DISSERTATION

HEALTH-RELEVANT POLLUTANTS IN US LANDSCAPE FIRE SMOKE: ABUNDANCE, HEALTH IMPACTS, AND INFLUENCE ON INDOOR AND OUTDOOR AIR QUALITY

Submitted by

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ABSTRACT

HEALTH-RELEVANT POLLUTANTS IN US LANDSCAPE FIRE SMOKE: ABUNDANCE, HEALTH IMPACTS, AND INFLUENCE ON INDOOR AND OUTDOOR AIR QUALITY

Landscape (wild, prescribed, and agricultural) fires have a significant impact on air quality in the United States (US). As anthropogenic emissions decline and emissions from landscape fires increase across the coming century, the relative importance of landscape fire smoke on US air quality and health will increase. Landscape fire smoke is a complex mixture of multiple gas- and particle-phase pollutants, which are harmful to human health. The health impacts of landscape fire smoke may differ from urban pollution as the seasonal and spatial distribution, particle size distribution and composition, and relative abundance of gas-phase species in landscape fire smoke are different from urban pollution sources. Epidemiology studies of smoke events, which often rely on particulate matter (PM) concentrations as a smoke exposure tracer, show smoke negatively impacts respiratory health. The contribution of gas-phase hazardous air pollutants (HAPs) to the health impacts of smoke has yet to be directly quantified. In addition, the implications of episodic landscape fire emissions on the sub-national temporal and spatial distribution of health events are not well characterized. Finally, a majority of the work on the health and air quality impacts of landscape fire smoke has focused on outdoor air. Recent works have shown that landscape fire smoke can impact indoor air quality, but there is large heterogeneity in both smoke events and the indoor environments impacted by smoke events. To date, no study of US wildfire smoke influence on indoor air quality has analyzed indoor fine particulate matter ($PM_{2.5}$) concentrations across multiple western US cities during multiple extreme smoke events.

In the first chapter of this dissertation, we combine aircraft-based *in-situ* smoke plume observations with interpolated regulatory surface monitor observations to quantify the health risk of HAPs in US smoke. Using observations from the Western Wildfire Experiment for Cloud Chem-

istry, Aerosol Absorption, and Nitrogen (WE-CAN), a 2018 aircraft-based field campaign that measured HAPs and PM in western US wildfire smoke plumes, we identify the relationships between HAPs and associated health risks, PM, and smoke age. We find the ratios between acute, chronic noncancer, and chronic cancer HAPs health risk and PM in smoke decrease as a function of smoke age by up to 72% from fresh (<1 day of aging) to old (>3 days of aging) smoke. We show that acrolein, formaldehyde, benzene, and hydrogen cyanide are the dominant contributors to gas-phase HAPs risk in smoke plumes. We use ratios of HAPs to PM along with annual average smoke-specific PM to estimate current and potential future smoke HAPs risks.

Next, we use a health impact assessment with observation-based smoke $PM_{2.5}$ to determine the sub-national distribution of mortality and the sub-national and sub-annual distribution of morbidity attributable to US smoke $PM_{2.5}$ from 2006-2018. We estimate disability-adjusted life years (DALYs) for $PM_{2.5}$ and 18 gas-phase HAPs in smoke using the HAPs to PM ratios developed in Chapter 2. Although the majority of large landscape fires occur in the western US, we find the majority of mortality (74%) and morbidity (on average 75% across 2006-2018) attributable to smoke $PM_{2.5}$ occurs outside the West due to a higher population density in the East. Across the US, smoke-attributable morbidity predominantly occurs in spring and summer. The number of DALYs associated with smoke $PM_{2.5}$ are approximately three orders of magnitude higher than DALYs associated with gas-phase smoke HAPs. These results indicate that awareness and mitigation of landscape fire smoke exposure is important across the US, not just in regions in proximity to large wildfires.

Finally, we use a large low-cost sensor network of indoor and outdoor $PM_{2.5}$ monitors to characterize the relationship between indoor and outdoor air quality during smoke events. We identify smoke-impacted regions of the western US with a high density of co-located (distance < 1000 m) indoor and outdoor PurpleAir monitors. In these regions, we calculate indoor $PM_{2.5}$/outdoor $PM_{2.5}$ ratios on smoke-impacted and smoke-free days and find this ratio is < 1 (indoor $PM_{2.5}$ less than outdoor $PM_{2.5}$) at 98% of the monitor pairs for smoke-impacted days, compared to 54% on smoke-free days. On smoke-impacted days, indoor $PM_{2.5}$ concentrations increase as outdoor $PM_{2.5}$ Air

Quality Index (AQI) increases by 25% per AQI bin, on average. However, the ratio of indoor $PM_{2.5}$ to outdoor $PM_{2.5}$ decreases by 28% per AQI bin. These results show that landscape fire smoke influences indoor air quality across many indoor environments in multiple cities, and this impact increases with smoke event intensity. In addition, this work highlights the utility of low-cost monitoring in quantifying indoor air quality during smoke events. However, we show that the present distribution of these indoor monitors suggests a bias towards census tracts of lower social vulnerability.

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DEDICATION

This dissertation is dedicated to my grandparents: Lillian Dean, Nancy O'Dell, Robert "Sonny" Dean, and Ronald "Razz" O'Dell, whose optimism, perseverance, and love will always inspire me. When I grow up, I want to be like them.

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CHAPTER 1

INTRODUCTION

1.1 Motivation and Background

Concern about pollutants in the air we breathe has a long history. There are multiple records of ancient societies seeking to control air pollution and reduce exposure after public concern. Ancient Romans complained of air pollution as "heavy heavens" and complaints on the pollution of air from coal burning in England are documented as early as the 1200s (Jacobson, 2012). Motivation for modern air quality regulation is often attributed to the infamous London Smog event and similar events across multiple cities in the United States (US). In the present day, the World Health Organization (WHO) sets global guidelines for ambient air quality, yet ambient air pollution remains a leading global contributor to poor health and mortality (GBD, 2019).

In the US, many health-relevant pollutants are regulated by the Environmental Protection Agency (EPA) under the Clean Air Act. Particulate matter with aerodynamic diameters smaller than 2.5 microns ($PM_{2.5}$), one such pollutant regulated by the EPA, is directly emitted from both natural and anthropogenic sources and secondarily produced in the atmosphere through photochemical processing of gas-phase emissions. Exposure to $PM_{2.5}$ has been linked with premature mortality and cardiopulmonary morbidity (Burnett et al., 2018; Pope III and Dockery, 2006; Dockery et al., 1993; Pope III, 2007; Krewski et al., 2009). Successful regulation of anthropogenic sources of $PM_{2.5}$ has significantly reduced national-average $PM_{2.5}$ (e.g., Malm et al., 2017; Hand et al., 2013; O'Dell et al., 2019). These reductions in anthropogenic-sourced $PM_{2.5}$ are projected to continue across the present century (Ford et al., 2018; Lam et al., 2011; Leibensperger et al., 2012). In addition to PM and the 5 other criteria air pollutants (i.e., carbon monoxide, lead, ozone, sulfur dioxide, and nitrogen dioxide), the EPA also regulates emissions of 187 Hazardous Air Pollutants (HAPs) under the 1990 Clean Air Act Amendments (EPA, 2015). HAPs are compounds known or suspected to lead to cancer or other severe health impacts (EPA, 2015). Compared to $PM_{2.5}$, HAPs are far less studied and monitored.

In many parts of the world, including the US, biomass burning is a major source of both $PM_{2.5}$ and volatile organic compounds (including several HAPs; Akagi et al., 2011; Crutzen and Andreae, 1990; Yokelson et al., 2008). Recent years have been plagued by recorded-breaking fire seasons in the Amazon, Australia, and western North America. The projected impacts of a changing climate on biomass burning vary by region (e.g., Moritz et al., 2012). In the western US, large fires have been increasing since the mid-1980s due to natural and anthropogenic climate change, as well as fuel accumulation from former land management strategies (Westerling et al., 2006; Westerling, 2016; Abatzoglou and Williams, 2016; Barbero et al., 2014; Marlon et al., 2012). This led to increases in extreme $PM_{2.5}$ event intensity (McClure and Jaffe, 2018) and possibly summer-mean $PM_{2.5}$ concentrations (increasing trends not statistically significant, O'Dell et al., 2019). Across the US, landscape (wild, prescribed, and agricultural) fires have a significant impact on air quality (e.g., Kaulfus et al., 2017; Brey et al., 2018b; David et al., 2021; Buysse et al., 2019) and are estimated to contribute over 40% of US primary $PM_{2.5}$ emissions and 20% of HAPs emissions (EPA, 2017). The impact of fires on US air quality and health is expected to increase in the future (Ford et al., 2018; Liu et al., 2016a; Neumann et al., 2021), where a warming, drying climate is projected to continue to increase large fire frequency and burn area in the western US (Barbero et al., 2015; Spracklen et al., 2009; Brey et al., 2021; Pechony and Shindell, 2010).

Given the large, increasing contribution of landscape fires to health-relevant pollutant emissions in the US, it is important to characterize the health impacts of exposure to landscape fire smoke. The health impacts of landscape fire emissions may differ from more historically studied anthropogenic emissions for several reasons. First, the chemical composition of fire-sourced $PM_{2.5}$ is different from urban $PM_{2.5}$ (e.g., Posfai et al., 2003) and several epidemiology and toxicology studies suggest fire-sourced $PM_{2.5}$ may be more toxic than urban $PM_{2.5}$ (Aguilera et al., 2021; DeFlorio-Barker et al., 2019; Wegesser et al., 2009). Second, compounds co-emitted with $PM_{2.5}$ (e.g., HAPs) may differ from urban emissions and lead to variable health impacts. Finally, landscape fires are highly episodic and follow seasonal cycles, which vary by US region (e.g., Brey et al., 2018a). Epidemiology studies of landscape fire smoke agree that acute exposure to smoke

$PM_{2.5}$ negatively impacts respiratory health (Reid et al., 2016a; Cascio, 2018; Liu et al., 2015, and references within). Cardiovascular impacts of acute smoke exposure are less certain (e.g., Reid et al., 2016a), but there is growing evidence to suggest landscape fire smoke negatively impacts cardiovascular health (e.g., Wettstein et al., 2018; Magzamen et al., 2021). The health impacts of chronic smoke exposure are currently unknown (e.g., Reid et al., 2016a). The contribution of gas-phase HAPs, which have health implications from both acute and chronic exposure, to the health impacts of smoke have yet to be directly quantified. In addition, the implications of regional differences in smoke seasonality and abundance on local healthcare systems is not well understood.

Quantifying exposure to landscape fire smoke is challenging due to the transient nature of smoke, sparse regulatory monitoring networks, lack of information on surface $PM_{2.5}$ air quality from satellites, and limitations of chemical transport models (e.g., uncertain smoke plume injection height; Paugam et al., 2016). Dense low-cost monitoring networks have the potential to aid in characterizing exposure to smoke if sensor limitations can be overcome (Landis et al., 2021). In particular, the rapidly growing PurpleAir monitor network, which relies on optical Plantower $PM_{2.5}$ sensors, provides real-time indoor and outdoor $PM_{2.5}$ observations (https://www.purpleair.com/). Low-cost monitoring networks, including the PurpleAir network, have been used to improve estimates of outdoor $PM_{2.5}$ during smoke events (Gupta et al., 2018; Huang et al., 2021; Mallia et al., 2020; Bi et al., 2020). The influence of landscape fire smoke on indoor air quality, where adults report spending a majority of their time (Klepeis et al., 2001), is an active area of research (Xiang et al., 2021; Kirk et al., 2018; Shrestha et al., 2019; Henderson et al., 2005; Messier et al., 2019; Barn et al., 2008; Reisen et al., 2019; Stauffer et al., 2020; Wheeler et al., 2021; Kaduwela et al., 2019). Low-cost monitoring networks, like the PurpleAir network, can help expand current understanding of the impacts of landscape fire smoke on indoor air quality to a large number of indoor environments across multiple cities and smoke events. Given the impacts of landscape fire smoke on US health (as we will show in Chapter 3), projected increases in the frequency and intensity of smoke events (Liu et al., 2016b), and the large amount of time people spend indoors, it is crucial

to characterize indoor air quality during smoke events to help inform smoke exposure mitigation measures across heavily smoke-impacted regions.

1.2 Scope of Dissertation

In this dissertation, we conduct a quantitative assessment of the influence of US landscape fire smoke on multi-pollutant exposure, health impacts, and indoor air quality with the goal of providing the knowledge necessary to prepare for future smoke events. To this aim, we identify the health risk associated with gas-phase HAPs in smoke, we quantify the acute and chronic effects of smoke at sub-national scales in the US, and quantify indoor air quality during smoke events in multiple western US cities. The following paragraphs provide an overview of the chapters in this dissertation.

Chapter 2 is a research article published in *Environmental Science and Technology* (O'Dell et al., 2020). In this chapter, we provide a quantitative assessment of the abundance and health risk of gas-phase HAPs in western US wildfire smoke using *in-situ* observations from an aircraft-based field campaign. HAPs abundance and health risk are assessed as a function of both smoke plume age and smoke PM concentration. These HAPs to PM ratios are applied to interpolated smoke PM estimates to determine the health risk from smoke-enhanced HAPs exposure in the western US.

Chapter 3 is a research article under review for *GeoHealth*. In this chapter, we conduct a health impact assessment of acute and chronic exposure to landscape fire smoke in the US. We present state, regional, and seasonal totals of the health impacts of smoke exposure. In our assessment of acute smoke exposure, we apply recently developed relative risks of acute smoke-specific $PM_{2.5}$ exposure in the US to estimate smoke-attributable asthma hospitalizations and emergency department visits. In our assessment of chronic smoke exposure, we quantify the disability-adjusted life years attributable to smoke $PM_{2.5}$ as well as multiple gas-phase HAPs, using the ratios of HAPs to PM developed in Chapter 2.

Chapter 4 is a research article in preparation for *GeoHealth*. In this chapter, we present a quantitative analysis of the influence of smoke on indoor air quality across the western US using the PurpleAir network of low-cost $PM_{2.5}$ monitors. We evaluate the relative concentrations of

indoor and outdoor $PM_{2.5}$ on smoke-impacted and smoke-free days across several smoke-impacted western US cities at large and at individual monitors within the cities. In addition, we determine the indoor $PM_{2.5}$ concentrations across these cities as a function of outdoor Air Quality Index (AQI), which is often used in air quality communication and public guidance during smoke events.

Finally, in Chapter 5, we provide a summary of the conclusions from the above chapters. We also discuss the overarching implications of this dissertation and areas for future research.

CHAPTER 2

HAZARDOUS AIR POLLUTANTS IN FRESH AND AGED WESTERN US WILDFIRE SMOKE AND IMPLICATIONS FOR LONG-TERM EXPOSURE

This work is published in *Environmental Science and Technology*[1].

2.1 Motivation and Background

Biomass burning is one of the largest global sources of both particulate matter (PM) and volatile organic compounds (VOCs) to the atmosphere (Akagi et al., 2011; Crutzen and Andreae, 1990; Yokelson et al., 2008). In the United States (US), many anthropogenic sources of these pollutants have been decreasing over the past few decades due to successful regulations (Hand et al., 2013; Malm et al., 2017; McCarthy et al., 2007; Ridley et al., 2018). Over this same time period, large wildfires have been increasing in both frequency and burned area in the western US (Abatzoglou and Williams, 2016; Barbero et al., 2014; Westerling, 2016). Western US wildfire burned area is expected to continue to increase over the coming century due to climate change and land management choices (Barbero et al., 2015; Spracklen et al., 2009; Yue et al., 2013). This trend, combined with anticipated continued decreases in anthropogenic emissions, is projected to increase the fraction of the national average $PM_{2.5}$ from fires to over 50% by the end of the century (Ford et al., 2018; Liu et al., 2016b). In the present day, biomass burning already has a major impact on US air quality. In heavily fire-impacted regions, fires drive interannual variability in summer PM concentrations (Jaffe et al., 2008; Spracklen et al., 2007), have increased the magnitude of extreme PM events (McClure and Jaffe, 2018), and increased summer-average PM (O'Dell et al., 2019).

Wildland fire smoke contains many health-relevant compounds beyond PM (Andreae, 2019; Wentworth et al., 2018; EPA, 2015). Emissions from fires can lead to enhancements in ozone (Brey and Fischer, 2016; Jaffe et al., 2013; Lindaas et al., 2017; Morris et al., 2018), a respiratory irritant. Many of the VOCs emitted by wildland fires also have known negative health effects

[1] 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 and Technology, 54(19), 11838-11847. https://doi.org/10.1021/acs.est.0c04497

and several have been classified as known or reasonably anticipated carcinogens by the National Toxicology Program (NTP, 2016). A recent list of emissions factors for fires contains over 20 gas-phase compounds that have been classified as HAPs by the US EPA (Andreae, 2019; EPA, 2005). HAPs are compounds known or suspected to lead to cancer or other severe health impacts. A list of 189 of these compounds was compiled by the EPA as part of the 1990 amendments to the Clean Air Act. Three of the top five often most abundant VOCs emitted from wildland fires and often other types of biomass burning (methanol, formaldehyde, and acetaldehyde) (Akagi et al., 2011; Liu et al., 2016c; Stockwell et al., 2015, 2014) are on the HAPs list. Previous studies of occupational smoke exposure have quantified a limited number of HAPs in firefighter exposure to wildland fire smoke (Barboni et al., 2010; MacSween et al., 2019; Reinhardt et al., 2000; Reinhardt and Ottmar, 2004; Romagnoli et al., 2014) compared to the number of HAPs that have now been quantified in smoke (Andreae, 2019; Wentworth et al., 2018; Koss et al., 2018).

Despite the potential health risk from HAPs in smoke, these compounds remain unquantified in nonoccupational studies of smoke exposure (Reid et al., 2016a). To directly include HAPs in smoke exposure assessments presents several challenges. Ground observations of HAPs are both spatially and temporally limited, which makes it difficult to assess exposure to highly transient wildfire smoke events. The atmospheric lifetimes vary substantially between different HAPs, from hours to weeks. Some HAPs are also produced secondarily from the oxidation of other species emitted by wildfires. Thus, the ratio between individual HAPs and PM or the relative abundances of different HAPs is a function of smoke age.

In this work, we use daytime wildfire smoke plume observations from the Western Wildfire Experiment for Cloud Chemistry, Aerosol Absorption, and Nitrogen (WE-CAN) aircraft-based field campaign to investigate the relationship between HAP-associated health risks and PM as a function of smoke age. We identify the health risk associated with exposure to HAPs in smoke as a function of plume age and determine which HAPs are the greatest contributors to this risk. We quantify the relationship between HAPs and PM, a common smoke tracer in many health studies, in smoke plumes as a function of smoke age. Finally, we use these relationships, along

with interpolated smoke-specific PM estimates, to identify the spatial distribution of chronic HAPs exposure and subsequent risk from smoke in the western US from 2006-2018. To our knowledge, this is the first multiyear, observation-based paper to estimate the risk of nonoccupational exposure to gas-phase HAPs in wildland fire smoke in the US. The relationships between HAPs and PM in smoke that we calculate have the potential to be used in future health studies and can help inform how variability in HAPs may impact current studies of smoke PM.

2.2 Materials and Methods

This work relies on HAPs and PM observations with diameters smaller than 1 μm (PM_1) from the WE-CAN campaign (https://www.eol.ucar.edu/field_projects/we-can). WE-CAN was an aircraft-based field campaign that sampled wildfire smoke in the western US from 24 July - 28 August, 2018 using the NSF/NCAR C-130. WE-CAN focused on daytime observations of fresh smoke outflow from large wildfires and short-term, < 1 day of aging, downwind plume evolution with a semi-lagrangian sampling technique. However, the aircraft also intercepted and sampled many additional lofted smoke plumes and large regional smoke mixtures, aged several days. Most of these smoke plumes were intercepted in the free troposphere and could have different composition from plumes that remain near ground-level where people are exposed to smoke (Burling et al., 2011). This is a limitation of using observations from aircraft platforms for health-focused analyses. However, the WE-CAN dataset includes measurements of many different smoke plumes emanating from highly active wildfires and a variety of fuels. Of particular relevance here, WE-CAN deployed a comprehensive VOC package designed to overcome the limitations of each individual technique to produce robust sampling of a large number of compounds.

WE-CAN measurements taken onboard the NSF/NCAR C-130 used in this study include: carbon monoxide (CO; Aerodyne Research Mini-QCL tunable diode laser infrared absorption spectrometer; Lebegue et al., 2016; and Picarro G2401-m WS-CRDS analyzer), aerosol composition (High Resolution Time-of-Flight Aerosol Mass Spectrometer; HR-ToF-AMS; DeCarlo et al., 2006; Garofalo et al., 2019) black carbon (Single Particle Soot Photometer; SP2; Schwarz et al., 2008), and VOCs (NCAR's Trace Organic Gas Analyzer; TOGA; Apel et al., 2015; and Proton-Transfer-

Reaction Time-of-Flight Mass Spectrometer; PTR-ToF-MS). Detailed information on these instruments is available in the supplement. VOC observations from TOGA were used to identify urban influence and estimate chemical ages of WE-CAN observations because TOGA speciated more tracers than the PTR-ToF-MS and had a lower detection limit. All data were averaged to the TOGA sampling frequency, the instrument with the lowest time resolution, of 28-second integrated sampling every 100 seconds for flights 1-12 (through 77030 seconds since midnight Coordinated Universal Time (UTC) on flight 12), and 33-second integrated sampling time every 105 seconds for flights 12 (starting at 77030 seconds since midnight UTC) -16. Instrument operators lengthened the sample time in later flights to prevent loss of higher-weight VOCs. This change does not impact any species in our analysis. For VOC observations below reported detection limits, we assumed one-half the limits of detection; however, assuming 0 or the limit of detection instead does not impact our conclusions. The 32 HAPs that were measured during WE-CAN by these instruments are listed in Table A.1 in the supplement.

Smoke-influenced data were identified using co-elevated CO > 85 ppb, hydrogen cyanide (HCN) > 275 ppt, and acetonitrile (CH_3CN) > 200 ppt (Schwarz et al., 2008). We identified and excluded 31 samples (3% of all smoke samples in our analysis) with urban influence because we were not able to accurately age these points with our method due to urban sources of acrolein. Urban-influenced points were defined as observations that met at least one of the following criteria: 2,2,4-trimethylpentane > 20 ppt (fuel additive), tetrachloroethene > 2 ppt (dry-cleaning and metal degreasing agent), HFC-134a > 125 ppt (refrigerant), or HCFC-22 > 275 ppt (refrigerant) (ATSDR, 2020; EPA, 2013; Xiang et al., 2014). For the remaining smoke-influenced HAPs and PM_1 observations, we present a background-corrected mass concentration at standard temperature and pressure (STP) as the difference between the in-smoke, non-urban-influenced data and background, where negative values were taken as zero enhancement, and background was defined as the median of all non-smoke-influenced WE-CAN observations. Our main conclusions are not sensitive to the decision to remove negative smoke-enhanced mass concentrations and are robust within the interquartile range of the background for each species.

Smoke-influenced data across all WE-CAN observations of different fuel types and burn conditions were grouped by an approximate chemical plume age. Grouping the data by plume age, rather than burn conditions or fuel type, allows us to probe changes in smoke HAPs as a function of age over the general range of emissions from western wildfires. However, our results may not be applicable to specific cases or to smoke other from western US wildfires. We estimate chemical plume age using three VOCs, for which OH is likely the dominant sink, with different 2^{nd}-order reaction rate constants for loss via reaction with OH: 2-methylfuran (k_{OH} = 7.31 x 10^{-11} cm^3 $molecule^{-1}$ s^{-1}; Aschmann et al., 2011), acrolein (k_{OH} = 1.96 x 10^{-11} cm^3 $molecule^{-1}$ s^{-1}; Atkinson, 1986; Manion et al., 2015) and acrylonitrile (k_{OH} = 4.04 x 10-12 cm^3 $molecule^{-1}$ s^{-1}; Manion et al., 2015; Harris et al., 1981). Although acrolein also has a chemical source from alkenes, this source is negligible in the free troposphere compared to the direct emissions of acrolein from fires (Apel et al., 2015). We assigned each in-smoke observation to an age category as follows: if 2-methylfuran > 0.7 ppt (95^{th} percentile of non-smoke background observations), smoke was designated as "young"; if 2-methylfuran was not elevated but acrolein was > 7.4 ppt, smoke-impacted data was designated as "medium"; if neither 2-methylfuran nor acrolein were elevated, but acrylonitrile was > 2.9 ppt, smoke-impacted data was designated as "old". If none of these age tracers are elevated, but the smoke tracers are elevated, the data was assigned an age of "older." This final "older" category contains 15 data points and does not present significantly different characteristics than the old category, so the two categories were combined and labeled as "old" throughout.

An approximate age range for each category was determined by assuming that reaction with OH was the main loss process for the age tracers. We assumed a near-source distribution of initial mixing ratios based on what was observed when the aircraft intercepted a set of 10 fires closest to the source; these samples were typically collected within minutes to hours of emission. An approximate loss rate for each species was then calculated as the loss rate by reaction with OH at an assumed OH concentration of 2 x 10^6 molecules cm^{-3}, within the range of typical summertime OH concentrations and elevated concentrations observed in smoke plumes (e.g., Akagi et al., 2012). The chemical age was estimated as the time required for these high near-source abundances to

decay to the 95^{th} percentile of that species in smoke-free background air intercepted during the WE-CAN flights. These initial near-source mixing ratios and the distribution of estimated times to background are shown in Figures A.1 and A.2, respectively. With this method, we found an approximate age of < 1 day for young smoke, 1-3 days for medium smoke, and > 3 days for old smoke.

We acknowledge that this method of assigning approximate chemical age is sensitive to both variability in emissions from fires and dilution. In Figure A.1, we show the range of mixing ratios of the age-tracers within the sampling transects closest to the WE-CAN fires, and in Figure A.2, we show the estimated time required for these age-tracers to reach the 95^{th} percentile of their respective background mixing ratios in non-smoke conditions. Dilution effects could cause our method of smoke aging to misplace younger, dilute smoke in an older category. We explore the potential impacts of dilution on age assignment in Figure A.3, where we show that the distribution of dilution-corrected tracers is similar to the non-dilution-corrected tracer distributions. Age uncertainties due to dilution are thus unlikely to significantly impact our conclusions. This method of age assignment is also sensitive to the assumed OH concentration and the assumption that OH is the dominant sink for each age tracer. Increasing our assumed OH concentration to 5 x 10^6 molecules cm^{-3}, the elevated OH concentration estimated for a young western US smoke plume in Akagi et al. (2012), reduces the median age for each category by 60%. In addition to OH, there are other oxidants in smoke plumes that can react with the age tracers. We provide estimates of the lifetime of each age tracer with O_3, NO_3 and OH using 2^{nd} order reaction rate coefficients in Table A.2 While OH is likely the dominant sink for acrolein and acrylonitrile (Grosjean, 1990; Munshi et al., 1989), 2-methylfuran is also highly reactive with NO_3 (Kind et al., 1996). Therefore, in dark plume centers where NO_3 chemistry is possible or in plumes existing through nighttime periods, the age associated with the young and medium age categories would decrease. These latter two assumptions in OH concentration and role as the dominant sink for each age tracer would only impact the associated quantitative ages while the qualitative "young," "medium," "old" categorization would not be affected. A final issue to note is that the measurements are averaged over the

TOGA sampling time. The TOGA sampling times can encompass both smoke-impacted and background conditions or steep gradients in concentration. This can impact the classification used here, and it is particularly relevant for narrow plumes. Despite these limitations of our aging method, we find general agreement between our chemical age categorization and physical age estimates of WE-CAN smoke plumes (Lindaas et al., 2021).

To estimate the health risk posed by HAPs exposure in wildfire smoke, we used acute (1 hour of exposure) and chronic (70 years of exposure) reference concentrations and chronic cancer unit risk estimates from the EPA Office of Air Quality Planning and Standards (OAQPS, EPA, 2014b). Acute reference concentrations are an estimate of the concentration below which exposures ranging from 15 minutes to multiple weeks leads to negligible to moderate risk. In this study we only use acute reference exposure levels (RELs) for one hour of exposure. Chronic reference concentrations are an estimate of the concentration below which lifetime exposure has no appreciable risk. Uncertainty in these reference concentrations is estimated to span approximately an order of magnitude (EPA, 2014b). The chronic cancer unit risk estimate is an approximate upper-bound on the additional lifetime cancer risk for chronic exposure per unit (e.g., 1 μg m^{-3}) of a given HAP (EPA, 2014b). Reference concentrations and cancer unit risk estimates for HAPs measured in WE-CAN are given in Table A.1.

We compared all observations to acute reference concentrations to investigate how frequently these limits are exceeded in smoke plumes of different ages. We calculated hazard quotients as the ratio of observed mass concentrations for each HAP to a reference concentration, and these were summed across HAPs to calculate the acute and chronic hazard index. Cancer risk is calculated as the product of the observed mass concentration and the cancer unit risk estimate, giving the risk of developing cancer per 1 million people. For each observation within the three age categories, mass concentrations at STP of each HAP were used to determine the acute hazard index, chronic hazard index, and cancer risk. We divided these values by co-sampled PM_1 mass concentrations at STP. We then calculated the median of the PM_1-weighted hazard indices and cancer risk for each HAP across the three age categories. We chose to normalize by PM_1, as opposed to an inert

tracer, because PM is a commonly-used health metric for smoke exposure (Reid et al., 2016a). However, normalizing by CO, a relatively inert tracer on these timescales, did not change our main conclusions (Figure A.4).

Individual HAP to PM_1 relationships were calculated as the median of observed HAPs to PM_1 ratios for each age category. To estimate HAPs mixing ratios and subsequent health risk across the western US from 2006-2018, we applied these HAPs to PM_1 ratios to estimates of ambient smoke-specific $PM_{2.5}$, therefore scaling the HAPs concentration with the ambient smoke-specific $PM_{2.5}$ concentration. Ambient, smoke-specific $PM_{2.5}$ concentrations were estimated following O'Dell et al. (2019). In brief, daily concentrations of $PM_{2.5}$ were calculated by interpolating $PM_{2.5}$ monitoring data from the EPA's Air Quality System to a 15 x 15 km grid. Smoke-specific $PM_{2.5}$ concentrations were calculated by subtracting seasonal, non-smoke background concentrations from ambient data on days identified as smoke impacted by the Hazard Mapping System Fire and Smoke product (Rolph et al., 2009; Brey et al., 2018a). We used the 13-year mean smoke-specific $PM_{2.5}$ concentrations along with the HAPs to PM_1 ratios to estimate chronic HAPs exposure and subsequent hazard ratios and cancer risk from wildland fire smoke under several assumptions. First, because there is higher spatial coverage of $PM_{2.5}$ monitors across the US, but only PM_1 was reliably measured during WE-CAN, we assumed the mass contribution of smoke particulate matter with diameters between 1 and 2.5 μm was negligible, allowing us to use HAPs ratios to PM_1 with $PM_{2.5}$ concentrations. This assumption artificially increases the ratio-estimated HAPs concentrations relative to if $PM_{2.5}$ had been available to use in the HAPs to PM ratios. Second, following the approximate smoke ages from Brey et al. (2018a), we assumed the medium-age ratios were applicable across the western US.

There are several limitations and assumptions made when using this method of risk assessment with WE-CAN observations of wildfire smoke. Concentrations of HAPs measured in WE-CAN may not be representative of personal exposure due to individual behavior (e.g., time spent indoors/outdoors) and, as mentioned previously, lofted smoke measured in WE-CAN may have a different composition than smoke that remains near the surface (Burling et al., 2011). The major-

ity of young and medium aged WE-CAN TOGA observations were sampled at altitudes between 3 and 5 km above sea level, while the majority of old smoke observations were sampled above 5 km (Calahorrano et al., 2021). For chronic risk assessments, we assumed that the 2006-2018 average concentrations of smoke-specific HAPs are continuous and constant over a lifetime (rather than imposing higher intermittent concentrations during smoke events). While constant exposure is not true of smoke, assuming constant exposure is common for long-term smoke $PM_{2.5}$ health assessments (e.g., Ford et al., 2018). It is currently uncertain how intermittent versus continuous exposures over a lifetime may lead to different chronic health outcomes. Further, although smoke plumes are a complex mixture of both HAPs and criteria air pollutants (CO, PM, O_3), we treat each HAP based on its individual risk and assume that their risks are linearly additive. The potentially nonlinear impacts of multi-pollutant exposure are not well understood, but could lead to synergies that elevate risk beyond what is presented here.

2.3 Results and Discussion

WE-CAN flight tracks and TOGA observations used in this study, colored by estimated chemical age, are plotted in Figure 2.1. The majority of WE-CAN smoke samples in this study were from young and medium aged plumes (346 and 463 TOGA samples, respectively, of the total 903 TOGA smoke samples with an assigned age and available PM_1 observations). Most smoke plumes were sampled in a pseudo-lagrangian fashion. Although WE-CAN was focused on observations of fresh smoke linked to specifically targeted fires, the plane often opportunistically intercepted older smoke (94 TOGA samples) when transiting to and from these fresh smoke plumes. Two flights, one through southern Oregon and another through the California Central Valley, specifically targeted smoke of mixed ages from multiple fires.

Distributions of the background-corrected mass concentrations of HAPs in wildfire smoke measured during WE-CAN for each smoke-age category are shown in Figure 2.2. The mass concentrations of the HAPs are plotted alongside their acute exposure limits (for one hour of exposure) from the California EPA, where available. Although there were 32 HAPs measured in WE-CAN, and over half of these species were occasionally elevated to mixing ratios > 100 ppt over background,

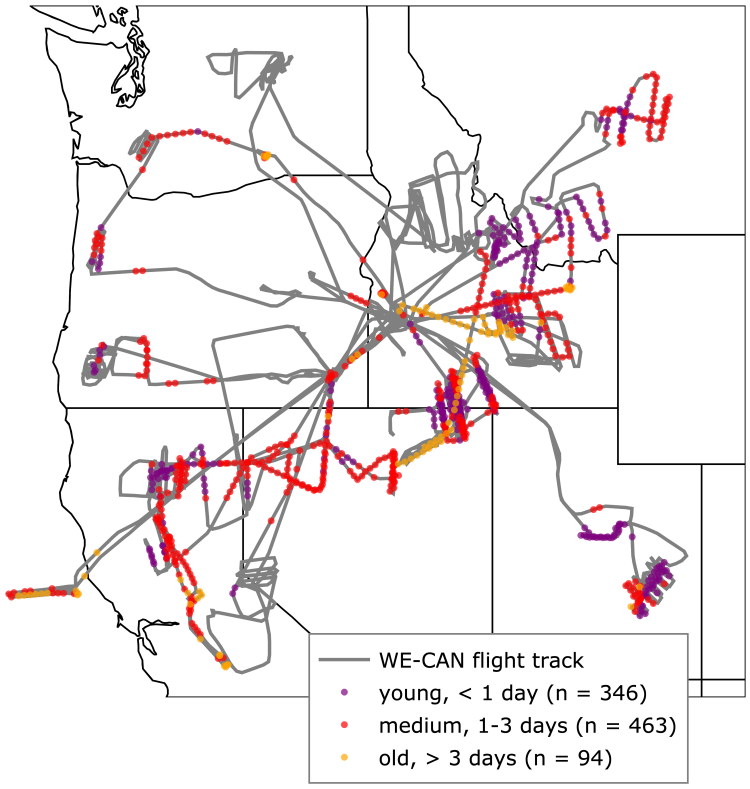

Figure 2.1: WE-CAN flight tracks (grey lines) with TOGA samples used in this study (filled dots) colored by estimated smoke plume chemical age. The number of data points in each smoke age category are given alongside the legend in parenthesis.

only a few exceeded acute exposure limits. In young smoke, formaldehyde, acrolein, and benzene exceeded the California EPA reference exposure levels for no adverse effects. These occurred most often in the highly concentrated fresh plumes with physical ages as young as 20 minutes (Lindaas et al., 2021). In medium and old smoke within the WE-CAN dataset, no HAPs were found to exceed acute exposure limits. The young age category is most likely relevant to firefighter exposure and those in close proximity to the fires. Previous studies of firefighter exposure to smoke from western US fires have observed formaldehyde, acrolein, and benzene concentrations approximately 6 times higher than the maximum concentrations observed in WE-CAN (Reinhardt and Ottmar, 2004). However, while we find these species exceeded reference concentrations for the general population, Reinhardt and Ottmar (2004) found those species did not exceed Occupational

Safety and Health Administration short-term exposure limits and permissible exposure limits for wildfire firefighters, which are set much higher than the reference concentrations used here.

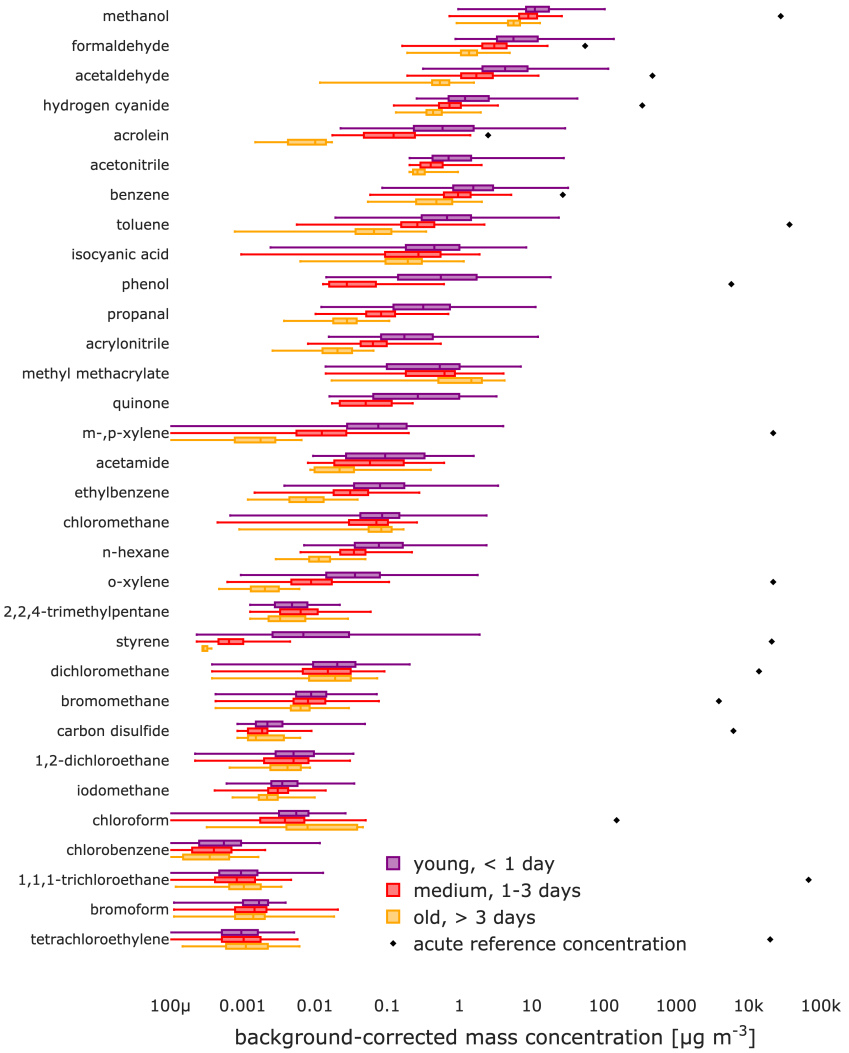

Figure 2.2: Boxplots of the enhancement of HAPs measured during WE-CAN in young, medium, and old smoke plumes. Species are listed in order of decreasing WE-CAN emissions ratio. Boxes extend from the 25^{th} to 75^{th} percentile and whiskers extend from the minimum non-zero value to the maximum value. Diamonds show the California EPA acute reference exposure level for one-hour average exposure to each HAP, where available.

Several species in Figure 2.2 do not have OAQPS reported acute or chronic reference concentrations nor cancer unit risk estimates. These are: isocyanic acid, quinone, iodomethane and 2,2,4-trimethylpentane. These compounds are removed from the remainder of our results and

discussion. The species with the highest in-smoke concentrations that does not have published reference concentrations nor cancer unit risk estimates in OAQPS, isocyanic acid, has previously been identified as a health-relevant compound in smoke (Roberts et al., 2011). Roberts et al. (2011) found smoke-influenced ambient mixing ratios above 200 pptv (0.4 μg m^{-3} STP), similar to the abundances observed in medium and old smoke during WE-CAN. Isocyanic acid exposure levels greater than 1 ppbv (2 μg m^{-3} STP) are suspected to negatively impact human health (Roberts et al., 2011). During WE-CAN, mass concentrations above 2 μg m^{-3} STP were observed in young smoke, implying isocyanic acid may be at hazardous levels for firefighters and those close to fires. In addition to the hazardous compounds listed in Figure 2.2, it is possible more of the many species emitted by fires and/or produced in smoke plumes could also be hazardous but are not identified as such by the EPA because they are less commonly emitted or produced. Further, while our study focuses on gas-phase HAPs, particle-phase HAPs, as well as varying particle composition and size distribution, could also impact overall smoke hazard.

In Figure 2.3, we show the median PM_1-weighted risk of gas-phase HAPs in smoke, colored by contributing HAP, for acute (panel a), chronic noncancer (panel b), and chronic cancer (panel c) outcomes for each smoke age category. Normalization by PM_1 is motivated by PM's use as the dominant smoke metric in epidemiology studies of smoke exposure (Reid et al., 2016a), and it approximately normalizes for smoke-plume dilution. In Figure A.4, we normalize gas-phase HAPs risk by CO, an inert tracer on these timescales, and find similar results to those presented in Figure 2.3. In every panel of Figure 2.3, the total PM_1-weighted HAPs risk decreases as the smoke ages, indicating that the total health risk of smoke varies for the same PM_1 concentration, depending on the smoke age. This decrease is most prevalent for acute outcomes in Figure 2.3a. We find 10 μg m^{-3} of PM_1 in young smoke (aged less than one day) is associated with a total acute hazard index of 0.085, while 10 μg m^{-3} of PM_1 in smoke aged 1-3 days is associated with an acute hazard index of 0.057, and smoke aged over 3 days has a hazard index of 0.024 per 10 μg m^{-3} of PM_1. This is a drop of 72% in the risk of gas-phase HAPs per PM_1 from young to old wildfire smoke.

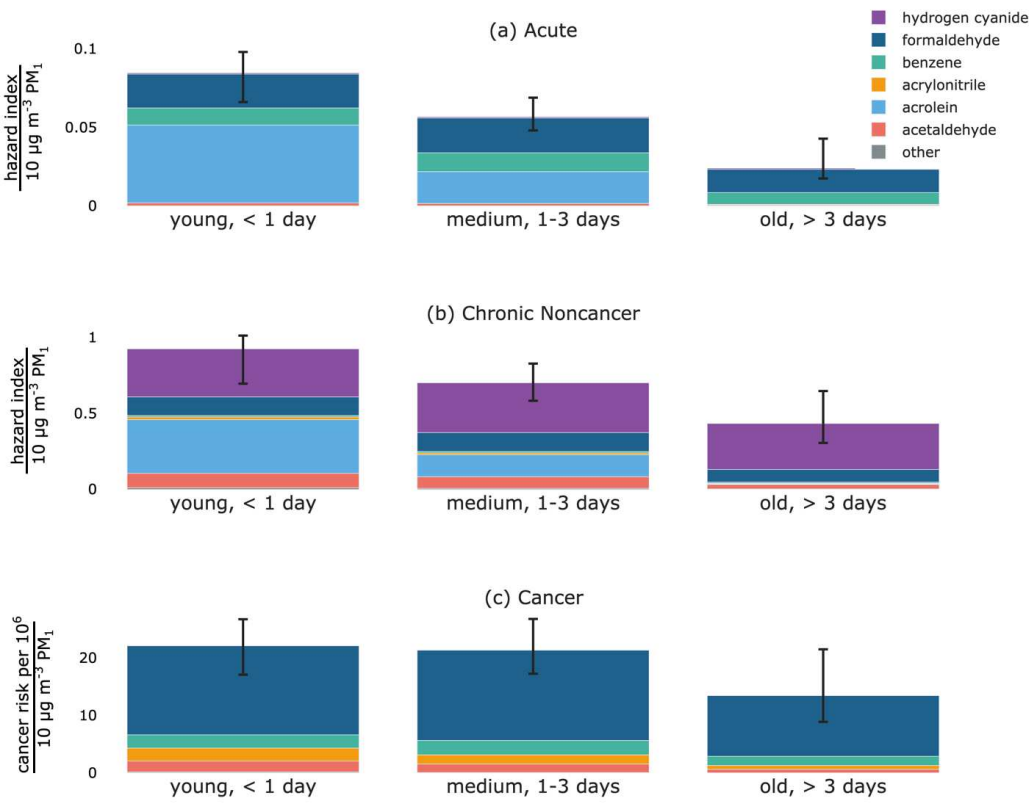

Figure 2.3: Median gas-phase HAPs acute hazard index (panel a), chronic noncancer hazard index (panel b), and cancer risk (panel c) ratio to 10 μg m^{-3} of smoke PM_1 for each smoke age category. Black bars span from the 25^{th} to 75^{th} percentile of the species-summed hazard index for each age category. "Other" contains any species with non-zero smoke elevation and an available acute 1-hour California EPA reference exposure level, chronic reference concentration, or cancer unit risk estimate. A full list of species included in this analysis and associated reference concentrations and cancer unit risk estimates are given in Table A.1

To reach an acute hazard index of 1 (threshold of potential risk) would require 118 μg m^{-3} of young smoke PM_1, but 175 μg m^{-3} of medium smoke PM_1, and 417μg m^{-3} of old smoke PM_1. In extreme smoke events, $PM_{2.5}$ concentrations of this magnitude have been observed at monitors near population centers (US EPA AQS). During the extreme smoke events in San Francisco, CA in November 2018, multiple AQS $PM_{2.5}$ monitors reported hourly $PM_{2.5}$ concentrations > 200 μg m^{-3} and several located outside the city reported hourly concentrations as high as 600 μg m^{-3} (US EPA AQS) Based on our ratios of HAPs hazard index to PM_1, these locations likely also experienced a gas-phase HAPs acute hazard index above 1 due to the wildfire smoke. Although observations of acrolein and formaldehyde are not available during this event, we find the observed

daily-average benzene concentrations from all sources in the Bay Area on 16 November 2018 (4.9 and 5.9 μg m^{-3}; US EPA AQS) from the EPA AQS network are within 20% of estimated smoke-specific benzene concentrations (5.1 μg m^{-3}) using our benzene to PM_1 ratio for young smoke with observed daily-average $PM_{2.5}$ (170 μg m^{-3}; US EPA AQS). While the ratios and the results presented in Figure 2.3 are broadly representative of a mixture of flaming and smoldering wildfires consuming a variety of western US fuels (the conditions sampled in WE-CAN), the ratios are likely not applicable to smoke from specific fuels and burn conditions, especially in regions with different climates and ecosystems.

In Figure 2.3b and 2.3c, similar decreases are observed for the PM-weighted chronic noncancer and cancer risks from gas-phase HAPs in smoke. Chronic noncancer risk per 10 μg m^{-3} of PM_1 (Figure 2.3b), drops from 0.93 to 0.43 (54%), and chronic cancer risk per 10 μg m^{-3} of PM_1 (Figure 2.3c) from 22 per million population to 13 per million, or 41% from young (< 1 day) to old (> 3 days of aging) smoke. Therefore, to reach a hazard index of 1 for chronic noncancer outcomes would require chronic exposure to 11 μg m^{-3} of young smoke PM_1, or 23 μg m^{-3} of old smoke PM_1. For chronic cancer outcomes to reach a level of 1 per million, which has been identified as a level of "potential concern" (McCarthy et al., 2009) would require chronic exposure to 0.45 μg m^{-3} of young smoke PM_1, or 0.77 μg m^{-3} of old smoke PM_1. Later, in Figure 2.4, we will estimate chronic exposure to smoke-specific $PM_{2.5}$ and implications for risk from chronic exposure to smoke-elevated gas-phase HAPs for the western US.

Figure 2.3 shows the contribution of each individual HAPs hazard quotient to the total hazard index. Acrolein is the dominant contributor to the health risk of acute exposure (Figure 2.3) to gas-phase compounds in young smoke, contributing 58% of the hazard index. The next highest contributors to total acute risk are formaldehyde and benzene, contributing 25% and 13% of the hazard index in young smoke, respectively. Due to the relatively short atmospheric lifetime of acrolein, its contribution drops drastically with smoke age to 36% in medium smoke and 0% in old smoke. Benzene and formaldehyde then contribute higher fractions of the total hazard index.

Acrolein and hydrogen cyanide are the dominant contributors to the hazard index of chronic exposure to HAPs in young smoke (Figure 2.3b), contributing 38% and 34% of the median hazard index, respectively. The contribution of acrolein to the total hazard index again decreases with smoke age, making hydrogen cyanide the dominant contributor to the hazard index of HAPs in medium (aged 1-3 days) and old (aged > 3 days) smoke, contributing 47% and 70% of the hazard index in medium smoke and old smoke, respectively. In Figure 2.3c, formaldehyde is the dominant contributor to cancer risk from HAPs in wildfire smoke for each smoke-age category. Formaldehyde accounts for over 50% of the cancer risk in young, medium, and old smoke. While formaldehyde has a relatively short atmospheric lifetime of several hours against oxidation by OH and photolysis during the day (Atkinson, 2000; Levy, 1972) it is produced during the oxidation of methane and other VOCs (Lee et al., 1998), and thus remains elevated in older smoke. Other dominant contributors to cancer risk from HAPs include: acrylonitrile, benzene, and acetaldehyde.

Figure 2.4 panels a and d show the mean smoke-specific $PM_{2.5}$ in the western US from 2006-2018 and annual mean for 2018, respectively. These data have been updated to recent years from a previous work (O'Dell et al., 2019). Figure 2.4 panels b and c show the estimated hazard index and cancer-risk, respectively, associated with chronic exposure to gas-phase HAPs in smoke. For this calculation, present-day chronic exposure is taken as the 2006-2018 mean mass concentration. In Figure 2.4d-e, we repeat these calculations using annual-mean 2018 smoke concentrations (high smoke year) as a proxy for potential future smoke concentrations if 2018-like years become the norm due to increasing fire burn area (Yue et al., 2013). For this calculation, we use the ratios of medium-aged smoke (aged 1-3 days) and assume the mass of particles with diameters between 1 and 2.5 μm is negligible. The former assumption may underestimate the HAPs risk for populations frequently exposed to younger smoke (or overestimate if the population is more frequently exposed to older smoke) and the latter assumption could also lead to an overestimation if a significant portion of the $PM_{2.5}$ mass is from particles larger than 1 μm in diameter.

Figure 2.4b shows that the chronic noncancer hazard index from chronic exposure to gas-phase HAPs in smoke is < 1 across the western US. A hazard index of 1 is used as the threshold to indicate

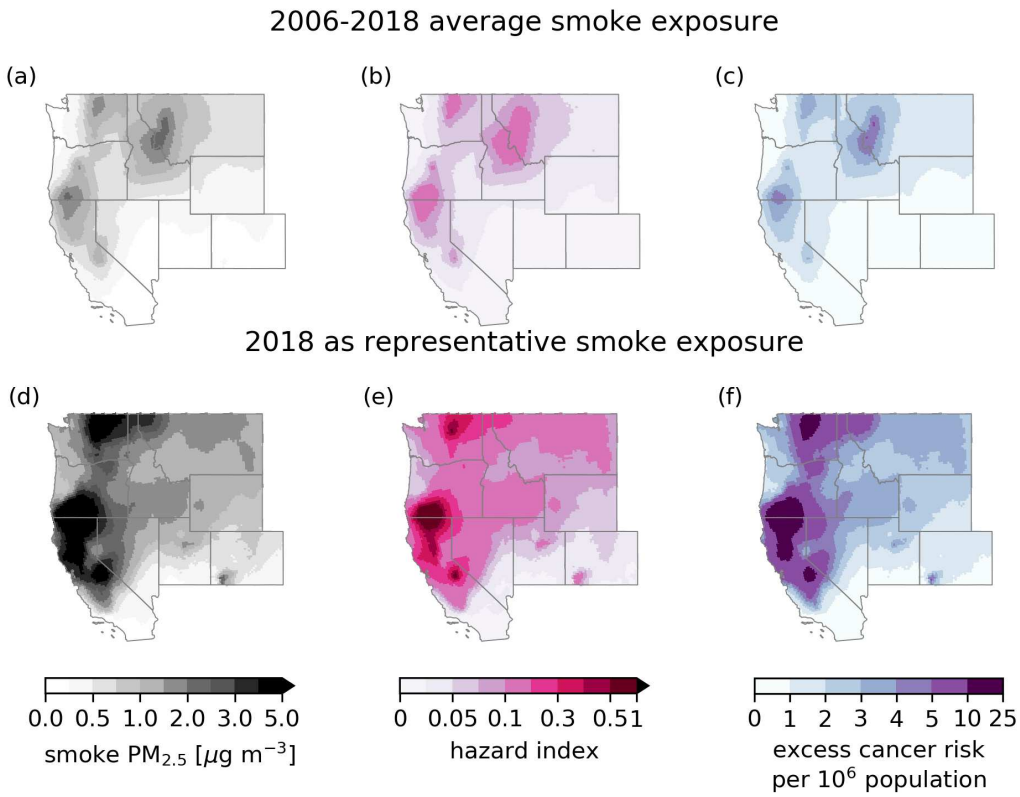

Figure 2.4: 2006-2018 mean smoke-specific $PM_{2.5}$ (a), hazard index (b) and excess cancer risk due to smoke exposure on a 15 x 15 km grid (c) for chronic exposure to gas-phase HAPs in wildland fire smoke when 2006-2018 mean smoke is used as chronic exposure. 2018 annual average smoke-specific $PM_{2.5}$ (d), hazard index (e), and excess cancer risk due to smoke exposure (f) on a 15 x 15 km grid for chronic exposure to gas-phase HAPs in wildland fire smoke when 2018 annual average smoke is assumed to be chronic exposure. Hazard indices and cancer risks assume ratios of gas-phase HAPs to PM_1 in medium smoke.

a potential health risk. Although smoke-specific hazard indices are below this value, the smoke HAPs may combine with other HAPs sources, or other air pollutants, to increase overall risks. Figure 2.4c shows the estimated cancer risk per 10^6 population at present-day smoke conditions. In previous studies of health risk from chronic exposure to urban HAPs in the US from the EPA, a cancer risk of 1 per 10^6 was used to identify locations and pollutants of "potential concern" (McCarthy et al., 2009). McCarthy et al. (2009) found 100% of urban monitors studied exceeded this threshold for both benzene and formaldehyde, which are also main contributors to chronic cancer risk due to gas-phase HAPs in wildfire smoke. Due predominantly to formaldehyde, heavily

fire-impacted regions of the western US exceed this threshold due to wildfire smoke alone, as shown in Figure 2.4c.

In panels d-f of Figure 2.4, we investigate how the potential chronic health impacts of smoke exposure would change if a fire year like 2018 became representative of future chronic exposure. Figure 2.4e shows the hazard index approaches 1 for noncancerous health impacts of chronic exposure to gas-phase HAPs under this scenario. Again, these wildfire smoke HAPs abundances could combine with HAPs from other sources and increase this risk further. In Figure 2.4f, the cancer risk due to gas-phase HAPs in smoke in parts of the western US exceeds 10 incidences per million population. Nearly the entire western US exceeds the national average cancer risk from HAPs in smoke of 0.71 per million (EPA, 2014a) and "potentially concerning" benchmark of 1 incidence per million population due to wildfire smoke alone in Figure 2.4f. While this estimated cancer mortality from HAPs in smoke is small compared to estimated all-cause mortality from chronic smoke $PM_{2.5}$ exposure of 15,000-20,000 deaths in the US annually (Ford et al., 2018), wildfire smoke is an important source of HAPs health risk in heavily fire-impacted areas. Our estimated cancer risk from gas-phase HAPs in smoke in heavily fire-impacted regions is nearly one third of the national average total cancer risk from ambient air toxics of 31 per million according to the EPA's 2014 national air toxics assessment (EPA, 2014a).

To our knowledge, this is the first multi-year, observation-based study to investigate the potential acute and chronic health risks of non-occupational exposure to gas-phase HAPs in wildfire smoke. This work adds to the growing literature on the health impacts of wildfire smoke exposure by investigating the potential health impacts of smoke constituents previously missing from smoke exposure assessments, and connecting them to a commonly used smoke tracer (PM). While gas-phase HAPs likely play a much smaller role in the health impacts of smoke compared to PM, the systematic evolution in the ratio of HAPs to PM with smoke age presented here highlights the importance of multi-pollutant assessment for wildfire smoke exposure. Future work should investigate how the variability observed in emissions of HAPs and PM from different fire conditions and fuels (Akagi et al., 2011; Andreae, 2019; Stockwell et al., 2014; Burling et al., 2010; Sekimoto

et al., 2018) contributes to systematic differences in smoke HAPs exposure in different regions. Exposure to HAPs during smoke events could be assessed using ground-based observations of the smoke-elevated HAPs highlighted in this study: acrolein, formaldehyde, benzene, and hydrogen cyanide. In order to understand how HAPs may play a role in health impact assessments of smoke exposure, a greater understanding of the health effects of individual HAPs and co-exposure to multiple pollutants is needed.

2.4 Acknowledgements and Data Availability

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CHAPTER 3

ESTIMATED MORTALITY AND MORBIDITY ATTRIBUTABLE TO SMOKE PLUMES IN THE US: NOT JUST A WESTERN US PROBLEM

This work is under review for *GeoHealth*[1].

3.1 Motivation and Background

Smoke from landscape (wild, prescribed, and agricultural) fires significantly degrades air quality across the US (Brey et al., 2018b; Brey and 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 ($PM_{2.5}$) in the US (EPA, 2017) and is responsible for a majority of non-anthropogenic exceedences 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 have led to observed increases in the intensity of extreme $PM_{2.5}$ events and summer mean $PM_{2.5}$, despite decreasing anthropogenic emissions (McClure and Jaffe, 2018; 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., 2016a; 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 et al., 2016a; 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 et al., 2016b). Impacts on mortality and cardiovascular mor-

[1]O'Dell, K., K. Bilsback, B. Ford, S. E. Martenies, S. Magzamen, E. V. Fischer and J. R. Pierce (under review), Estimated Mortality and Morbidity Attributable to Smoke Plumes in the US: Not Just a Western US Problem, GeoHealth, M.S. no: 2021GH000457.

bidity are less certain (e.g., Reid et al., 2016a), but evidence for these outcomes of acute smoke exposure is growing (e.g., Magzamen et al., 2021; Wettstein et al., 2018; Doubleday et al., 2020). Aguilera et al. (2021) and DeFlorio-Barker et al. (2019) 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. 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 et al., 2018b; 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; EPA, 2015) which are compounds known or suspected to lead to serious health impacts (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 health impact assessments (HIA) to quantify: (1) the seasonal and spatial distribution of US asthma hospital admissions and emergency department (ED) visits attributable to acute smoke $PM_{2.5}$ expo-

sure, (2) the mortality from chronic smoke $PM_{2.5}$ exposure by state, and (3) 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 smoke $PM_{2.5}$ exposure (Borchers Arriagada et al., 2019) to estimate the hospital admissions and 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.

3.2 Materials and Methods

3.2.1 Smoke $PM_{2.5}$ and HAPs Concentration Estimates

To conduct this HIA on smoke in the US, we estimated observation-based daily smoke $PM_{2.5}$ 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 2006-2018. 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 acknowledgements section. Daily-average $PM_{2.5}$ observations from the surface monitors in the EPA Air Quality System (AQS) were interpolated to a 15 x 15 km grid using ordinary kriging. Daily smoke plume information was obtained from NOAA Hazard Mapping System (HMS) smoke plume polygons (Brey et al., 2018a; 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 $PM_{2.5}$ concentrations, we estimated a non-smoke background $PM_{2.5}$ as the seasonal median of the gridded $PM_{2.5}$ on days without an overlapping HMS smoke plume. We also conduct our analysis using the seasonal mean of the gridded $PM_{2.5}$ on days without an overlapping HMS smoke plume and find this choice does not

impact our main conclusions. The smoke $PM_{2.5}$ was then calculated as the difference between the kriged $PM_{2.5}$ and the non-smoke background $PM_{2.5}$ on smoke-impacted days. On non-smoke-impacted days, smoke $PM_{2.5}$ was set to zero. These data have been previously used in atmospheric science, epidemiological, and economic studies of US smoke $PM_{2.5}$ (Abdo et al., 2019; Burkhardt et al., 2019a,b; Gan et al., 2020; Lipner et al., 2019; Magzamen et al., 2021; O'Dell et al., 2020, 2019)

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_1 (particulate matter with aerodynamic diameters smaller than 1 μm) were developed using observations from the Western Wildfire 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_1 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 $PM_{2.5}$ by grid cell for a gridded estimate of chronic smoke HAPs exposures. To perform this calculation, we made several assumptions. First, we assumed the mass concentration of PM with diameters between 1 μm and 2.5 μm was negligible. The WE-CAN ratios were to smoke PM_1 mass concentrations while our kriged estimates were of smoke $PM_{2.5}$ mass concentrations. Volume size distributions of smoke aerosol from Bian et al. (2020) indicate that <5% of $PM_{2.5}$ volume (and hence mass) exists in the diameter range of 1-2.5 μm, thus errors due to this assumption are <5%, smaller than errors in exposure and the health impact functions. Further, we assumed that the WE-CAN HAPs to $PM_{2.5}$ ratios are representative of all US smoke plumes. However, smoke HAPs concentrations may vary by fuel type (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 of smoke $PM_{2.5}$, such that the overall health estimates of smoke are not greatly influenced by our assumptions in the HAPs calculation.

3.2.2 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 et al., 2016a) and have been included in a meta-analysis of acute smoke $PM_{2.5}$ exposure (Borchers Arriagada et al., 2019). We estimated asthma hospitalizations and ED visits attributable to acute smoke $PM_{2.5}$ exposure with the following health impact function,

$$\Delta Events = Population \times (Y_0/365) \times \left(1 - \mathrm{e}^{-\beta \times \Delta PM_{2.5}}\right), \tag{3.1}$$

from Anenberg et al. (2010, 2014) for chronic $PM_{2.5}$ exposure. We assumed the acute smoke health impact function follows the same functional form (e.g., Pratt et al., 2019). In Eq. 3.1, Y_0 is the annual baseline asthma hospital admission rate or asthma ED visit rate, $PM_{2.5}$ is the daily smoke $PM_{2.5}$ concentration, and β is defined,

$$\beta = \ln(RR)/\Delta X, \tag{3.2}$$

where *RR* is the relative risk per ΔX 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 et al., 2016b; 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 B.1. As shown in Figure B.1., we found the meta-analysis central estimate and 95^{th} percent confidence interval (CI) lies within the much wider 95^{th} percent CI of our pooled RRs estimate. Thus, we used the meta-analysis RR in our calculations due to its tighter CI.

We applied the smoke-specific RRs in Eq. 3.1 and 3.2 along with daily kriged smoke $PM_{2.5}$. We used 2.5 arc-minute gridded population estimates for 2010 from the National Space Administration's Socioeconomic Data and Applications Center (SEDAC, 2018), which we regridded to the 15 x 15 km kriged $PM_{2.5}$ grid. National annual baseline rates, also for 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). RRs and baseline rates used in this work are provided in Table B.1. These data were applied in Eq. 3.1 to estimate daily, gridded asthma hospital admissions and asthma ED visits attributable to smoke $PM_{2.5}$ at lag day 0. We represent uncertainty in the 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 9 US regions following Brey et al. (2018a). 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 supplemental Table B.1. Seasons were defined as follows: Winter: January, February, March; Spring: April, May June; Summer: July, August, September; Fall: October, November, December. 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 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 B.5-B.8 in the supplement.

3.2.3 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,

$$\Delta Events = Population \times Y_0 \times (1 - 1/\mathrm{HR})\,, \tag{3.3}$$

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 μg m^{-3} , the lowest observed concentration in the cohort studies used to develop the GEMM. In Table B.3., 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 Eq. 3.4 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 (± 2 standard error) in the GEMM concentration response function coefficients.

3.2.4 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 B.1). 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.

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

$$DALYs = C_i \times 365 \times V \times \left[(\delta DALY_{cancer}/\delta intake)_i + (\delta DALY_{noncancer}/\delta intake)_i \right], \quad (3.4)$$

similar to Logue et al. (2012). In Eq. 3.4, C_i is the concentration of smoke HAP *i* described in Section 2.1, *V* is 14.4 m^3 day^{-1}, an estimated population-mean volume of air inhaled per day from Logue et al. (2012), and $(\delta DALY_{cancer}/\delta intake)_i$ is the estimated DALYs due to cancer effects, and $(\delta DALY_{noncancer}/\delta intake)_i$ 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^{th} and 2.5^{th} 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 $(\delta DALY/\delta intake)_i$ x k_i^{-1} to $(\delta DALY/\delta intake)_i$ x 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. We apply these upper and lower bounds on the 95^{th} percent CI into equation 3.4 to estimate the uncertainty bounds for our DALY estimates.

3.3 Results

3.3.1 Landscape Fire Smoke $PM_{2.5}$

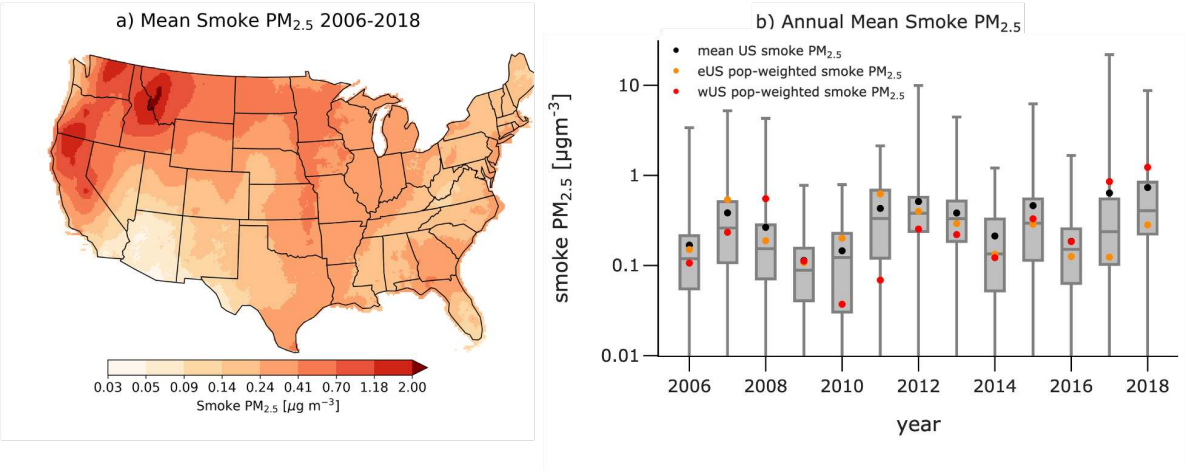

Figure 3.1: 2006-2018 mean smoke $PM_{2.5}$ on a 15 x 15 km grid (panel a) and the distribution of annual average smoke $PM_{2.5}$ across all grid cells for each year (panel b). Boxes in panel (b) extend from the 25^{th} to 75^{th} percentile, with a bar across the box indicating the median value. Box 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.

Observation-based smoke $PM_{2.5}$ estimates across the study period are presented in Figure 3.1a and b. Mean total $PM_{2.5}$ from 2006-2018 and the long-term smoke $PM_{2.5}$ fraction are shown in Figure B.2 in the supplement. The 2006-2018 mean smoke $PM_{2.5}$, Figure 3.1a, reaches over 2 μg m^{-3} in heavily fire-impacted regions of the western US. The box plots in Figure 3.1b show the distribution of annual average smoke $PM_{2.5}$ across all 15 x 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 μg m^{-3} . Across all US grid cells, the area-weighted mean annual smoke $PM_{2.5}$ (black points in Figure 3.1b) is much lower ranging from 0.11 μg m^{-3} in 2009 to 0.73 μg m^{-3} in 2018. We find the area-weighted mean smoke $PM_{2.5}$ across the US is often similar to the population-weighted mean smoke $PM_{2.5}$ in the eastern and western US (orange and red points in Figure 3.1b, respectively). The mean population-weighted smoke $PM_{2.5}$ across the full time period is higher in the western US (0.33 μg m^{-3}) than the eastern US (0.26 μg 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 average observation-based smoke $PM_{2.5}$ is lower than model-based estimates used in previous HIAs (Fann et al., 2018). 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. However, these studies estimated chronic smoke $PM_{2.5}$ exposure over different time periods and, 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) and our observation-based estimates (e.g., lack of information on vertical smoke distribution, Brey et al., 2018a) that lead to uncertainties in total smoke $PM_{2.5}$ concentrations.

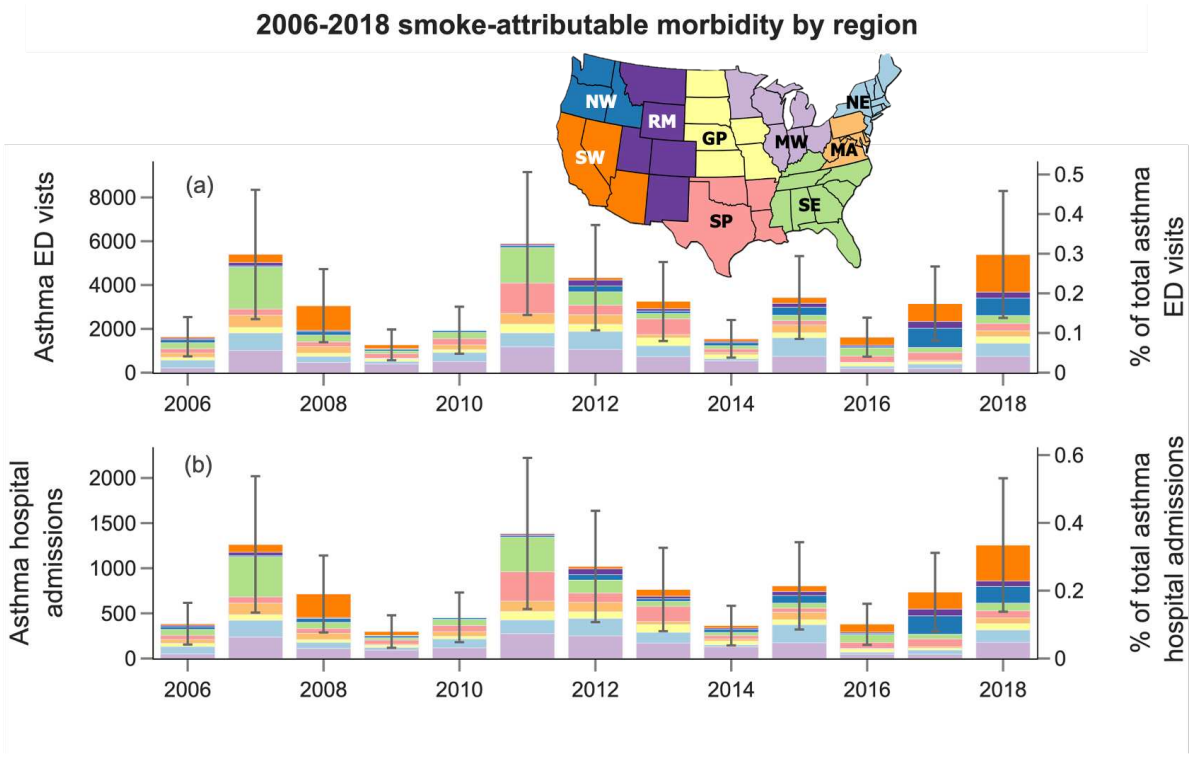

Figure 3.2: Asthma emergency department (ED, panel a) and asthma hospital admissions (panel b) attributable to smoke $PM_{2.5}$ across the contiguous US for each year from 2006-2018. Colors represent different US regions, as indicated by the map, where darker colors represent regions in the western US 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.

3.3.2 Spatial Distribution of Morbidity Attributable to Smoke $PM_{2.5}$

In Figure 3.2, we show the contribution of each region to the total number and percent of asthma ED visits (Figure 3.2a) and asthma hospital admissions (Figure 3.2b) attributable to smoke $PM_{2.5}$ in the US by year from 2006-2018. There is high inter-annual variability in the total number of 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. We find a lower total number (300-1,400) of smoke $PM_{2.5}$ attributable asthma hospital admissions than ED visits, due to a lower baseline rate in the former.

The asthma hospital admissions attributable to smoke $PM_{2.5}$ contribute a similar percent (0.08 - 0.37%) of total annual asthma hospital admissions. Total numbers and percent of asthma ED visits and hospital admissions attributable to smoke $PM_{2.5}$ by year are given in Table 3.1 alongside previous estimates of 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).

Table 3.1: 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 HIAs.

Outcome	This Work	Ford et al. (2018)[2]	Fann et al. (2018)[3]	Neumann et al. (2021)[4]
All Respiratory Hospital Admissions	-	-	3,900 - 8,500	350
Asthma Hospital Admissions	300 - 1,400[5]	-	-	68
Asthma ED Visits	1,300 - 5,900[5]	-	-	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	-	-

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 3.2, including the Midwest (MW), Great Plains (GP), Southern Plains (SP), Northeast (NE), Mid Atlantic (MA), and Southeast (SE) regions). There are only two 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 3.2, 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 re-

[2]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.

[3]Fann et al., 2018 results are 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.

[4]Neuman 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.

[5]Range across all years in this analysis (2006-2018).

gions contribute on average 19% of US asthma morbidity attributable to smoke $PM_{2.5}$. The high inter-annual variability in the total number of asthma ED visits and hospitalizations attributable to smoke $PM_{2.5}$ is also not exclusively driven by the western states. In fact, in the year with the most 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 μg m^{-3}) than the eastern states (0.26 μg 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 Figure 3.3, 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 non-western regions such as the Northeast, Southeast, and Midwest (Figure 3.3c). 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, 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.

3.3.3 Seasonality of Morbidity Attributable to Smoke $PM_{2.5}$

In Figure 3.3, we also show the seasonality of 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 B.5-B.8 in the supplement. In Figure B.9, we show the percent contribution of each season to regional morbidity attributable to smoke $PM_{2.5}$ summed across all years. Across the US, most 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 2006-2018 occur. Notably, the total number of asthma ED visits and hospitalizations are at an annual nadir over the summer (Pendergraft et al., 2005; Silverman et al., 2003).

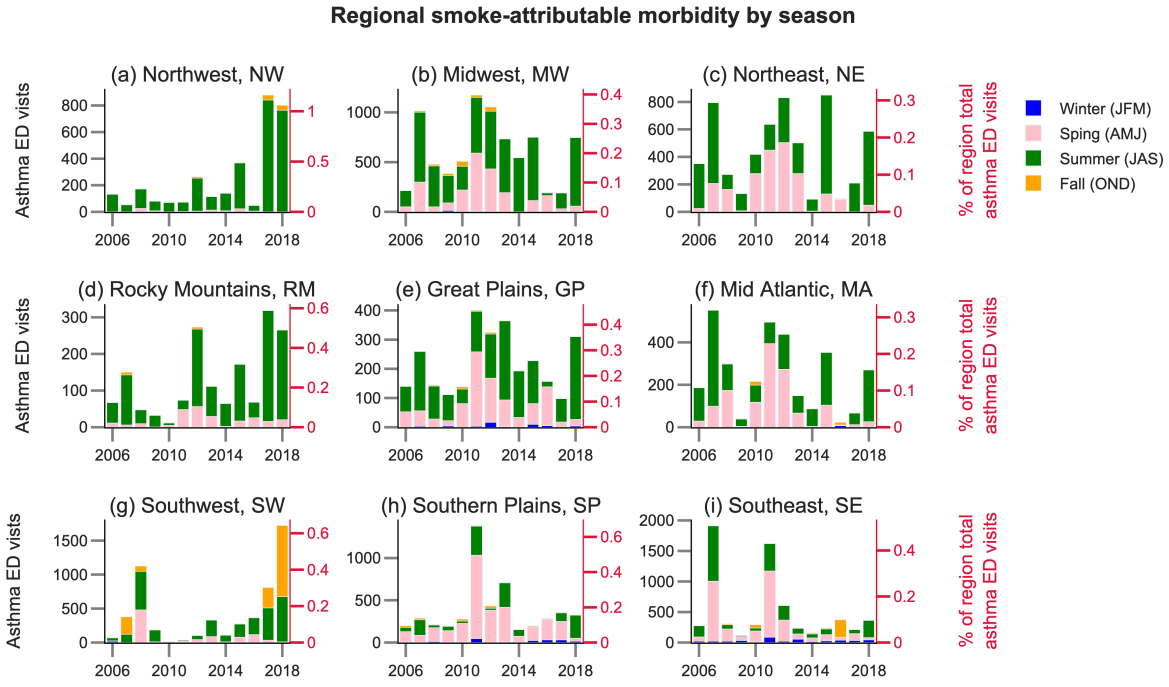

Figure 3.3: Asthma emergency department (ED) visits attributable to smoke $PM_{2.5}$ by each US region colored by season. The left y-axis represents the total number of asthma ED visits attributable to smoke $PM_{2.5}$ in each region and the right y-axis represents the percent of region total asthma ED visits attributable to smoke $PM_{2.5}$. Region names and abbreviations follow the same regions from the map in Figure 3.2.

The seasonality of asthma ED visits attributable to smoke $PM_{2.5}$ varies by US region. In the western regions shown in Figure 3.3a, d, and g, most asthma ED visits attributable to smoke $PM_{2.5}$ occur in the summer (see also Figure B.9). This is largely driven by the timing of large wildfires (e.g., Brey et al., 2018b; 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 2006-2018 occur in the spring (see Figure 3.3i and h). This partially aligns with observed timing of local landscape fires (Brenner, 1991; Brey et al., 2018a; Dennis et al., 2002; McCarty et al., 2009). However, there is additional prescribed and agricultural burning in these regions in other seasons (Brey et al., 2018a; McCarty et al., 2009; Dennis et al., 2002), which is generally missed in our smoke $PM_{2.5}$ method and hence is not reflected in the 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 Figure 3.3b, c, e, and f). Smoke in these regions is likely a combination of local landscape fires and transported smoke from other US regions and Canada (Brey et al., 2018a; DeBell et al., 2004; Le et al., 2014; Rogers et al., 2020; Wu et al., 2018).

The timing and intensity of morbidity attributable to smoke $PM_{2.5}$ across the US presented here from 2006-2018 is likely to change in the future due to climate-driven changes in fire regimes (Abatzoglou and Williams, 2016; Barbero et al., 2014; Goss et al., 2020; Spracklen et al., 2009; Williams et al., 2019), human-fire interactions (Balch et al., 2017; Kupfer et al., 2020), population, and land-management strategies (Ford et al., 2018). 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 (Abatzoglou and Williams, 2016; Barbero et al., 2014; Spracklen et al., 2009). Notably, in 2018 and more recently in the 2020 fire season (not included in our dataset), large fires

had extended, dramatic impacts on air quality in multiple large cities. For example, in Figure 3.3a and 3.3g, 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.

3.3.4 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 3.1. 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 2008-2012, 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 3.1, 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 3.4, we show the total annual number and percent of all-cause mortality attributable to total $PM_{2.5}$ (Figure 3.4a, c) and smoke $PM_{2.5}$ (Figure 3.4b, d) by US state. The fraction of all mortality attributable to total

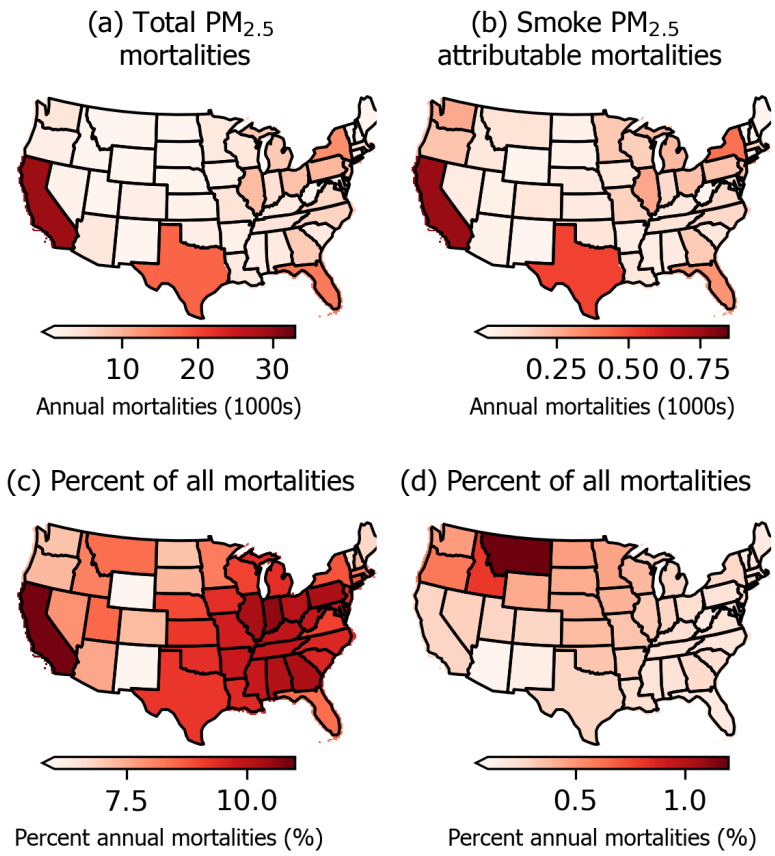

Figure 3.4: Total annual mortalities (in 1000s) attributable to (a) total $PM_{2.5}$ and (b) smoke $PM_{2.5}$ in each state. Panels c and d show the percent of all mortalities in each state attributable to total $PM_{2.5}$ and smoke $PM_{2.5}$, respectively.

$PM_{2.5}$ 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 B.2a). California has the highest 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 2006-2018 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 mortalities in eastern states with high population density, but lower long-term population-average smoke $PM_{2.5}$ exposure.

3.3.5 Smoke-Enhanced Hazardous Air Pollutants Chronic HIA

Figure 3.5 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 3.5, 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 3.5. Figure B.10 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 didn't have established DALY factors.

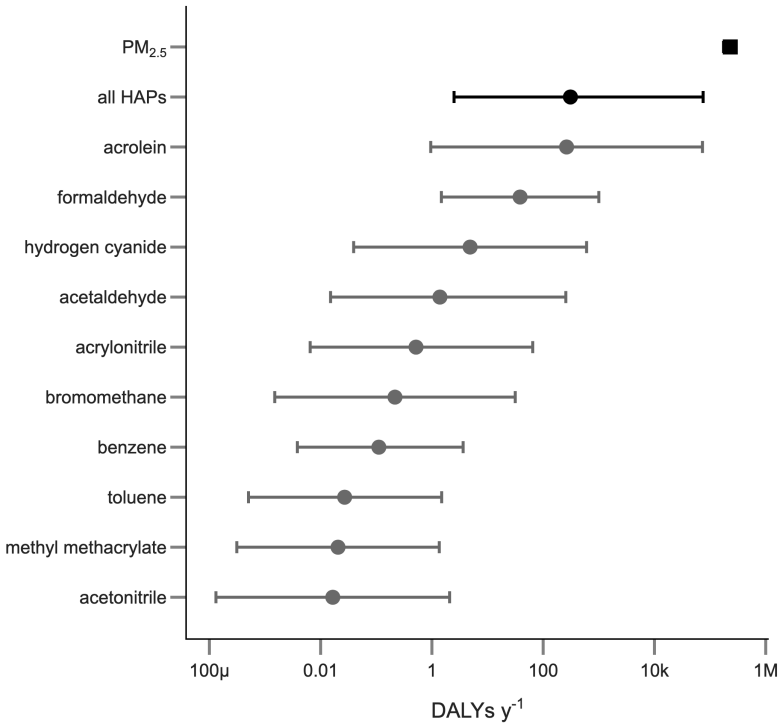

Figure 3.5: US-total disability adjusted life years (DALYs) attributable to smoke $PM_{2.5}$ and gas-phase smoke Hazardous Air Pollutants (HAPs) each year. $PM_{2.5}$ DALY error bars represent the range of DALYs estimated using $\pm$ 2 times the standard error for the coefficients in the GEMM health 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.

The relatively low number of DALYs attributable to smoke HAPs in Figure 3.5 is due, in part, to the low estimated HAPs concentrations. The 2006-2018 mean smoke-enhanced concentrations of acrolein (4.4 x 10^{-3} μg m^{-3}) and formaldehyde (4.2 x 10^{-2} μg m^{-3}) in the US estimated here are approximately 80-96% 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 B.11). 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 3.5 are representative of the high uncertainty in the DALYs per HAPs intake estimates, which are approximated from animal toxicology studies (Huijbregts et al., 2005). There is additional uncertainty, not represented in Figure 3.5, 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^{th} and 97.5^{th} 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 $PM_{2.5}$ 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 $PM_{2.5}$ are estimated from an epidemiologically based concentration response function, the smoke $PM_{2.5}$ DALY totals (and mortalities) presented here may already incorporate health impacts of compounds co-emitted with $PM_{2.5}$. Smoke $PM_{2.5}$ may also include particle-phase HAPs, such as polycyclic aromatic hydrocarbons (PAHs; Andreae, 2019), which may impact the overall toxicity of $PM_{2.5}$ mass concentrations in smoke.

3.3.6 Limitations

There are several limitations of our observation-based smoke $PM_{2.5}$ 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 et al., 2018b). 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-hour 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.

3.4 Conclusions

In the present work, we used an HIA as a tool to understand (1) the distribution of health events due to acute and chronic smoke exposure across US states and EPA regions, and (2) 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 $PM_{2.5}$ (Neumann et al., 2021; Fann et al., 2018; Ford et al., 2018) to conduct the first HIA of smoke with observation-based smoke $PM_{2.5}$,

sub-annual temporal resolution of 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 $PM_{2.5}$ continues to increase (Ford et al., 2018; Neumann et al., 2021; Li et al., 2020; Liu et al., 2016a; 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 wildfire 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. A greater understanding of the seasonality of these acute events by region may help states prepare for the potential increasing burden posed to the healthcare system by smoke. It will be important to understand how this seasonality may change by region in the future due to climate change and human influence on fire ignition and suppression.

3.5 Acknowledgements and Data Availability

Thank you to my co-authors on this paper: Kelsey Bilsback, Bonne Ford, Sheena E. Martenies, Sheryl Magzamen, Emily V. Fischer, and Jeffrey R. Pierce for their helpful feedback. 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. The kriged $PM_{2.5}$ data used in this analysis can be accessed at https://doi.org/10.25675/10217/230602. Python codes used for the analysis presented here and in the supplementary material are available at https://github.com/kaodell/smoke_HIA.

CHAPTER 4

INFLUENCE OF WILDFIRE SMOKE ON INDOOR AIR QUALITY IN SEVERAL WESTERN US CITIES

This work is in preparation for *GeoHealth*[1]

4.1 Introduction and Background

In the western United States (US), wildfires significantly degrade outdoor air quality (e.g., Brey et al., 2018b; Kaulfus et al., 2017; Buysse et al., 2019; Ford et al., 2018) and are a major contributor to primary fine particulate matter ($PM_{2.5}$) emissions (US EPA NEI). Population exposure to wildfire smoke has been associated with negative impacts on respiratory health (Reid et al., 2016a; Cascio, 2018; Liu et al., 2015, and references within). Due to natural and anthropogenic climate change and historical fire-suppression tactics, large wildfires have been increasing in frequency and burned area in the western US since the mid-1980s (Westerling et al., 2006; Westerling, 2016; Abatzoglou and Williams, 2016; Barbero et al., 2014; Marlon et al., 2012). These increases are projected to continue across the 21^{st} century in a warming and drying climate (Barbero et al., 2015; Spracklen et al., 2009; Brey et al., 2021; Pechony and Shindell, 2010). Increasing extreme $PM_{2.5}$ event intensity and summer-average $PM_{2.5}$ concentrations in the western US have been attributed to wildfires (McClure and Jaffe, 2018; O'Dell et al., 2019) and, due to increases in large wildfires, smoke events are projected to increase in frequency and intensity in the western US in the coming decades (Liu et al., 2016b).

Previous works on the air quality and health impacts of wildfire smoke have largely focused on the influence of smoke on ambient air quality. However, surveys suggest adults spend approximately 90% of their time indoors (Klepeis et al., 2001). Characterizing indoor air quality during wildland fire smoke events is an emerging issue (Xiang et al., 2021; Kirk et al., 2018; Shrestha et al., 2019; Henderson et al., 2005; Messier et al., 2019; Barn et al., 2008; Reisen et al., 2019; Stauffer et al., 2020; Wheeler et al., 2021; Kaduwela et al., 2019). Previous works have identified

[1] O'Dell, K., B. Ford, J. Burkhardt, J. Bayham, E. V. Fischer and J. R. Pierce (in prep), Influence of Wildfire Smoke on Indoor Air Quality in Several Western US Cities.

an influence of wildland fire smoke events on indoor particle concentrations in US residences (Xiang et al., 2021; Kirk et al., 2018; Shrestha et al., 2019; Henderson et al., 2005; Messier et al., 2019; May et al., 2021), schools (Kaduwela et al., 2019; May et al., 2021), and an office space (Stauffer et al., 2020). Case studies suggest indoor $PM_{2.5}$ concentrations are lower than outdoor concentrations during smoke events, but this is dependent on building features (windows open/closed, HVAC systems, etc.) and occupant activity (i.e., cooking) (Kirk et al., 2018; Shrestha et al., 2019; Henderson et al., 2005; Barn et al., 2008; Reisen et al., 2019; Xiang et al., 2021; Mott et al., 2002; May et al., 2021).

There is large heterogeneity in both smoke events and indoor environments potentially impacted by wildfire smoke events. To date, no study of US wildfire smoke influence on indoor air quality has analyzed indoor $PM_{2.5}$ concentrations across multiple western US cities during multiple extreme smoke events. As smoke $PM_{2.5}$ concentrations and subsequent health impacts are projected to increase in the future (Ford et al., 2018; Liu et al., 2016a; Neumann et al., 2021), it is crucial to quantify exposure to smoke indoors in multiple smoke-impacted areas. Such a characterization of indoor $PM_{2.5}$ concentrations during multiple smoke events across many indoor environments could help inform mitigation strategies in areas heavily impacted by wildland fire smoke.

While there are networks of regulatory monitors for ambient air quality, there are no regulatory monitoring networks for indoor air quality. The vast PurpleAir low-cost sensor monitoring network (https://www.purpleair.com/) provides a novel opportunity to evaluate the influence of smoke on indoor air quality over a significantly larger set of indoor environments than previous studies. The network provides real-time measurements of indoor or outdoor $PM_{2.5}$ concentrations from over 20,000 monitors across the globe. The Plantower low-cost optical PM sensors (PMS5003 and PMS1003) used in the PurpleAir network have been extensively evaluated against regulatory monitors and generally have high precision, but low accuracy, compared to more robust measurement techniques (Malings et al., 2020; Delp and Singer, 2020; Sayahi et al., 2019; Barkjohn et al., 2020; Tryner et al., 2020a; Holder et al., 2020; Mehadi et al., 2020). Thus, PurpleAir monitor

performance can often be improved with a simple correction factor (Malings et al., 2020; Delp and Singer, 2020; Barkjohn et al., 2020; Tryner et al., 2020a; Holder et al., 2020; LRAPA, 2018). For example, Barkjohn et al. (2020) found PurpleAir monitors across the US overestimate outdoor $PM_{2.5}$ concentrations by around 40%, but a correction factor reduces the overall root mean square error from 8 to 3 μg m^{-3}. There is heterogeneity in monitor performance due to variability in aerosol optical properties and atmospheric conditions (e.g., Tryner et al., 2020a,b; Sayahi et al., 2019), variable (potentially non-ideal) monitor placement by owners, lack of regular calibration and maintenance leading to issues such as baseline drift (e.g., Sayahi et al., 2019), etc. Overcoming these issues with data cleaning and correction factors for quantitative research applications of the PurpleAir observations is an active area of research.

The PurpleAir network has been used previously to study the relationship between indoor and outdoor air quality and the influence of smoke on air quality. Monitors in the PurpleAir network have been used in conjunction with regulatory monitors to capture outdoor air quality impacts of wildfire smoke (Gupta et al., 2018; Mallia et al., 2020; Bi et al., 2020). In addition, the network has recently been used to identify increases in indoor $PM_{2.5}$ during COVID-19 lockdowns (Mousavi and Wu, 2021), annual and diurnal patterns in relationships between indoor and outdoor $PM_{2.5}$ (Krebs et al., 2021), and outdoor $PM_{2.5}$ infiltration factors/penetration rates (Bi et al., 2021; Krebs et al., 2021). Indoor and outdoor PurpleAir monitors have also been used to evaluate low-cost filter effectiveness during an intense smoke event (May et al., 2021). It is currently unclear how the relationships between indoor and outdoor $PM_{2.5}$ concentrations during smoke events may differ from smoke-free periods over a large number of indoor environments. The PurpleAir low-cost sensor network has strong potential to further our currently limited understanding of the impacts of smoke on indoor air quality.

In this work, we use co-located (within 1000 m) indoor and outdoor $PM_{2.5}$ monitors from the PurpleAir network to identify the relationship between indoor and outdoor $PM_{2.5}$ during smoke-impacted periods and smoke-free periods in 2020. We focus our analysis on five smoke-impacted areas with high monitor density to characterize city-wide patterns of smoke influence on indoor

air across heterogeneity in indoor environments. We evaluate the census-tract level socioeconomic representation of co-located indoor and outdoor low-cost sensors in these regions. For each monitor in the five areas, we determine the change in daily-average indoor to outdoor $PM_{2.5}$ ratios on smoke-impacted days, relative to smoke-free days. Finally, we evaluate the influence of smoke-impacted outdoor air on indoor air quality as a function of the outdoor Air Quality Index (AQI). To our knowledge, this is the first multi-city study on the influence of smoke events and smoke-event intensity on indoor air quality, including an unprecedented number of indoor environments. The results presented here could help inform public guidance on exposure mitigation strategies during smoke events, which are likely to increase in the future (Liu et al., 2016b; Ford et al., 2018).

4.2 Methodology

4.2.1 PurpleAir Dataset Overview, Cleaning, and Scaling

For this work, we use low-cost (<$300) monitors available in the public PurpleAir monitor network. Sensor lists were downloaded from the PurpleAir API (https://api.purpleair.com/), and archived data was downloaded from the ThingSpeak API. The network consists of two main types of monitors: PA-II, designed for outdoor or indoor use, and PA-I-Indoor, designed for indoor use only. The PA-II monitor contains two Plantower PMS5003 sensors, while the PA-I-Indoor monitor contains one Plantower PMS1003 sensor (https://www.purpleair.com/). The two Plantower sensor models are nearly identical but have slightly different laser wavelengths (650 +/- 10 nm for PMS1003, and 680 +/- 10 nm for PMS5003) and air flow pathways within the sensor (Kelly et al., 2017; Sayahi et al., 2019). Plantower sensors estimate particle mass concentrations every 80 seconds by converting observations of particle number via light attenuation to particle mass concentrations. Plantower reports two mass concentration estimates using different conversion factors "cf_1" and "cf_atm" recommended for the factory environment and outdoor use, respectively, according to the Plantower manuals. Each PurpleAir monitor also measures current temperature and relative humidity. These data are available to download for public monitors on the PurpleAir webpage. Monitors are labelled as located indoors or outdoors by the user during the set-up process.

As of 13 April 2021, 6,081 indoor and 18,634 outdoor monitors from the PurpleAir network had data available for download (although not all are currently active).

From the PurpleAir monitors available, we identified pairs of indoor and outdoor monitors by first locating the nearest outdoor monitor to every indoor monitor. If the nearest outdoor monitor was greater than 1000 m away, the indoor monitor was removed from our analysis. With this criteria, we identified 5,069 monitor pairs (5,069 unique indoor monitors and 3,352 unique outdoor monitors), globally. A single outdoor monitor can be paired to multiple indoor monitors (i.e., it is the closest outdoor monitor to multiple indoor monitors). We downloaded 10-minute average observations from 2017 - 2020 for these monitor pairs from the PurpleAir network. We focus our analysis on 2020 in the contiguous western US (west of 100^o W, 3,206 monitor pairs), due to greater monitor availability and a high number of intense smoke events in several major western cities. We note the COVID-19 pandemic likely led to unique building occupancy and behavioral patterns in 2020 that may influence $PM_{2.5}$ concentrations (e.g., Mousavi and Wu, 2021), and we discuss potential implications of this on our analysis in the limitations section.

We follow suggested sensor performance guidelines from the Plantower manuals to clean data from the PurpleAir sensors. First, we remove monitors with a reported "cf_1" $PM_{2.5}$ concentration outside 0-500 μg m^{-3}, the sensor effective range reported by the Plantower manual, removing 82,755 (0.06%) of the 10-minute observations. For the PA-II and PA-II-SD monitors, which contain two Plantower PMS5003 sensors (labelled "A" and "B"), we filtered observations for agreement between the two sensors following the reasonable agreement reported by the Plantower manual : For "cf_1" $PM_{2.5}$ observations < 100 μg m^{-3}, we remove data where the absolute difference between A and B sensor-reported "cf_1" $PM_{2.5}$ is > 10 μg m^{-3}. For "cf_1" $PM_{2.5}$ observations > 100 μg m^{-3}, we remove data where the percent difference between A and B sensor-reported "cf_1" $PM_{2.5}$ is > 10%. This data cleaning reduced the total 10-minute $PM_{2.5}$ observations from the co-located monitor pairs by 3,427,987 (2.50% of original total). Overall, 2.55% of the original 137,434,817 observations were removed by the data cleaning, leaving 133,924,075 10-minute ob-

servations in total, or 44,203,374 paired observations (removing the observations where either the indoor or outdoor $PM_{2.5}$ observation is missing from monitor pairs).

Many previous studies have shown that PurpleAir monitors, and Plantower sensors in general, have high precision but low accuracy compared to Federal Reference Method monitors, and thus should have a correction factor applied for analysis. Because the PurpleAir monitors rely on light-scattering, which is sensitive to particle size, composition, and hygroscopicity, PurpleAir performance and correction factors vary for different particle sources (Singer and Delp, 2018; Tryner et al., 2020b) and ambient conditions (Sayahi et al., 2019; Malings et al., 2020; Tryner et al., 2020a; Barkjohn et al., 2020). Several correction factors are available directly on the PurpleAir monitor website. At the time of this writing, these include: "US EPA" (developed for outdoor monitors across the US; Barkjohn et al., 2020); "AQandU" (winter in Salt Lake City Utah; https://aqandu.org/); "LRAPA" (woodsmoke in Oregon; http://www.lrapa.org/); and "WOODSMOKE" (woodsmoke in Australia; Robinson, 2020). While there are several woodsmoke-specific correction factors (LRAPA, Robinson, 2020; Holder et al., 2020; Delp and Singer, 2020; Mehadi et al., 2020), we chose to adjust both indoor and outdoor data using the correction factor developed by Barkjohn et al. (2020).

The Barkjohn et al. (2020) correction model was developed by comparison of outdoor PA-II monitors to $PM_{2.5}$ monitors in the Environmental Protection Agency's Air Quality System, which follow a Federal Reference Method. The application of this model to indoor PurpleAir monitors is an extrapolation as the model has not yet been tested for PA-I-Indoor monitors, designed for indoor use, which contain a different Plantower sensor. We discuss this further in the limitations section. The Barkjohn et al. (2020) correction factor is:

$$PM_{2.5} = 0.524 \times PM_{cf\_1} - 0.0852 \times RH + 5.72, \tag{4.1}$$

where PM_{cf_1} is the average of the A and B sensor-reported $PM_{2.5}$ with the "cf_1" conversion factor, and RH is relative humidity in percent. The correction leads to $PM_{2.5}$ estimates similar to those produced by smoke-specific correction models at high concentrations (Holder et al., 2020;

Barkjohn et al., 2020). Finally, we calculated daily averages of the corrected $PM_{2.5}$ observations, removing times when less than 50% of the day (72 10-minute averages) reported $PM_{2.5}$ measurements. This removed nearly 2.6% of the daily, co-located $PM_{2.5}$ observations.

4.2.2 Identification of Smoke-Impacted Observations

We identified smoke-impacted time periods by combining satellite-based smoke-plume estimates with the PurpleAir surface $PM_{2.5}$ observations. The National Oceanic and Atmospheric Administration (NOAA) Hazard Mapping System (HMS) produces a smoke plume product that identifies locations where there is smoke somewhere in the atmospheric column based on the inspection of visible satellite imagery by trained satellite analysts (Ruminski et al., 2006; Rolph et al., 2009). HMS smoke plumes may miss dilute smoke, cannot determine the vertical distribution of smoke, and are limited only to daylight hours (Brey et al., 2018a; Rolph et al., 2009). Because HMS smoke plumes cannot distinguish the vertical extent of smoke plumes, co-located PurpleAir indoor and outdoor monitors were labeled as smoke-impacted when both of the following criteria were met: (1) an HMS smoke plume is identified at the monitor location, and (2) the daily outdoor $PM_{2.5}$ concentration is > 1 sigma above the monitor's 2020 annual-mean outdoor $PM_{2.5}$ concentration. We repeated our analysis using only HMS plumes to identify smoke-impacted observations and found that removing the $PM_{2.5}$ > 1 sigma requirement does not impact our main conclusions. Smoke-free observations were defined as all observations at monitors on days without an HMS smoke plume aloft. With this method, we identified 17,399 smoke-impacted monitor-days and 240,032 smoke-free monitor-days across year 2020 within the contiguous western US. The 2020 annual median smoke-impacted and smoke-free indoor and outdoor $PM_{2.5}$ concentrations are calculated for all monitor pairs.

4.2.3 Regional Analysis

We focus the remainder of our analysis on several regions with a sufficient number of indoor and outdoor monitor pairs and which experienced smoke-impacted air quality events in 2020. With this criteria, we selected the following regions: San Francisco, CA; Los Angeles, CA; Seattle, WA

and Portland, OR; Salt Lake City, UT; and the Colorado Front Range containing 2340, 184, 84, 63, and 39 indoor-outdoor monitor pairs reporting in 2020, respectively. Within each region, we identified the representation of socioeconomic status monitored by these co-located monitor pairs. However, we do not know whether the indoor monitors within each census tract are located in residential or commercial buildings. We counted the number of monitor pairs by census tract (based on the monitor location provided in the PurpleAir data download) and identified the social vulnerability of each census tract with the 2018 Social Vulnerability Index from the Center for Disease Control (CDC)/ Agency for Toxic Substances and Disease Registry (CDC/ATSDR, 2018). The CDC's Social Vulnerability Index is a national percentile ranking of each census tract by social vulnerability across multiple indicators; higher values indicate higher vulnerability. Distributions of monitors by Social Vulnerability Index were compared to the region's population distribution by Social Vulnerability Index.

In each region, we investigated the relationship between indoor and outdoor $PM_{2.5}$ concentrations on smoke-impacted and smoke-free days across the region at large and at individual monitor pairs within the region. To evaluate the relationship across the region at large, we grouped all daily indoor and outdoor observation pairs within each region by smoke-influence and smoke-intensity (denoted by outdoor $PM_{2.5}$ Air Quality Index, AQI). The $PM_{2.5}$ AQI bins, as defined by the EPA for 24-hour average $PM_{2.5}$ concentrations, are as follows, "good": $PM_{2.5} < 12$ μg m^{-3}, "moderate": 12 μg m^{-3} < $PM_{2.5} < 35$ μg m^{-3}, "unhealthy for sensitive groups": 35 μg m^{-3} < $PM_{2.5} <$ 50 μg m^{-3}, "unhealthy": 50 μg m^{-3} < $PM_{2.5} < 150$ μg m^{-3}, "very unhealthy": 150 μg m^{-3} < $PM_{2.5} < 250$ μg m^{-3}, and "hazardous": 250 μg m^{-3} < $PM_{2.5}$. In the outdoor distributions, we weighted outdoor observations by the number of indoor monitors assigned as its pair. Binning all monitors in each region allowed us to investigate potential regional differences in relative indoor and outdoor $PM_{2.5}$ concentrations across indoor environment heterogeneity within the region. We also evaluated the indoor and outdoor relationship at each individual monitor pair in the regions by calculating the ratio of daily-average indoor $PM_{2.5}$ concentrations to daily-average outdoor $PM_{2.5}$ concentrations (indoor/outdoor ratio).

4.3 Results

4.3.1 Indoor and Outdoor $PM_{2.5}$ Concentrations at Western US PurpleAir Monitors

The 2020 annual-median $PM_{2.5}$ concentrations on smoke-impacted and smoke-free days for co-located indoor and outdoor PurpleAir monitors in the western US are shown in Figure 4.1. A majority of the co-located monitor pairs with sufficient samples are located in California with additional clusters of monitors in the Colorado Front Range, Salt Lake City, and Seattle. By definition of the smoke-impacted days (HMS smoke plume aloft and outdoor $PM_{2.5}$ greater than 1 sigma above the annual mean), the outdoor $PM_{2.5}$ on smoke-free days is lower than outdoor $PM_{2.5}$ on smoke-impacted days, on average (Figure 4.1a and b). We also find the median indoor $PM_{2.5}$ is, on average, higher on smoke-impacted days compared to smoke-free days (Figure 4.1c and d). Across all monitors, median indoor $PM_{2.5}$ is, on average, 123% (interquartile range, IQR: 61% - 247%) or 5.9 μg m^{-3} (IQR: 2.7 - 13.0 μg m^{-3}) higher on smoke-impacted days. Thus, smoke events degrade indoor air quality across a large number of indoor environments in the western US.

Median indoor $PM_{2.5}$ concentrations on smoke-impacted days (Figure 4.1d) are lower than median outdoor $PM_{2.5}$ on smoke-impacted days (Figure 4.1b) at 470 of 480 monitors, which is not as strongly reflected on smoke-free days (only 286 monitors of 480 monitors showed lower indoor concentrations, Figures 4.1 a and c). For smoke-impacted days, median indoor $PM_{2.5}$ is 64% (IQR: 44% to 80%) lower than median outdoor $PM_{2.5}$ across the monitors. For smoke-free days, median indoor $PM_{2.5}$ is only 6.7% (IQR: -10%, 22%) lower than median outdoor $PM_{2.5}$. Further, at 40% of the monitor pairs, median indoor $PM_{2.5}$ is greater than median outdoor $PM_{2.5}$ on smoke-free days. Finally, we find a slightly higher correlation between daily-average indoor and outdoor $PM_{2.5}$ on smoke-impacted days (Spearman's r of 0.69) compared to smoke-free days (0.60). However, at nearly half (41%) of the monitors, there is a higher correlation between daily-average indoor and outdoor $PM_{2.5}$ on smoke-free days than smoke-impacted days, Figure C.1.

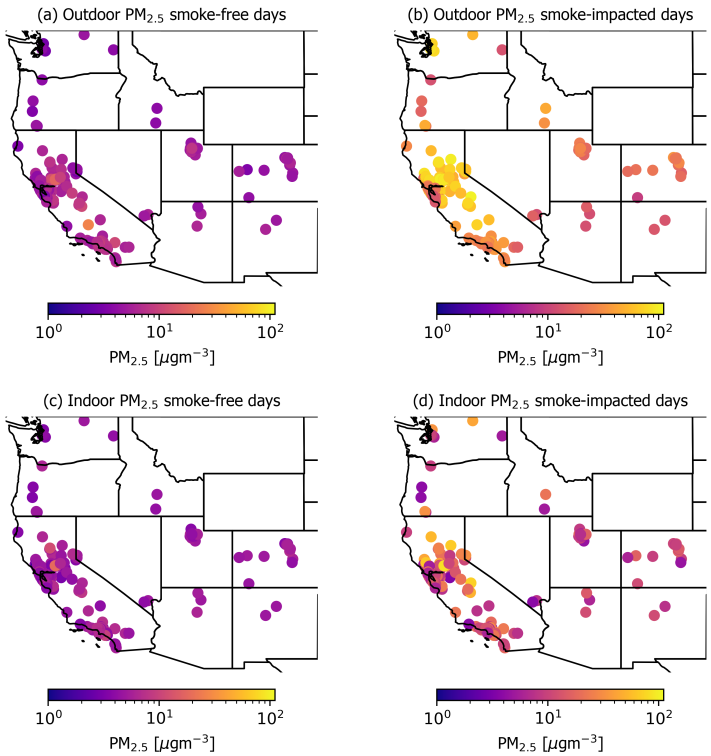

Figure 4.1: 2020 median daily-average $PM_{2.5}$ on smoke-impacted and smoke-free days at co-located (d < 1000 m) outdoor (panels a and b) and indoor (panels c and d) PurpleAir monitors. Monitor pairs are excluded from the figure if they contain fewer than 10 smoke-impacted or smoke-free daily-average $PM_{2.5}$ observations in 2020.

4.3.2 Influence of Wildfire Smoke on Indoor $PM_{2.5}$ Concentrations Across Several Western US Cities

In Figure 4.2, we show a map of co-located PurpleAir monitors in each region and the representation of the co-located monitor locations in terms of Social Vulnerability by census tract. Figures 4.2a-d show the monitors selected to represent each region of interest. A large number of monitor pairs shown in Figure 4.2 were installed directly after smoke events occurred in the region, and hence not every point in Figure 4.2 provides data during smoky time periods. A large increase in installed PurpleAir monitors also occurred after severe smoke events in California in 2018 and 2019 (Delp and Singer, 2020; Krebs et al., 2021). Figure 4.2f shows the cumulative distribution of the population and number of co-located PurpleAir monitor pairs for each region and the full US by the CDC's Social Vulnerability Index (higher values indicate higher vulnerability). Across all

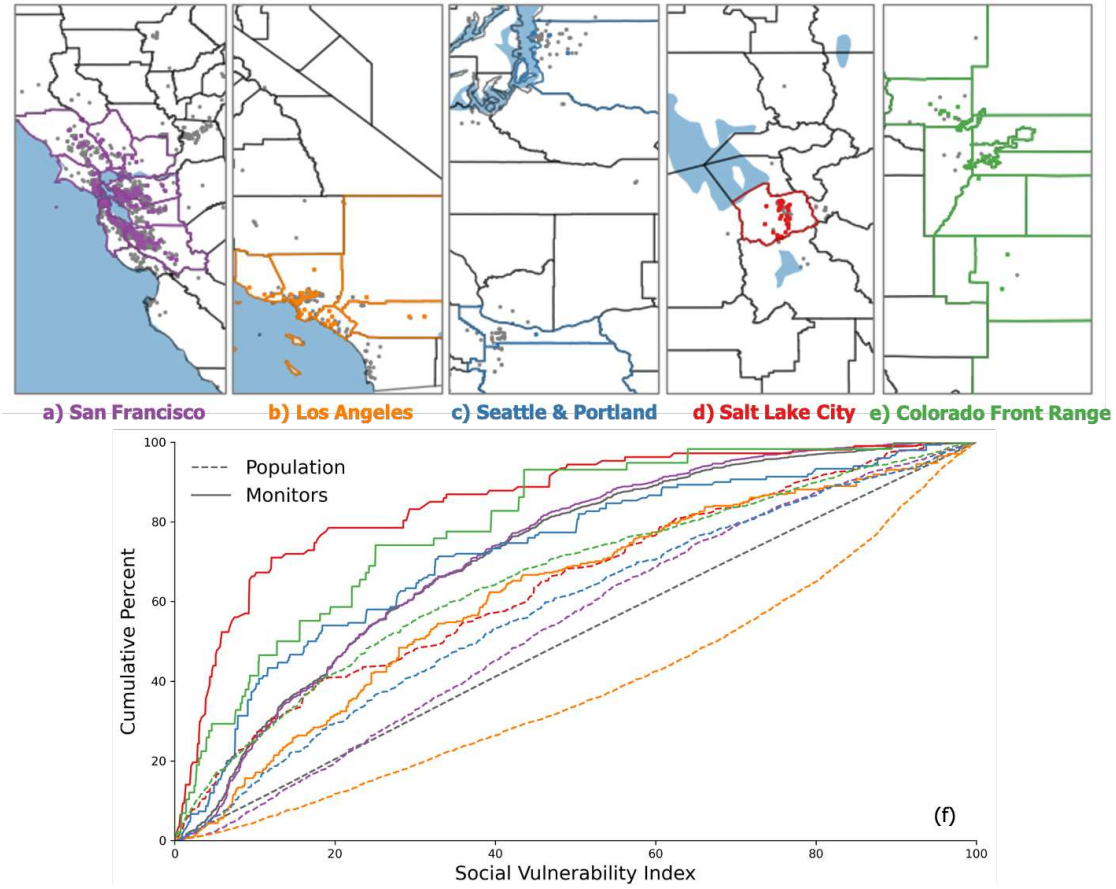

Figure 4.2: Maps of co-located PurpleAir monitors in San Francisco (panel a), Los Angeles (panel b), Seattle and Portland (panel c), Salt Lake City (panel d), and the Colorado Front Range (panel e). Colored county lines denote counties included in each area. Colored monitors indicate monitors within the selected counties that have sufficient data for individual monitor analysis (at least 10 smoke-impacted and smoke-free days). Panel f shows the normalized cumulative distribution function of population and the number of co-located (d<1000m) indoor and outdoor PurpleAir monitor pairs by Social Vulnerability Index from the CDC for each region (same colors as top panels) and the US at large (in gray). Higher index values indicate higher vulnerability.

regions, and the US at large, there is a higher number of co-located monitors in census tracts of lower social vulnerability compared to the population in those census tracts. Thus, those of higher social vulnerability, which are typically exposed to higher levels of outdoor air pollution (Hajat et al., 2015), are underrepresented by these co-located PurpleAir monitors. This is an important limitation of the present work and the PurpleAir network in general.

Figures 4.3a and b show the indoor and outdoor distribution of daily $PM_{2.5}$ concentrations for smoke-free observations and smoke-impacted observations, respectively, for each region. Indoor

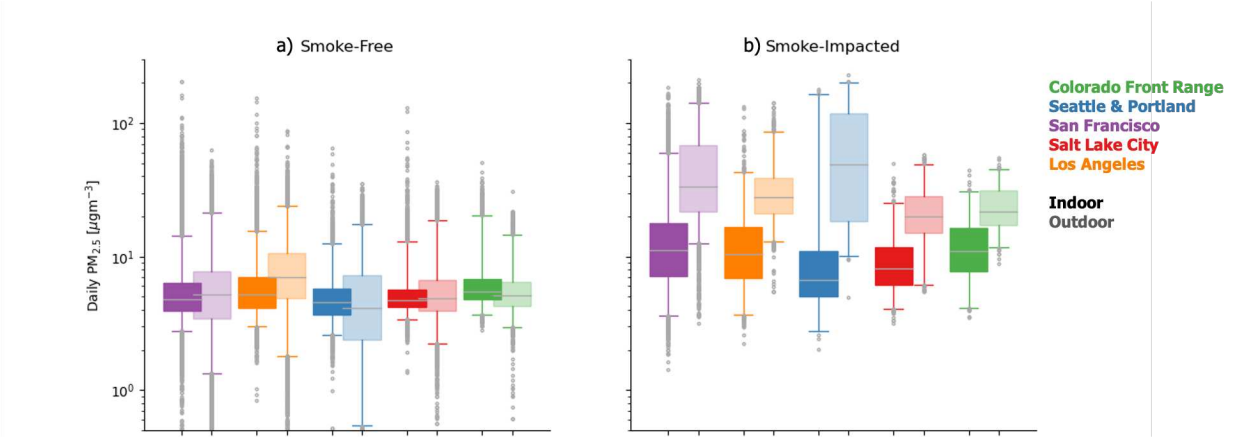

Figure 4.3: Distributions of indoor (darker boxes) and outdoor (lighter boxes) daily-average $PM_{2.5}$ for smoke-free (panel a) and smoke-impacted (panel b) observations in each region. Lines across each box indicate the median value, whiskers extend from the 2.5^{th} to the 97.5^{th} percentile, and values outside this percentile range are shown as gray points. The y-axis is truncated at 0.5 μg m^{-3}.

and outdoor concentrations in smoke-free and smoke-impacted conditions occasionally exceed 100 μg m^{-3} which may be due to indoor pollution events, highly localized outdoor sources, dense smoke plumes, or monitor malfunction that was not removed during data cleaning. The median and IQR for indoor $PM_{2.5}$ during smoke-free conditions across the five regions is remarkably similar, where the median indoor $PM_{2.5}$ concentrations for San Francisco, Los Angeles, Seattle and Portland, Salt Lake City, and the Colorado Front Range are 4.82 μg m^{-3}, 5.23 μg m^{-3}, 4.52 μg m^{-3}, 4.70 μg m^{-3}, and 5.49 μg m^{-3}, respectively. Outdoor concentrations show slightly more variability. Across the four regions, Los Angeles has the highest median daily outdoor $PM_{2.5}$ concentration across all area monitors (7.04 μg m^{-3}), while the Seattle and Portland area has the lowest concentration (4.10 μg m^{-3}). Across all regions, daily indoor $PM_{2.5}$ is similar to outdoor $PM_{2.5}$ on smoke-free days. There is a notable difference for the Los Angeles area monitors, which show the largest shift between the distributions of indoor and outdoor $PM_{2.5}$ on smoke-free days, where the indoor $PM_{2.5}$ is lower. In contrast, there is a smaller shift in the distribution of indoor and outdoor $PM_{2.5}$ on smoke-free days in Seattle and Portland and the Colorado Front Range, where the indoor $PM_{2.5}$ is slightly higher.

On smoke-impacted days, there is a clear difference between the distributions of indoor and outdoor $PM_{2.5}$ in each region, where the outdoor $PM_{2.5}$ distribution is shifted towards higher con-

centrations. Median smoke-impacted indoor $PM_{2.5}$ concentrations in the five regions are 49-86% lower than median smoke-impacted outdoor $PM_{2.5}$ concentrations. The distributions of smoke-impacted daily7 indoor $PM_{2.5}$ are again similar across the five regions. There are regional differences in daily outdoor $PM_{2.5}$ on smoke-impacted days. Median daily outdoor $PM_{2.5}$ is highest in the Seattle and Portland area (48.80 μg m^{-3}), followed by San Francisco (33.70 μg m^{-3}) and Los Angeles (27.87 μg m^{-3}). Although the Seattle and Portland area has the highest outdoor $PM_{2.5}$, it has the lowest indoor $PM_{2.5}$ amongst the regions. However, of the regions in Figure 4.3, the Seattle and Portland area has the fewest $PM_{2.5}$ observations. In all five regions, indoor $PM_{2.5}$ is generally lower than outdoor $PM_{2.5}$ on smoke-impacted days.

Comparing Figures 4.3a and b shows that the area-wide distributions of both indoor and outdoor $PM_{2.5}$ concentrations are shifted towards higher concentrations on smoke-impacted days, compared to smoke-free days. Area-median indoor $PM_{2.5}$ concentrations are 32-57% higher on smoke-impacted days compared to smoke-free days across the regions. However, relative indoor and outdoor $PM_{2.5}$ concentrations during smoke events can vary significantly across different indoor environments (Kirk et al., 2018; Henderson et al., 2005; Shrestha et al., 2019). We show the median ratio of daily-average indoor to outdoor $PM_{2.5}$ for smoke-impacted and smoke-free observations at each monitor in the five regions in Figure 4.4. Black dashed lines across the figure indicate where the smoke-impacted and smoke-free ratios are equal to 1. Smoke-impacted ratios for nearly all monitors (364 of 371) in the five areas lie below the 1 line, indicating that for these indoor environments, indoor $PM_{2.5}$ is, in general, lower than outdoor $PM_{2.5}$ on smoke-impacted days. Monitors are more evenly distributed about the 1 line for smoke-free observations, where 54% of monitors have a smoke-free indoor/outdoor ratio <1. In Figures C.2-C.6, we show two-dimensional histograms of hourly indoor $PM_{2.5}$ versus hourly outdoor $PM_{2.5}$ for smoke-impacted and smoke-free observations in each city. The figures, in agreement with Figures 4.3 and 4.4, show that on smoke-impacted days, indoor $PM_{2.5}$ concentrations are predominantly lower than outdoor $PM_{2.5}$, while on smoke-free days, the indoor and outdoor $PM_{2.5}$ concentrations are often of a similar magnitude.

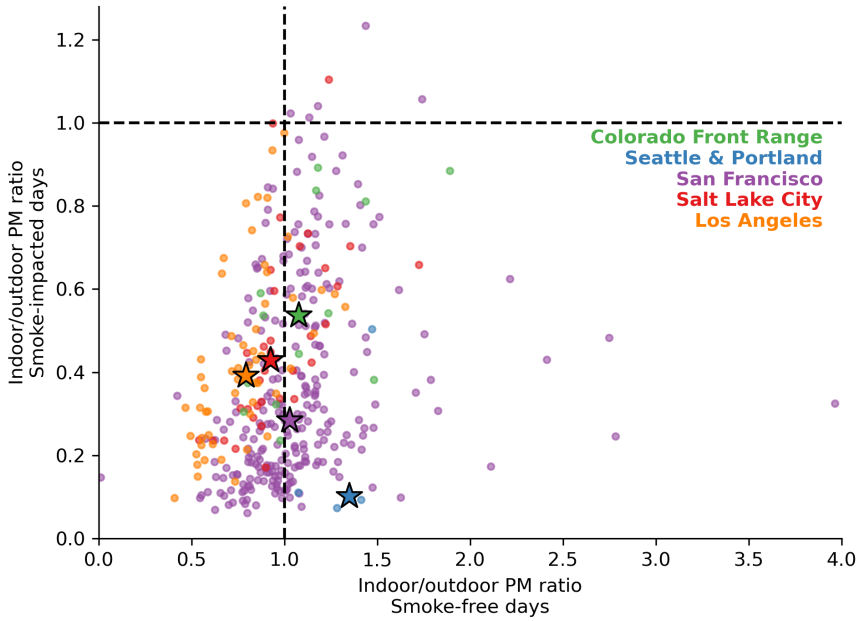

Figure 4.4: Median ratios of daily-average indoor to outdoor $PM_{2.5}$ on smoke-impacted and smoke-free days. Dashed lines indicate where ratios are equal to 1. Monitors are colored by location. Stars represent the region-median ratio of indoor to outdoor $PM_{2.5}$ on smoke-impacted and smoke-free days. The x-axis is truncated above an indoor/outdoor ratio of 4 for clarity. There are two points off the scale of this graph, both monitors in San Francisco, at (7.57, 1.37) and (5.28, 0.54).

The indoor/outdoor ratios on smoke-free days show some patterns by city. The monitors in the Bay Area are evenly distributed about the 1 line for smoke-free indoor/outdoor ratios. In contrast, a majority of monitors in Los Angeles (88%) and Salt Lake City (66%) have indoor/outdoor ratios < 1 on all days. Finally, although there are only a few monitors in Seattle, they all report relatively low indoor to outdoor ratios on smoke-impacted days (<0.51). Regional differences may also be driven by differences in outdoor, rather than indoor $PM_{2.5}$ concentrations in these areas, as indicated by the higher intra-regional variability in outdoor $PM_{2.5}$ compared to indoor $PM_{2.5}$ in Figure 4.3. However, without knowledge of occupant behavior and the type of indoor environment in which these monitors are located, both of which impact indoor $PM_{2.5}$ concentrations, it is challenging to distinguish regional differences. It is especially challenging in locations with few monitor pairs, like Seattle and Portland and the Colorado Front Range. Although it is difficult to establish regional differences in indoor/outdoor ratios, nearly all indoor environments in these regions have an average indoor/outdoor ratio < 1 on smoke-impacted days.

4.3.3 Relationship Between Indoor and Outdoor $PM_{2.5}$ as a Function of Smoke Intensity

We explore the relationship between indoor $PM_{2.5}$ concentrations and indoor/outdoor ratios as a function of outdoor $PM_{2.5}$ for summer and fall smoke-impacted observations in Figure 4.5. Figure 4.5 panels a-e show daily-average outdoor $PM_{2.5}$ concentrations binned and colored according to the $PM_{2.5}$ levels associated with the EPA's AQI levels. The EPA's AQI is widely used across the US for public communication on air quality and mitigation strategies during pollution episodes, including recommendations to remain indoors. PurpleAir monitors have been found to show a linear response to $PM_{2.5}$ up to around 200 μg m^{-3}, and thus are likely reliable for $PM_{2.5}$ AQI levels up to the 150-250 μg m^{-3} bin (Mehadi et al., 2020; Holder et al., 2020). In San Francisco, there were 236 monitor days with daily-average $PM_{2.5}$ concentrations associated with the "very unhealthy" (150 μg m^{-3} < $PM_{2.5}$ < 250 μg m^{-3}) AQI. These occurred at 277 unique monitor pairs on 09/10, 09/11, 10/01, and 10/02. In the Seattle and Portland area, there were 13 monitor days with daily-average $PM_{2.5}$ concentrations associated with the "very unhealthy" AQI, occurring between 9/11 and 9/17 at 7 unique monitor pairs. Across all regions, the majority of the smoke-impacted outdoor $PM_{2.5}$ concentrations are in the 12-35 μg m^{-3} range. The indoor $PM_{2.5}$ concentrations generally increase as the outdoor $PM_{2.5}$ concentrations increase across each AQI bin in each region. The only exception is for the <12, 12-35, and 35-55 μg m^{-3} outdoor $PM_{2.5}$ bins in Seattle and Portland, where there is no distinct change in the indoor $PM_{2.5}$ distributions. On average, median indoor $PM_{2.5}$ increases by 25% per AQI bin across the regions. The daily-average indoor $PM_{2.5}$ concentrations rarely reach the same AQI level as the outdoor $PM_{2.5}$, especially for outdoor $PM_{2.5}$ AQI levels above the 12-35 μg m^{-3} bin. Above this "moderate" $PM_{2.5}$ level, only 7.6% of indoor $PM_{2.5}$ concentrations are at or above the outdoor $PM_{2.5}$ AQI level. However, the daily-average indoor $PM_{2.5}$ concentrations are frequently elevated above the "healthy" (< 12 μg m^{-3}) AQI level for outdoor AQI above the "moderate" (12-35 μg m^{-3}) level.

Figure 4.5 panels f-j show the ratio of daily-average indoor to outdoor $PM_{2.5}$ on smoke-impacted days, binned by outdoor $PM_{2.5}$ AQI. Although the indoor $PM_{2.5}$ increases across each AQI bin, the ratio of indoor to outdoor $PM_{2.5}$ generally decreases. On average, the median in-

door/outdoor ratio across the regions decreases by 28% per bin. As shown in Figure 4.5f-j, the absolute decrease in the indoor/outdoor ratio is larger for the lower AQI bins, compared to the higher AQI bins in Los Angeles, San Francisco, and the Colorado Front Range. However, in the Seattle and Portland area (up to the 55-150 μg m^{-3} bin) and Salt Lake City, the decrease across AQI bins appears more linear (on the logarithmic scale). For all AQI bins above the <12 μg m^{-3} bin, the indoor/outdoor ratio is < 1 for 98% of the observations. Thus, although the indoor $PM_{2.5}$ increases as outdoor $PM_{2.5}$ increases on smoke-impacted days, the absolute increase in the indoor $PM_{2.5}$ is smaller than the increase in outdoor $PM_{2.5}$. Further, the relatively smaller increase in indoor $PM_{2.5}$ does not typically result in indoor $PM_{2.5}$ concentrations at the same AQI level as the outdoor concentrations but can result in indoor $PM_{2.5}$ concentrations above the "healthy" AQI level.

4.3.4 Limitations

Our analysis on the influence of smoke on indoor air quality is limited by a lack of detail on indoor environment characteristics and occupant behavior. There are multiple potential confounding variables that we are unable to properly control for when using monitors in the PurpleAir network. These confounding variables include characteristics of the indoor environment such as home age, home type, whether the building is rented or owned, stove type, level of air filtration, location of monitor, among other factors, which may impact indoor air quality (Shrestha et al., 2019; Allen et al., 2003) observed in this study. In addition, we do not have a record of occupant behavior such as cooking times, opening windows, etc., which may have also impacted indoor air quality (e.g., Farmer et al., 2019; Patel et al., 2020). In addition, due to the COVID-19 pandemic, indoor behavior in 2020 may differ from a typical year. The stay-at-home orders and increased remote work likely led to higher-than-normal occupancy of residential spaces and lower-than-normal occupancy of non-residential spaces. There were observed 17-24% increases in indoor $PM_{2.5}$ during the March 2020 COVID-19 lockdowns for PurpleAir monitors in California (Mousavi and Wu, 2021). However, these values were found to return to normal levels post-lockdown (Mousavi and Wu, 2021). Because we are unaware of the type of indoor environment (e.g. office space, res-

idential building, etc.) and the abnormal work and life circumstances created by the COVID-19 pandemic, it is challenging to control for these confounding variables through other indicators such as weekend/weekday variables. Despite this lack of detail on the indoor environment and unique circumstances created by the COVID-19 pandemic, we find a consistent impact of smoke on indoor air quality.

There are likely biases present in the indoor environments monitored in the PurpleAir network, which may impact this work. Individuals who purchase PurpleAir monitors for their personal residences are likely invested in the air quality in their home and may be likely to take extra steps to improve their indoor air quality compared to the general public. In addition, community groups, government agencies, and scientists also purchase and deploy PurpleAir monitors. Placement of these monitors may also add a bias depending on project goals. Lastly, as shown in Figure 4.2, census tracts of higher social vulnerability are not well represented in the PurpleAir indoor and outdoor monitor pairs used here. These areas can be subject to higher levels of outdoor air pollution (Hajat et al., 2015).

Regional differences in indoor/outdoor ratios observed here may be related to differences in infrastructure, socioeconomic status, and/or meteorology. We did not explore these variables directly in the present work. In the conclusions chapter, we discuss a potential future analysis to investigate the relationship between these variables and PurpleAir indoor/outdoor $PM_{2.5}$ ratios.

There are additional challenges with measuring $PM_{2.5}$ concentrations with the PurpleAir network and low-cost sensors. PurpleAir monitors are often purchased and installed by citizens at their homes. This may impact data quality as the monitors are not calibrated in the local environment and may not be located in ideal sampling locations. Data availability may also be inhibited as the PA-II and PA-I-Indoor monitors store data through a wireless internet connection, which may be unstable. Because PurpleAir monitors rely on light-scattering, reported $PM_{2.5}$ mass concentrations are sensitive to particle size, composition, and hygroscopicity. Therefore, PurpleAir performance can change by aerosol source type (Tryner et al., 2020a; Singer and Delp, 2018) and the ambient environment (Tryner et al., 2020b; Sayahi et al., 2019), which can differ indoors

and outdoors. Mehadi et al. (2020) found PurpleAir performance is also sensitive to woodsmoke composition. The Barkjohn et al. (2020) correction factor used here was developed for all-source outdoor $PM_{2.5}$ observations across the US and agrees with woodsmoke correction factors at high concentrations (Holder et al., 2020). There has been less validation of sensors in an indoor environment, but Delp and Singer (2020) found a factor of 2 overprediction for an indoor sensor during smoke events, consistent with the typical offset found for outdoor monitors. We repeated our analysis using the indoor-developed scaling from Delp and Singer (2020) for indoor monitors and with the LRAPA correction factor, developed in an area impacted by woodsmoke from home heating in winter, and found lower indoor concentrations by AQI bin and a smaller decrease (or no decrease) in the indoor/outdoor ratios with increasing outdoor AQI bins. These correction factors also resulted in lower indoor/outdoor ratios on smoke-free days. However, our main conclusions do not change. At present, these limitations are inherent with any use of the indoor $PM_{2.5}$ observations from PurpleAir.

4.4 Discussion and Implications

The present study has expanded upon previous works on the influence of smoke on indoor air quality through use of a large network of low-cost indoor and outdoor $PM_{2.5}$ monitors. We find indoor $PM_{2.5}$ concentrations are elevated on smoke-impacted days, indicating an influence of smoke on indoor air quality in many indoor environments. Further, indoor $PM_{2.5}$ concentrations are typically lower than outdoor concentrations on smoke-impacted days, which is less typical of non-smoke impacted days. Previous studies in the US have similarly reported higher indoor $PM_{2.5}$ concentrations during smoke events relative to non-smoke-impacted periods (Henderson et al., 2005; Kirk et al., 2018; Shrestha et al., 2019). With a larger dataset, we were able to identify how the relationship between indoor and outdoor $PM_{2.5}$ changes as a function of smoke intensity across multiple indoor environments and regions. We show as outdoor $PM_{2.5}$ concentrations increase on smoke-impacted days, indoor $PM_{2.5}$ concentrations also increase, but the ratio of indoor to outdoor $PM_{2.5}$ decreases. This is in agreement with Shrestha et al. (2019), who showed that indoor $PM_{2.5}$ number concentrations increased in low-income Denver homes across the "low," "medium," and

"high," concentration classifications of HMS plumes. In contrast, Wheeler et al. (2021) found, "unexpectedly," a higher infiltration of $PM_{2.5}$ during low smoke periods compared to high smoke periods in a local library during the Australian bushfires of 2019. Although we did not remove indoor-generated $PM_{2.5}$ events from our ratio calculation, as was done in Wheeler et al. (2021) and Shrestha et al. (2019), our reported decrease in indoor/outdoor $PM_{2.5}$ ratios across AQI bins indicate a relative decrease in infiltration (or increase in air filter use) for denser smoke plumes.

Our work shows the utility of a large low-cost sensor network in understanding the impact of wildfire smoke, a projected increasing source of US $PM_{2.5}$ (Ford et al., 2018; Yue et al., 2013; Li et al., 2020), on indoor air quality. The PurpleAir monitor network has grown substantially over the past several years. As we note here for 2020 and has been reported in previous studies for 2018 and 2019, the number of PurpleAir monitors in several western US cities increases significantly after a large smoke event (Delp and Singer, 2020; Krebs et al., 2021). This result not only indicates citizens and local actors are aware of the impact of smoke on air quality, but care to monitor their exposure during pollution events. Low-cost sensors, such as the PurpleAir monitors, could be deployed in future citizen-science programs where indoor air could be monitored in a large number of homes for a longer period of time with a record of building characteristics and occupant activity. Such a study would combine the advantages of this work (large numbers of indoor environments and extended time periods) with previous short-term studies that had more information on indoor environment characteristics. Future work with PurpleAir monitoring of indoor air quality should focus on a greater representation of low income, high vulnerability locations, which are currently underrepresented in co-located indoor and outdoor PurpleAir monitors in smoke-impacted western US cities and the US at large.

As smoke $PM_{2.5}$ is predicted to increase in the future in the western US (Yue et al., 2013; Ford et al., 2018; Li et al., 2020; Neumann et al., 2021; Liu et al., 2016a), it will be important to ensure indoor $PM_{2.5}$ concentrations do not increase beyond healthy levels during smoke events when the public is advised to remain indoors. The relationships between indoor and outdoor $PM_{2.5}$ concentrations presented here show that for many indoor environments, remaining indoors is currently an

effective, but limited, strategy to reduce exposure during smoke events. As shown in Figure 4.5 a-e, indoor concentrations are not always at "healthy" AQI levels during smoke events and continue to increase as outdoor $PM_{2.5}$ concentrations increase. We show this was the case across many indoor environments in multiple western US cities during 2020 smoke events. Therefore, additional actions to reduce exposure in these spaces may be needed beyond simply remaining indoors on smoke-impacted days. Previous studies of indoor air during smoke events have found filters can further reduce indoor $PM_{2.5}$ concentrations during smoke events (Barn et al., 2008; Henderson et al., 2005; Kirk et al., 2018; Shrestha et al., 2019; Xiang et al., 2021) and subsequent health impacts (Fisk and Chan, 2017; Mott et al., 2002).

4.5 Acknowledgements and Data Availability

Thank you to my co-authors on this paper: Jude Bayham, Jesse Burkhardt, Bonne Ford, Emily V. Fischer, and Jeffrey R. Pierce for their helpful feedback. 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. Python codes used for the analysis presented here and in the supplementary material will be made available at https://github.com/kaodell/purple_air_smoke.

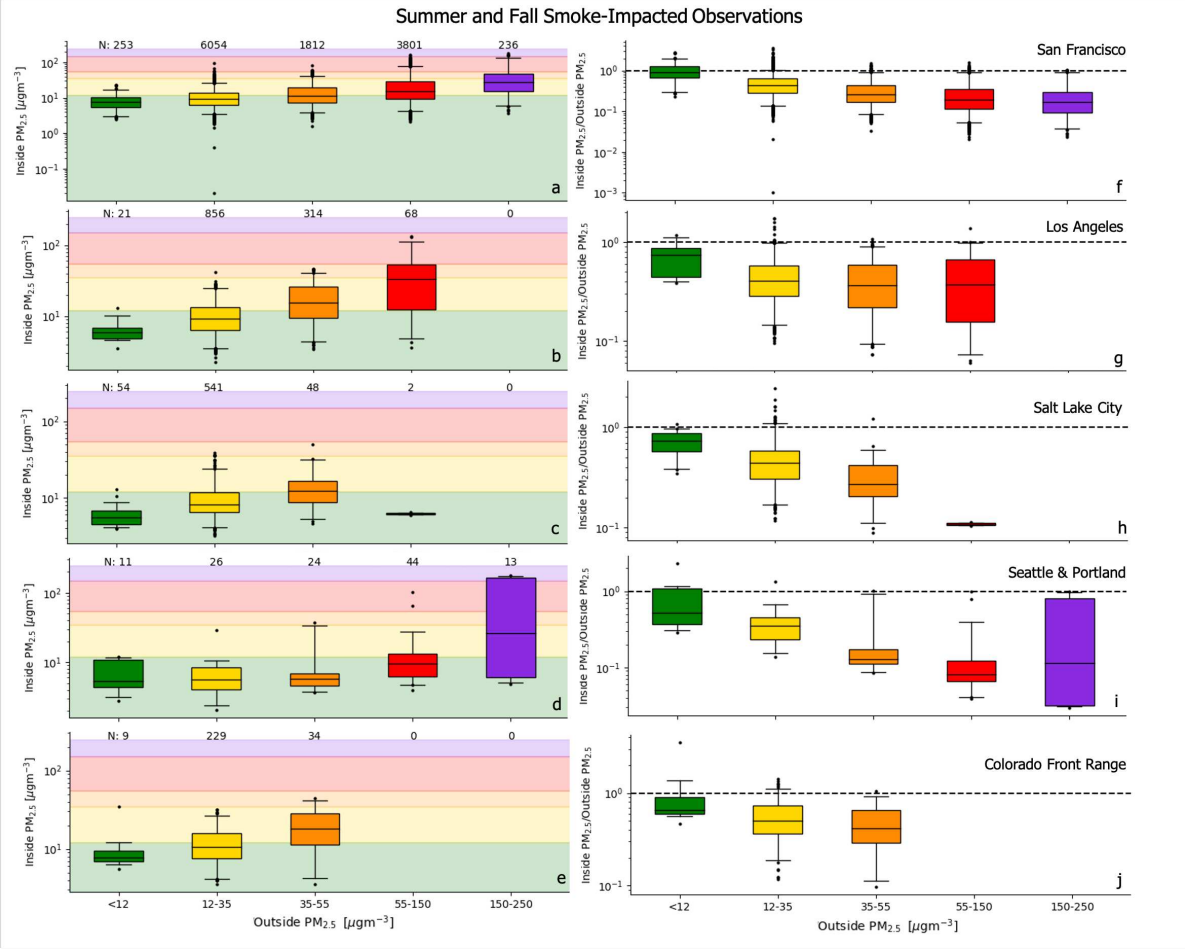

Figure 4.5: Daily-average indoor $PM_{2.5}$ concentrations (panels a-e) and ratios of daily-average indoor to daily-average outdoor $PM_{2.5}$ (panels f-j) as a function of Air Quality Index (AQI) level associated with the outdoor $PM_{2.5}$ concentrations for each of the five regions. Colors of boxes indicate outdoor $PM_{2.5}$ AQI level. Background colors on the left panels indicate AQI level associated with the indoor $PM_{2.5}$ concentrations. Median values are indicated as a black line across each box, whiskers extend from the 2.5^{th} to 97.5^{th} percentiles, and values outside this range are shown as black points. The black dashed line in panels f-j indicates where the indoor/outdoor ratio is equal to 1.

CHAPTER 5

SUMMARY, IMPLICATIONS, AND FUTURE WORK

5.1 Summary and Implications

This dissertation presents a quantitative assessment of the influence of US landscape fire smoke on multi-pollutant exposure, health impacts, and indoor air quality. A greater understanding of US smoke exposure, to which this work contributes, can support individuals and local decision makers with the necessary knowledge to prepare for future smoke events. This final chapter summarizes the key findings of this dissertation within the context of this goal and presents recommendations for future work.

In Chapter 2, we applied a risk-assessment to identify health-relevant gas-phase HAPs in western US wildfire smoke. We found the ratios between acute, chronic noncancer, and chronic cancer HAPs health risk and PM in smoke decrease as a function of smoke age by up to 72% from fresh (<1 day of aging) to old (>3 days of aging) smoke. Acrolein, formaldehyde, benzene, and hydrogen cyanide are the dominant contributors to this health risk of gas-phase HAPs in smoke plumes. We used ratios of HAPs to PM along with annual average smoke-specific PM to show if heavy smoke years like 2018 become the norm, smoke HAPs may pose a modest excess cancer risk. This work suggests fresh smoke plumes contain more toxic gas-phase HAPs than aged smoke plumes; however, the relationship between smoke age and toxicity remains an open question (e.g., Magzamen et al., 2021). During smoke events, HAPs exposure could be monitored with ground-based observations of the key health-relevant smoke HAPs identified in this work.

In Chapter 3, we applied the HAPs to PM ratios, developed in Chapter 2, in a national health impact assessment of exposure to landscape fire smoke and estimated DALYs from both smoke $PM_{2.5}$ and smoke HAPs. This work built upon the initial health assessment of gas-phase HAPs in Chapter 2, quantifying health impacts of HAPs on the same scale as smoke $PM_{2.5}$. We found DALYs attributable to HAPs in smoke are approximately three orders of magnitude lower than DALYs associated with smoke $PM_{2.5}$. However, there is high uncertainty in the DALYs attributable to HAPs largely due to uncertainty in HAPs health impacts in humans. Further, although the majority of

large landscape fires occur in the western US, the majority of mortality (74%) and morbidity (on average 75% across 2006-2018) attributable to smoke $PM_{2.5}$ occurs outside the West, an effect largely driven by higher population density in the East. Across the US, smoke-attributable morbidity predominantly occurs in spring and summer. These results indicate awareness of landscape fire smoke exposure is important across the US, not just in regions in proximity to large wildfires. In addition, the regional seasonality of the health impacts of smoke presented in this work can guide local messaging and preparedness for future smoke events.

In Chapter 4, we analyzed data from a network of indoor and outdoor low-cost $PM_{2.5}$ monitors to quantify the influence of western wildfire smoke events on indoor air quality. We found indoor $PM_{2.5}$ concentrations increased during smoke-impacted periods as outdoor $PM_{2.5}$ increased. While indoor $PM_{2.5}$ concentrations often remained below outdoor $PM_{2.5}$ concentrations during smoke events, indoor $PM_{2.5}$ concentrations still rose to levels greater than the EPA 24-hour standard of 35 μg m^{-3}. We found this occurred across multiple western US cities in 2020. These results suggest remaining indoors during smoke events is an effective, but limited, strategy in reducing smoke exposure. Additional action to reduce indoor smoke exposure, such as running portable air cleaners, during severe smoke events may be needed.

5.2 Recommendations for Future Work

The work presented in this dissertation has taken steps towards a greater understanding of several key knowledge gaps on wildfire smoke and health. However, there remain many open questions in these areas. These include: (1) the impact of smoke plume composition and age on plume toxicity, (2) population health impacts of multiple pollutants in smoke, and (3) personal smoke exposure. The following sections outline outstanding questions in these areas, next steps towards tackling them, and applications beyond US landscape fire smoke.

5.2.1 The Impact of Smoke Plume Composition and Age on Toxicity

Particle Composition

Toxicology studies suggest landscape fire smoke composition influences toxicity, where smoke plumes of different chemical compositions can differentially impact health (e.g., Kim et al., 2018, 2019; Rager et al., 2021). In Chapter 2 of this dissertation, we assessed the relationship between gas-phase HAPs in western US wildfire smoke, smoke age, and plume toxicity. In Chapter 3 we estimated that smoke $PM_{2.5}$ contributes a greater health risk than gas-phase smoke HAPs, using existing concentration response functions for all-source $PM_{2.5}$ and risk factors for species-specific gas-phase HAPs. However, current understanding of $PM_{2.5}$ composition in smoke plumes and relationships with toxicity are limited. Previous field and lab campaigns show that particle composition evolves with age, leading to higher oxygen to carbon ratios (Hodshire et al., 2019). Detailed smoke particle composition is currently a challenge even for well-instrumented lab studies (Jen et al., 2019). Data from field campaigns of smoke, including the WE-CAN campaign data used in this work, rarely contain a detailed speciation of organic particles in smoke, which comprise approximately 90% of smoke fine particle mass (e.g., Garofalo et al., 2019). Future works should aim to further characterize smoke particle composition as a function of fuel type, burn conditions, and smoke age.

For increased information on particle composition to inform potential impacts on smoke toxicity and health, a greater understanding of the relationship between particle composition and health is also needed. This information could be used to adjust the EPA AQI scale and associated health-impact warnings (currently based on anthropogenic $PM_{2.5}$), not only for smoke-specific $PM_{2.5}$, but even by different smoke ages, fire types, and burn conditions. For such an understanding of the influence of smoke composition and age on toxicity and health, a convergence of expertise from the three disciplines of atmospheric chemistry, epidemiology, and toxicology will be required.

Smoke in Urban and Indoor Environments

Many assessments of the chemical composition of landscape fire smoke, including the assessment presented in Chapter 2 of this dissertation, focus on lofted smoke mixing into the free troposphere or smoke in clean lab environments. These smoke conditions may not be representative of the mixture of smoke and either urban air or indoor air to which people are often exposed. In the recent 2020 wildfire season, smoke inundated urban areas across several western states and impacted indoor air quality, as we showed in Chapter 4 of this dissertation. There are few observations of the chemical composition of these surface-level urban/smoke mixtures and indoor/smoke mixtures. Urban/smoke and indoor/smoke mixtures may contain higher levels of HAPs (Messier et al., 2019; McCarthy et al., 2009) and lead to additional chemical processing of the smoke plume, which may impact toxicity. In addition, smoke plumes that remain at the surface may have a different composition than lofted smoke measured in aircraft-based field campaigns (Burling et al., 2011). Few studies have assessed these differences. A potential difference between lofted and surface-level smoke plume composition is a large limitation of the HAPs assessment in this work. The lack of chemical composition assessments of smoke in urban and indoor environments remains a large disconnect between atmospheric chemistry assessments of landscape fire smoke composition and the smoke composition information that would be most relevant for epidemiology and toxicology studies.

Multi-Pollutant Exposure

Although we assessed the relative health impacts from multiple pollutants in smoke in this dissertation, the possible nonlinear impacts of co-exposure to multiple pollutants remains largely unknown. In order to better characterize the health impacts of smoke plumes, which contain many health-relevant pollutants, a greater understanding of the health impacts of individual HAPs and multi-pollutant exposure, including particle-phase species, is needed. This will require a health-relevant chemical speciation of the smoke plumes to which people are exposed (described above).

Further, there are additional health-relevant pollutants in smoke not included in this work, including nitrogen dioxide and ozone, which should also be included in future works.

Next Steps

Future field campaigns should aim to sample smoke mixtures to which people are exposed including urban/smoke mixtures and indoor/smoke mixtures with more comprehensive particle-phase speciation. The methodology in Chapters 2 and 3 of this dissertation outline how results from such campaigns could be applied to assess important health-relevant components and subsequent health impacts of these air mixtures. Given a speciation of particle-phase HAPs in smoke, the methodology to determine health impacts of gas-phase HAPs could also be applied to quantify impacts of speciated particles in smoke. However, such an assessment is currently limited by a limited chemical speciation of smoke particles and lack of DALY factors for many particle-phase species.

5.2.2 Population Health Impacts of Multiple Pollutants in Landscape Fire Smoke

Application to Smoke-Impacted Regions Beyond the Western US

This dissertation has focused on assessing exposure and subsequent health impacts of multiple pollutants in US landscape fire smoke. However, there are many other parts of the world that are significantly impacted by landscape fire smoke, including Brazil, Indonesia, and Australia, among others (Johnston et al., 2012). This work relied on HAPs to PM ratios developed from western wildfire smoke and concentration response functions with relative risks derived predominately from epidemiological studies of western wildfire smoke. Smoke-enhanced pollutant abundance and health impacts of smoke in other parts of the world may differ from those studied in the western US for several reasons. First, smoke composition is a function of fuel type and burn conditions (Andreae, 2019; Akagi et al., 2011; Stockwell et al., 2014; Burling et al., 2010; Sekimoto et al., 2018) which differ across these heavily fire-impacted regions. The chemical composition of smoke also changes as the smoke ages in transit (Palm et al., 2020). Second, there are differences across the populations in each region. Differences in baseline health risk and social

vulnerability (CDC/ATSDR, 2018) (GBD) across the populations may lead to different population health implications of smoke exposure. The methods in this dissertation could be applied to assess the abundance and health impacts of multiple pollutants in smoke with additional data from these different regions.

First, estimates of smoke-specific $PM_{2.5}$ for the regions are needed. At present, the methods used to estimate observation-based smoke-specific $PM_{2.5}$ used here could not be applied to estimate smoke-specific $PM_{2.5}$ in other regions (apart from Canada) due to either a lack of sufficient monitors for kriging or unavailability of the HMS smoke plume product, which is only available over North America. Rapidly increasing low-cost monitoring networks, such as PurpleAir, may be able to fill spatial gaps of regulatory monitors (Gupta et al., 2018). Satellite-based surface $PM_{2.5}$ estimates could be used in place of kriging for estimates of total $PM_{2.5}$, but there is no direct substitute for the HMS product globally to identify smoke-impacted $PM_{2.5}$ concentrations. Visible satellite imagery, used to develop the HMS smoke plume product, is available globally. Thus, the HMS dataset, in theory, could be expanded to other fire-impacted regions around the globe. Model-based estimates of smoke-specific $PM_{2.5}$ can, and have, been used to estimate smoke-specific $PM_{2.5}$ around the globe (Johnston et al., 2012). However, estimated health impacts are sensitive to smoke $PM_{2.5}$ estimates (Cleland et al., 2021; Gan et al., 2017). Thus, it will be important to improve estimates of smoke $PM_{2.5}$ in these regions for future health studies through application and combination of multiple data sources.

In order to estimate smoke-enhanced ambient concentrations of HAPs in smoke, several approaches could be taken. To follow the methods in this dissertation, smoke composition assessments, like those discussed in Section 5.2.1, of fires in these regions would be needed to determine relative PM and HAPs abundance. Currently available estimates of emissions from different fire types and fuel burned could be applied to assess this relationship in fresh smoke, but as we show in Chapter 2, this relationship changes as the smoke ages, and as discussed above, may be different for ground-level urban/smoke and indoor/smoke mixtures. Ground-based monitoring of key health-relevant pollutants identified in this work could also be used and compared to the HAPs-

estimation methods used in Chapters 2 and 3. However, this method of assessing HAPs exposure is also currently limited. Monitoring networks of HAPs exist, but available data from these networks are temporally and spatially sparse (McCarthy et al., 2009). For future assessments of multiple pollutant exposure in smoke, estimates of exposure to HAPs should be improved either by developing smoke-specific HAPs to PM ratios for different fire types or by improved monitoring of key smoke HAPs. Again, it will also be important to incorporate other health-relevant pollutants into future assessments of multi-pollutant exposure including ozone and nitrogen dioxide.

Finally, epidemiological studies of smoke exposure for local fire types and populations are needed to inform the local concentration response function.

Additional Applications

The quantification of DALYs from gas-phase HAPs presented in Chapter 3 could also be applied to other significant sources of global air pollution. In particular, this analysis could be applied to lab and field assessments of emissions from cookstoves, which are likely a large contributor to household air pollution worldwide (GBD, 2019; Kodros et al., 2018). A recent lab study identified many particle- and gas-phase HAPs in cookstove emissions (Bilsback et al., 2019). Although not assessed in this dissertation, the methods used here could be applied to estimate DALYs from speciated particle-phase HAPs. Such an assessment could help identify cookstove models with less harmful emissions.

Next Steps

The work presented in this dissertation suggests landscape fire smoke significantly impacts health in the eastern US. Previous studies project an increase in smoke in parts of the eastern US (e.g., Ford et al., 2018). However, the health impacts of smoke in the eastern US are severely understudied. HAPs to PM ratios estimated here from the WE-CAN campaign data may not be representative of smoke composition in the eastern US. Further, to our knowledge, there are only four epidemiology studies of smoke exposure in the eastern US: three on peat fires in North Carolina (Rappold et al., 2011, 2012; Tinling et al., 2016), and one on elderly response to transported

Canadian wildfire smoke in New York (Le et al., 2014). Future epidemiology studies of smoke exposure should aim to characterize health impacts of other types of landscape fire smoke in different parts of the eastern US. Additional field campaign data (Fire Influence on Regional and Global Environments and Air Quality (FIREX-AQ) and Studies of Emissions and Atmospheric Composition, Clouds and Climate Coupling by Regional Surveys (SEC^{4}ARS)) and data from laboratory studies (Fire Lab at Missoula Experiment (FLAME), and FIREX) can provide emissions estimates of HAPs and PM across different fuel types and burn conditions in the US. A PM-weighted risk assessment could be applied across these data for a more comprehensive understanding of HAPs risk in US landscape fire smoke.

5.2.3 Personal Smoke Exposure

Currently, epidemiological studies of landscape fire smoke exposure rely on ambient concentrations of smoke-specific $PM_{2.5}$. Very little is known about personal smoke exposure. Because adults spend a majority of their time indoors (Klepeis et al., 2001), assessments of indoor smoke exposure are needed to estimate personal smoke exposure. In this dissertation, we show the utility of low-cost monitoring for assessing indoor air quality across a large number of indoor environments. The methodology used here can be applied to other significantly smoke-impacted regions given sufficient monitoring. In future works, it will be important to investigate potential predictors of indoor air quality during smoke events so that patterns can be established. If sufficient variability is explained by these predictors, indoor $PM_{2.5}$ concentrations during smoke events could be estimated for locations without indoor monitors. Such a dataset, in conjunction with maps of outdoor estimates of smoke-specific $PM_{2.5}$, could be used to estimate "personal" smoke exposure at the census-tract or zip-code level. These data could be used to identify locations where local efforts to reduce smoke exposure, such as supplying portable air filters and establishing clean air shelters, should be focused.

At the time of this writing, there are few locations with a high density of indoor monitors that could be used in such an analysis outside the western US. Additional low-cost monitors will be needed in other heavily smoke-impacted areas. Further, this work found census tracts of high

vulnerability are underrepresented in the PurpleAir co-located monitors used here. Indoor environment characteristics, which influence outdoor air infiltration, may systematically differ by socioeconomic vulnerability (for example, presence of air filtration). Future work with low-cost air quality sensors should aim to increase monitoring efforts in low-income, high-vulnerability populations.

Next Steps

As mentioned previously in Chapter 4, there are several confounding variables which may impact the relationship between indoor and outdoor air quality which were unaccounted for in the present work. Additional variables could be incorporated and may explain some of the variability in indoor/outdoor ratios. In particular, there are potential differences in socioeconomic status and infrastructure between the five regions in this work which may affect the indoor environment characteristics and, subsequently, indoor/outdoor ratios. These include age of building, local rate of home ownership, type of building (single family vs. multi-unit), and availability of air conditioning. These data are available from the American Housing Survey at the census-tract level (Bureau, 2019). In addition, differences in meteorology and time of day may also impact indoor/outdoor ratios (i.e., more moderate temperatures may lead to increased infiltration via open windows). Indoor/outdoor ratios could be binned by these different variables to determine if they explain any additional variability. Although the addition of these data could provide some insight on indoor environment characteristics and possible occupant behavior, it will not fill all data gaps. In dense urban environments with mixed zoning, it may be difficult to determine if monitors are in residential or commercial spaces. Further, the American Housing Survey does not provide information on individual homes, but rather census-tract level data. Thus, we will remain uncertain of individual indoor environment characteristics and occupant behavior.

Information on these confounding variables may also be able to be obtained from PurpleAir monitor owners themselves. A large increase in PurpleAir monitors in the wake of significant smoke events suggests communities are interested in monitoring their air quality. Some owners of PurpleAir monitors have been willing to participate in short-term studies of indoor air quality

during smoke events (May et al., 2021). Future research should aim to collaborate with these citizen scientists for a comprehensive picture of the influence of smoke on indoor air quality across a larger set of indoor environments and multiple smoke events. A study of this nature could combine the advantages of a large network of monitors with key information on building characteristics and occupant behavior, which can have a significant impact on indoor air quality during smoke events (e.g., Kirk et al., 2018).

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APPENDIX A

SUPPLEMENTAL INFORMATION FOR CHAPTER 2

A.1 Detailed WE-CAN Sampling Methodology and Smoke Plume Aging

Carbon monoxide (CO) was measured by an Aerodyne Research Mini-QCL tunable diode laser infrared absorption spectrometer (Lebegue et al., 2016) and Picarro G2401-m WS-CRDS analyzer. To eliminate variable baseline contribution of ambient pressure absorbances, and thereby optimize accuracy of CO quantifications, the optical bench of the Mini-QCL spectrometer was purged with dry zero air from which residual CO had been catalytically scrubbed. The measured concentration of purge gas N_2O was used in the spectral fit of the spectral baseline. One-sigma precision for CO observations from the two instruments are 100 ppt and 30 ppb, respectively. The instruments were calibrated during research flights by overfilling the inlet with working standard gases comprised of ultra zero air containing a known mixture of measured gases. CO observations from the Picarro G2401-m were substituted for the single flight when observations from the Mini-TILDAS were not available.

Observations of volatile organic compounds (VOCs) were made with a proton-transfer-reaction time-of-flight mass spectrometer (PTR-ToF-MS 4000, Ionicon Analytic, Innsbruck, Austria) and NCAR's Trace Organic Gas Analyzer (TOGA; Apel et al., 2015). During WE-CAN the PTR-ToF-MS measured ion m/z from 15-400 at 2 or 5 Hz frequency through a 3 m, 60 C heated inlet. The PTR-ToF-MS was calibrated each flight using certified gas standard mixtures containing 25 distinct VOCs (Apel-Riemer Environmental Inc., Miami, FL). Sensitivities for all other measured ions were estimated using the method described by Sekimoto et al. (2018). Measurement uncertainties are 15-50% and mostly contributed by error in the sensitivity estimates. For flights 1 through 12 (at 77030 UTC) the TOGA instrument took 28-second integrated samples every 100 seconds, and then 33-second integrated samples every 105 seconds for flights 12 (at 77030 UTC) through 16. Uncertainties for the species measured by TOGA discussed in this work range from 15% to 50%.

Sub-micron particle mass was estimated using observations from two instruments: total non-refractory mass from a High Resolution Time-of-Flight Aerosol Mass Spectrometer (HR-ToF-

AMS; DeCarlo et al., 2006; Garofalo et al., 2019) and black carbon mass from a single particle soot photometer (SP2, Schwarz et al., 2008). During WE-CAN, the AMS was operated in standard mass spectrometry mode with a pressure-controlled inlet and time resolution of 5 s (2.5 s open, 2.5 s closed). The AMS has an aerodynamic diameter cutoff of 1 micron and a collection efficiency of 0.65 was applied. The uncertainty (2 sigma) in AMS observations, dominated by an uncertainty in the collection efficiency, is 35%. Further details on the operation procedure and data processing for AMS observations during WE-CAN is available in Garofalo et al. (2019). The SP2 uses a 1064 nm Nd:YAG laser to heat absorbing material, primarily refractory black carbon, to its vaporization temperature and measures the resulting incandescence, which is proportional to mass (Schwarz et al., 2008). Particles smaller than about 70 nm in diameter will not heat up sufficiently to incandesce and be detected. During WE-CAN, HEPA-filtered ambient air was used to dilute the SP2 sample to prevent signal saturation. Observations from both instruments were placed on the same time resolution as the TOGA observations and then summed to estimate total submicron particle mass.

Table A.1 - HAPs measured during WE-CAN and their cancer unit risk estimate (URE), chronic noncancer reference concentration (RfC), and acute RfC taken from the Office of Air Quality Planning and Standards (OAQPS) database.

Name	Chemical Formula	CAS num-ber	Instrument	Cancer URE[a] [ug^{-1} m^3]	Cancer URE source	Chronic noncancer RfC[a] [mg m^{-3}]	Chronic noncancer RfC source	Acute RfC[b] [mg m^{-3}]	Acute RfC source[c]
acetaldehyde	C_2H_4O	75-07-0	TOGA	0.0000022	IRIS	0.009	IRIS	0.47	REL
acetonitrile	CH_3CN	75-05-8	TOGA	NA	NA	0.06	IRIS	22	AEGL-1
acrolein	C_3H_4O	107-02-8	TOGA	NA	NA	0.00035	CAL	0.0025	REL
acrylonitrile	C_3H_3N	107-13-1	TOGA	0.000068	IRIS	0.002	IRIS	3.7	AEGL-2
benzene	C_6H_6	71-43-2	TOGA	0.0000078	IRIS	0.03	IRIS	0.027	REL
bromoform	$CHBr_3$	75-25-2	TOGA	0.0000011	IRIS	NA	NA	NA	NA
bromomethane	CH_3Br	74-83-9	TOGA	NA	NA	0.005	IRIS	3.9	REL
carbon	CS_2	75-	TOGA	NA	NA	0.7	IRIS	6.2	REL

disulfide		15-0							
chlorobenzene	C_6H_5Cl	108-90- 7	TOGA	NA	NA	1	CAL	46	AEGL-1
chloromethane	CH_3Cl	74-87-3	TOGA	NA	NA	0.09	IRIS	1900	AEGL-2
dichloromethane	CH_2Cl_2	75-09-2	TOGA	0.00000001	IRIS	0.6	IRIS	14	REL
ethylbenzene	C_8H_{10}	100-41-4	TOGA	0.0000025	CAL	1	IRIS	140	AEGL-1
formaldehyde	CH_2O	50-00-0	TOGA	0.000013	IRIS	0.0098	ATSDR	0.055	REL
hydrogen cyanide	HCN	74-90-8	TOGA	NA	NA	0.0008	IRIS	0.34	REL
iodomethane	CH_3I	74-88-4	TOGA	NA	NA	NA	NA	130	AEGL-1
methanol	CH_3OH	67-56-1	TOGA	NA	NA	20	IRIS	28	REL
n-hexane	C_6H_{14}	110-54-3	TOGA	NA	NA	0.7	IRIS	10000	AEGL-2
propanal	C_3H_6O	123-38-6	TOGA	NA	NA	0.008	IRIS	110	AEGL-1
styrene	C_8H_8	100-	TOGA	NA	NA	1	IRIS	21	REL

		42-5							
tetrachloroethene	C_2Cl_4	127-18-4	TOGA	0.00000026	IRIS	0.04	IRIS	20	REL
toluene	C_7H_8	108-88-3	TOGA	NA	NA	5	IRIS	37	REL
chloroform	$CHCl_3$	67-66-3	TOGA	NA	NA	0.098	ATSDR	0.15	REL
o-xylene	C_8H_{10}	95-47-6	TOGA	NA	NA	0.1	IRIS	22	REL
m-,p-xylene	C_8H_{10}	108-38-3, 106-42-3	TOGA	NA	NA	0.1	IRIS	22	REL
phenol	C_6H_6O	108-95-2	PTR	NA	NA	0.2	CAL	5.8	REL
acetamide	C_2H_5NO	60-35-5	PTR	0.00002	CAL	NA	NA	NA	NA
quinone	$C_6H_4O_2$	106-51-4	PTR	NA	NA	NA	NA	NA	NA
methyl methacrylate	$C_5H_8O_2$	80-62-6	PTR	NA	NA	0.7	IRIS	70	AEGL-1

isocyanic acid	HNCO	75-13-8	PTR	NA	NA	NA	NA	NA	NA
1,1,1-trichloroethane	CH_3CCl_3	71-55-6	TOGA	NA	NA	5	IRIS	68	REL
1,2-dichloroethane	$C_2H_4Cl_2$	107-06-2	TOGA	0.000026	IRIS	2.4	ATSDR	NA	NA
2,2,4-trimethylpentane	C_8H_{18}	540-84-1	TOGA	NA	NA	NA	NA	NA	NA

[a]Assumed chronic exposure for these values is continuous exposure for 70 years.

[b]Assumed acute exposure for these values is one hour.

[c]The OAQPS database has multiple values for acute exposure. In our estimates of risk from acute exposure we only use species with 1 hour RELs.

Table A.2 – Reaction rates and estimated lifetimes for second order reaction of 2-methylfuran, acrolein, and acrylonitrile with OH[6–9], O_3[7,10,11], and NO_3[12–14]. *k* is given in units of cm^3 molecules s^{-1} and all lifetimes are reported in hours.

age tracer	k_{OH}	τOH[a] (h)	k_{O3}	τO_3[b] (h)	k_{NO3}	τNO_3[c] (h)
2-methylfuran	7.31×10^{-11}	1.9	-[d]	-	2.57×10^{-11}	0.1
acrolein	1.96×10^{-11}	7.1	2.81×10^{-19}	5.8×10^{2}	1.11×10^{-15}	3.1×10^{3}
acrylonitrile	4.04×10^{-12}	34	1.38×10^{-19}	1.2×10^{3}	6.83×10^{-18}	5.1×10^{5}

a. Assumed OH concentration of 2.0×10^{6} molecules cm^{-3}

b. Assumed O_3 concentration of 1.7×10^{12} molecules cm^{-3} (65 ppb at STP, median O_3 mixing ratio in smoke-impacted data points in our analysis)

c. Assumed nighttime NO_3 concentration of 8.0×10^{7} molecules cm^{-3} (3 ppt at STP)[15].

3-methylfuran, structurally similar to 2-methylfuran, has a k_{O3} of 2.05×10^{-17} cm^3 molecules s^{-1} and an estimated lifetime against loss by reaction with O_3 of 8.0 hours[16]

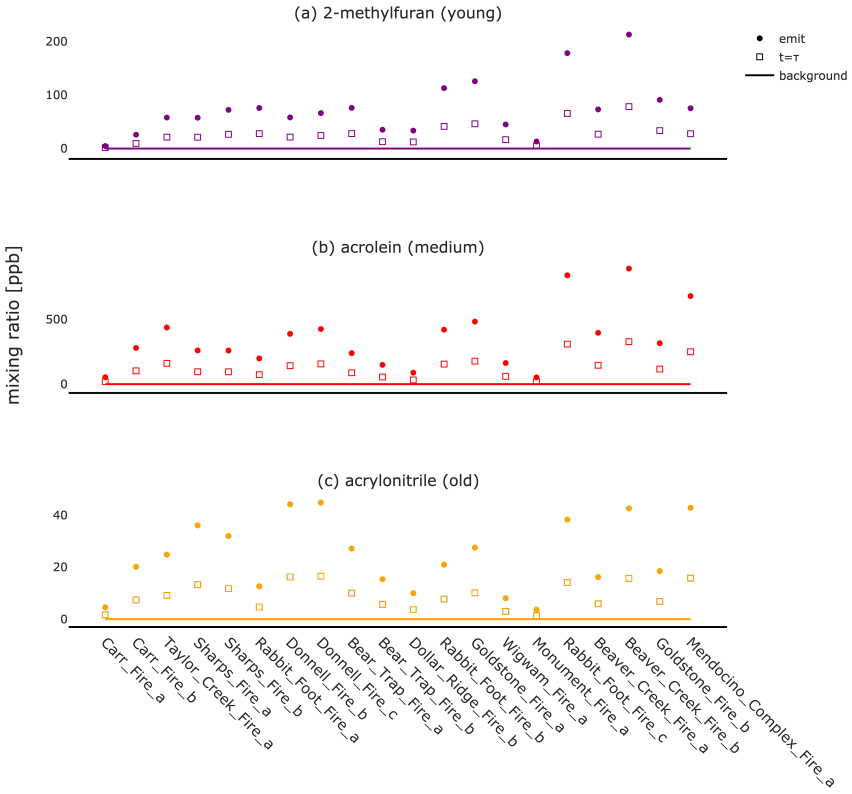

Figure A.1: Plume-integrated mixing ratios during emissions passes (the plume transects that are closest to the fire) ("emit," calculated from observations by the PTR-ToF-MS), mixing ratios at the e-folding time (τ) of each species for 2^{nd} order reaction with OH ("t=τ", calculated using the k_{OH} values in the main text and an OH concentration of 2x10^6 molecules cm^{-3}), and estimated western United States no-smoke background mixing ratios during WE-CAN ("background", calculated from observations by TOGA) for each age tracer. Fire designations "a, b, and c" indicate transects through a smoke plume from a single fire more than 30 minutes apart. Plume-integrated 2-methylfuran mixing ratios are calculated as 85% of the PTR combined mixing ratio of 2-methylfuran and 3-methylfuran, calculated from Koss et al. (2018)

A.2 VOC to PM Ratios and Comparison to Carbon Monoxide (CO) for Each Age Group

Median ratios, the 25^{th} and 75^{th} percentiles, the number of observations, and percent of observations above the detection limit for each HAP to PM_1 in young, medium, old, and older smoke are given in the supplemental excel file available online with O'Dell et al. (2020).

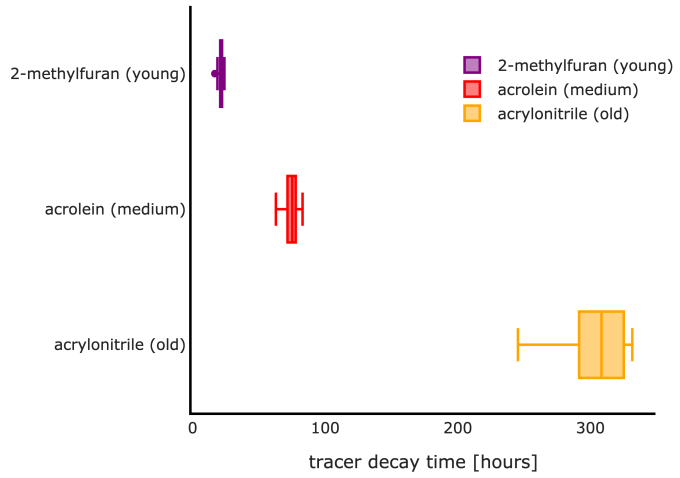

Figure A.2: Distribution of the times required for initial plume-integrated mixing ratios of age tracers to reach the 95^{th} percentile of smoke-free background observations for each species via reaction with OH (at an assumed concentration of $2x10^6$ molecules cm^{-3}) for each emissions pass (the plume transect that is closest to the fire) during WE-CAN.

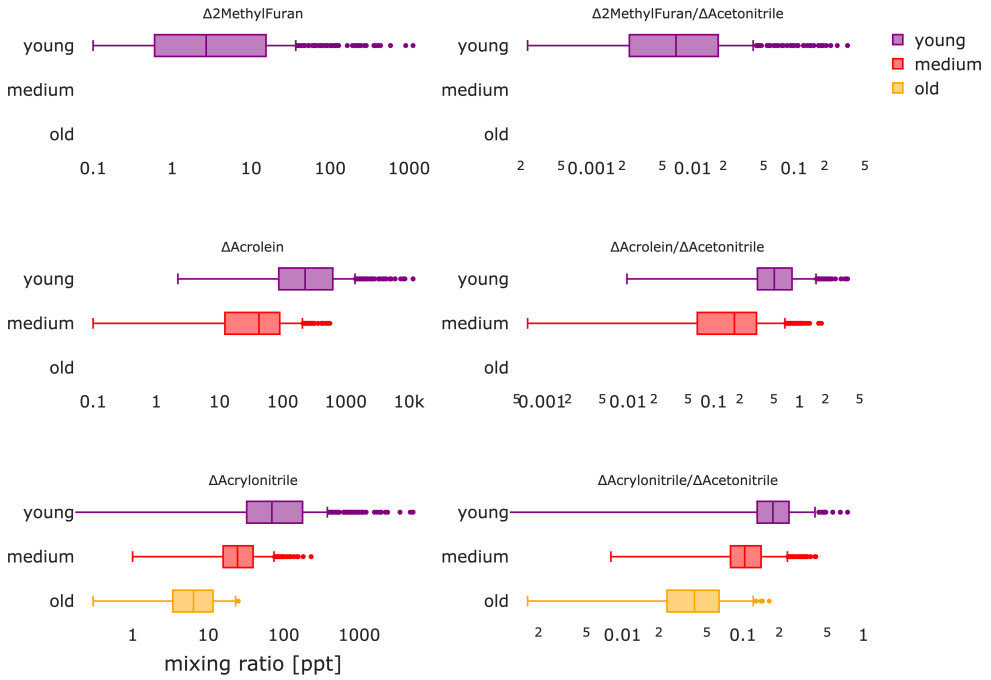

Figure A.3: Box plots of smoke-elevated age tracer mixing ratios in ppt (left panel) and dilution-corrected smoke-elevated age tracer ratios (right panel) for each age group.

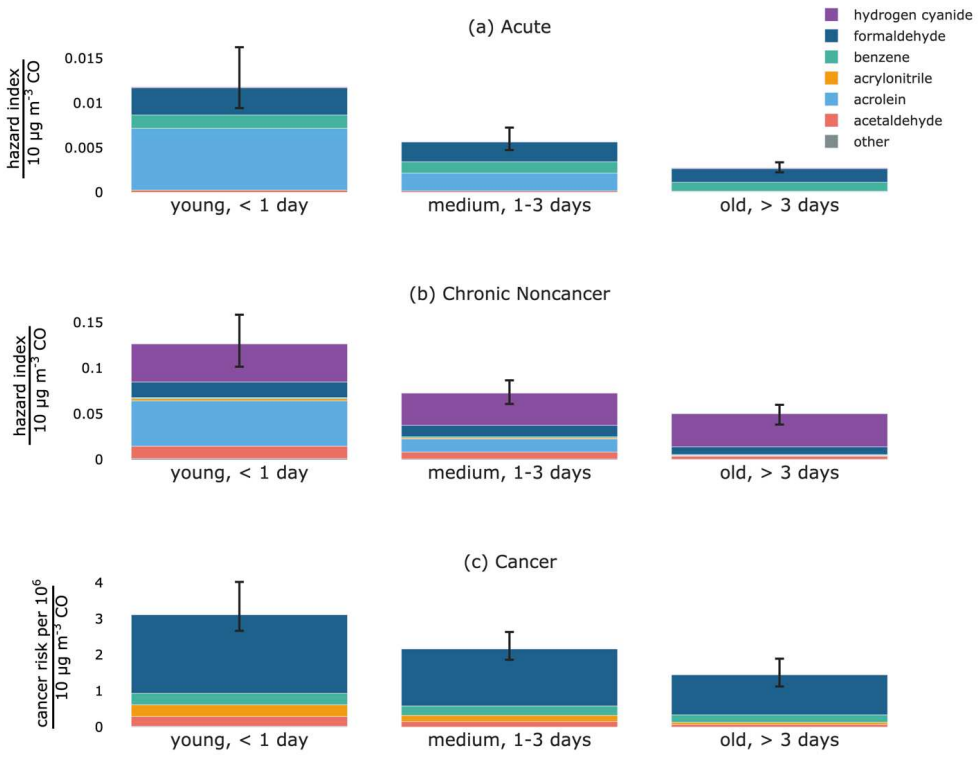

Figure A.4: Similar to Figure 2.2 but normalized by CO rather than PM_1. Median gas-phase HAPs acute hazard index (panel a), chronic noncancer hazard index (panel b), and cancer risk (panel c) ratio to 10 μg m^{-3} of smoke-elevated CO for each smoke age category. Black bars span from the 25^{th} to 75^{th} percentile of the CO-weighted hazard index for each age category. "Other" contains any species with non-zero smoke elevation and an available acute 1-hour California EPA reference exposure level, chronic reference concentration, or cancer unit risk estimate. A full list of species included in this analysis and associated reference concentrations and cancer unit risk estimates are given in Table A.1.

APPENDIX B

SUPPLEMENTAL INFORMATION FOR CHAPTER 3

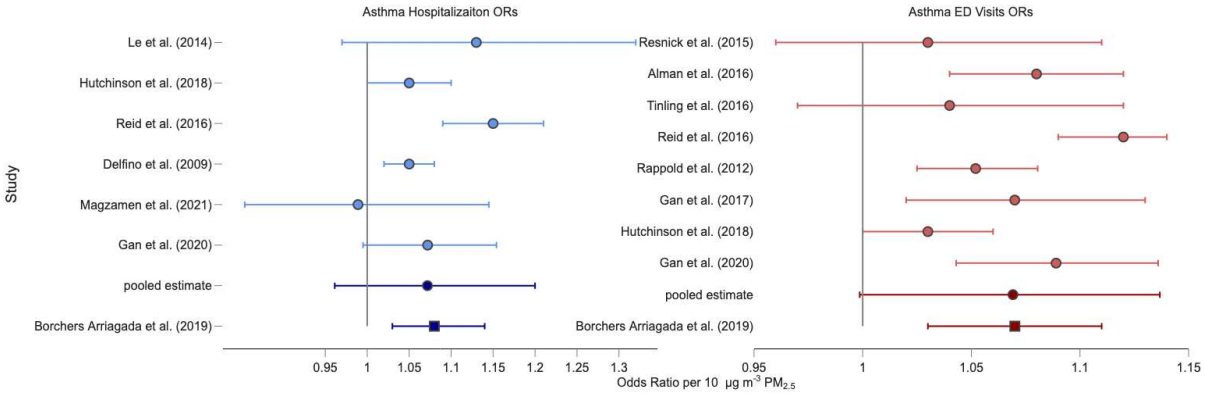

Figure B.1: Forest plot for monte-carlo pooled estimate of smoke-specific odds ratios per 10 μg m^{-3} smoke $PM_{2.5}$ for asthma hospitalizations (left) and asthma emergency department (ED) visits (right). Lighter colored circles and error bars indicate individual study odds ratios, with 95% confidence intervals, included in the pooled estimate. Darker diamonds and error bars indicate the central estimate and uncertainty around the monte-carlo pooled odds ratio. Darker squares and error bars indicate the meta-analysis estimate at 95% confidence interval from Borchers Arriagada et al. (2019). See main text for full references for each study.

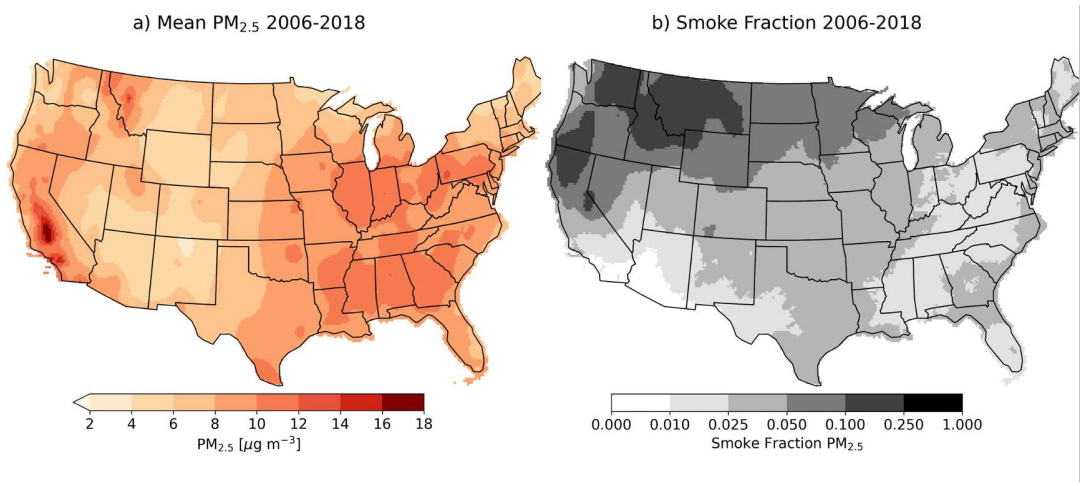

Figure B.2: Panel (a) shows the 2006-2018 mean total $PM_{2.5}$ estimated on a 15 x 15 km grid using kriged daily $PM_{2.5}$ observations from the Environmental Protenction Agency (EPA) Air Quality System (AQS) Federal Reference Method (FRM) and non-FRM monitors. Panel (b) shows the ratio of 2006-2018 mean smoke $PM_{2.5}$ (see Figure 1a in the main text) to 2006-2018 total $PM_{2.5}$.

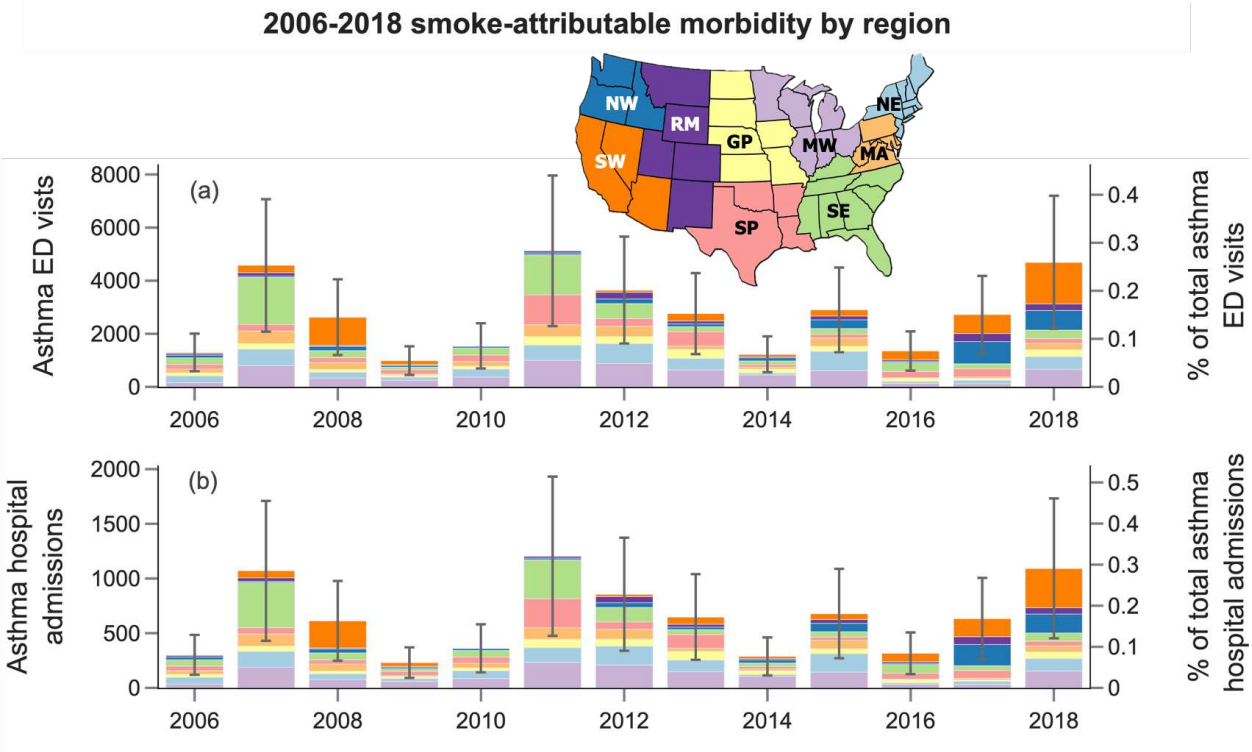

Figure B.3: Same as Figure 3.2 in the main text, but using a seasonal mean non-smoke $PM_{2.5}$ background estimate.

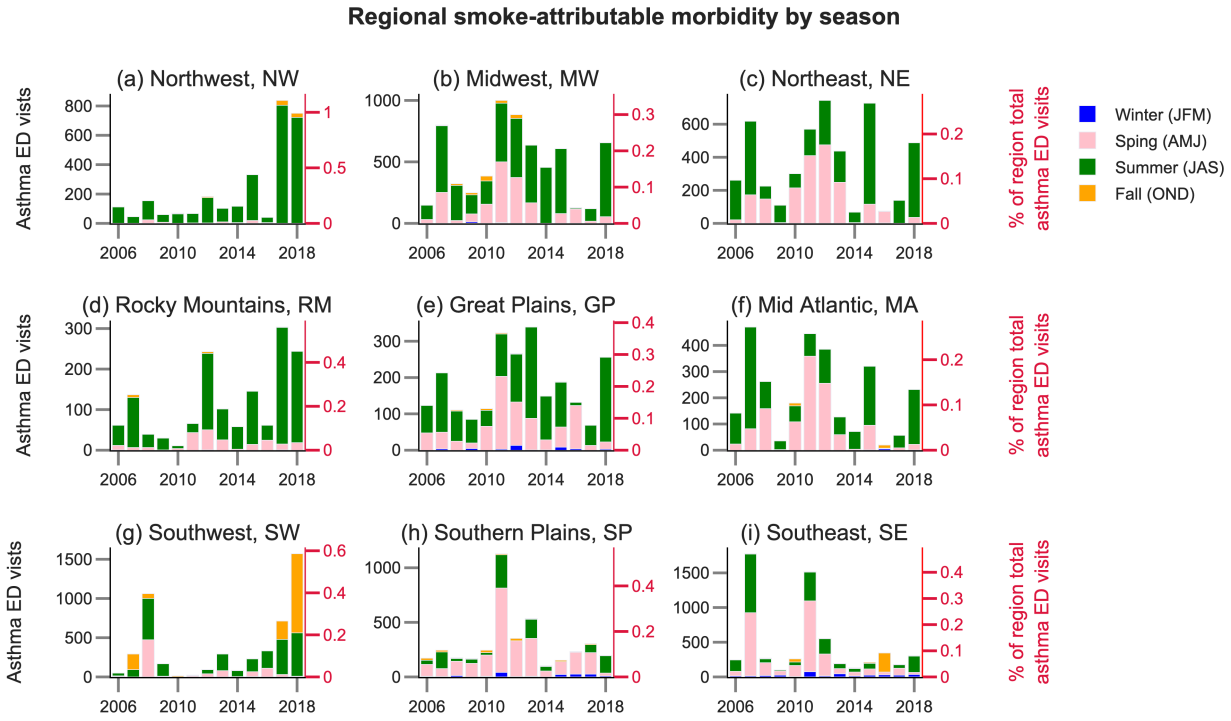

Figure B.4: Same as Figure 3.3 in the main text, but using a seasonal mean non-smoke $PM_{2.5}$ background estimate.

Table B.1: Relative risks and baseline rates used in health impact functions. Asthma hospitalization and emergency department visit baseline rates from HCUP NEDS and NIS (AHRQ, 2006). Baseline rates for mortality are from the GBD (GBD, 2019). Relative risks for asthma hospitalizations and emergency department visits are from Borchers Arriagada et al. (2019)

	Asthma hospitalizations	Asthma emergency department visits	All-cause (non-communicable diseases + lower respiratory infections) mortality	All-cause (non-communicable diseases + lower respiratory infections) DALYs
Baseline rate	129.9 per 100,000 persons	625.7 per 100,000 persons	732.93 per 100,000 persons	26688.87 per 100,000 persons
Relative risk per 10 μg m^{-3}	1.08 (95% CI: 1.03,1.14)	1.07 (95%CI: 1.03,1.11)	See Burnett et al. (2018) for hazard ratio equation.	See Burnett et al. (2018) for hazard ratio equation.

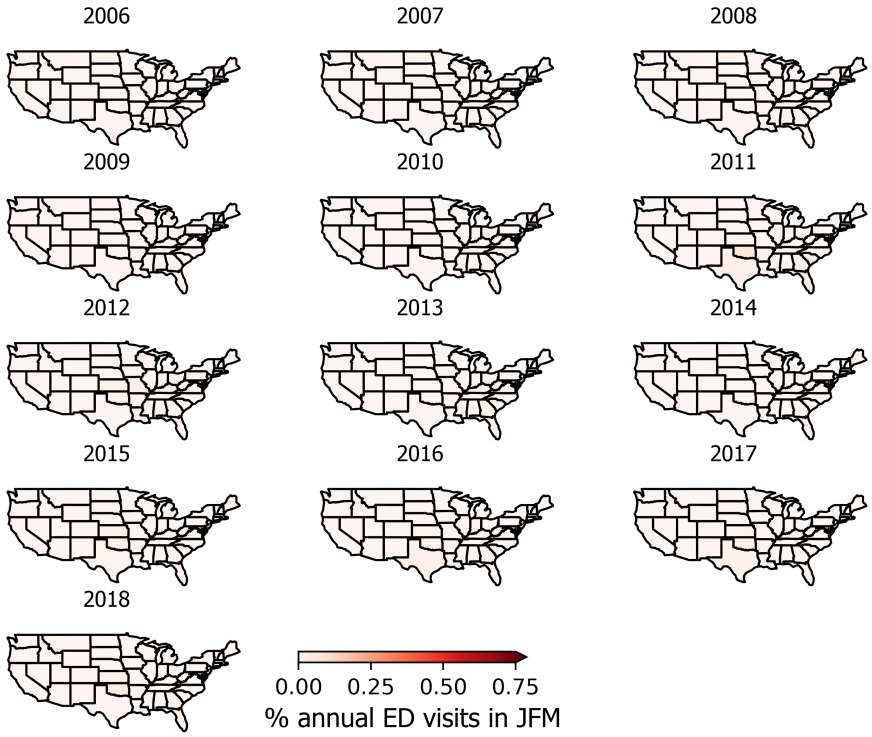

Figure B.5: Percent of annual total asthma emergency department (ED) visits by state attributable to smoke occurring in the winter (JFM) season.

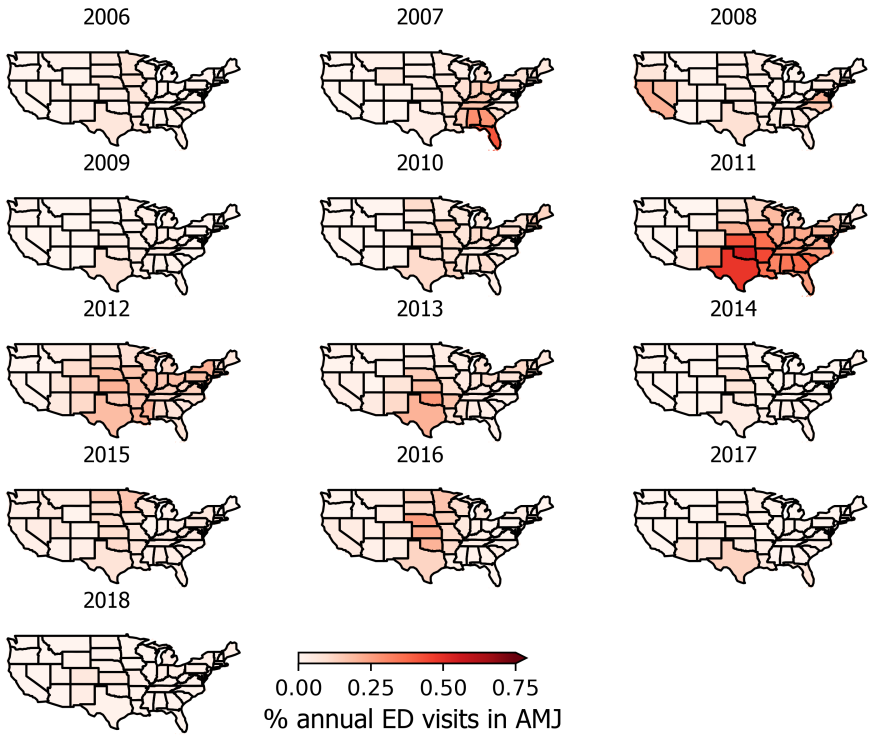

Figure B.6: Percent of annual total asthma emergency department (ED) visits by state attributable to smoke occurring in the spring (AMJ) season.

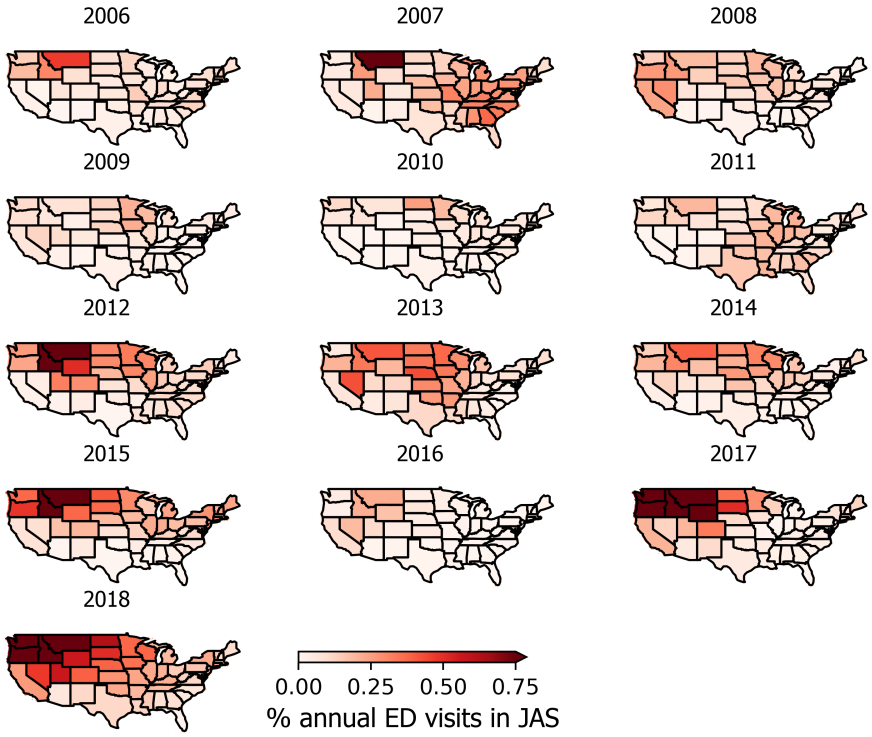

Figure B.7: Percent of annual total asthma emergency department (ED) visits by state attributable to smoke occurring in the summer (JAS) season.

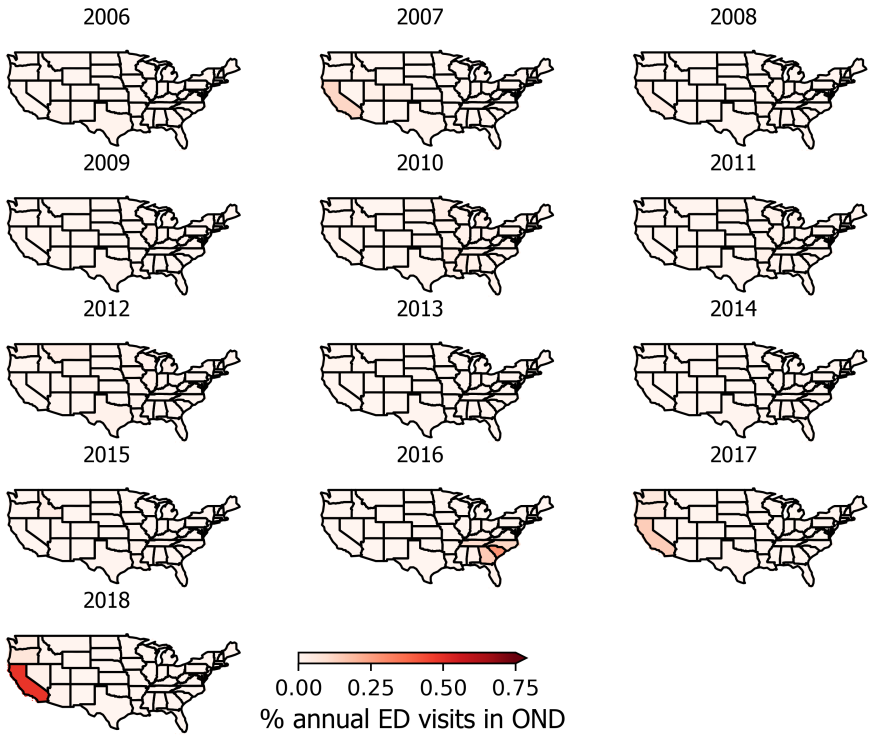

Figure B.8: Percent of annual total asthma emergency department (ED) visits by state attributable to smoke occurring in the fall (OND) season.

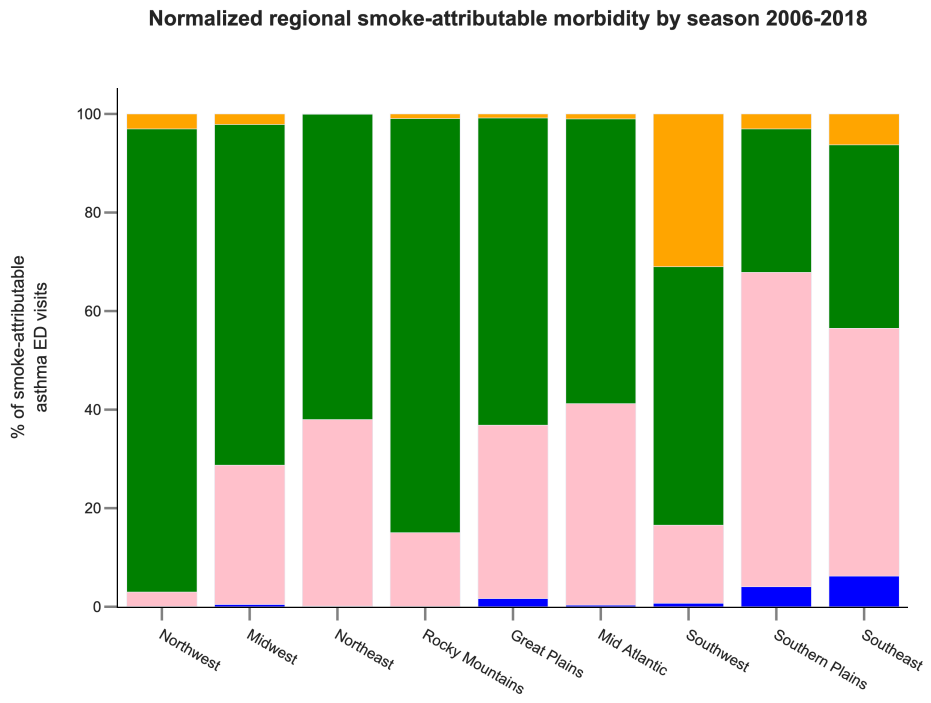

Figure B.9: The percent of all smoke-attributable asthma emergency department (ED) visits from 2006-2018 that occurred in each season: winter (JFM) in blue, spring (AMJ) in pink, summer (JAS) in green, and fall (OND) in orange.

Table B.2: Definition of regions used in morbidity analysis.

Region Name	Region Abbreviation	States
Northwest	NW	WA, OR, ID
Midwest	MW	OH, IL, IN, MI, WI, MN
Northeast	NE	ME, NH, RI, VT, CT, MA NJ, NY
Rocky Mountains	RM	MT, WY, UT, CO, NM
Great Plains	GP	ND, SD, IA, KS, NE, MO
Mid Atlantic	MA	PA, WV, VA, MD, DE, DC
Southwest	SW	AZ, NV, CA
Southern Plains	SP	TX, AR, OK, LA
Southeast	SE	KY, TN, SC, NC, GA, AL MS, FL

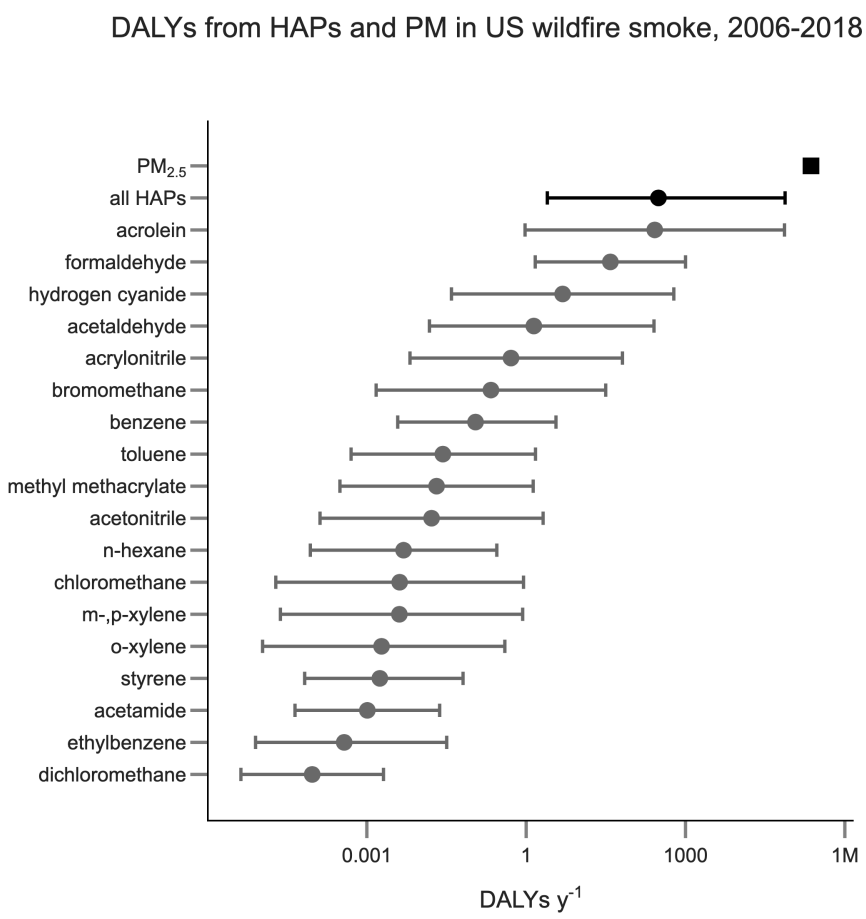

Figure B.10: Same as Figure 3.5 in the main text, but for all HAPs included in the present analysis with non-zero DALYs per year. 14 additional HAPs were included in the analysis but resulted in 0 additional DALYs due to a zero smoke-enhanced concentration (bromoform, carbon disulfide, chlorobenzene, tetrachloroethylene, chloroform, quinone, 1,1,1-trichloroethane, 1,2-dichloroethane, 2,2,4-trimethylpentane) and/or no available DALY factor from Huijbregts et al. (2005) (1,1,1-trichloroethane, 2,2,4-trimethylpentane, isocyanic acid, phenol, propanal, methanol, iodomethane).

Table B.3: Sensitivity analysis for the smoke-attributable mortality health impact assessment. The values presented in the main text are in bold.

	All-cause	5 Leading Causes
Mortalities from Chronic Smoke Exposure (median smoke background)	**6,300 (4,800 - 7,800)**	4,200 (3,100 - 5,200)
Mortalities from Chronic Smoke Exposure (mean smoke background)	5,400 (4,000 - 6,700)	3,600 (2,700 - 4,400)

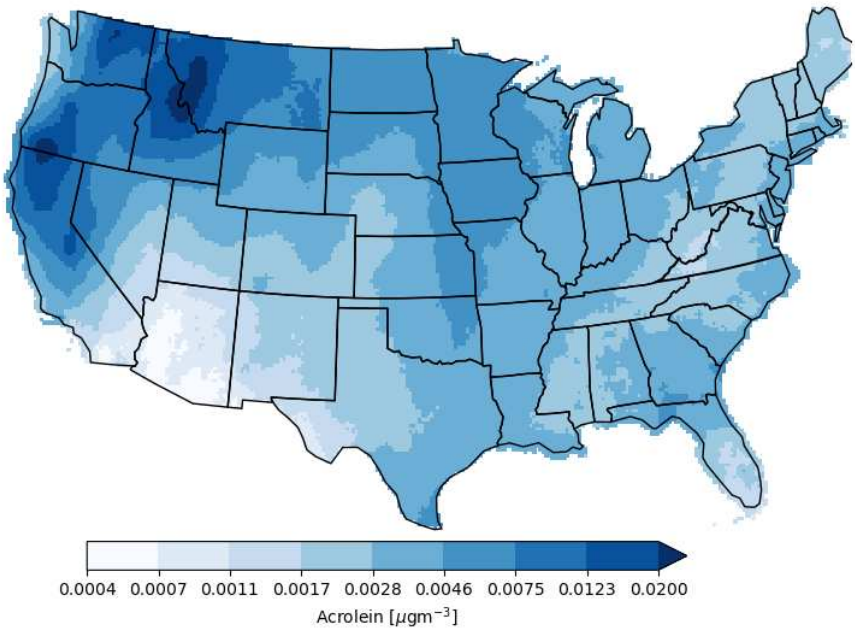

Figure B.11: 2006-2018 mean smoke-specific acrolein concentrations using the acrolein to PM ratio for young smoke.

APPENDIX C

SUPPLEMENTAL INFORMATION FOR CHAPTER 4

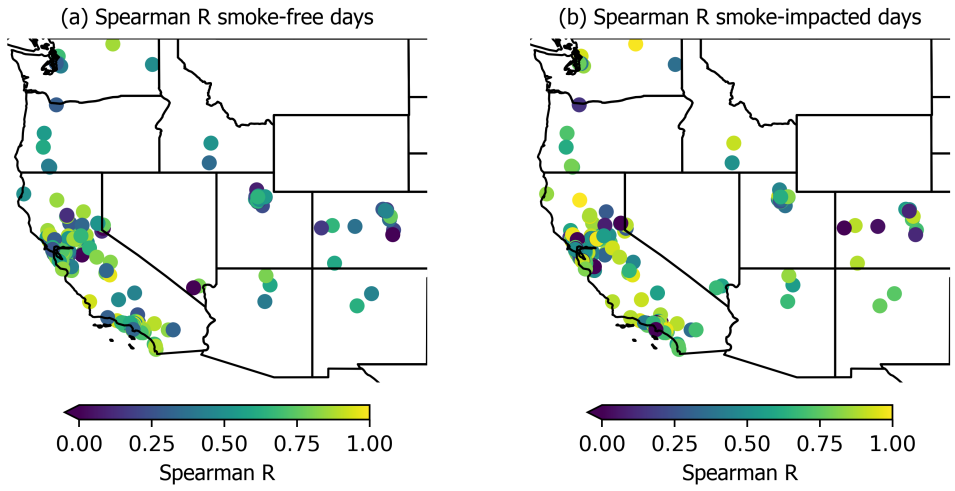

Figure C.1: Spearman's R on (a) smoke-free and (b) smoke-impacted days in 2020.

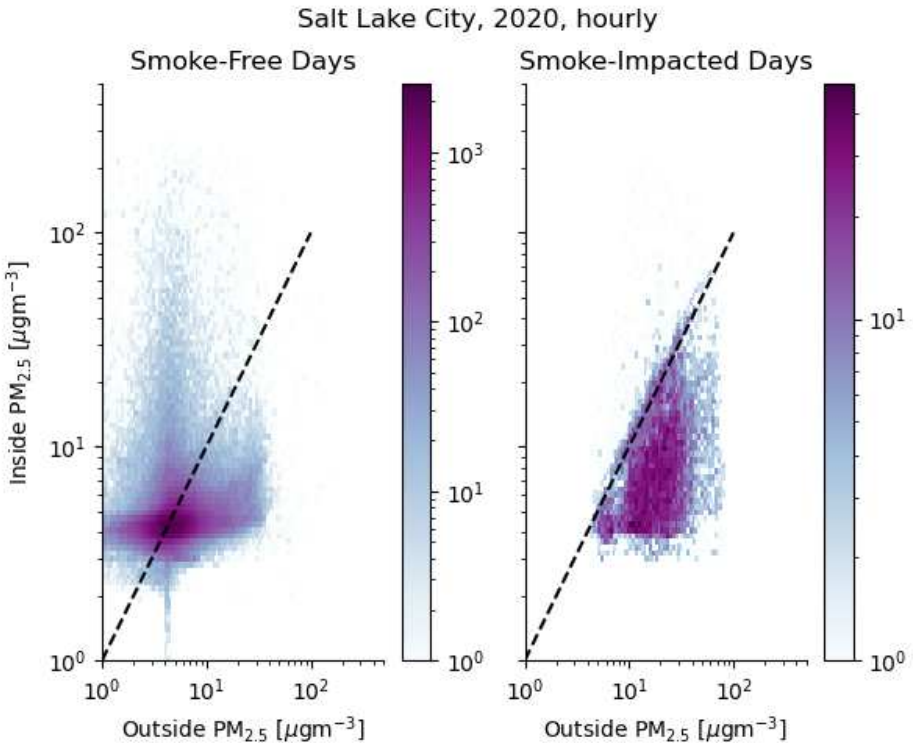

Figure C.2: Hourly indoor $PM_{2.5}$ versus hourly outdoor $PM_{2.5}$ for Salt Lake City for smoke-free (left) and smoke-impacted (right) observations in 2020.

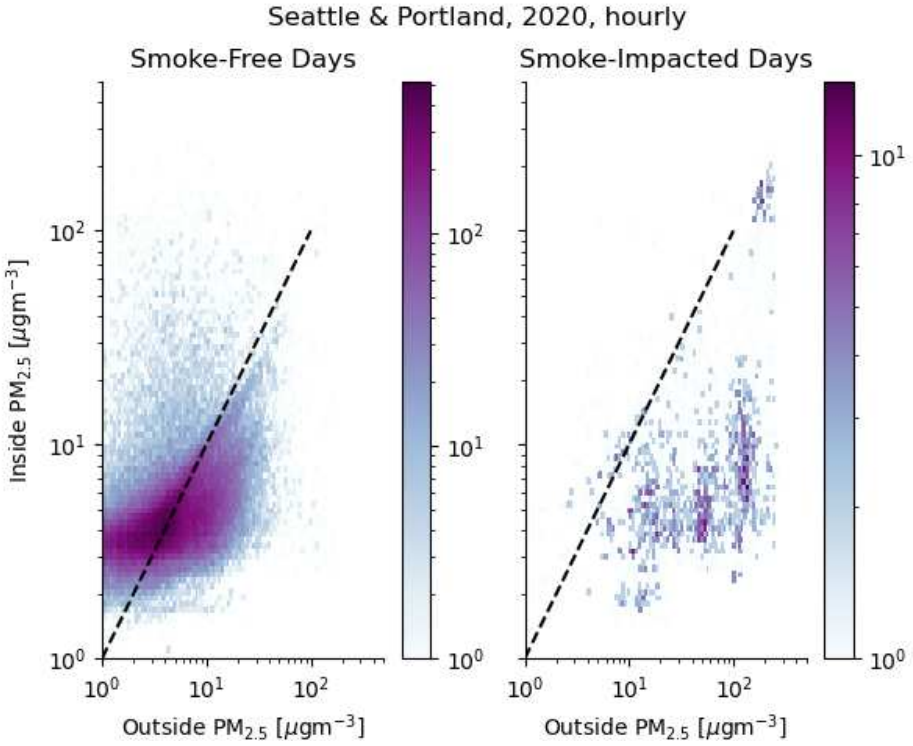

Figure C.3: Hourly indoor $PM_{2.5}$ versus hourly outdoor $PM_{2.5}$ for the Portland and Seattle area for smoke-free (left) and smoke-impacted (right) observations in 2020.

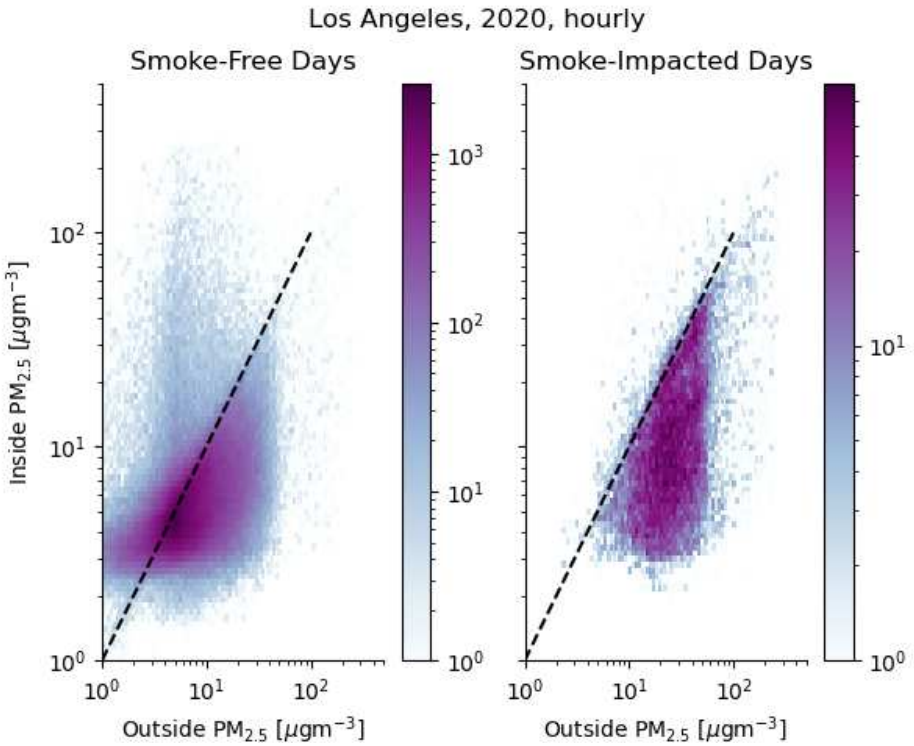

Figure C.4: Hourly indoor $PM_{2.5}$ versus hourly outdoor $PM_{2.5}$ for Los Angeles for smoke-free (left) and smoke-impacted (right) observations in 2020.

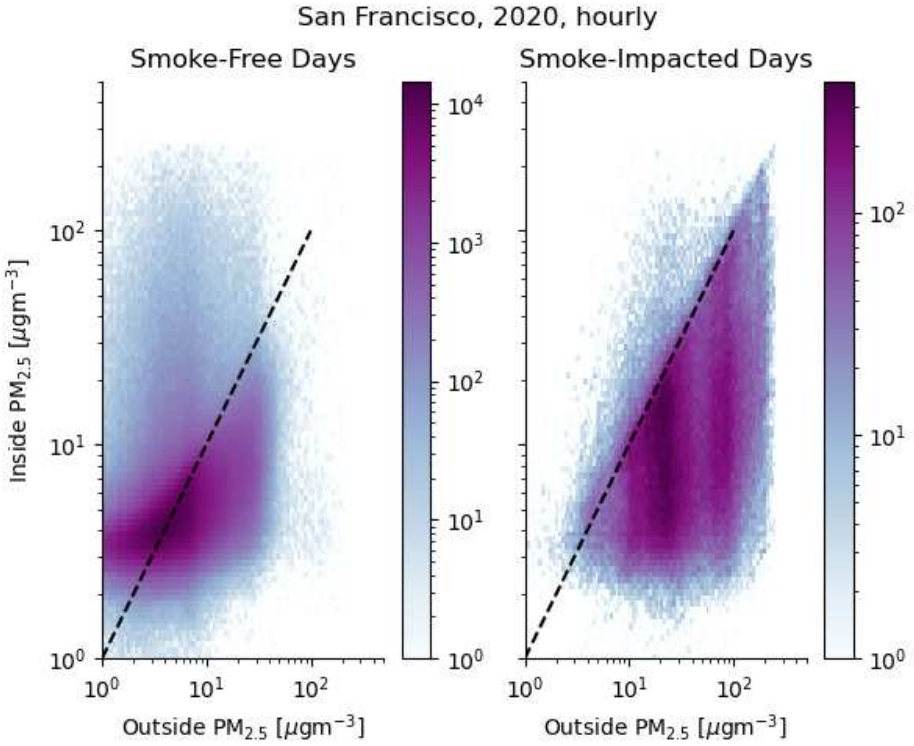

Figure C.5: Hourly indoor $PM_{2.5}$ versus hourly outdoor $PM_{2.5}$ for San Francisco for smoke-free (left) and smoke-impacted (right) observations in 2020.

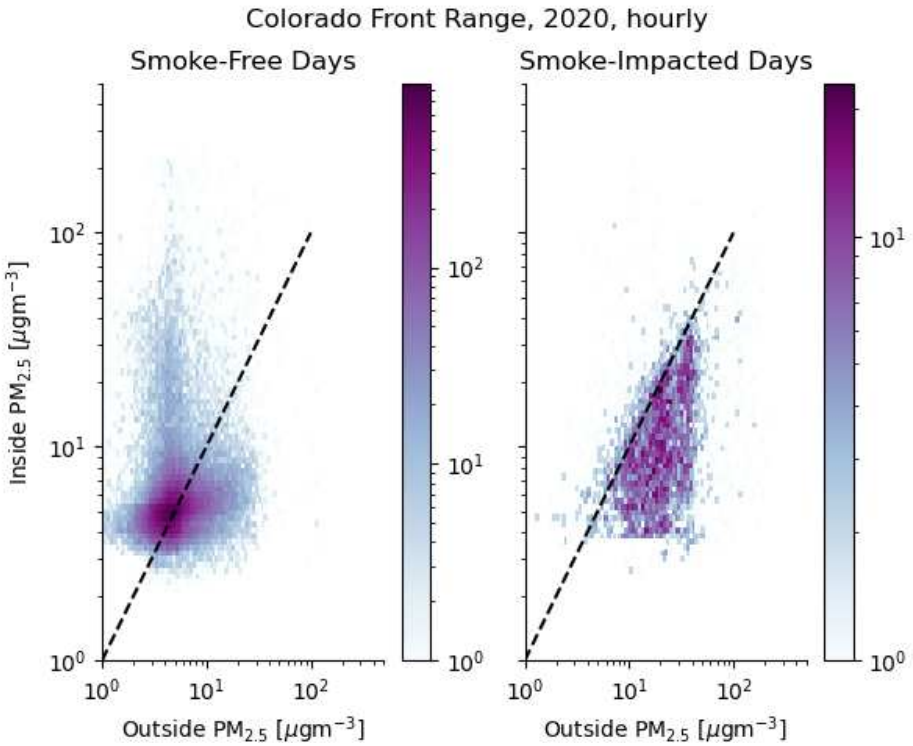

Figure C.6: Hourly indoor $PM_{2.5}$ versus hourly outdoor $PM_{2.5}$ for the Colorado Front Range for smoke-free (left) and smoke-impacted (right) observations in 2020.