$ . The majority of DALYs from exposure to gas-phase smoke HAPs is attributable to acrolein (85%), followed by formaldehyde (12%). However, the abundance and relative contribution of individual HAPs to overall HAPs health risk is known to change with smoke age (O'Dell et al., 2020). In addition to the HAPs listed in Figure 5, we also calculated DALYs for eight additional gas-phase smoke HAPs, however the DALYs due to exposure to these HAPs was  $<0.01$  DALYs  $y^{-1}$ , so we removed them from Figure 5. Figure S11 shows the DALYs estimated for all smoke HAPs in this study. In addition to the HAPs included here, there are additional smoke HAPs in the gas and particle phase that either were not measured in the WE-CAN campaign or did not have established DALY factors.

The relatively low number of DALYs attributable to smoke HAPs in Figure 5 is due, in part, to the low estimated HAPs concentrations in smoke. The 2006–2018 mean smoke-enhanced concentrations of acrolein ( $4.4 \times 10^{-3} \mu g m^{-3}$ ) and formaldehyde ( $4.2 \times 10^{-2} \mu g m^{-3}$ ) in the US estimated here are approximately 80% lower than estimated background concentrations in North America (McCarthy et al., 2006). For both acrolein and formaldehyde, these long-term average smoke-enhanced concentrations are below the California Office of Environmental Health Hazard Assessment (OEHHA) chronic reference exposure level at which health impacts might be expected (OEHHA, 2016; Figure S12). However, these smoke HAPs concentrations are likely present with additional HAPs sources which may lead to higher overall chronic exposures (e.g., McCarthy et al., 2006).

We acknowledge there is a high degree of uncertainty in the estimates of DALYs from HAPs and several assumptions made in the comparison of DALYs from speciated gas-phase HAPs to DALYs from  $PM_{2.5}$  mass concentrations. The large uncertainty bars for DALYs from HAPs in Figure 5 are representative of the high uncertainty in the DALYs per HAPs intake estimates, which are approximated from animal toxicology

### DALYs from HAPs and PM in US wildfire smoke, 2006-2018



**Figure 5.** US-total disability-adjusted life years (DALYs) attributable to smoke PM<sub>2.5</sub> and gas-phase smoke Hazardous Air Pollutants (HAPs) each year. PM<sub>2.5</sub> DALY error bars represent the range of DALYs estimated using  $\pm 2$  times the standard error for the coefficients in the GEMM health impact function. HAPs DALY error bars represent the range of DALYs estimated using the upper and lower bounds of the 95% CI for the DALY factor for each pollutant. HAPs associated with fewer than 0.01 DALYs per year have been removed from this figure. The full figure with all HAPs is available in the supplement.

studies (Huijbregts et al., 2005). There is additional uncertainty, not represented in Figure 5, from the smoke HAPs concentration estimates from O'Dell et al. (2020) discussed in Section 2.1. We are unable to quantify the magnitude of this uncertainty. However, we tested the sensitivity of our results to uncertainty in HAPs concentration estimates by re-calculating DALYs from HAPs using the 2.5<sup>th</sup> and 97.5<sup>th</sup> percentile of HAPs to PM ratios from O'Dell et al. (2020), which resulted in estimates of 93 and 635 DALYs from HAPs in smoke, respectively. This range in estimated DALYs is much smaller than the uncertainty range in DALYs due to uncertainty in the DALYs per HAP intake from Huijbregts et al. (2005). There is a large difference between the methods used to estimate HAPs DALYs, which are toxicology-based, and PM<sub>2.5</sub> DALYs, which rely on an epidemiology-based concentration response function for mortality, that may impact DALY estimates and uncertainties. In addition, because the DALYs attributable to smoke PM<sub>2.5</sub> are estimated from an epidemiologically-based concentration response function, the smoke PM<sub>2.5</sub> DALY totals (and mortalities) presented here may already incorporate health impacts of compounds co-emitted with PM<sub>2.5</sub>. Smoke PM<sub>2.5</sub> may also include particle-phase HAPs, such as polycyclic aromatic hydrocarbons (PAHs; Andreae, 2019), which may impact the overall toxicity of PM<sub>2.5</sub> mass concentrations in smoke.

### 3.6. Limitations

There are several limitations of our observation-based smoke PM<sub>2.5</sub> and HAPs exposure estimates which may impact the present study. First, the HMS smoke product can omit the smoke from small short-lived



fires, likely leading to an underestimate of smoke in the Southeast and Midwest where small fires contribute a large fraction of burned area (Brey, Ruminski, et al., 2018). In addition, HMS relies on visible satellite imagery, which is only available during daylight hours, and dilute smoke is more difficult to identify visually than concentrated smoke, thus the HMS analysis is a lower bound on daytime smoke extent across the US. As there is no HMS information overnight, the overnight portion of our 24-h average  $PM_{2.5}$  is more uncertain. Smoke mixed with PM from other sources is similarly difficult to positively identify. This issue is of particular relevance in the southeastern US. In our smoke  $PM_{2.5}$  estimates, small or dilute smoke plumes could be incorporated into the “non-smoke” days and artificially increase the non-smoke  $PM_{2.5}$  background estimate. Finally, our method of estimating smoke  $PM_{2.5}$  has no independent concentration information where there are no monitoring sites. However, monitors are typically in locations with a high population density, thus this limitation would have less impact on a national HIA.

There are also several limitations to our health impact assessment of smoke  $PM_{2.5}$  and smoke HAPs. As mentioned previously, smoke  $PM_{2.5}$  and  $PM_{2.5}$  from urban sources have a different toxicity (Wegesser et al., 2009). It is currently unclear how this may affect health outcomes of chronic exposure. The differential long-term impacts of consistent (e.g., ambient urban) versus episodic (e.g., smoke plumes) exposures are also currently unknown. There are additional challenges with separating mortalities due to short-term exposure versus long-term exposure for an episodic source, like landscape-fire smoke. Smoke  $PM_{2.5}$  DALYs (and mortalities) may already incorporate health impacts from co-emitted species including HAPs, thus DALYs attributable to smoke  $PM_{2.5}$  and smoke HAPs are not mutually exclusive. In addition, the two methods used to estimate DALYs attributable to non-speciated smoke  $PM_{2.5}$  and speciated gas-phase smoke HAPs are very different with unique uncertainties and assumptions, which may differentially impact estimated DALYs. There are many uncertainties in any HIA due to uncertainties in baseline rates, exposure, and health impact functions, among other factors.

#### 4. Conclusions

In the present work, we used an HIA as a tool to understand (a) the distribution of health events due to acute and chronic smoke exposure across US states and EPA regions, and (b) the relative contribution of gas-phase smoke HAPs and smoke  $PM_{2.5}$  to chronic-exposure health outcomes. In this study, we built on previous HIAs of US smoke  $PM_{2.5}$  (Fann et al., 2018; Ford et al., 2018; Neumann et al., 2021) to conduct the first HIA of smoke with observation-based smoke  $PM_{2.5}$ , sub-annual temporal resolution of asthma morbidity attributable to smoke  $PM_{2.5}$ , and chronic impacts of smoke HAPs. We show, by number, that more asthma morbidities due to acute smoke exposure occur in non-western US regions in most years. In heavily fire impacted years, there is a higher contribution of smoke  $PM_{2.5}$  to asthma morbidities in the West (over 1% of asthma ED visits) compared to the East (maximum of 0.3%–0.6%). The seasonality of these morbidities varies by region, but nationwide morbidities attributable to smoke  $PM_{2.5}$  predominantly occur in spring and summer. We show the highest number of deaths for smoke  $PM_{2.5}$  occur in the most populous states, while the highest fraction of deaths attributable to smoke  $PM_{2.5}$  (up to 1% of all mortality) occur in the northwestern states. In addition, we provide the first, to our knowledge, estimates of DALYs from smoke  $PM_{2.5}$  and speciated gas-phase HAPs. We show smoke  $PM_{2.5}$  is associated with approximately  $10^3$  times the number of DALYs from gas-phase smoke HAPs concentrations, but there remains high uncertainty in the health implications of HAPs exposure.

Smoke plumes contain many health-relevant pollutants. Based on our results, smoke  $PM_{2.5}$  remains an important indicator of smoke-specific health impacts. However, there is a high degree of uncertainty in the potential human health impacts of many HAPs in smoke. Further, in addition to the HAPs included in our study, there may be many HAPs in smoke in the gas and particle phase not included in our work that contribute to the observed health impacts of smoke exposure. More research is needed to understand the concentration and health impacts of these speciated compounds in smoke as well as the subsequent impacts of multi-pollutant exposure.

As wildfires and smoke-attributable  $PM_{2.5}$  continue to increase (Ford et al., 2018; Li et al., 2020; Liu et al., 2016; Neumann et al., 2021; Yue et al., 2013), it is important to understand and prepare for health impacts of smoke. Our results indicate the impacts of smoke on public health extend across the US and are

not constrained to the western states during the typical fire season. Therefore, it is important for the entire US population to have increased awareness of wildfire smoke and knowledge of when/how to mitigate exposure. This is especially important for those in states not typically thought of as fire-impacted and/or far downwind of large fires, who may be less aware of the presence of smoke. Messaging and preparedness for smoke in each region should focus on local seasonality in smoke-attributable health events. The assessment of the seasonality of these acute events by region presented here may help states prepare for the potential increasing burden posed to the healthcare system by smoke. Our findings also underscore the need for continued development and more active use of national scale smoke forecasts to increase awareness and capacity to prepare for the impact of smoke in downwind regions. It will be important to understand how smoke sources and seasonality may change by region in the future due to climate change and human influence on fire ignition and suppression.

## Conflict of Interest

The authors declare no conflicts of interest relevant to this study.

## Data Availability Statement

The kriged PM<sub>2.5</sub> data used in this analysis can be accessed at DOI <https://doi.org/10.25675/10217/230602>. Python codes used for the analysis presented here and in the Supporting Information S1 are available at <https://doi.org/10.5281/zenodo.5164901>.

## Acknowledgments

We thank the anonymous reviews for their helpful and insightful feedback on this work. This work was supported by the National Science Foundation (NSF) grant number GRFP-006784-00003 and National Aeronautics and Space Administration (NASA) Health and Air Quality Applied Sciences Team grant number 80NSSC21K0429.

## References

- Abatzoglou, J. T., & Williams, A. P. (2016). Impact of anthropogenic climate change on wildfire across western US forests. *Proceedings of the National Academy of Sciences*, 113(42), 11770–11775. <https://doi.org/10.1073/pnas.1607171113>
- Abdo, M., Ward, L., O'Dell, K., Ford, B., Pierce, J. R., Fischer, E. V., & Crooks, J. L. (2019). Impact of wildfire smoke on adverse pregnancy outcomes in Colorado, 2007–2015. *International Journal of Environmental Research and Public Health*, 16(19), 3720. <https://doi.org/10.3390/ijerph16193720>
- Aguilera, R., Corringham, T., Gershunov, A., & Benmarhnia, T. (2021). Wildfire smoke impacts respiratory health more than fine particles from other sources: Observational evidence from Southern California. *Nature Communications*, 12(1), 1493. <https://doi.org/10.1038/s41467-021-21708-0>
- AHRQ. (2006). *HCUP Databases. Healthcare Cost and Utilization Project (HCUP)*. Retrieved from [www.hcup-us.ahrq.gov/databases.jsp](http://www.hcup-us.ahrq.gov/databases.jsp)
- Alman, B. L., Pfister, G., Hao, H., Stowell, J., Hu, X., Liu, Y., & Strickland, M. J. (2016). The association of wildfire smoke with respiratory and cardiovascular emergency department visits in Colorado in 2012: A case crossover study. *Environmental Health*, 15, 64. <https://doi.org/10.1186/s12940-016-0146-8>
- Andreae, M. O. (2019). Emission of trace gases and aerosols from biomass burning—An updated assessment. *Atmospheric Chemistry and Physics*, 19(13), 8523–8546. <https://doi.org/10.5194/acp-19-8523-2019>
- Anenberg, S. C., Horowitz, H. L., Tong, D. Q., & West, J. J. (2010). An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. *Environmental Health Perspectives*, 118(9), 1189–1195. <https://doi.org/10.1289/ehp.0901220>
- Anenberg, S. C., West, J. J., Yu, H., Chin, M., Schulz, M., Bergmann, D., et al. (2014). Impacts of intercontinental transport of anthropogenic fine particulate matter on human mortality. *Air Quality, Atmosphere & Health*, 7(3), 369–379. <https://doi.org/10.1007/s11869-014-0248-9>
- Balch, J. K., Bradley, B. A., Abatzoglou, J. T., Nagy, R. C., Fusco, E. J., & Mahood, A. L. (2017). Human-started wildfires expand the fire niche across the United States. *Proceedings of the National Academy of Sciences*, 114(11), 2946–2951. <https://doi.org/10.1073/pnas.1617394114>
- Barbero, R., Abatzoglou, J. T., Larkin, S., Kolden, C. A., & Stocks, B. (2015). Climate change presents increased potential for very large fires in the contiguous United States. *International Journal of Wildland Fire*, 24(7), 892–899. <https://doi.org/10.1071/WF15083>
- Bell, M. L., Francesca, D., Keita, E., Zeger Scott, L., & Samet Jonathan, M. (2007). Spatial and temporal variation in PM<sub>2.5</sub> chemical composition in the United States for health effects studies. *Environmental Health Perspectives*, 115(7), 989–995. <https://doi.org/10.1289/ehp.9621>
- Bian, Q., Ford, B., Pierce, J. R., & Kreidenweis, S. M. (2020). A decadal climatology of chemical, physical, and optical properties of ambient smoke in the Western and Southeastern United States. *Journal of Geophysical Research: Atmospheres*, 125(1), e2019JD031372. <https://doi.org/10.1029/2019JD031372>
- Bilsback, K. R., Baumgartner, J., Cheeseman, M., Ford, B., Kodros, J. K., Li, X., et al. (2020). Estimated aerosol health and radiative effects of the residential coal ban in the Beijing-Tianjin-Hebei region of China. *Aerosol and Air Quality Research*, 20(11), 2332–2346. <https://doi.org/10.4209/aaqr.2019.11.0565>
- Borchers Arriagada, N., Horsley, J. A., Palmer, A. J., Morgan, G. G., Tham, R., & Johnston, F. H. (2019). Association between fire smoke fine particulate matter and asthma-related outcomes: Systematic review and meta-analysis. *Environmental Research*, 179, 108777. <https://doi.org/10.1016/j.envres.2019.108777>
- Brenner, J. (1991). Southern oscillation anomalies and their relationship to wildfire activity in Florida. *International Journal of Wildland Fire*, 1(1), 73–78. <https://doi.org/10.1071/wf9910073>
- Brey, S. J., Barnes, E. A., Pierce, J. R., Wiedinmyer, C., & Fischer, E. V. (2018). Environmental conditions, ignition type, and air quality impacts of wildfires in the Southeastern and Western United States. *Earth's Future*, 6(10), 1442–1456. <https://doi.org/10.1029/2018EF000972>

- Brey, S. J., & Fischer, E. V. (2016). Smoke in the city: How often and where does smoke impact summertime ozone in the United States? *Environmental Science & Technology*, 50(3), 1288–1294. <https://doi.org/10.1021/acs.est.5b05218>
- Brey, S. J., Ruminski, M., Atwood, S. A., & Fischer, E. V. (2018). Connecting smoke plumes to sources using Hazard Mapping System (HMS) smoke and fire location data over North America. *Atmospheric Chemistry and Physics*, 18(3), 1745–1761. <https://doi.org/10.5194/acp-18-1745-2018>
- BRFSS/CDC. (2019). *Table L1: Adult self-reported lifetime asthma prevalence and number by state or territory*. Retrieved from <https://www.cdc.gov/asthma/brfss/2019/tableL1.html>
- Burkhardt, J., Bayham, J., Wilson, A., Berman, J. D., O'Dell, K., Ford, B., et al. (2020). The relationship between monthly air pollution and violent crime across the United States. *Journal of Environmental Economics and Policy*, 9(2), 188–205. <https://doi.org/10.1080/21606544.2019.1630014>
- Burkhardt, J., Bayham, J., Wilson, A., Carter, E., Berman, J. D., O'Dell, K., et al. (2019). The effect of pollution on crime: Evidence from data on particulate matter and ozone. *Journal of Environmental Economics and Management*, 98, 102267. <https://doi.org/10.1016/j.jeem.2019.102267>
- Burnett, R., Chen, H., Szyszkowicz, M., Fann, N., Hubbell, B., Pope, C. A., et al. (2018). Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. *Proceedings of the National Academy of Sciences*, 115(38), 9592–9597. <https://doi.org/10.1073/pnas.1803222115>
- Burnett, R. T., Pope, C. A., Ezzati, M., Casey, O., Lim Stephen, S., Mehta, S., et al. (2014). An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environmental Health Perspectives*, 122(4), 397–403. <https://doi.org/10.1289/ehp.1307049>
- Buysse, C. E., Kaulfus, A., Nair, U., & Jaffe, D. A. (2019). Relationships between particulate matter, ozone, and nitrogen oxides during urban smoke events in the western US. *Environmental Science & Technology*, 53, 12519–12528. <https://doi.org/10.1021/acs.est.9b05241>
- Cascio, W. E. (2018). Wildland fire smoke and human health. *The Science of the Total Environment*, 624, 586–595. <https://doi.org/10.1016/j.scitotenv.2017.12.086>
- Cleland, S. E., Serre, M. L., Rappold, A. G., & West, J. J. (2021). Estimating the acute health impacts of fire-originated PM<sub>2.5</sub> exposure during the 2017 California Wildfires: Sensitivity to choices of inputs. *GeoHealth*, 5(7), e2021GH000414. <https://doi.org/10.1029/2021GH000414>
- Cohen, A. J., Brauer, M., Burnett, R., Anderson, H. R., Frostad, J., Estep, K., et al. (2017). Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: An analysis of data from the Global Burden of Diseases Study 2015. *The Lancet*, 389(10082), 1907–1918. [https://doi.org/10.1016/S0140-6736\(17\)30505-6](https://doi.org/10.1016/S0140-6736(17)30505-6)
- Crouse, D. L., Pinault, L., Balram, A., Brauer, M., Burnett, R. T., Martin, R. V., et al. (2019). Complex relationships between greenness, air pollution, and mortality in a population-based Canadian cohort. *Environment International*, 128, 292–300. <https://doi.org/10.1016/j.envint.2019.04.047>
- David, L. M., Ravishankara, A. R., Brey, S. J., Fischer, E. V., Volckens, J., & Kreidenweis, S. (2021). Could the exception become the rule? “Uncontrollable” air pollution events in the U.S. due to wildland fires. *Environmental Research Letters*. <https://doi.org/10.1088/1748-9326/abef13>
- DeBell, L. J., Talbot, R. W., Dibb, J. E., Munger, J. W., Fischer, E. V., & Frolking, S. E. (2004). A major regional air pollution event in the northeastern United States caused by extensive forest fires in Quebec, Canada. *Journal of Geophysical Research*, 109(D19). <https://doi.org/10.1029/2004JD004840>
- DeFlorio-Barker, S., Crooks, J. L., Reyes, J., & Rappold, A. G. (2019). Cardiopulmonary effects of fine particulate matter exposure among older adults, during wildfire and non-wildfire periods, in the United States 2008–2010. *Environmental Health Perspectives*, 127(3), 037006. <https://doi.org/10.1289/EHP3860>
- Delfino, R. J., Brummel, S., Wu, J., Stern, H., Ostro, B., Lipsett, M., et al. (2009). The relationship of respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003. *Occupational and Environmental Medicine*, 66(3), 189–197. <https://doi.org/10.1136/oem.2008.041376>
- Dennis, A., Fraser, M., Anderson, S., & Allen, D. (2002). Air pollutant emissions associated with forest, grassland, and agricultural burning in Texas. *Atmospheric Environment*, 36(23), 3779–3792. [https://doi.org/10.1016/S1352-2310\(02\)00219-4](https://doi.org/10.1016/S1352-2310(02)00219-4)
- Doubleday, A., Schulte, J., Sheppard, L., Kadlec, M., Dhammapala, R., Fox, J., & Busch Isaksen, T. (2020). Mortality associated with wildfire smoke exposure in Washington State, 2006–2017: A case-crossover study. *Environmental Health*, 19(1), 4. <https://doi.org/10.1186/s12940-020-0559-2>
- Fann, N., Alman, B., Broome, R. A., Morgan, G. G., Johnston, F. H., Pouliot, G., & Rappold, A. G. (2018). The health impacts and economic value of wildland fire episodes in the U.S: 2008–2012. *The Science of the Total Environment*, 610, 802–809. <https://doi.org/10.1016/j.scitotenv.2017.08.024>
- Ford, B., Burke, M., Lassman, W., Pfister, G., & Pierce, J. R. (2017). Status update: Is smoke on your mind? Using social media to assess smoke exposure. *Atmospheric Chemistry and Physics*, 17(12), 7541–7554. <https://doi.org/10.5194/acp-17-7541-2017>
- Ford, B., Martin, M. V., Zelasky, S. E., Fischer, E. V., Anenberg, S. C., Heald, C. L., & Pierce, J. R. (2018). Future fire impacts on smoke concentrations, visibility, and health in the contiguous United States. *GeoHealth*, 2, 229–247. <https://doi.org/10.1029/2018GH000144>
- Gan, R. W., Ford, B., Lassman, W., Pfister, G., Vaidyanathan, A., Fischer, E., et al. (2017). Comparison of wildfire smoke estimation methods and associations with cardiopulmonary-related hospital admissions. *GeoHealth*, 1(3), 2017GH000073. <https://doi.org/10.1002/2017GH000073>
- Gan, R. W., Liu, J., Ford, B., O'Dell, K., Vaidyanathan, A., Wilson, A., et al. (2020). The association between wildfire smoke exposure and asthma-specific medical care utilization in Oregon during the 2013 wildfire season. *Journal of Exposure Science and Environmental Epidemiology*, 30(4), 618–628. <https://doi.org/10.1038/s41370-020-0210-x>
- GBD. (2019). *Global burden of disease collaborative Network Global burden of disease study 2019 (GBD 2019) results*. Institute for Health Metrics and Evaluation (IHME). Retrieved from <http://ghdx.healthdata.org/gbd-results-tool>
- Gilman, J. B., Lerner, B. M., Kuster, W. C., Goldan, P. D., Warneke, C., Veres, P. R., et al. (2015). Biomass burning emissions and potential air quality impacts of volatile organic compounds and other trace gases from fuels common in the US. *Atmospheric Chemistry and Physics*, 15(24), 13915–13938. <https://doi.org/10.5194/acp-15-13915-2015>
- Goss, M., Swain, D. L., Abatzoglou, J. T., Sarhadi, A., Kolden, C. A., Williams, A. P., & Diffenbaugh, N. S. (2020). Climate change is increasing the likelihood of extreme autumn wildfire conditions across California. *Environmental Research Letters*, 15(9), 094016. <https://doi.org/10.1088/1748-9326/ab83a7>
- Huijbregts, M. A. J., Rombouts, L. J. A., Ragas, A. M. J., & Meent, D. van de. (2005). Human-toxicological effect and damage factors of carcinogenic and noncarcinogenic chemicals for life cycle impact assessment. *Integrated Environmental Assessment and Management*, 1(3), 181–244. <https://doi.org/10.1897/2004-007R.1>

- Hutchinson, J. A., Vargo, J., Milet, M., French, N. H. F., Billmire, M., Johnson, J., & Hoshiko, S. (2018). The San Diego 2007 wildfires and Medi-Cal emergency department presentations, inpatient hospitalizations, and outpatient visits: An observational study of smoke exposure periods and a bidirectional case-crossover analysis. *PLoS Medicine*, 15(7), e1002601. <https://doi.org/10.1371/journal.pmed.1002601>
- Jin, Y., Goulden, M. L., Faivre, N., Veraverbeke, S., Sun, F., Hall, A., et al. (2015). Identification of two distinct fire regimes in Southern California: Implications for economic impact and future change. *Environmental Research Letters*, 10(9), 094005. <https://doi.org/10.1088/1748-9326/10/9/094005>
- Kaulfus, A. S., Nair, U., Jaffe, D., Christopher, S. A., & Goodrick, S. (2017). Biomass burning smoke climatology of the United States: Implications for particulate matter air quality. *Environmental Science & Technology*, 51(20), 11731–11741. <https://doi.org/10.1021/acs.est.7b03292>
- Kiser, D., Metcalf, W. J., Elhanan, G., Schnieder, B., Schlauch, K., Joros, A., et al. (2020). Particulate matter and emergency visits for asthma: A time-series study of their association in the presence and absence of wildfire smoke in Reno, Nevada, 2013–2018. *Environmental Health*, 19(1), 92. <https://doi.org/10.1186/s12940-020-00646-2>
- Kodros, J. K., Wiedinmyer, C., Ford, B., Cucinotta, R., Gan, R., Magzamen, S., & Pierce, J. R. (2016). Global burden of mortalities due to chronic exposure to ambient PM 2.5 from open combustion of domestic waste. *Environmental Research Letters*, 11(12), 124022. <https://doi.org/10.1088/1748-9326/11/12/124022>
- Krewski, D., Jerrett, M., Burnett, R. T., Ma, R., Hughes, E., Shi, Y., et al. (2009). *Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality* (Research Report) (pp. 5–114). Health Effects Institute.
- Kupfer, J. A., Terando, A. J., Gao, P., Teske, C., & Hiers, J. K. (2020). Climate change projected to reduce prescribed burning opportunities in the south-eastern United States. *International Journal of Wildland Fire*, 29(9), 764–778. <https://doi.org/10.1071/WF19198>
- Lam, Y. F., Fu, J. S., Wu, S., & Mickley, L. J. (2011). Impacts of future climate change and effects of biogenic emissions on surface ozone and particulate matter concentrations in the United States. *Atmospheric Chemistry and Physics*, 11(10), 4789–4806. <https://doi.org/10.5194/acp-11-4789-2011>
- Le, G. E., Breyse, P. N., McDermott, A., Eftim, S. E., Geyh, A., Berman, J. D., & Curriero, F. C. (2014). Canadian forest fires and the effects of long-range transboundary air pollution on hospitalizations among the elderly. *ISPRS International Journal of Geo-Information*, 3(2), 713–731. <https://doi.org/10.3390/ijgi3020713>
- Leibensperger, E. M., Mickley, L. J., Jacob, D. J., Chen, W.-T., Seinfeld, J. H., Nenes, A., et al. (2012). Climatic effects of 1950–2050 changes in US anthropogenic aerosols—Part 1: Aerosol trends and radiative forcing. *Atmospheric Chemistry and Physics*, 12(7), 3333–3348. <https://doi.org/10.5194/acp-12-3333-2012>
- Li, Y., Mickley, L. J., Liu, P., & Kaplan, J. O. (2020). Trends and spatial shifts in lightning fires and smoke concentrations in response to 21st century climate over the forests of the Western United States. *Atmospheric Chemistry and Physics Discussions*, 1–26. <https://doi.org/10.5194/acp-2020-80>
- Lipner, E. M., O'Dell, K., Brey, S. J., Ford, B., Pierce, J. R., Fischer, E. V., & Crooks, J. L. (2019). The Associations between clinical respiratory outcomes and ambient wildfire smoke exposure among pediatric asthma patients at National Jewish Health, 2012–2015. *GeoHealth*, 3(6), 146–159. <https://doi.org/10.1029/2018GH000142>
- Liu, J. C., Mickley, L. J., Sulprizio, M. P., Dominici, F., Yue, X., Ebisu, K., et al. (2016). Particulate air pollution from wildfires in the Western US under climate change. *Climatic Change*, 138(3–4), 655–666. <https://doi.org/10.1007/s10584-016-1762-6>
- Liu, J. C., Pereira, G., Uhl, S. A., Bravo, M. A., & Bell, M. L. (2015). A systematic review of the physical health impacts from non-occupational exposure to wildfire smoke. *Environmental Research*, 136, 120–132. <https://doi.org/10.1016/j.envres.2014.10.015>
- Logue, J. M., Price, P., Sherman, S. M., & Singer, B. C. (2012). A method to estimate the chronic health impact of air pollutants in U.S. residences. *Environmental Health Perspectives*, 120(2), 216–222. <https://doi.org/10.1289/ehp.1104035>
- Magzamen, S., Gan, R. W., Liu, J., O'Dell, K., Ford, B., Berg, K., et al. (2021). Differential cardiopulmonary health impacts of local and long-range transport of wildfire smoke. *GeoHealth*, 5(3), e2020GH000330. <https://doi.org/10.1029/2020GH000330>
- McCarthy, M. C., Hafner, H. R., & Montzka, S. A. (2006). Background concentrations of 18 air toxics for North America. *Journal of the Air & Waste Management Association*, 56(1), 3–11. <https://doi.org/10.1080/10473289.2006.10464436>
- McCarty, J. L., Korontzi, S., Justice, C. O., & Loboda, T. (2009). The spatial and temporal distribution of crop residue burning in the contiguous United States. *The Science of the Total Environment*, 407(21), 5701–5712. <https://doi.org/10.1016/j.scitotenv.2009.07.009>
- McClure, C. D., & Jaffe, D. A. (2018). US particulate matter air quality improves except in wildfire-prone areas. *Proceedings of the National Academy of Sciences*, 115, 7901, 7906. <https://doi.org/10.1073/pnas.1804353115>
- NASA SEDAC. (2018). *Documentation for the gridded population of the world, version 4 (GPWv4), revision 11 data sets*. Retrieved from <https://link.springer.com/article/10.1007/s11869-010-0125-0>
- Neumann, J. E., Amend, M., Anenberg, S. C., Kinney, P. L., Sarofim, M., Martinich, J., et al. (2021). Estimating PM2.5-related premature mortality and morbidity associated with future wildfire emissions in the western U.S. *Environmental Research Letters*, 16, 035019. <https://doi.org/10.1088/1748-9326/abe82b>
- O'Dell, K., Ford, B., Fischer, E. V., & Pierce, J. R. (2019). Contribution of wildland-fire smoke to US PM2.5 and its influence on recent trends. *Environmental Science & Technology*, 53(4), 1797–1804. <https://doi.org/10.1021/acs.est.8b05430>
- O'Dell, K., Hornbrook, R. S., Permar, W., Levin, E. J. T., Garofalo, L. A., Apel, E. C., et al. (2020). Hazardous air pollutants in fresh and aged western US wildfire smoke and implications for long-term exposure. *Environmental Science & Technology*, 54(19), 11838–11847. <https://doi.org/10.1021/acs.est.0c04497>
- OEHHA. (2016). *OEHHA acute, 8-hour and chronic reference exposure level (REL) summary [Text]*. Retrieved from <https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary>
- Paugam, R., Wooster, M., Freitas, S., & Val Martin, M. (2016). A review of approaches to estimate wildfire plume injection height within large-scale atmospheric chemical transport models. *Atmospheric Chemistry and Physics*, 16(2), 907–925. <https://doi.org/10.5194/acp-16-907-2016>
- Pendergraft, T. B., Stanford, R. H., Beasley, R., Stempel, D. A., & McLaughlin, T. (2005). Seasonal variation in asthma-related hospital and intensive care unit admissions. *Journal of Asthma*, 42(4), 265–271. <https://doi.org/10.1081/JAS-200057893>
- Pope, C. A., Ezzati, M., & Dockery, D. W. (2009). Fine-particulate air pollution and life expectancy in the United States. *New England Journal of Medicine*, 360(4), 376–386. <https://doi.org/10.1056/NEJMSa0805646>
- Pratt, J. R., Gan, R. W., Ford, B., Brey, S., Pierce, J. R., Fischer, E. V., & Magzamen, S. (2019). A national burden assessment of estimated pediatric asthma emergency department visits that may be attributed to elevated ozone levels associated with the presence of smoke. *Environmental Monitoring and Assessment*, 191(2), 269. <https://doi.org/10.1007/s10661-019-7420-5>



- Rappold, A. G., Cascio, W. E., Kilaru, V. J., Stone, S. L., Neas, L. M., Devlin, R. B., & Diaz-Sanchez, D. (2012). Cardio-respiratory outcomes associated with exposure to wildfire smoke are modified by measures of community health. *Environmental Health*, 11(1), 71. <https://doi.org/10.1186/1476-069X-11-71>
- Reid, C. E., Brauer, M., Johnston, F. H., Jarrett, M., Balmes, J. R., & Elliott, C. T. (2016). Critical review of health impacts of wildfire smoke exposure. *Environmental Health Perspectives*, 124(9), 1334–1343. <https://doi.org/10.1289/ehp.1409277>
- Reid, C. E., Jerrett, M., Tager, I. B., Petersen, M. L., Mann, J. K., & Balmes, J. R. (2016). Differential respiratory health effects from the 2008 northern California wildfires: A spatiotemporal approach. *Environmental Research*, 150, 227–235. <https://doi.org/10.1016/j.envres.2016.06.012>
- Resnick, A., Woods, B., Krapfl, H., & Toth, B. (2015). Health outcomes associated with smoke exposure in Albuquerque, New Mexico, during the 2011 Wallow Fire. *Journal of Public Health Management and Practice*, 21, S55–S61. <https://doi.org/10.1097/PHH.0000000000000160>
- Rogers, H. M., Ditto, J. C., & Gentner, D. R. (2020). Evidence for impacts on surface-level air quality in the northeastern US from long-distance transport of smoke from North American fires during the Long Island Sound Tropospheric Ozone Study (LISTOS) 2018. *Atmospheric Chemistry and Physics*, 20(2), 671–682. <https://doi.org/10.5194/acp-20-671-2020>
- Ruminski, M., Kondragunta, S., Draxler, R. R., & Zeng, J. (2006). Recent changes to the hazard mapping system. In *Presented at the 15th Int. Emiss. Inventory Conf. (Reinventing Inventories)*. Retrieved from [https://www.researchgate.net/publication/228625934\\_Recent\\_changes\\_to\\_the\\_Hazard\\_Mapping\\_System](https://www.researchgate.net/publication/228625934_Recent_changes_to_the_Hazard_Mapping_System)
- Sekimoto, K., Koss, A. R., Gilman, J. B., Selimovic, V., Coggon, M. M., Zarzana, K. J., et al. (2018). High- and low-temperature pyrolysis profiles describe volatile organic compound emissions from western US wildfire fuels. *Atmospheric Chemistry and Physics*, 18(13), 9263–9281. <https://doi.org/10.5194/acp-18-9263-2018>
- Silverman, R. A., Stevenson, L., & Hastings, H. M. (2003). Age-related seasonal patterns of emergency department visits for acute asthma in an urban environment. *Annals of Emergency Medicine*, 42(4), 577–586. [https://doi.org/10.1067/S0196-0644\(03\)00410-4](https://doi.org/10.1067/S0196-0644(03)00410-4)
- Sleiman, M., Logue, J. M., Luo, W., Pankow, J. F., Gundel, L. A., & Destailhats, H. (2014). Inhalable constituents of thirdhand tobacco smoke: Chemical characterization and health impact considerations. *Environmental Science & Technology*, 48(22), 13093–13101. <https://doi.org/10.1021/es5036333>
- Spracklen, D. V., Logan, J. A., Mickley, L. J., Park, R. C., Yevich, R., Westerling, A. L., & Jaffe, D. A. (2007). Wildfires drive interannual variability of organic carbon aerosol in the western U.S. in summer. *Geophysical Research Letters*, 34(16). <https://doi.org/10.1029/2007GL030037>
- Spracklen, D. V., Mickley, L. J., Logan, J. A., Hudman, R. C., Yevich, R., Flannigan, M. D., & Westerling, A. L. (2009). Impacts of climate change from 2000 to 2050 on wildfire activity and carbonaceous aerosol concentrations in the western United States. *Journal of Geophysical Research*, 114(D20), D20301. <https://doi.org/10.1029/2008JD010966>
- Tagaris, E., Manomaiphiboon, K., Liao, K.-J., Leung, L. R., Woo, J.-H., He, S., et al. (2007). Impacts of global climate change and emissions on regional ozone and fine particulate matter concentrations over the United States. *Journal of Geophysical Research*, 112(D14). <https://doi.org/10.1029/2006JD008262>
- Tinling, M. A., West, J. J., Cascio, W. E., Kilaru, V., & Rappold, A. G. (2016). Repeating cardiopulmonary health effects in rural North Carolina population during a second large peat wildfire. *Environmental Health*, 15, 12. <https://doi.org/10.1186/s12940-016-0093-4>
- US EPA. (2015). *Hazardous air pollutants [collections and lists]*. Retrieved from <https://www.epa.gov/haps>
- US EPA. (2017). *2017 National Emissions Inventory (NEI) data [Other policies and guidance]*. Retrieved from <https://www.epa.gov/air-emissions-inventories/2017-national-emissions-inventory-nei-data>
- Val Martin, M., Heald, C. L., Lamarque, J. F., Tilmes, S., Emmons, L. K., & Schichtel, B. A. (2015). How emissions, climate, and land use change will impact mid-century air quality over the United States: A focus on effects at national parks. *Atmospheric Chemistry and Physics*, 15, 2805–2823. <https://doi.org/10.5194/acp-15-2805-2015>
- Verdier, M. G. de, Gustafson, P., McCrae, C., Edsbäcker, S., & Johnston, N. (2017). Seasonal and geographic variations in the incidence of asthma exacerbations in the United States. *Journal of Asthma*, 54(8), 818–824. <https://doi.org/10.1080/02770903.2016.1277538>
- Wegesser, T. C., Pinkerton, K. E., & Last, J. A. (2009). California Wildfires of 2008: Coarse and fine particulate matter toxicity. *Environmental Health Perspectives*, 117(6), 893–897. <https://doi.org/10.1289/ehp.0800166>
- Westerling, A. L., Gershunov, A., Brown, T. J., Cayan, D. R., & Dettinger, M. D. (2003). Climate and wildfire in the Western United States. *Bulletin of the American Meteorological Society*, 84(5), 595–604. <https://doi.org/10.1175/BAMS-84-5-595>
- Wettstein, Z. S., Hoshiko, S., Fahimi, J., Harrison, R. J., Cascio, W. E., & Rappold, A. G. (2018). Cardiovascular and cerebrovascular emergency department visits associated with wildfire smoke exposure in California in 2015. *Journal of the American Heart Association*. <https://doi.org/10.1161/jaha.117.007492>
- Williams, A. P., Abatzoglou, J. T., Gershunov, A., Guzman-Morales, J., Bishop, D. A., Balch, J. K., & Lettenmaier, D. P. (2019). Observed impacts of anthropogenic climate change on wildfire in California. *Earth's Future*, 7(8), 892–910. <https://doi.org/10.1029/2019EF001210>
- Wu, Y., Arapi, A., Huang, J., Gross, B., & Moshary, F. (2018). Intra-continental wildfire smoke transport and impact on local air quality observed by ground-based and satellite remote sensing in New York City. *Atmospheric Environment*, 187, 266–281. <https://doi.org/10.1016/j.atmosenv.2018.06.006>
- Yue, X., Mickley, L. J., Logan, J. A., & Kaplan, J. O. (2013). Ensemble projections of wildfire activity and carbonaceous aerosol concentrations over the western United States in the mid-21st century. *Atmospheric Environment*, 77, 767–780. <https://doi.org/10.1016/j.atmosenv.2013.06.003>