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Multiple Approaches to Understanding the Intersection of
Climate Change, Air Quality & Public Health

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Abstract

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By Jennifer D. Stowell

An overwhelming majority of climate scientists have declared the validity of climate change and its potential threat to the environment and to living organisms on the planet. Though it is still taken lightly by some, temperatures across the globe are rising—especially near the poles where sheets of ice help to balance the conditions we experience in the troposphere. In fact, temperature anomalies have been tracked for many years by different entities, including both governmental and scientific organizations. Overwhelmingly, the temperature anomalies they have tracked deviate little from one another and follow nearly the exact same trend of warming in our atmosphere. Many skeptics consider the major cause of changes in climate to be natural increases of energy from the sun. While it is true that the sun has a natural, oscillating pattern of high and low energy, it has been operating on the same 11-year cycle for hundreds of years and with only slight increases in trend. However, global temperatures began to deviate from normal patterns in the 1950s and have continued to rise ever since. Hence, there must be something else causing temperatures to accelerate to such high levels. Aside from human development and some natural depletion of vegetation and forests, little else has changed in the total environment, save human activity. So, while there could be something at play of which we are unaware, it seems very likely that human activity is contributing to the problem in a significant way. This is evident when looking at levels of carbon dioxide (CO_2). Since the industrial revolution, CO_2 has continued to surpass natural levels that were seen prior to large-scale fossil fuel use. These CO_2 concentrations, similar to temperature, are also trending higher with little to no decrease.

An important issue that we must consider when approaching the climate change issue, is the effect that it can have on living organisms. One of the most readily apparent ways in which climate change affects living things is through large decreases in air quality. Climate change can have both direct and indirect paths of affecting the air that we breathe. For instance, a direct path involves the direct formation of ozone (O_3) in the troposphere where it can harm human health. With respect to indirect pathways, the effects of climate change on wildfire activity have been evident in the past few decades. This is considered an indirect effect of climate on air quality because rising temperatures can increase the incidence of wildfires—which in turn can dump toxic chemicals and particles into the atmosphere through the dissipation of smoke plumes.

In this dissertation, the effect of climate change on air quality is approached in three different ways and on three different spatial scales. The first objective looks at the future changes in harmful tropospheric O_3 on a national scale. This approach separates differences in concentration according to source: climate change or emissions policies. Using modeling methods to separate and predict future O_3 by source adds to our understanding of the potential dangers to human health that we could experience in future years. With future O_3 concentrations predicted by source, we can then project the impact on multiple morbidities and premature mortality. Results from this analysis showed that while the effect on morbidity varies between locations, it was evident that climate change could impact future mortality via O_3 exposure. However, the real culprit of excess mortality due to O_3 was emissions policy. Looking at two

different predicted emissions scenarios, there was a significant difference in effects on mortality for scenarios in which allowed emissions are not restricted by emissions policies.

Another objective estimates the association of present-day wildfire activity on cardiorespiratory events on a statewide scale. Using health records form the state of Colorado during the fire seasons (May-August) of 2011-2014, we can estimate the association between smoke $PM_{2.5}$ exposure and adverse health outcomes. Using a two-stage modeling approach, we calculated the contribution of smoke $PM_{2.5}$ to total $PM_{2.5}$. Separating the contributions allowed us to examine the effects due solely to smoke $PM_{2.5}$. It was evident from our results that smoke $PM_{2.5}$ was associated with many respiratory morbidities, but not associated with cardiovascular disease. We also conducted stratified analyses on both age and sex. While no significant difference was observed for sex, several differences were apparent for age. One of the most striking results was the odds ratio or expected increase in risk of asthma exacerbation due to smoke $PM_{2.5}$ exposure. The results suggest that risk increases by over 8% (95% CI: 1.06, 1.11) for every 1 $\mu g/m^3$ increase in smoke $PM_{2.5}$ exposure. This result is higher than risk reported in previous publications. One conclusion that we might draw is that smoke $PM_{2.5}$ may be more toxic than background, ambient $PM_{2.5}$.

A third objective builds upon the results from the Colorado wildfire study and attempts to estimate future wildfire health impacts on a regional scale in the western US. Using complicated chemical transport models with and without included fire sources, we were again able to separate out smoke $PM_{2.5}$, but in this instance, we are investigating potential future changes and health burden due to additional smoke $PM_{2.5}$ exposure in the 2050s. This involved taking the difference between future smoke $PM_{2.5}$ and present smoke $PM_{2.5}$ in order to estimate potential smoke $PM_{2.5}$ increases we could expect in addition to our present exposure. Through this process and adopting the risk measurement and the incidence of emergency department visits from respiratory outcomes in the Colorado study, we were able to project the future health burden from smoke $PM_{2.5}$.We observed a few hotspots that seem to be highly affected by future smoke $PM_{2.5}$ concentrations. These areas included northern Idaho, Nevada, and the coast of Oregon. However, it was also important to keep in mind the population distribution in comparison with the increased effects on human health. When looking at the results compared to changes in population, Montana stood as another area for concern. This was due to its relatively high increase in wildfire $PM_{2.5}$ events and an overall decline in population that is expected by the 2050s.

Taken together, these three aims help us understand more about the relationship between air quality and climate change. And, in turn, this allows for us to draw out potential risk to human health that could be seen in the future. Looking at 3 different approaches with each on a different spatial scale allowed us to explore some of the assumptions that we might draw from future exposures to O_3 and wildfire smoke $PM_{2.5}$. Moving forward, it will be important to expand on these future impacts and find ways to attach monetary and other important values to these expected changes. This type of analysis could be beneficial in that it can be a tool for both informing policy and emergency response plans as we look to the changes that may be expected in the future.

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1. INTRODUCTION

1.1 Impact of climate change on air quality and human health through multiple pathways

Climate change, while contested politically, receives overwhelming support from the scientific community, with over 97% of climate scientists acknowledging the threat it poses.[1] In addition, many scientific entities have issued statements stating that climate change is a real concern.[2] Climate change is defined as changes in long-term meteorological trends. These changes can affect weather patterns, disaster susceptibility, and migration of humans. Such changes have become apparent in the last few decades as we see regular record-breaking temperatures in many regions. Multiple countries and organizations have pledged to reduce their contributions to the known causal components of climate change, but extensive resistance in some groups is still a major issue.

Perhaps one of the reasons that climate change is such an inciting topic is the fact that some of it is natural.[3] This complicates the issue by making it difficult to separate the anthropogenic effects from the natural. Changes in long-term weather patterns have undergone natural cycles of heat and ice. This is still the case and accounts for some of the changes we are experiencing.[4] Some of these changes are natural responses to solar activity. Some would argue that the warming seen is due to changes in the sun's energy emittance. However, the sun emits energy on a predictable, oscillating cycle of ~11 years and climate records continue to show this general pattern with only slight deviances.[5] Additionally, if the conditions we are experiencing now were due solely to energy emitted by the sun, we would expect all layers of the atmosphere to be warming. However, that is not the case. The troposphere (level of the atmosphere in which we live) has seen continued, accelerated heating while other layers have not.[6]

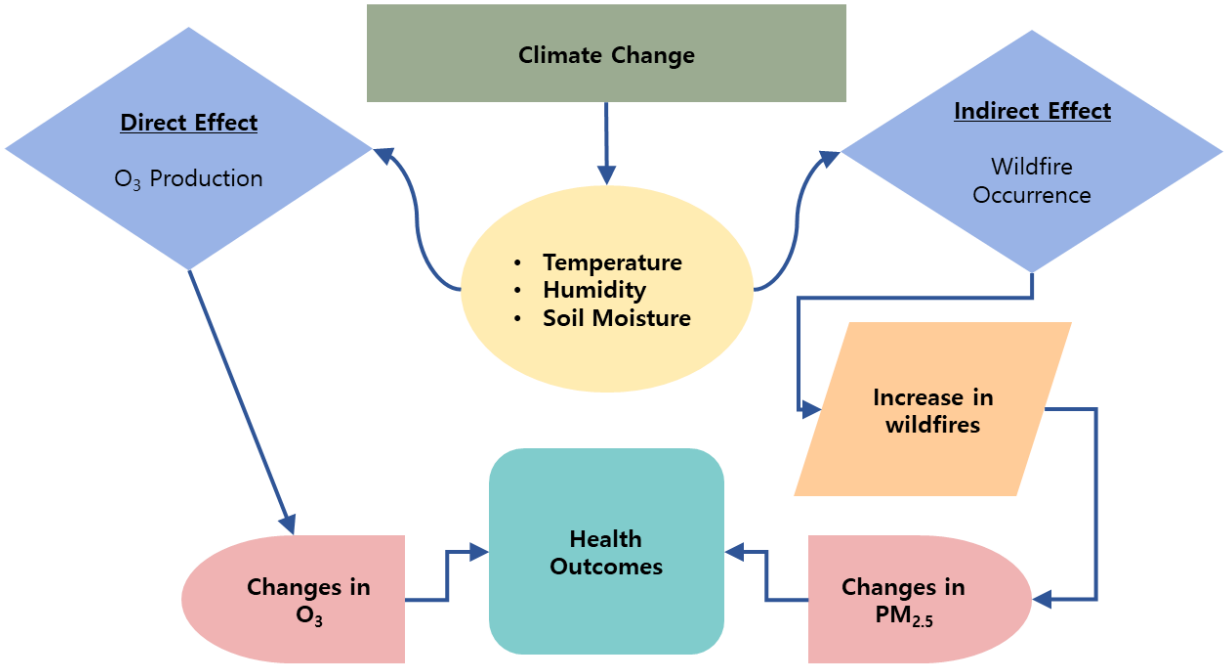
Another reason for which climate change has an uphill battle is the lag in detectable affects. On average, there is a 30-40-year gap between human behavior and its effects seen in current climate conditions.[4] For example, the temperature increases we are experiencing today are likely due to emissions from the 1970s and 1980s. This delay in resulting effects such as temperature increase, contributes significantly to the debate and reluctance of human populations of enacting change. This is of course a huge obstacle and difficult to communicate because of the complexity of the issue. Hence, inciting change becomes a struggle between scientists who know the mechanics behind the processes and society—that finds it difficult to draw these connections. Another example of this complication is that, even if we were to cut our emissions to zero tomorrow, temperatures and extreme events will continue to increase for multiple decades.

While there could be something we are unaware of, the likeliest explanation is that changes in climate are occurring due to increased human activity. CO_2 has been rapidly rising since the dawn of the Industrial Revolution (about 1750).[7] Levels of CO_2 are approximately 45% higher than they were previously. In the climate research arena, levels of CO_2 are used as a gauge for tracking the increases and decreases in most greenhouse gases (GHG). When we examine the tropospheric amounts of CO_2 a consistent pattern arises. These levels have been consistently rising since the 1950s with only seasonal oscillations. Many studies have been done to show that CO_2 and the other GHGs cause warming.[8,9] This is known without contestation. In fact, certain levels of GHG are necessary elements in the atmosphere that help to keep the troposphere habitable. For example, if the atmosphere were to instantly lose all GHGs tomorrow, the average annual temperature on earth would be close to -20° C or, roughly 0° F—our present average is near 60° F.[10]

Naturally occurring CO_2 has been tracked actively since 1958 and levels from fossils and ice cores have aided in our understanding of the history of CO_2 in our air. Historical data shows, when charted as far back as possible, CO_2 levels remained much the same across the globe with a natural seasonal oscillating pattern around 280 parts per million (ppm). Levels today are above 400 ppm. This suggests that something drastic has been increasingly raising the CO_2 levels in the atmosphere. While there has been some loss of forest and increased developed land, there is nothing natural (that scientists are aware of) that could raise the levels that high in such a relatively short period of time.[11] So, the likeliest explanation is that human activity changed drastically in the mid-18th century due to the burning of fossil fuels. Thus, while natural rises in GHGs have been seen—they tend to follow a seasonal oscillating pattern, scientists have yet to find a natural cause for the stark rises we see today.

One way that changing climate conditions have affected life on earth is the worsening of regional air quality. With the passing of the Clean Air Act of 1970, the public became more aware of ways that certain activities (i.e. exhaust from motor vehicles) might be contributing to pollution.[12] Air quality is determined by a multitude of factors—relying, in part, on changes in harmful chemicals such as ozone (O_3) and combinations of particulate matter (PM). It is important to note that changes in climatic conditions can have both direct and indirect influence on the overall air quality in a region. Potential resulting increase in air pollution is, therefore, concerning for many reasons—some of which may be in the form of potential positive feedback-loops between increasing temperature and worsening air quality. Higher temperatures tend to increase the presence and formation of many of these components, and, rising levels of at least some pollutants will contribute to continued temperature rise.

Along with the detrimental effects to visibility and adaptation, climate changes can have serious effects on human health. Indeed, climate change can be linked to multiple pathways that influence human health. Air quality, as is our focus here, is greatly affected by a host of climate conditions such as temperature, humidity, and storm patterns. In this body of work, we investigate the direct effect of change on tropospheric O_3 production and the indirect effects of change on air quality due to increased wildfire activity (see Figure 1-1 below).



Intro Figure 1- 1

Adverse Health Effects of Climate Change. *Examples of the impact of climate change on health through multiple paths.*

1.2 Direct effects of climate change: tropospheric ozone

Ozone (O_3) is a common secondary pollutant found in earth's atmosphere. In the stratosphere, O_3 is a necessary element to protect the planet from harmful ultraviolet (UV) radiation that would otherwise damage life found in the lower portion of the atmosphere known as the troposphere. The chemical constituents of the tropospheric atmosphere play a vital role in sustaining life. Chemicals like O_3 in the troposphere, however, can have detrimental effects on humans and other life. O_3 is considered a secondary pollutant, meaning that it is not directly emitted but is formed by other emissions. O_3 is produced by various combinations of precursor pollutants (i.e., volatile organic carbons (VOCs) and nitrogen oxides (NOx)) in the presence of sunlight and heat.[13] This is why O_3 values tend to be higher in the summer, often with visible smog in the atmosphere. O_3 in the lower levels of the atmosphere are expected to increase in future decades due to this reliance on sunlight for formation, continued heating, increases in solar radiation and production of precursory chemicals.

1.2.1 Toxicology of tropospheric O_3

As far back as the 1950s, the scientific community began to study adverse effects of O_3 exposure.[14] The primary toxic effects come from oxidation and generation of free radicals. Additionally, O_3 can decrease levels of compounds that are necessary in multiple human systems. These primary compounds include multiple antioxidants, such as ascorbate. Increases of protein oxidation is also present—resulting in what is referred to as oxidative stress.[15] Multiple in-vitro and in-vivo studies support these statements. For example, in rats, exposure to O_3 induced body weight loss, lung lesions, and oxidative stress. Additionally, early studies found that O_3 had the potential to affect other organ systems.[16,17] In conjunction with other studies, the push for cleaner air culminated in the Clean Air Act of 1970 and, since then, studies have become increasingly

more focused and complex.[18,19] Early studies in human subjects began in the 1960s investigating the impact of O_3 exposure on forced expiratory volume. In these controlled studies, an increase in shallow breaths was documented. It was also shown that increased respiration during exercise compounded the effects of exposure. While there are many pathways which O_3 can affect human health, the most prominent is the increase in reactive oxygen species—which, in turn, can cause oxidative stress on the respiratory system. This is generally seen through increased airway responsiveness, with more extreme responses seen in asthmatics and other individuals with pre-existing respiratory conditions.

1.3 Direct effects of climate change on wildfire

Wildfire activity has been steadily increasing across the globe for the last few decades. These increases are evident in the heightened frequency, size and severity of the fires.[20-22] There are several meteorological and land use patterns that contribute to enhanced wildfire conditions, including higher temperatures, lower humidity, lower soil moisture content, and higher winds. Similarly, fuel sources (i.e., vegetation) and forest encroachment (converting forest space to agricultural or developed land) are large components of wildfire activity. As seen in recent history, wildfires have increased and worsened in response to changes in these and other factors.

Wildfires require a myriad of elements in order to start—we have touched on temperatures and soil moisture content; however, fires also require an igniting source. Some of these include lightning and human error, and, increased contact with plentiful fuel sources can complicate the human error component. For example, population continues to expand and inhabit more and more area on the globe. This is evident in deforestation and continued encroachment into forested or fuel-rich areas. This “intrusion” can be a result of the expansion of both urban and rural

communities closer to previously natural areas. This has been exceptionally displayed in the last few fire seasons in the state of California, where, despite the ignition source, property and human life were greatly impacted due to proximity to large fuel sources.[23]

1.3.1 Indirect effects of climate change: $PM_{2.5}$ from smoke

The indirect effects of climate change on air quality via increasing wildfire activity has been recorded in past years. One of the many negative effects of wildfire activity is the resulting smoke plumes. Generally, the air surrounding a fire has high levels of material potentially toxic to living organisms[24,25]. A primary component of smoke is a mixture of particles (including both solids and liquid droplets) suspended in the air known as particulate matter (PM). PM constitutes a mixture of various chemical species including metals, nitrates, sulfates, and organic compounds.[26] A general distinction used in delineating PM is particle size. Coarse PM or PM10 denotes particles with an aerodynamic diameter between 2.5 and 10 micrometers (μm) and $PM_{2.5}$ (or "fine PM") refers to particles that measure less than 2.5 μm in aerodynamic diameter. PM exposure is associated with a myriad of health problems, including asthma and other respiratory complaints.[27-30] The extent of the exposure is related to the size of the particle—which determines how deep the particle can travel into the respiratory tract and bloodstream.[26,30-34] Generally, smaller particles travel further into the respiratory system and have a stronger association with adverse health outcomes, including asthma exacerbation, decreased lung function, and even cardiovascular complaints.[34] Hence, a consideration of $PM_{2.5}$ exposures is necessary to better define potential future impacts of air quality on human health.

States in the Rocky Mountain Region of the US (including Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, and Wyoming) tend to be highly affected, with smoke plumes from individual fires often crossing multiple states.[35] These states have experienced this

heightened activity coupled with steady increases in temperature, reduced rainfall, and soil moisture deficits.[34,36-38] Again, this has been evident in the recent past and, with climate change, worsening conditions will continue to feed the ignition of wildfires.

One of the biggest issues, however, with studying wildfire exposure is the difficulty of assigning actual exposure amounts due solely to smoke sources. Lessons learned from studies involving satellite-derived PM can aid in understanding the effects of smoke exposure. Previously, many studies have substituted ambient urban PM exposures for wildfire exposures.[20,29] This is a potentially flawed practice since wildfire particulate matter can differ in composition from urban PM composition.[20,39] While there is a wide range of $PM_{2.5}$ found in ambient air at any given time, smoke from wildfires can produce significantly higher exposures to harmful compounds (specifically organic compounds) than are normally found in non-fire, urban settings.[40-44] Each of these substances can affect the body differently depending on the dose or concentration of the exposure. These toxicological differences may lead to differences in how particulate matter from wildfire affects the human body. As stated previously, a large concern is how far these substances penetrate human biological systems.

1.3.2 Toxicology of wildfire $PM_{2.5}$

Multiple toxicological studies indicate that wildfire $PM_{2.5}$ may have different constituents than urban $PM_{2.5}$ and this suggests potential differences in health outcomes from this source.[39,45,46] Small particles such as organic carbon may be responsible for stimulation of macrophage activity in the alveolar sacs, causing the release of proinflammatory cytokines. This action can eventually lead to increased oxidative stress.[47,48] Wegesser et al. (2009) observed significant changes in macrophage and neutrophil counts in mouse lung samples exposed to wildfire PM compared to ambient air PM.[44] An additional study by the same group expounded their findings, showing that

the concentration of PAHs (polycyclic aromatic hydrocarbons) in fire smoke could be 50-fold higher in fine PM over coarse PM.[49] Coarse PM particles accounted for more of the inflammatory response and coarse PM from wildfire was shown to induce greater oxidative stress than ambient conditions. Additionally, wildfire PM caused significant antioxidant depletion over the ambient samples.[49] In a toxicology study at the University of Southern California, Verma et al. (2009) found that the overall number concentration of particles increased by at least 2-fold during a fire event. Concentrations of CO and NO also increased (3-fold) with ozone and nitrogen dioxide minimally affected. Trace elements of manganese, potassium, phosphorus, and magnesium were all statistically elevated in wildfire samples. Verma et al. (2009) also documented higher concentrations of organic carbon during the fire event.[43] Franzi et al. (2011) and Wong et al. (2011) looked specifically at the inflammatory responses due to wildfire smoke exposure. Wildfire PM was approximately 5 times more toxic to lung macrophages than ambient PM. Both papers show significant changes in reactive oxygen species and subsequent oxidative stress. Overwhelmingly, the additional oxidative stress placed on the human physiology by wildfire PM (both fine and coarse) leads to significantly higher cell degeneration and apoptosis.[45,46]

1.4 Intersection between health, climate, air quality and wildfire events

While the effects of climate change alone may not have a large impact on ambient particulate matter (PM), a link between climate change and an increase in wildfire episodes has been identified.[50] This relationship can greatly affect the air quality on any given day during fire seasons. Taken together, the potentially complicated intersection between climate change, wildfire potential, and that of human health poses a unique concern for both environmental and health professionals. Specifically, while physical changes are usually the most readily apparent damage

from wildfires, smoke exposure can be very hazardous to human health and has yet to receive the same attention. Thus, moving forward, researchers need to explore options to better quantify wildfire smoke exposure and links to specific health outcomes. This combination of quantifying exposure and understanding the resultant health responses is critical to future policy, emergency response planning, and strategic access to health care during these events.

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2. SIGNIFICANCE

This project investigates potential climate-change driven effects on $PM_{2.5}$ and O_3 with an emphasis on the effects of wildfires. Through a combination of climate modeling and downscaling methods, this research builds connections between ambient air pollution, anthropogenic sources, wildfire smoke exposure, and predicted climate change. The results of this approach may help to answer key questions and to fill gaps in our current knowledge and predictive capabilities regarding wildfire exposure.

2.1 Study Rationale

Poor air quality poses a substantial health burden that spans all ages. While great strides have been made in certain air pollutants, others continue to increase. The WHO has estimated that more than 9 out of 10 people worldwide consistently breathe potentially toxic air. This exposure leads to an estimated 7 million deaths per year (https://www.who.int/airpollution/en/). Also, more than one third of deaths from stroke, heart disease, and lung cancer has been associated with toxic air exposures.[1] In addition to this issue, there is the complication that climate change poses as the world gets hotter, more populated, and more industrialized. Increases in factors such as these feed back into the climate-health process and compounds the issue further.

Understanding the importance of improving our air quality requires observations and investigations into the pollutants and their sources. While tackling *all* air pollution at once seems daunting, it is more manageable to delve into the effects of individual pollutants or sources. The following aims were pursued to better understand the effects of O_3 (from both emissions and climate change) and increasing wildfire smoke.

2.2 Description of aims

Aim 1: Define the state of the science and the potential viability of alternative methods for separating emissions and climate change effects using climate modeling under multiple emissions scenarios. Hypothesis 1: Increased ozone concentrations under a higher anthropogenic emissions scenario will have a greater impact on human mortality in the future. Additionally, the separation of climate change and emissions effects is possible using a hybrid method of both statistical and dynamical downscaling and will offer a metric for the impact of planned emissions mitigation.

Aim 2: Describe the impacts of wildfires and urban pollution on air quality and population health in the state of Colorado during the fire seasons of 2011-2014. Assess the impact of wildfire smoke based on current regulations and policies. Hypothesis 2: Wildfires related $PM_{2.5}$ is associated with multiple health outcomes. Differences in effect will be seen when stratified on age.

Aim 3: Estimate the impacts of increased future wildfires and urban pollution on air quality and population health in the Rocky Mountains Region. Assess the impact of future wildfire smoke predictions based on current regulations and policies. Hypothesis 3: Future wildfires and urban air pollution will increase in number and/or severity and will have a negative impact on the overall public health of the Rocky Mountain Region.

2.3 Synopsis of purpose and intent

The following chapters represent the body of work designed to achieve the above aims. Chapters 3 explores the complexities of scale, separation of climate and emissions policy, and specific modeling parameters that are prominent concerns when dealing with the study of air

quality and human health. Chapter 4 expands on the methods from these chapters to investigate the potential link between wildfire events and adverse health outcomes. Chapter 5 addresses the potential future impact of smoke $PM_{2.5}$ exposure using what we have learned from the previous chapters.

3. SEPARATING THE EFFECTS OF CLIMATE CHANGE AND EMISSIONS

[Manuscript 1]

The Impact of Climate Change and Emissions Control on Future Ozone Levels: Implications for Human Health

Jennifer D. Stowell, Young-min Kim, Yang Gao, Joshua S. Fu, Howard H. Chang, Yang Liu

3.1 Abstract

Overwhelming evidence has shown that, from the Industrial Revolution to the present, human activities influence ground-level ozone (O_3) concentrations. Past studies demonstrate links between O_3 exposure and health. However, knowledge gaps remain in our understanding concerning the impacts of climate change mitigation policies on O_3 concentrations and health. Using a hybrid downscaling approach, we evaluated the separate impact of climate change and emission control policies on O_3 levels and associated excess mortality in the US in the 2050s under two Representative Concentration Pathways (RCPs). We show that, by the 2050s, under RCP4.5, increased O_3 levels due to combined climate change and emission control policies, could contribute to an increase of approximately 50 premature deaths annually nationwide in the US. The biggest impact, however, is seen under RCP8.5, where rises in O_3 concentrations are expected to result in over 2,200 additional premature deaths annually. The largest increases in O_3 are seen in RCP8.5 in the Northeast, the Southeast, the Central, and the West regions of the US. Additionally, when O_3 increases are examined by climate change and emissions contributions separately, the benefits of emissions mitigation efforts may significantly outweigh the effects of climate change mitigation policies on O_3-related mortality.

3.2 Background

Since the Clean Air Act of 1970, atmospheric ozone (O_3) concentrations have declined in the US. Nevertheless, the American Lung Association reported that, as of 2013, over 138 million people in the US (~44%) continue to live in areas where O_3 levels exceed regulatory standards.[1] Among common air pollutants that impact public health, O_3 is one of the most detrimental. Risk of O_3-related adverse outcomes is a public health concern due to widespread O_3 exposure, which is ubiquitous in industrialized regions. Research has consistently linked O_3 exposure to a variety of adverse health outcomes including increased emergency room (ER) visits and hospitalizations, asthma exacerbation, cardiovascular stress, impaired lung function, and premature death.[2-12] Multiple studies have demonstrated the connections between climate change to O_3 concentrations and these potential health outcomes. For example, Tagaris et al. found the highest climate-induced O_3 increases coincided with the most densely populated areas in the US and increases in national premature mortality of approximately 300 additional deaths annually.[8] Bell et al. also showed that climate change-induced O_3 increases are associated with significant increases in premature mortality and ER/hospital admissions.[3,4] Additionally, by comparing future O_3 concentrations and associated adverse health outcomes from seven published studies, Post et al. showed substantial heterogeneity in the projections when different models and methods were considered.[7] One such example found in this comparison of studies demonstrated a large discrepancy in O_3-related excess mortality due to climate change among the studies examined (ranging from -600 deaths to over 2,500 deaths annually).

The primary drivers of ground-level O_3 generation are precursor emissions (nitrogen oxides (NOx) and volatile organic compounds (VOCs)), presence of methane, and favorable meteorological conditions.[13-15] Because both emissions and meteorology vary in space, O_3

concentrations can be spatially heterogeneous at the scale of a few kilometers to tens of kilometers.[16] Therefore, spatially-resolved estimates of O_3 levels are important when evaluating its potential impact on air quality and human health as well as developing applicable mitigation and adaptation policies. However, as Post et al. reported, the coarse spatial resolution of global climate models (GCMs) cannot resolve the fine-scale features in future O_3 levels.[7]

Both dynamical and statistical downscaling approaches have been developed to address this resolution incongruence. Dynamical downscaling involves executing high-resolution regional climate models (RCMs) and air quality models using GCM outputs as boundary conditions. This method integrates atmospheric chemistry composition, allowing for extrapolation of future atmospheric conditions.[15] However, the high computational demand (due to high-resolution, full-chemistry simulations) limits the application to multiple GCM outputs and reduces the availability of these methods.[17-19] Previous studies have used dynamical downscaling methods to study the impact of climate change on future O_3 and air quality. At 36 km resolution, Nolte et al. used dynamical downscaling methods to show significant increases in summer O_3 and a lengthening of the O_3 season under a high emissions scenario as well as substantial decreases during the summer season under a lower emissions scenario.[15]

Statistical downscaling methods use efficient statistical methods based on historical atmospheric patterns to relate coarse-resolution GCM simulations to finer grid results, which is much less computationally demanding.[17] Previous studies have investigated the relationship between O_3 and changes in meteorological conditions using statistical models. For example, Cox and Chu examined 100 meteorological variables for potential effects on ambient O_3, and found that maximum surface temperature, wind speed, relative humidity, mixing layer, and cloud cover were significant. Both Dawson et al. and Camalier et al. found similar statistically significant

results showing that daily maximum temperature, relative and absolute humidity, wind speeds, and mixing height greatly affect O_3 concentration.[13,20,21] The limitations of statistical downscaling are mainly due to the assumption that the statistical association between O_3 levels and meteorological conditions will remain the same in the future, which may not be realistic given potential future variations in atmospheric chemistry and emissions.[22]

In addition to air pollution levels estimated at fine spatial scales, the impacts on future O_3 levels due to climate change and future emissions need to be assessed separately for effective mitigation measures. Above all, the impact of air pollution emissions control can have a more immediate effect on air quality and subsequent human health than the effects from slowing down climate change.[23] Previously utilized emission scenarios, however, do not allow for such separation of O_3 levels due to climate change and emissions. The latest Representative Concentration Pathways (RCPs) differ from previous emission scenarios such as the Special Report on Emissions Scenarios (SRES) by integrating current and planned environmental policies.[24-26] As a result, RCP-based climate model simulations reflect the combined impact of both climate change and planned emission control on air pollutant levels.[24] This integrated combination provides a platform to develop methods to examine the separate contributions of climate change and emissions. There are multiple RCP scenarios with underlying population growth, economic, and emissions assumptions. RCP2.6, 4.5 and 6.0 all represent some form of improvement upon our current trajectory of growth and environmental policy. RCP8.5, however, represents a “business-as-usual” scenario in which nations choose to retain current economic, environmental, and social tracks. For example, RCP4.5 represents a future scenario with medium to low greenhouse gas emissions, medium-level air pollution, less crop land, and low population growth. RCP8.5, on the other hand, is characterized by high population growth, low to medium

crop land use, increasing trends for methane and nitrous oxide, and higher concentrations of almost all air pollutants.[26]

The objective of this study is to estimate the contribution of climate change and emissions control to future O_3 levels separately at high spatial resolution in the Continental US. We present a hybrid dynamical-statistical downscaling approach to project and separate the impacts of climate change and air pollution emissions control on future O_3 levels under both RCP4.5 and 8.5. Additionally, we expand our analysis and estimate county-level excess mortality due to projected O_3 exposure in the 2050s and evaluate the spatial and temporal patterns of associated estimated health risks. The 2050s were selected for the future projected years based on the IPCC common use of 2050 as a threshold for major global temperature divergence (i.e. potential to rise above 2°C).[27]

3.3 Data & Methods

Our four-step hybrid health impact projection approach is shown in Figure 3-1. Step 1 involves a dynamical downscaling framework following two RCPs respectively. This framework is composed of a GCM, a RCM, and an atmospheric chemistry model, which estimates county-level O_3 concentrations in the 2050s due to the combined effects of climate change and environmental policies as described in RCPs. Step 2 develops a statistical downscaling model to estimate future changes in O_3 concentrations from climate change, which uses both real-world historical climate conditions and high-resolution future climate conditions simulated by the RCM in step 1. Step 3 estimates the future change in O_3 concentrations due to emissions only by subtracting the statistical downscaling results (Step 2) from the dynamical results (Step 1). Finally,

in step 4, the results from steps 1-3 are placed in a human health context by estimating the future excess mortality due to projected changes in O_3 concentrations.

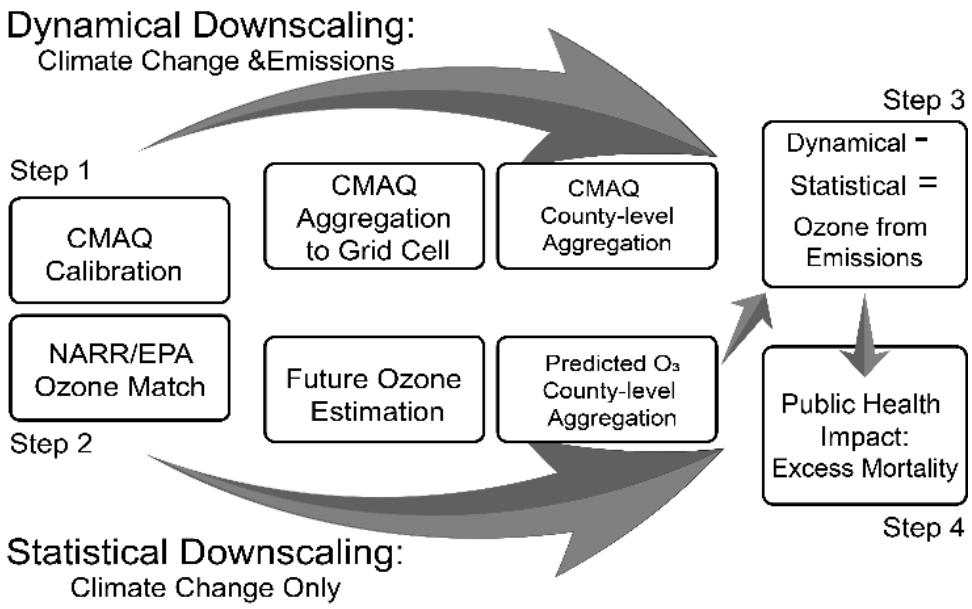

Figure 3- 1

Study Methods Flow. *Flow of study methods depicting both dynamical and statistical results. Isolation of O_3 attributable to emissions accomplished by taking the difference between the two illustrated downscaling methods. Excess deaths calculated by source for climate change only, and combined climate and emissions changes. Isolation of O_3 attributed to emissions is achieved by taking the difference between the two methods.*

3.3.1 Dynamical downscaling for O_3 due to changes in climate and air pollution emissions

The Community Earth System Model version 1.0 (CESM 1.0) is a state-of-the-art global climate model developed by the National Center for Atmospheric Research (NCAR).[28] As a fully coupled earth system model, there is a total of four components in CESM[29]: 1) the land surface component - Community Land Model (CLM4)[30]; 2) the ocean model and sea ice component -

Parallel Ocean Program version 2 (POP2)[31] and Los Alamos National Laboratory Sea Ice Model, version 4 (CICE4)[32]; 3) the atmospheric chemistry module - adapted from the Model for Ozone And Related chemical Tracers version 4 (MOZART-4)[33]; and 4) the bulk aerosol model (coupled to the atmospheric component Community Atmosphere Model, CAM4), referred to as CAM-Chem.[33,34] More details regarding the configurations of CAM-Chem have been described in previous studies.[17,35] CESM/CAM-Chem was continuously run from 2001-2059 under both RCP4.5 and RCP8.5 with spatial resolution of 0.9 degrees by 1.25 degrees.

The dynamical downscaling framework was developed to conduct high resolution simulations (12 km) at two time slices from 2001 to 2004 for the baseline historical period and 2055 to 2059 for future scenarios under RCP 4.5 and RCP 8.5.[17,18] The Weather Research and Forecasting regional climate model (WRF, version 3.2.1) and the Community Multi-scale Air Quality Model chemistry model (CMAQ, version 5.0),[36] were used in this study and detailed information on model configurations and dynamical downscaling technique was described in section 2 and 3 of Gao et al. (2013). Meteorological parameters such as hourly surface temperature, surface relative humidity, precipitation, zonal (U) and meridional (V) wind , planetary boundary layer height and pressure were generated by the WRF model whereas air pollutant concentrations such as O_3 was simulated from CMAQ.[37] The historical emissions (2001-2004) were based on US EPA's National Emission Inventory, whereas the future emissions of O_3 precursors were scaled based on RCP4.5 and RCP8.5, and more details can be found in Gao et al. 2013.[17] Thus, the CESM/WRF-CMAQ system simulates O_3 concentrations in the 2050s that reflect the influence of both climate change (i.e., changes in future meteorology) and changes in anthropogenic emissions at 12km spatial resolution.[24] The combined effect of climate and emissions on future ozone

changes was investigated in Gao et al. (2013), and this study focuses on separating the effect of climate on future ozone concentrations. The detailed method is described in a subsequent section.

We first computed differences in maximum daily average eight-hour (MDA8) O_3 between the 2000s and the 2050s for each 12 km grid cell and aggregated values to the 3,109 counties to obtain annual county-level changes. To reduce the bias of model simulation, we calibrated future CMAQ MDA8 O_3 levels based on the ratio of observed concentrations measured by the USEPA-AQS and the results of the year-round CMAQ-modeled historic MDA8 O_3 levels. A ratio method for calibration was preferred over the use of an additive bias correction. This technique was chosen primarily because, 1) the methods in this study design calculate changes between future and historical periods and an additive correction would be cancelled out, and 2) bias correction in this study is done spatially and ratio calibrations are more appropriate for capturing potential non-linearity. Each county was assigned a population-weighted centroid based on the centroids from the 2010 US Census. Using 40km radius buffers, the five closest CMAQ points to each county centroid are identified and average O_3 values were calibrated using the ratios mentioned above.[38] More details about the calibration method have been described elsewhere.[39]

3.3.2 Statistical downscaling for O_3 changes due to climate change

In order to estimate changes in O_3 levels between the 2050s and 2000s caused by climate change alone, we first developed a regression model to predict O_3 concentrations with meteorological variables from the North America Regional Reanalysis (NARR) dataset. The NARR dataset provides the base year (2001-2004) meteorological parameters for the statistical model. NARR is produced by the National Centers for Environmental Prediction and provides a wide range of observed climate parameters over North America on a 32 km grid.[40,41] Prior to modeling and analysis, we compared the CESM-WRF simulations against NARR values at the

daily level, using a 30-day moving average. Strong correlations of key variables between the two datasets confirmed the appropriateness of combining NARR and CESM-WRF in our approach (see Supplemental Table 1). For purposes of prediction, we computed annual medians of daily mean values for temperature, relative humidity, wind speed and direction, planetary boundary layer height, surface pressure and total annual precipitation for each 32 km (NARR) and 12 km (WRF) grid. We also calculated air stagnation which is defined as a day with surface daily wind speed < 3.2 m/s, wind speed at 500 hPa < 13 m/s, and slight or no precipitation (< 0.1 mm/day).[42] We then linked the MDA8 O_3 concentrations with the NARR meteorological data by selecting the nearest NARR cell to the closest USEPA O_3 monitoring site. Model development included all sites having at least two years of data (1,334 sites). In order to minimize impacts of short-term fluctuations and to focus on longer-term trends, we used a 30-day moving average window for all meteorological variables and MDA8 O_3.

To establish the associations between meteorological variables and MDA8 O_3, we developed a multiple linear regression (MLR) model. We included natural cubic splines of time (Julian day) to control for the long-term trend of O_3 concentration.[43] Usage of natural cubic splines greatly improves the coefficients of determination (R^2) for the model.[44] The basic form of the model is as below:

$$y = \beta_0 + \sum_{k=1}^{8} \beta_k x_k + ns(time) + \varepsilon$$

Equation 3- 1

where y is the 30 day moving-average MDA8 O_3 concentration; x_k is the 30 day moving-average value of the meteorological variables (temperature, relative humidity, planetary boundary height,

pressure, precipitation, and two horizontal wind components); ns(time) is the natural cubic splines of time (Julian day: four degrees of freedom), and ε is model error. We fitted this model for each EPA O_3 monitoring site. We matched the estimated regression coefficients (β_0 and β_k's) of the MLR model with the changes in meteorological variables between the 2050s and 2000s to obtain projected changes in O_3 levels. We then interpolated the site-specific O_3 changes to all 3,109 counties using a nearest-neighbor approach. The NARR-MLR model estimates the average annual amount of change in O_3 attributable to climate change alone. We then demonstrated the appropriateness of the chosen model using a 10-fold cross validation.

3.3.3 Future O_3 changes due to changes of air pollution emissions

In order to isolate changes in O_3 concentration attributable to future air pollution emissions alone, we calculated the differences between the concentrations generated in the previous two steps. The hybrid dynamical downscaling model involving the CMAQ-simulated O_3 values represents the changes in future concentration attributable to a combination of climate change and change of anthropogenic emissions (ΔO_3 climate change + emissions). The statistical downscaling model, on the other hand, is an estimation of changes in concentration due to climate change alone (ΔO_3 climate change). Thus, subtracting the statistical model (climate change only) from the dynamical model (climate change and emissions) we are left with an estimation of the average annual contributions (ppb) from air pollution emissions control policies alone ((ΔO_3 emissions; see Equation (2)).

$$\Delta O_{3\ climate\ change+emissions} - \Delta O_{3\ climate\ change} = \Delta O_{3\ emissions}$$

Equation 3- 2

3.3.4 Population health impact of future O_3 changes

Population and mortality rate estimates, as well as concentration response function (CRF) coefficients are required to estimate the excess mortality (EM) due to future changes in MDA8 O_3.[1,7,45] We utilize the four population projections developed by the Integrated Climate and Land-Use Scenarios (ICLUS) project: ICLUS A1, B1, A2 and B2. ICLUS converts the global Special Report on Emissions Scenarios (SRES) settings into county-level projections.[1,7,46,47] The SRES A1 storyline represents a scenario of rapid development, and slow population growth, while the A2 scenario represents regional economic development and much higher fertility rates. The B1 scenario assumes similar conditions to A1, with a larger emphasis on sustainable growth and lower domestic migration. The B2 scenario includes regional growth similar to A1 with moderate population growth, and much lower migration.[46] A comparison between the previous SRES projections and the new RCP projections has shown that climate conditions under RCP8.5 fall between the previous SRES A1 and A2 projections and RCP4.5 closely resembles atmospheric conditions under SRES B1.[48] Each projected population was applied to both RCP4.5 and RCP8.5 scenarios to reflect the differences between low and high emissions scenarios with varying population conditions. It is important to note that ICLUS scenarios have varying spatial resolutions due to differing projections in land and economic growth and, therefore, absolute deaths are difficult to compare across scenarios.[49] Therefore, we chose to normalize each ICLUS scenario independently from one another in order to compare national impact and across counties within each ICLUS scenario.

For the calculation of baseline mortality incidence, we used the predicted mortality rate for the year of 2050 at county level which is available from the Environmental Benefits Mapping and Analysis Program Community Edition 1.0.8 (BenMAP-CE) developed by the US Environmental

Protection Agency.[50] The BenMAP-CE provides county-specific mortality rates derived from projected age-specific ratios of 2050 mortality rates to 2005 mortality rates.

We based CRFs on the association between non-accidental, all-cause mortality and short-term exposure to MDA8 O_3 as estimated by Bell et al., (RR = 1.0064 (95% CI: 1.0041-1.0086) per 15 ppb).[4] The estimate from Bell et al. comes from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) dataset and cover 95 major US cities.[4] We estimated changes in EM at the county level using the following equation: [7,45]

$$\Delta y_i = POP_i \times MR_i \times [e^{\gamma \times \Delta C_i} - 1]$$

Equation 3- 3

where Δy is the expected number of deaths per year that may be attributed to changing air pollution levels (i.e., O_3) at county i, POPi is population of county i; MRi is population mortality rate; γ is the concentration-response coefficient for MDA8 O_3; and ΔCi is the difference in concentrations of MDA8 O_3 between future (2050s) and baseline (2000s) levels of MDA8 O_3.

To evaluate the uncertainty of EM estimates attributable to the ranges of the CRF coefficients and mortality rates, we applied Monte Carlo simulations (10,000 random samples) for each county, assuming a normal distribution of independent county-specific means, mortality rates and standard errors of the population and concentration variables. We then estimated climate-region and national level EM estimates by summing the county-level EMs. We also estimate 95% confidence intervals (CIs) of the EMs based on the mean and standard deviation of the Monte Carlo simulations at both levels.

3.4 Results

3.4.1 Future O_3 changes due to climate change

CESM/WRF simulations indicate wide spatial variations in the meteorological variables used as the future model inputs (step 2) (Supplemental Figure 3-1). Annual medians of daily mean temperature show an increase of approximately 1.2°C and 2.3°C across the continental US under RCP4.5 and RCP8.5, respectively, showing greater increases in the northeast, southeast, central, northwest, and southern climate regions than in the west and southwest regions (see Figure 3-2A for NOAA-defined climate regions). Annual average relative humidity (RH) could increase 0.45% under RCP4.5and 1.1% under RCP8.5, with higher increases in the Central region. Averages of planetary boundary layer height are projected to decrease by 24.0 m under RCP4.5 and 25.2 m under RCP8.5. Meridional (N/S) wind speed will decrease in most inland areas of the US under both RCPs, with highest decreases in the Northwest region. Zonal (E/W) wind speed will decrease in much of the U.S. with some increase seen in the West and Southwest regions.

Figure 3-2 depicts the MLR-estimated change in annual mean MDA8 O_3 concentrations (2050s vs. 2000s) for both RCP4.5 and RCP8.5. For all 1,334 O_3 monitoring sites, the MLR model performs well with relatively high R^2 values (average R^2=0.74). Figures 3-2C and 2D show the MDA8 O_3 changes between the 2000s and the 2050s from the statistical downscaling model.

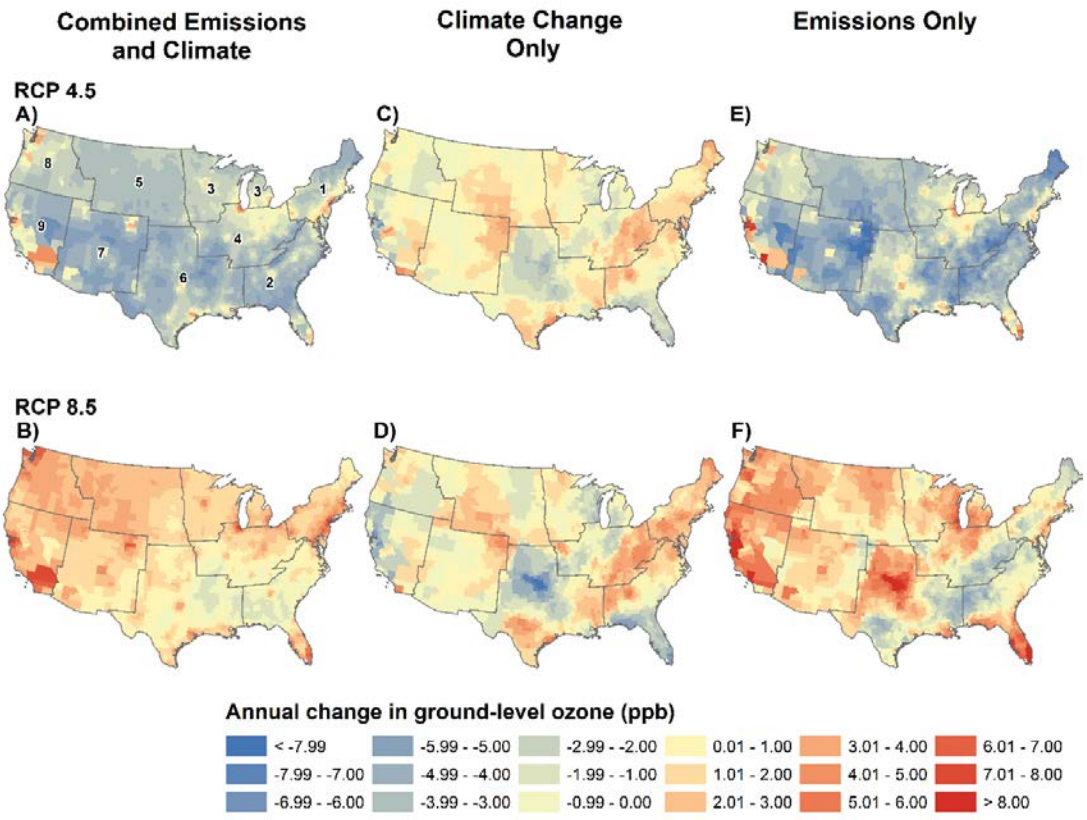

Figure 3- 2

Average Annual Change in Tropospheric Ozone*. Changes in O_3 Concentrations between 2000s and 2050s. (A) O_3 difference from combined climate change and emissions under RCP4.5; (B) O_3 difference from combined climate change and emissions under RCP8.5; (C) O3 difference from climate change under RCP4.5; (D) O_3 difference from climate change under RCP8.5; (E) O_3 difference from emissions only under RCP4.5; and (F) O_3 difference from emissions only under RCP8.5. Numbers represent US Climate Regions as defined by the National Climatic Data Center: 1. Northeast, 2. Southeast, 3. East North Central, 4. Central, 5. West North Central, 6. South, 7. Southwest, 8. Northwest, and 9. West.*

Climate change alone appears to cause some increase in MDA8 O_3 average annual (ppb) concentration in most of the continental US except for some counties in the West and South regions. Overall, increases in MDA8 O_3 due to climate change is projected to be 0.34 ppb (std. error 0.03) and 0.50 ppb (std. error: 0.04) under RCP4.5 and RCP8.5, respectively. The model performance was confirmed using a 10-fold cross validation comparing the results of the study model to the results from a series of models configured using both training and testing data. The validation resulted in less than 1% difference in root mean square error (RMSE, ~0.00001%).

3.4.2 Future O_3 levels due to climate change and changes in emissions

As simulated by the CESM/WRF-CMAQ system, modest climate change and strict emissions control under RCP4.5 result in a nationwide decrease in future MDA8 O_3 levels (on average 2.85 ppb, std. error: 0.03) except in a few large urban centers including Los Angeles, Chicago, and New York (Figure 3-2A). These hotspots of high O_3 under RCP4.5 are likely caused by NO_x decreases in large urban areas, resulting in reduced O_3 titration and higher concentrations of O_3.[17] Under RCP8.5, national mean O_3 level is projected to increase by ~1.33 ppb annually (std. error: 0.03, Figure 3-2B). With greater temperature rise, climate change only caused O_3 levels to increase in more regions under RCP8.5 than under RCP4.5, but the spatial patterns of O_3 changes are similar under these two scenarios (Figure 3-2D).

3.4.3 Future O_3 change due to changes of air pollution emissions

Under RCP4.5, average national MDA8 O_3 due to emissions alone decreases by 3.19 ppb (std. error: 0.04, Figure 3-2E). Comparing Figure 3-2C and 2E, it is clear that the O_3 reduction due to the assumed lower precursor emissions outweighs the O_3 increase due to higher temperature under RCP4.5. On the other hand, changes in future emissions alone under RCP8.5 would cause O_3 levels to increase in the US except in the mid-Atlantic and Southeastern region (Figure 3-2F).

Despite the emission reduction of O_3 precursors across all RCPs (including CO, NO_x and MVOCs), nationally averaged MDA8 O_3 may increase by 0.93 ppb (std. error: 0.05) in the 2050s under RCP8.5.

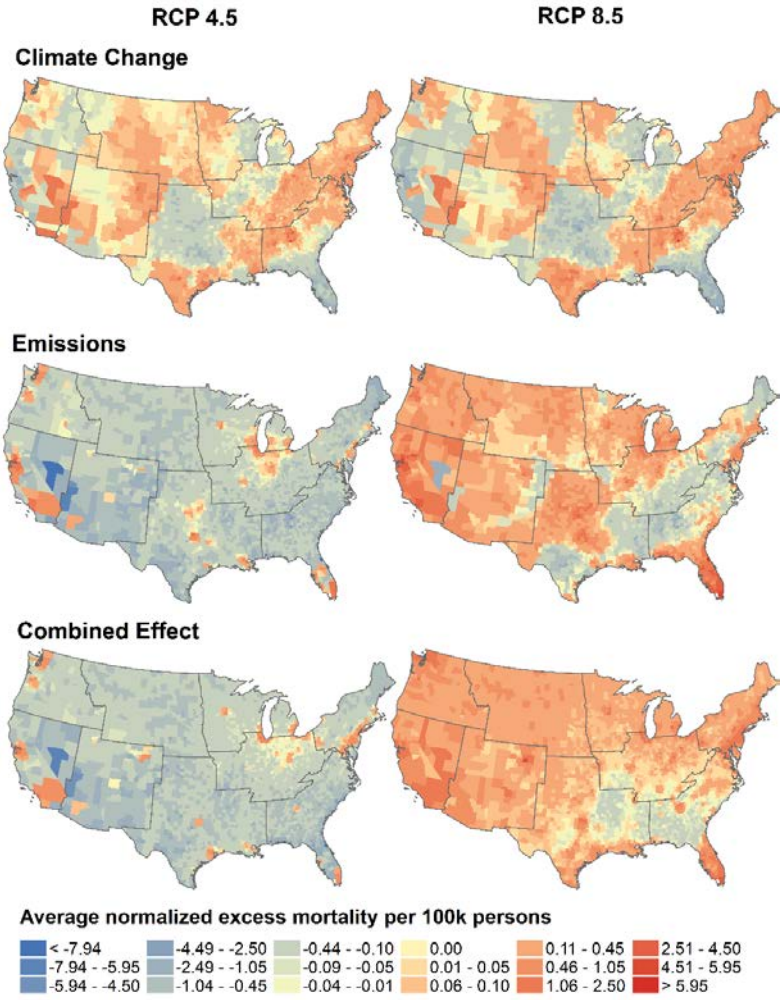

Figure 3- 3

Change in Mortality. *RCPs 4.5 and 8.5 using ICLUS A2 Scenario. Annual averaged, county-level excess mortality normalized by population. RCP4.5 (low emissions scenario) and RCP8.5 (high emissions scenario) results displayed by contributing source (combined effects, climate change*

and anthropogenic emissions). The combined effects represent the effects of both climate change and emissions.

	Climate change only		Emissions control only		Combined effect	
REGION	***RCP4.5***	***RCP8.5***	***RCP4.5***	***RCP8.5***	***RCP4.5***	***RCP8.5***
National	72 (SE=456)	47 (SE=525)	-41 (SE=1037)	2167 (SE=1386)	50 (SE=615)	2217 (SE=900)
Northeast	204 (SE=12)	330 (SE=17)	-2 (SE=28)	365 (SE=43)	208 (SE=40)	678 (SE=59)
Southeast	-47 (SE=10)	-100 (SE=17)	-186 (SE=14)	405 (SE=35)	-237 (SE=5)	298 (SE=19)
East North Central	-18 (SE=1)	-35 (SE=1)	22 (SE=1)	161 (SE=3)	4 (SE=1)	126 (SE=3)
Central	76 (SE=4)	87 (SE=4)	-42 (SE=7)	161 (SE=12)	30 (SE=11)	239 (SE=15)
West North Central	5 (SE=1)	5 (SE=1)	-20 (SE=1)	9 (SE=1)	-15 (SE=1)	13 (SE=1)
South	-11 (SE=3)	-13 (SE=3)	-80 (SE=3)	169 (SE=6)	-91 (SE=4)	152 (SE=6)
Southwest	20 (SE=7)	18 (SE=7)	-71 (SE=7)	98 (SE=15)	-52 (SE=3)	114 (SE=13)
Northwest	-12 (SE=2)	-5 (SE=2)	46 (SE=6)	122 (SE=8)	34 (SE=5)	112 (SE=9)
West	-143 (SE=154)	-170 (SE=182)	292 (SE=355)	678 (SE=468)	165 (SE=199)	475 (SE=287)

Table 1- 1

Short Term Excess Mortality under the ICLUS A2 Population Scenario. *Projected excess deaths using ICLUS A2 population scenarios attributable to climate change only, anthropogenic*

emissions only, and combined effects of both climate change and emissions for 2050s from baseline 2000s by US climatic region. (SE: standard error).

3.4.4 Population health impact of future O_3

Figure 3-3 displays the annual average, population-normalized county estimates for excess mortality in 2055-2059 for the ICLUS A2 scenario (high population growth) for both RCP4.5 and RCP8.5 per 100,000 persons. Emissions sources appear to play a significant role, especially in RCP8.5, with large increases in O_3-related mortality for much of the West, Midwest, and Eastern US. Table 1 shows the estimated O_3-related excess deaths by climate region for the ICLUS A2 scenario under RCP4.5 and RCP8.5. The highest excess deaths are found from emissions-only sources (compared to climate change only sources) under RCP8.5 with the West, Southeast, and Northeast regions showing the largest impact. Similar patterns are found for other ICLUS population scenarios and data can be found in Supplemental Tables 2 and 3.

Figure 3-4 highlights the state of California—an area of the US known for its pollution and related health issues. Shown together are the county-level O_3 and mortality results for RCP8.5. Hot spots of O_3 concentration increases and O_3-related EM can be seen in areas surrounding the San Francisco Bay and Los Angeles County. Notably, changes in O_3 concentration due to emissions appear to be highest in counties such as Los Angeles, Monterey, Orange, and San Joaquin. Meanwhile, O_3 concentration due to emissions appear to be lowest in the upper counties and central valley. Overall, under ICLUS A2 and RCP8.5, O_3-related EM due to climate change alone may increase by 180 deaths/year (std. error: 23.92) while under RCP4.5, 110 (std. error: 21.63) excess deaths may be expected due to climate change. However, emissions may have a greater impact on estimated excess mortality in California exhibiting an increase of approximately

315 (std. error: 21.21) deaths under RCP8.5. Under RCP4.5 excess deaths due to emissions can be expected to increase at much lower rates with only 87 (std. error: 8.98) deaths/year in the 2050s. In predictions including both sources, excess deaths under RCP8.5 for the state of California may exceed 486 deaths/year (std. error: 44.13) while scenarios using RCP4.5 predict lower increases of 230 deaths/year (std. error: 30.55).

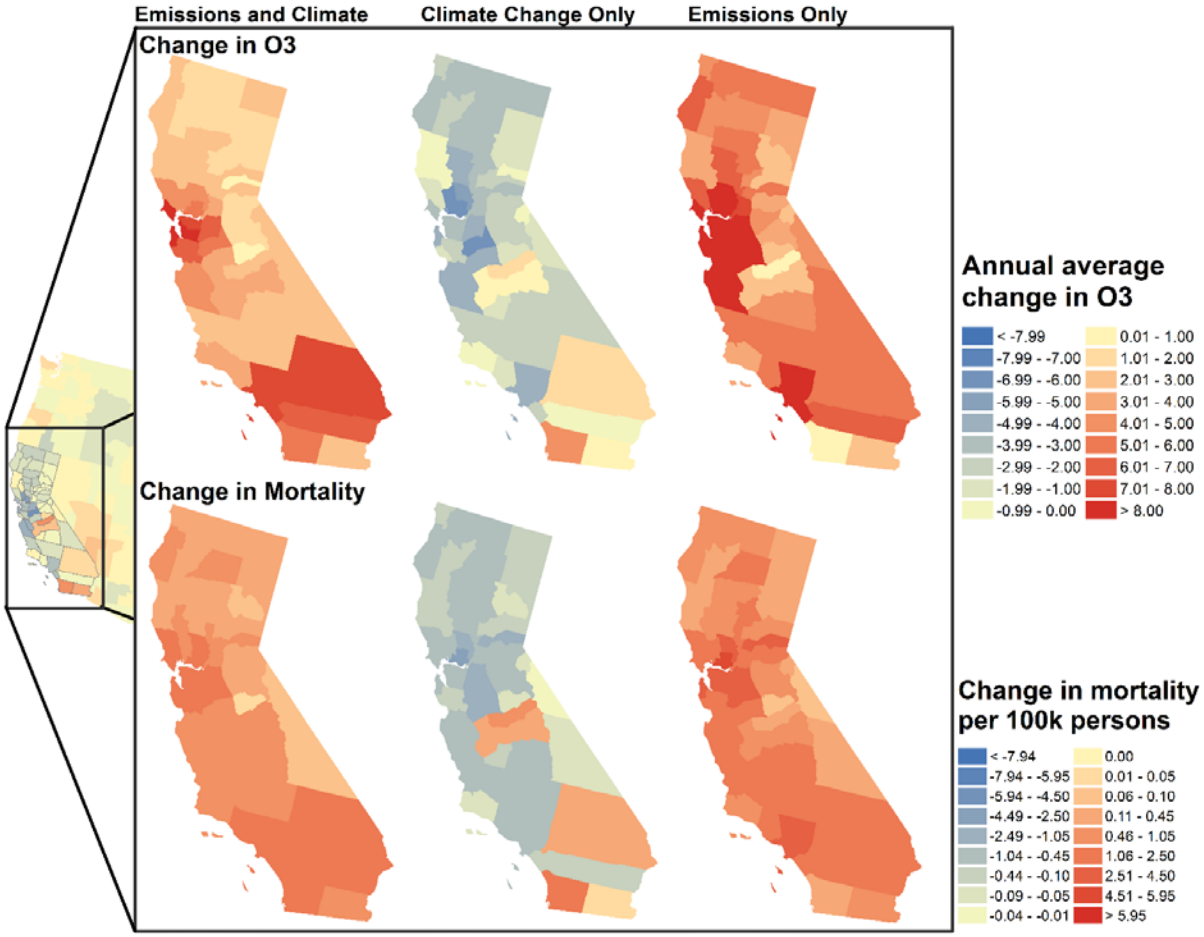

Figure 3- 4

California Case Study. *Annual averaged, county-level changes in O_3 and excess mortality normalized by population depicted for the state of California under RCP8.5.*

3.5 Discussion

3.5.1 Summary and impact of results

Our results point to significant differences in the contributions of climate and emissions mitigation to future O_3. Under both RCP scenarios, future emissions control policy will likely have a substantial impact on O_3 levels and its associated health effects. Our hybrid downscaling approach suggests that changes in emissions may be the source of the main incongruities between RCP4.5 and RCP8.5. Thus, while climate change alone may cause some adverse health effects due to poorer air quality, substantial and more immediate health benefits may be achieved by emission mitigation of O_3 precursors regardless of changing climate conditions especially under RCP8.5.

In all RCPs, most emissions of O_3 precursors are expected to decrease in the US due to nearly worldwide implementation of stricter environmental policies. However, the results under RCP8.5 suggest a rise in future O_3 concentrations. It is important to note that, with the climate change effect removed, the predictions still include background O_3 conditions. As is explained in Gao et al. 2013, the O_3 changes in RCP8.5 primarily occurs in spring and winter, especially over the western US, due to increases in global methane emissions (60% by the end of 2050s). In summer and fall, particularly over the eastern US, the increase of methane emissions is offset by the large reduction in anthropogenic VOC and NOx emissions, leading to decrease of O_3 concentrations.[17] Increases of O_3 in RCP 8.5 were also found across a majority of the troposphere in Young et al. [2013], which attributes the higher ozone concentration to large increase of methane and greater stratospheric influx.[52] The tropospheric ozone increase in RCP 8.5 was shown to enhance background ozone, further impacting regional modeling results through the boundary conditions. This enhanced background ozone can lead to elevated spring ozone in the western US and has also been documented by Lin et al. (2012).[53]

3.5.2 Comparison with current literature

Several recent studies have demonstrated findings comparable to our results regarding the ozone changes in RCP 4.5 and RCP 8.5.[54-56] In particular, as is shown in Gao et al. (2013; Figure 3-5c), the ozone increases largely disappear when the boundary conditions were cleaner and methane emission increases were removed in RCP 8.5. The effect of global background ozone increase on regional downscaling results through boundary conditions was further shown by a more recent sensitivity study (Yahya et al. (2017), Figure 7i vs. Figure 7iii), showing clearly that the high ozone boundary conditions inherited from global models under RCP 8.5 contributed to majority of the ozone increases in US in regional model results.[56] Thus, the combination of global background ozone increases and methane emission increases may be the main contributing factor for increases in O_3 and O_3-related EM under RCP8.5. Researchers have evaluated and proposed that the control of methane emissions may be an efficient way to reduce both tropospheric O_3 and radiative forcing.[15,57,58]

It is important to note that the estimated EM attributable to O_3 changes vary significantly, showing both negative and positive results by region, as seen in Table 1 and Supplemental Tables 2 and 3. Additionally, relatively high standard deviations are captured for most of the regional predictions. These reflect the varying EM from county-to-county within the regions. Further uncertainty is introduced with the CRF values which have been derived from short-term O_3 exposure (robust long-term estimates remain unavailable in the literature) with the assumption that county specific CRF coefficients are normally distributed. However, despite these limitations, the high-resolution hybrid downscaling system presented here allows the examination of EM at the county level, which is a major strength of our current study. As expected, county-level O_3-related

EM is high in counties with higher populations. However, U.S. counties, in general, stand to benefit from emission changes under RCP4.5 scenarios.

Since climate change can have important ramifications in California such as more severe and frequent wildfires and air pollution episodes, it has been the focus of extensive air pollution and climate research.[22,59-61] For example, Mahmud et al. performed statistical downscaling methods in the state of California using temperature data from the National Center for Atmospheric Research (NCAR) Reanalysis and 1-hour maximum O_3 values from two ground monitors.[22] The air temperature data have a coarse spatial resolution (2.5° x 4°), which makes it difficult to be directly associated with daily O_3 levels. Instead, linear regression models were developed between 850-hPa air temperature and quartiles of O_3 concentrations, posing an obstacle when linking O_3 exposure with population-specific concentration-response functions. Fujita et al., while using a high resolution (5 km) chose to utilize a chemical box model in which only one parameter could change at a time. While the methods vary significantly from those used in our study, the general trend of increasing O_3 and emissions is still evident.[61] Additionally, He et al. designed a study at a relatively low resolution (30 km) using multiple scenarios: climate change only, emissions only, and a boundary effect. Using CMAQ and the Sparse Matrix Operator Kernel Emissions Model (SMOKE), these scenarios were based on SRES A1B and A1FI. Generally, A1B is like the RCP4.5 scenario used in our study in terms of greenhouse gas emission increases and anthropogenic emission decrease, however, A1FI is very different from RCP8.5. In A1fi, all anthropogenic emissions are projected to increase. In RCP8.5, VOC/NOx is actually projected to decrease with large increases in methane leading to consequent rises in ozone levels.[60] Taken together, overall conclusions of these case studies are consistent with our results, but the use of hybrid downscaling

and the updated RCP scenarios strengthens our current study and lends more insight into the nuances of future ozone changes.

3.5.3 Strengths and limitations

While we have sought to improve on previous methods, some limitations remain. Uncertainties may lie in the estimation of the future mortality rate, CRF, population projection, the potential effect of interactions between temperature and O_3, and O_3 concentration predictions.[62-66] We attempt to account for some uncertainty by evaluating EM estimates using a robust Monte Carlo method. We acknowledge that other modeling frameworks have been proposed, however, we deemed our approach reasonable due to sufficient high model performance.[63,65] Exploration of additional techniques, though a future direction of study, was beyond the scope of this analysis. Another drawback lies in the cubic splines of time used in the model as they may underestimate the contribution of climate change to O_3 concentrations due to the removal of long-term trends. Additionally, the use of county-level resolution, while more precise than previous studies, may still cause some loss in detail of future O_3 predictions. However, it is necessary to keep the resolution of this data consistent with the resolution of the health data for analysis purposes (i.e., health data kept at county level for privacy protection). Future efforts to improve on this analysis could include enhancements in pollutant data collection locations, the addition of co-pollutant effects, and the effects of pollutants on human morbidity.

3.6 Study conclusions and future directions

The results of this study demonstrate that potential increases in premature death and in adverse health effects of climate change induced O_3 increases in the US may be substantially offset by the effect of emission reductions planned under RCP4.5. However, even with the reduction of

O_3 precursors, O_3-related excess mortality may still increase in the US, due to increases in methane emissions under RCP8.5. Thus, with responsible emissions policy, the effects of emission reduction of O_3 precursors is poised to significantly offset the adverse health effects of O_3 due to climate change. To prevent adverse health effects of this potential driver, it is important to continue to intensify mitigation efforts towards both GHGs and O_3 precursor emissions. These efforts are likely to avoid great cost to human health and quality of life.

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4. HEALTH EFFECTS OF SMOKE EXPOSURE

[Manuscript 2]

Associations of Wildfire Smoke $PM_{2.5}$ Exposure with Cardiorespiratory events in Colorado 2011-2014

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4.1 Abstract

Substantial increases in wildfire activity have been recorded in recent decades. Wildfires influence the chemical composition and concentration of particulate matter ≤2.5μm in aerodynamic diameter ($PM_{2.5}$). However, relatively few epidemiologic studies focus on the health impacts of wildfire smoke $PM_{2.5}$ compared with the number of studies focusing on total $PM_{2.5}$ exposure. We estimated the associations between cardiorespiratory acute events and exposure to smoke $PM_{2.5}$ in Colorado using a novel exposure model to separate smoke $PM_{2.5}$ from background ambient $PM_{2.5}$ levels. We obtained emergency department visits and hospitalizations for acute cardiorespiratory outcomes from Colorado for May-August 2011-2014, geocoded to a 4km geographic grid. Combining ground measurements, chemical transport models, and remote sensing data, we estimated smoke $PM_{2.5}$ and non-smoke $PM_{2.5}$ on a 1km spatial grid and aggregated to match the resolution of the health data. Time-stratified, case-crossover models were fit using conditional logistic regression to estimate associations between fire smoke $PM_{2.5}$ and non-smoke $PM_{2.5}$ for overall and age-stratified outcomes using 2-day averaging windows for cardiovascular disease and 3-day windows for respiratory disease. Per 1 $\mu g/m^3$ increase in fire smoke $PM_{2.5}$, statistically significant associations were observed for asthma (OR=1.081 (1.058, 1.105)) and combined respiratory disease (OR=1.021 (1.012, 1.031)). No significant relationships were

evident for cardiovascular diseases and smoke $PM_{2.5}$. Associations with non-smoke $PM_{2.5}$ were null for all outcomes. Positive age-specific associations related to smoke $PM_{2.5}$ were observed for asthma and combined respiratory disease in children, and for asthma, bronchitis, COPD, and combined respiratory disease in adults. No significant associations were found in older adults. This is the first multi-year, high-resolution epidemiologic study to incorporate statistical and chemical transport modeling methods to estimate $PM_{2.5}$ exposure due to wildfires. Our results allow for a more precise assessment of the population health impact of wildfire related $PM_{2.5}$ exposure in a changing climate.

4.2 Introduction

4.2.1 Increasing threat of wildfires

Climate change, defined as the long-term change in global and regional weather patterns, has been extensively documented since the mid-to-late 20th century.[1-4] Despite politically charged debates regarding the cause of the change, it is clear that climate change and its resulting extreme weather events may severely impact the health and well-being of populations across the globe.[5-8] One area that reflects the synergistic impact of climate change and human activity is the occurrence of wildfires. Notably, the Western US has seen consistent and rapid increases in wildfire activity since the 1980s. This increase has been characterized by rises in the frequency, severity, size, and total burned area associated with wildfires.[9-11] Fire effects are often seen at great distances from the events due to large smoke plumes, sometimes extending across multiple counties or states. States in the Rocky Mountain region continue to exhibit climatic factors conducive to fire activity—including high temperatures, low soil moisture, decreased rainfall, and increased solar radiation.[12-16] Conditions may become more suitable to large wildfires over time due to climate change.[17-19] Consequently, wildfires place significant burdens on the human, economic, and environmental systems in areas surrounding and downwind from the burn zone. This is of particular concern given the impact that wildfire events can have on regional air quality and, subsequently, human health.[20,21]

4.2.2 Particulate matter from smoke differs from ambient concentrations

Wildfire smoke can produce significantly higher exposures to harmful compounds than are normally found in non-fire urban settings.[22-24] Fine particulate matter ($PM_{2.5}$, airborne particles less than 2.5 μm in aerodynamic diameter) is of particular concern due to its ability to travel deep into the human respiratory system and enter the blood stream.[20,21,25-29] Smoke particles differ in

both size and composition from particles found in typical ambient PM from non-wildfire sources. It has been shown that organic compounds, such as methanol or formaldehyde, make up a significantly higher proportion of smoke $PM_{2.5}$ when compared with ambient PM.[23,20] These distinctions could have differing effects on human health outcomes and may vary by fuel source. This has been shown in both in vivo and in vitro studies using human cells and mice.[31-33] While much is left to be understood about the toxicological differences, current literature has begun to elucidate potential differences between smoke and ambient PM sources. It is, therefore, important to differentiate between smoke and non-smoke $PM_{2.5}$ when assessing the health impact of wildfires.

4.2.3 Epidemiological approaches to studying the effects of wildfire smoke exposure

While numerous epidemiological studies have established the associations between ambient $PM_{2.5}$ and human health, relatively few studies have focused specifically on wildfire smoke.[34-37] For example, Reid et al. published a study showing significant results for asthma during fire events (previous 2-day moving average) for a 5 $\mu g/m^3$ change in $PM_{2.5}$ concentration.[20] While Reid et al. included satellite and chemical transport data, they were limited to the use of fire day and fire distance parameters to account for smoke PM instead of directly estimating smoke PM concentrations. Additionally, many studies are restricted to the use of ambient urban air pollution measurements, coupled with fire day indicators, to represent fire-related exposures. In addition, current guidelines for public health response to wildfire events rely heavily on changes of ambient total PM measurements due to a lack of information in wildfire-specific air quality.[38] A few studies have distinguished among sources on larger scales.[39-41] For example, Liu et al. derived metrics of smoke waves for distinguishing fire activity and evaluated the health impacts of smoke $PM_{2.5}$.[41] Their chemical transport model simulations, however, were on a spatial grid of 0.5 x 0.67 degrees,

which may be too coarse to capture finer-scale spatial gradients of exposure, see Supplemental Figure 4-1.

4.2.4 Importance of isolating smoke-related particulate matter

Though there is consistent evidence for associations between wildfire events and disease, questions remain regarding the relationship between wildfire smoke $PM_{2.5}$ and both respiratory and cardiovascular outcomes given the difficulty in estimating smoke $PM_{2.5}$ exposure. Developing robust methods for understanding this complex relationship is vital to understand the potential future impacts of climate and wildfire events on human health. Building upon previous studies, the goal of our study is to estimate the associations for multiple respiratory and cardiovascular acute health events in relation to wildfire smoke $PM_{2.5}$ in Colorado during the fire seasons of 2011-2014 using novel, high-resolution methods to separate wildfire smoke $PM_{2.5}$ from background ambient $PM_{2.5}$.

4.3 Data & Methods

4.3.1 Health data

We obtained individual-level health data on daily hospitalizations and emergency department (ED) visits at all public and private hospitals for the fire seasons (May-August) of 2011-2014 from the Colorado Department of Public Health and Environment. Information included in the patient records are dates of admission, residential address, age, sex, payer information and International Classification of Diseases version 9 (ICD9) codes for primary and secondary diagnoses. Patients admitted to the hospital through the ED were only counted once, and those with elective hospitalizations were excluded from analysis.

We analyzed multiple endpoints for primary cardiovascular and respiratory diagnoses. Respiratory outcomes include asthma (ICD9: 493), bronchitis (ICD9: 490), chronic obstructive pulmonary disease (ICD9: 491, 492, and 496), upper respiratory infection (ICD9: 460-465 and 466.0), and combined respiratory disease (ICD9: 460-465, 466.0, 466.1, 466.11, 466.19, 480-486, 487, 488, 490, 491, 492, 496, and 493). Cardiovascular outcomes include ischemic heart disease (ICD9: 410-414), acute myocardial infarction (ICD9: 410), congestive heart failure (ICD9: 428), dysrhythmia (ICD9: 427), peripheral/cerebrovascular disease (ICD9: 433-437, 440, 443, 444, 451-453), and combined cardiovascular disease (ICD9: 410-414. 427, 428, 433-437, 440, 443, 444, 451-453). Due to inadequate numbers, events in children were not analyzed for COPD or any cardiovascular outcomes.

4.3.2 $PM_{2.5}$ and meteorological data

We sought to separate smoke $PM_{2.5}$ from ambient sources. To accomplish this, daily mean $PM_{2.5}$ concentrations were adopted and improved from our previous study by adding new data.[42] Briefly, mean concentrations were estimated using a two-model approach to combine information from high-resolution satellite AOD derived from the Multi-angle Implementation of Atmospheric Correction (MAIAC) algorithm, model simulations from the Community Multiscale Air Quality Modeling System (CMAQ), and ground measurements obtained from the U.S. Environmental Protection Agency (USEPA) for fire seasons in the state of Colorado (April-September, 2011-2014). The first model (i.e. AOD model) utilized random forest modeling to incorporate MAIAC AOD, smoke mask, meteorological fields and land-use variables. The second model (i.e. CMAQ model) utilized statistical downscaling to calibrate the CMAQ $PM_{2.5}$ simulations. Additional exposure modeling specifics can be found in Supplemental 2 and Supplemental Figure 4-2. The output exposure data have full coverage in space and time and can capture the large fire events at

a resolution of 1km x 1km (CV R^2 =0.81 and RMSE =1.85 μg/m^3). Compared to Geng et al. (2018), major improvements include new observation data from the National Park Service to capture $PM_{2.5}$ enhancement near wildfires, allowing for a better representation of high values found during fire events (Supplemental 4-2 and Supplemental Figure 4-2).[43,44] Additionally, a random forest approach was utilized instead of the original statistical downscaler for the AOD model. This improved the R^2 of the AOD model from 0.65 to 0.92 and the gap-filled R^2 from 0.66 to 0.81.[42] $PM_{2.5}$ exposure values were then aggregated to a 4km x 4km grid to match the resolution of the health data.

Fire count data were obtained using the MODIS fire count product to specify fire days for each grid cell.[45] Wildfire and prescribed fire emissions were obtained from the US EPA emissions inventory for the study period. To calculate the wildfire smoke $PM_{2.5}$ fractions, we used two CMAQ model scenarios–with and without smoke and dust particles. The differences between these scenarios were then divided by the total $PM_{2.5}$ scenario to calculate the smoke $PM_{2.5}$ fractions. The smoke $PM_{2.5}$ fractions were then multiplied by the total satellite based $PM_{2.5}$ exposure to get the smoke $PM_{2.5}$ concentrations.

4.3.3 Epidemiological modeling methods

We estimated associations between short-term changes in air quality and ED visits and hospital admissions using a case-crossover study design.[46] Each individual's event day (i.e., date of ED visit or hospitalization) was matched with up to four non-event days, with matching based on grid location, day of week, and calendar month.[47] Exposure and meteorology were assigned to each event day and corresponding non-event days based on the 4km x 4km grid cell in which the patient's address is located. The 4km grid was chosen a priori through collective agreement

between the researchers and the Colorado State Health Department. This resolution was deemed the finest resolution we could use while still conserving confidentiality. We then used conditional logistic regression to estimate the associations between ED visits and hospitalizations for each outcome and exposure to non-smoke $PM_{2.5}$ and smoke $PM_{2.5}$. The final models for respiratory outcomes are shown in model specification 1 & 2 below:

$$\boldsymbol{logit\ P(Y) = \beta(total_{3day}\ PM_{2.5}) + \beta(temp_{3day}) + ns(doy)}$$

Equation 4- 1

$$\boldsymbol{logit\ P(Y) = \beta(smoke_{3day}\ PM_{2.5}) + \beta(nonsmoke_{3day}\ PM_{2.5}) + \beta(temp_{3day}) + ns(doy)}$$

Equation 4- 2

where $total_{3day}\ PM_{2.5}$ represents the 3-day moving average for total $PM_{2.5}$ (i.e., smoke + non-smoke), $temp_{3day}$ is the 3-day moving average temperature, *ns(doy)* is a spline for day of year (two internal nodes per year), $smoke_{3day}\ PM_{2.5}$ represents the three-day moving average smoke $PM_{2.5}$; and $nonsmoke_{3day}\ PM_{2.5}$ denotes three-day moving average $PM_{2.5}$ not related to wildfires. Cardiovascular outcome models were conducted using the same models shown in model specifications 1 and 2, but with 2-day averaging windows. Exposure windows of 3-day average PM for respiratory outcomes and 2-day average PM for cardiovascular outcomes were decided a priori based on published studies and consensus information found in the latest Integrated Science Assessment from the USEPA.[20,41,48-53] Sensitivity analyses were conducted using lag 0, lag 0-1 and seven-day exposure windows for respiratory outcomes and lag 0 and three-day exposure windows for cardiovascular outcomes.

Other potential confounders were assessed (relative humidity, boundary layer height, heat index, wind speed). However, these parameters did not influence the results and were omitted in the final model. Analyses to examine the presence of potential effect modification were completed using sex and age-stratification. Age-stratified categories included children (0-18 years), adults (19-64 years), and older adult (65+ years). We conducted all analyses in R 3.4.3 (2017) and SAS© 9.4.

4.4 Results

4.4.1 Exposure modeling and smoke contribution to $PM_{2.5}$ levels

A time series plot for modeled statewide daily mean $PM_{2.5}$ concentrations is shown in Figure 4-1. Modeled total $PM_{2.5}$ values ranged from close to 0 to 47.48 μg/m^3, with an overall mean value of 4.67 μg/m^3. The exposure model was also used to separate smoke $PM_{2.5}$ from non-smoke $PM_{2.5}$. This separation is based on the CMAQ fraction, with total $PM_{2.5}$ equal to the sum of non-smoke $PM_{2.5}$ and smoke $PM_{2.5}$. Ratios of smoke $PM_{2.5}$ to total $PM_{2.5}$ ranged from 0 to 99.56% (mean=0.006%), with smoke $PM_{2.5}$ levels ranging from 0 to 37.34 μg/m^3. The statewide daily mean smoke vs. total $PM_{2.5}$ ratio is also shown for the entire study period (See Figure 4-2). As shown, concentrations varied year-to-year and between stations. This is likely due to the spatial variability of wildfires and varied smoke plume behavior due to factors such as prevailing wind speed and direction. To illustrate $PM_{2.5}$ concentrations and ratios attributable to fire, Figure 4-3 shows the domain-wide average total $PM_{2.5}$ on fire days (smoke $PM_{2.5}$ >1%) compared with the domain-wide average ratio of smoke $PM_{2.5}$. For the entire study period, total $PM_{2.5}$ averaged 7.87 μg/m^3 with average fire $PM_{2.5}$ ratios at 28%. Figure 4-4 shows locations on a fire day near two

major fires that occurred during our study period. As shown in Figure 4-4A, high levels of smoke PM can be seen despite more moderate total $PM_{2.5}$ concentrations. Figure 4-4B depicts a fire day with much higher total $PM_{2.5}$ concentrations and the subsequent contributions of smoke PM. Additional analysis showed relatively little correlation between smoke $PM_{2.5}$ and non-smoke $PM_{2.5}$ (Pearson correlation coefficient $r=0.11$, $p<0.0001$). The peaks of highest smoke $PM_{2.5}$ ratios tended to correspond with active fire days. Figure 4-5 illustrates the modeled total $PM_{2.5}$ and smoke $PM_{2.5}$ ratio for June 22, 2013, a peak fire day during the West Fork Fire Complex. As depicted, when compared to satellite imaging, the modeled smoke $PM_{2.5}$ appears to capture the apparent visible smoke plume adequately.

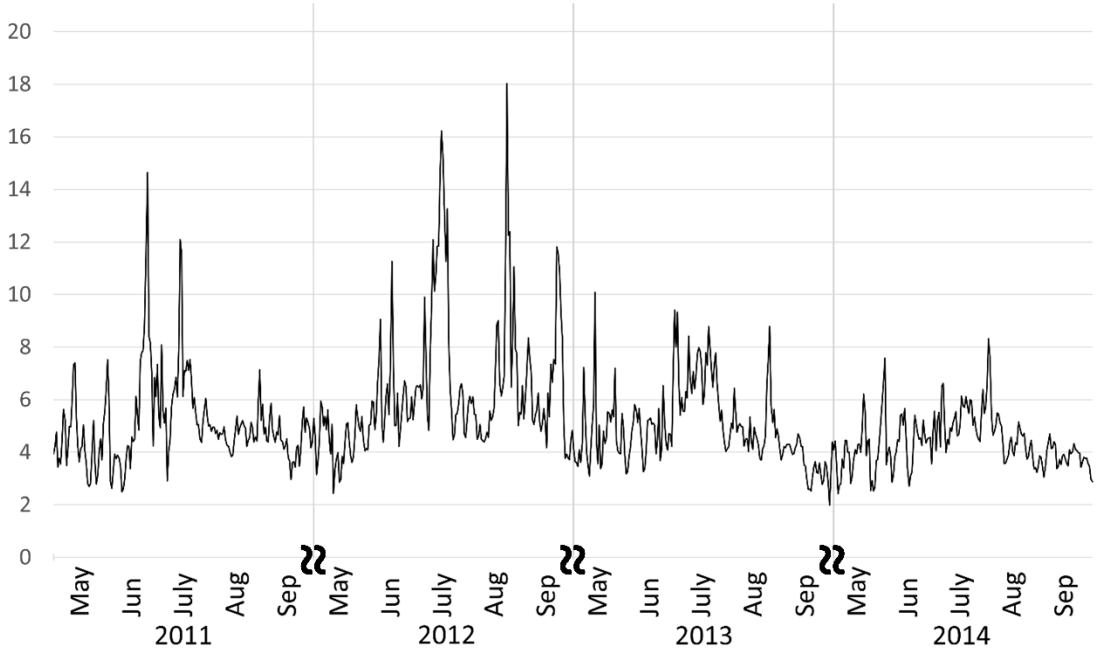

Figure 4- 1

Daily Mean Modeled $PM_{2.5}$ for Fire Seasons 2011-2014 in Colorado. *State-averaged time series data for fire seasons (May-August) 2011-2014 show total modeled $PM_{2.5}$ levels by day, month, and year.*

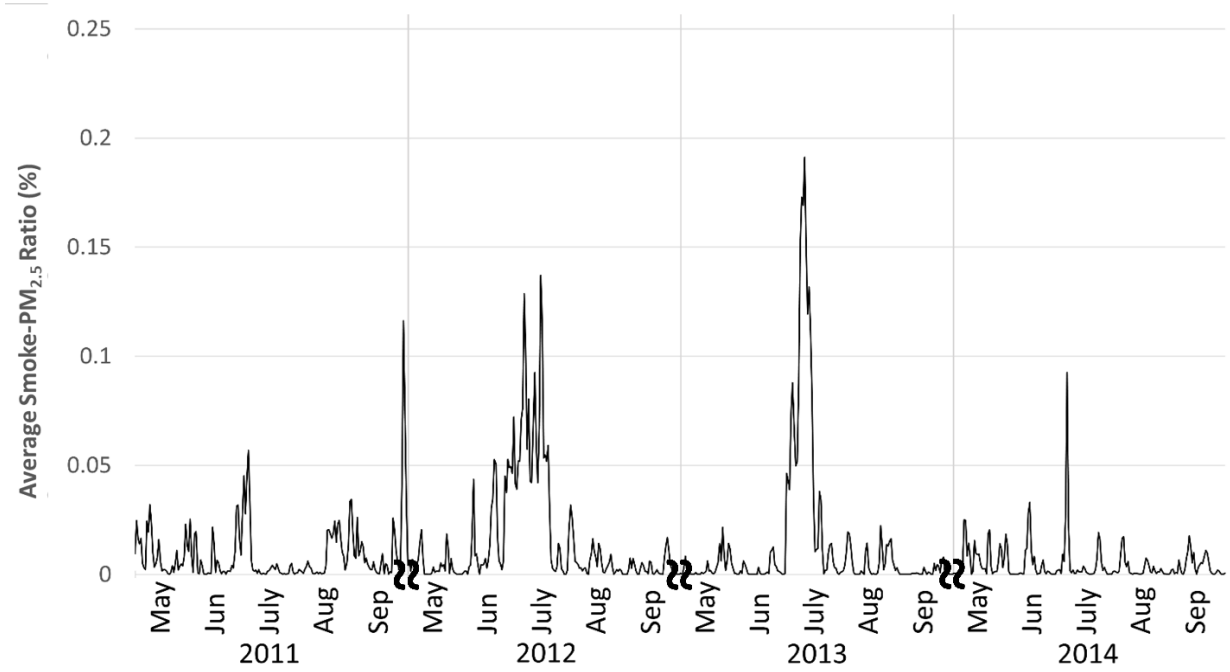

Figure 4- 2

Daily Mean Ratio of $PM_{2.5}$ Attributed to Wildfire. *State-averaged time series data for fire seasons (May-August) 2011-2014 depicting ratio of modeled smoke $PM_{2.5}$ to total modeled $PM_{2.5}$.*

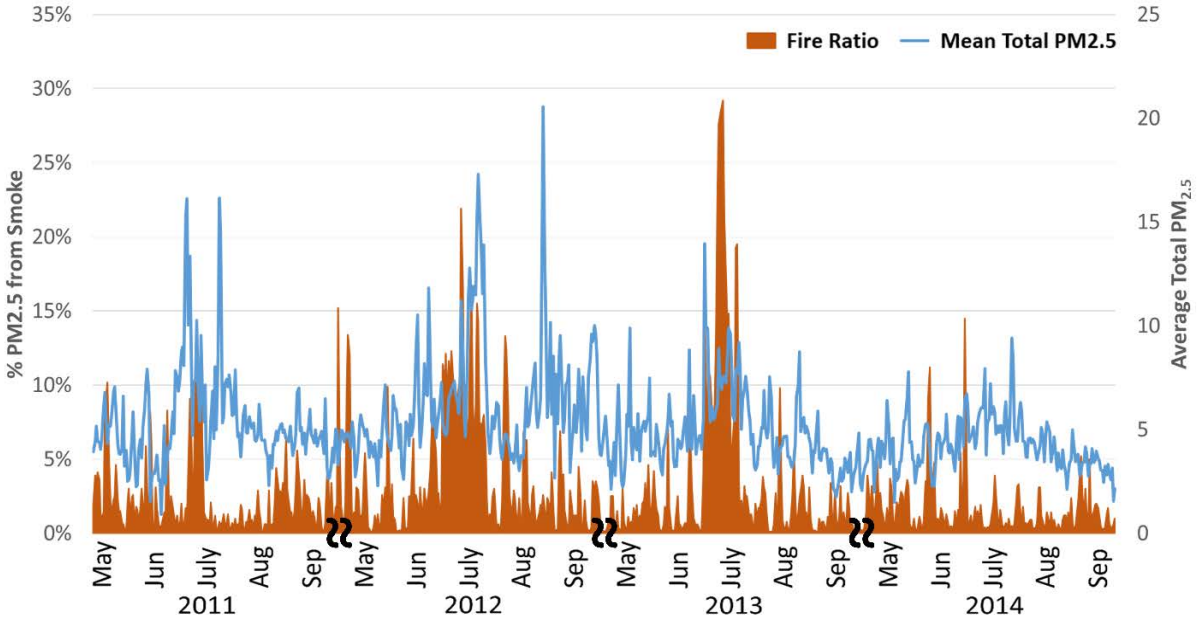

Figure 4- 3

Domain-wide Daily Mean Total $PM_{2.5}$ and Mean Ratio of $PM_{2.5}$ on Fire Days (Fire PM >1%).

Time series depicting both total and ratio of modeled smoke $PM_{2.5}$ to total modeled $PM_{2.5}$.

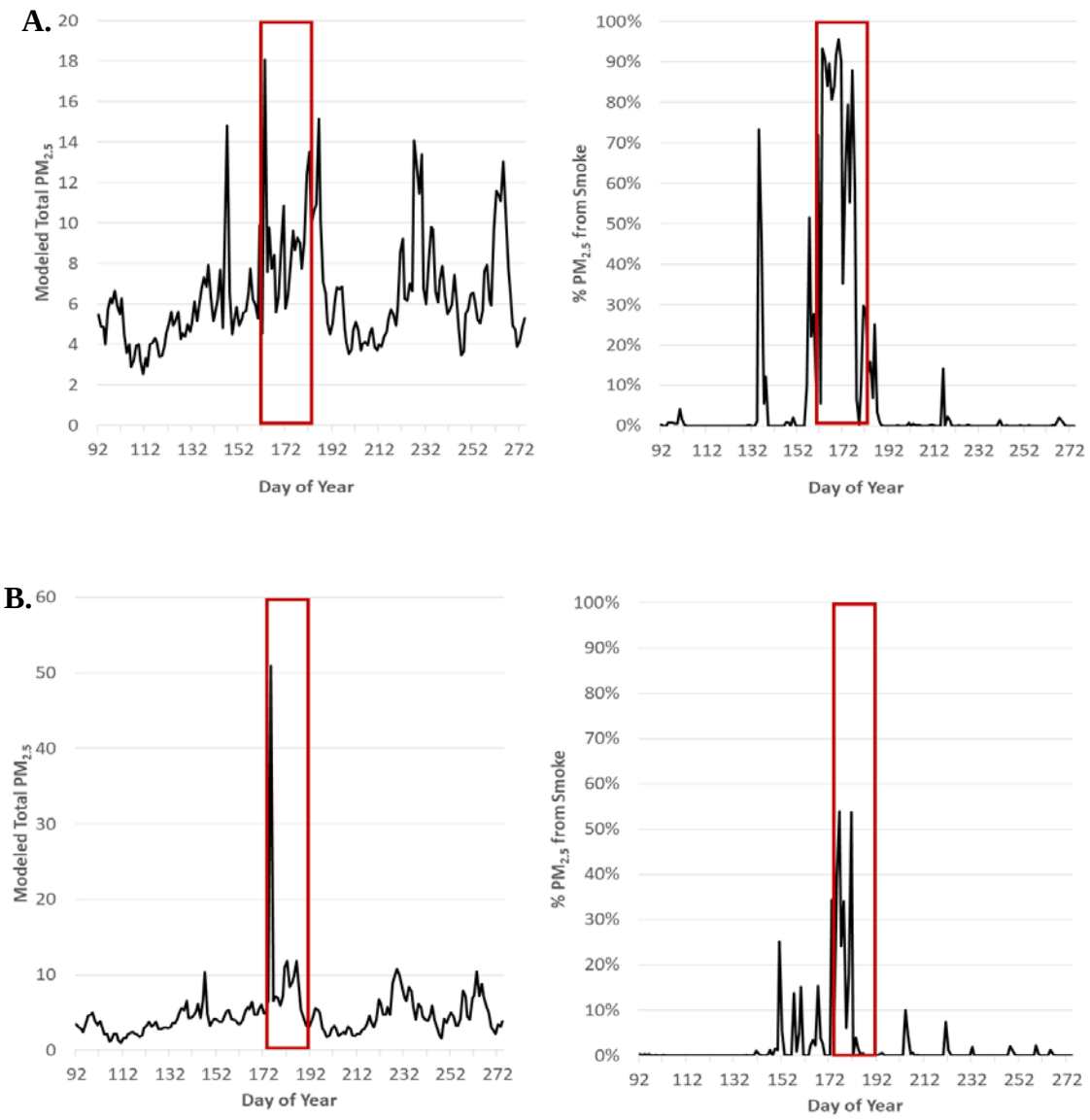

Figure 4- 4

Daily Mean Total $PM_{2.5}$ and Mean Ratio of $PM_{2.5}$ Attributed to Wildfire at Two Locations. *Time series depicting both total and ratio of modeled smoke $PM_{2.5}$ to total modeled $PM_{2.5}$. A) Location near the High Park Fire (June 9-30, 2012) and B) Location near Waldo Canyon Fire (June 23-July 10, 2012). Red boxes indicate active fire days.*

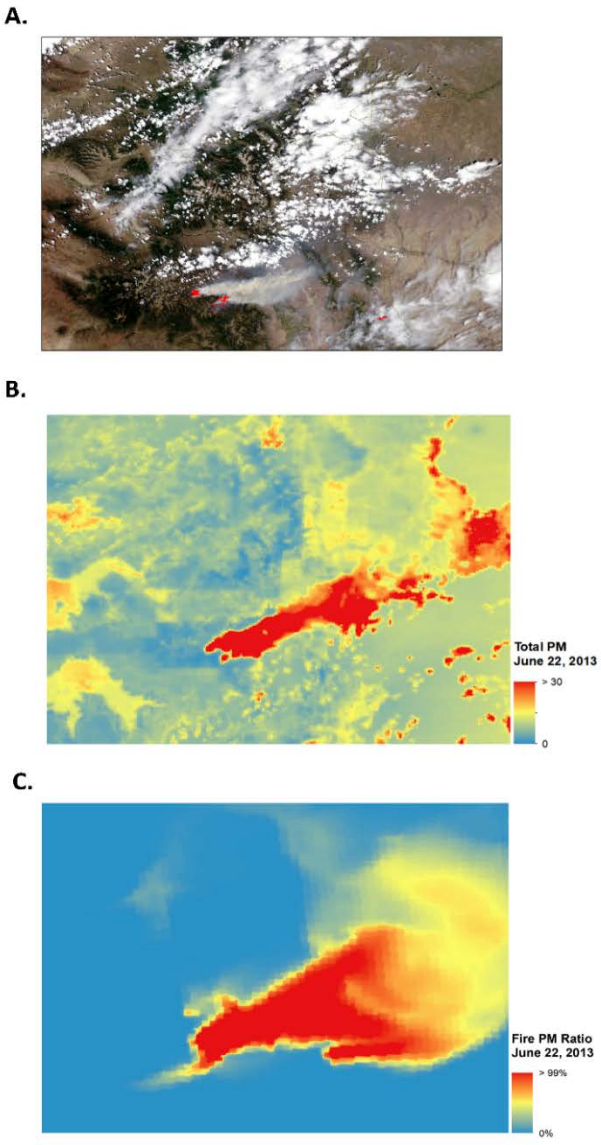

Figure 4- 5

Satellite Smoke Plume, Modeled Total $PM_{2.5}$ and Smoke $PM_{2.5}$ for West Fork Fire Complex, June 22, 2013. *Modeled data corresponds to visible smoke plume as shown in A-C. A) Satellite image from June 22, 2013 with active West Fork Complex Fire.*[54] *B) Total $PM_{2.5}$ for Colorado on June 22, 2013. C) Amount of $PM_{2.5}$ attributed to fire on June 22, 2013.*

4.4.2 Epidemiological modeling

After excluding duplicate events and events with non-geocoded addresses, 44,262 of 490,368 (9%) of cases were excluded from the analysis. A total of 446,106 ED visit and hospitalization events were analyzed from the Colorado Department of Public Health and Environment. Of those included, there were 204,823 male and 241,283 female cases. The lowest case count occurred in 2011 (n=102,318), with the highest number of cases in 2014 (n=129,477). While many reasons could exist, the large increase seen in 2014 could be explained by changes in health seeking behavior due to wider Medicaid coverage resulting from the implementation of the Affordable Care Act.[55] Other summary statistics on age groups and events per year are found in Table 1.

Using conditional logistic regression models, we estimated the odds ratio for exposure to smoke $PM_{2.5}$ and individual health outcomes. As shown in Figure 4-6 and Supplemental Table 1, we observed significant positive associations between 1 $\mu g/m^3$ increases in 3-day moving average fire exposures and both asthma (OR 1.081, 95% CI (1.058, 1.105)) and combined respiratory disease (OR 1.021, 95% CI (1.012, 1.031)) in a model that adjusted for $PM_{2.5}$ from other sources. There were no significant positive associations linked to cardiovascular outcomes and 2-day smoke $PM_{2.5}$ exposures (see Figure 4-7 and Supplemental Table 2). However, some inverse associations were shown to be protective for cardiovascular outcomes. This could possibly be due to random error, or it may be that individuals with pre-existing cardiovascular disease stay indoors on days with fire activity.

Table 2- 1

Epidemiologic data descriptive statistics.

	Case Count
Total Records	490,368
Geocoded	446,106
Non-geocoded	44,262
Year of Event	
2011	102,318
2012	102,574
2013	111,737
2014	129,477
Age Ranges	
0-18 y	94,022
19-64 y	202,665
65+ y	149,419
Sex	
Female	241,282
Male	204,823

The models were also run using total $PM_{2.5}$ for both cardiovascular and respiratory outcomes. Overall, the majority of the respiratory odds ratios for 3-day average total $PM_{2.5}$ were either null or trending to positive (Supplemental Table 3). The odds ratios for ischemic heart disease, acute myocardial infarction, and dysrhythmia also suggest a trend toward a positive association (see Supplemental Table 4). The cardiovascular results for total $PM_{2.5}$ included significant negative results for congestive heart failure, peripheral/cerebrovascular disease, and cardiovascular disease.

We conducted sensitivity analyses for additional exposure windows. Using lag 0 for both respiratory and cardiovascular outcomes, similar results were seen with smoke $PM_{2.5}$ exposure,

with notable differences in overall upper respiratory infection (OR 1.015, 95% CI (1.005, 1.026) and upper respiratory infection in children (OR 1.018, 95% CI (1.004, 1.003), see Supplemental Figures 4-3 and 4-4. Using lag 0-1 for all respiratory outcomes, the results were again similar to the initial analysis with changes for overall and child-only upper respiratory infections; see Supplemental Figure 4-5. Using a 7-day averaging window for respiratory outcomes, asthma was the only outcome to have a significant positive association with smoke $PM_{2.5}$ exposure (OR 1.081, 95% CI (1.051, 1.112), see Supplemental Table 5). The associations for asthma, upper respiratory infection, bronchitis, and combined respiratory disease trended positive but not significant for 7-day averaged total $PM_{2.5}$ exposure (see Supplemental Table 6). A 3-day averaging window used for cardiovascular outcomes also yielded either null or negative results (Supplemental Tables 7 and 8).

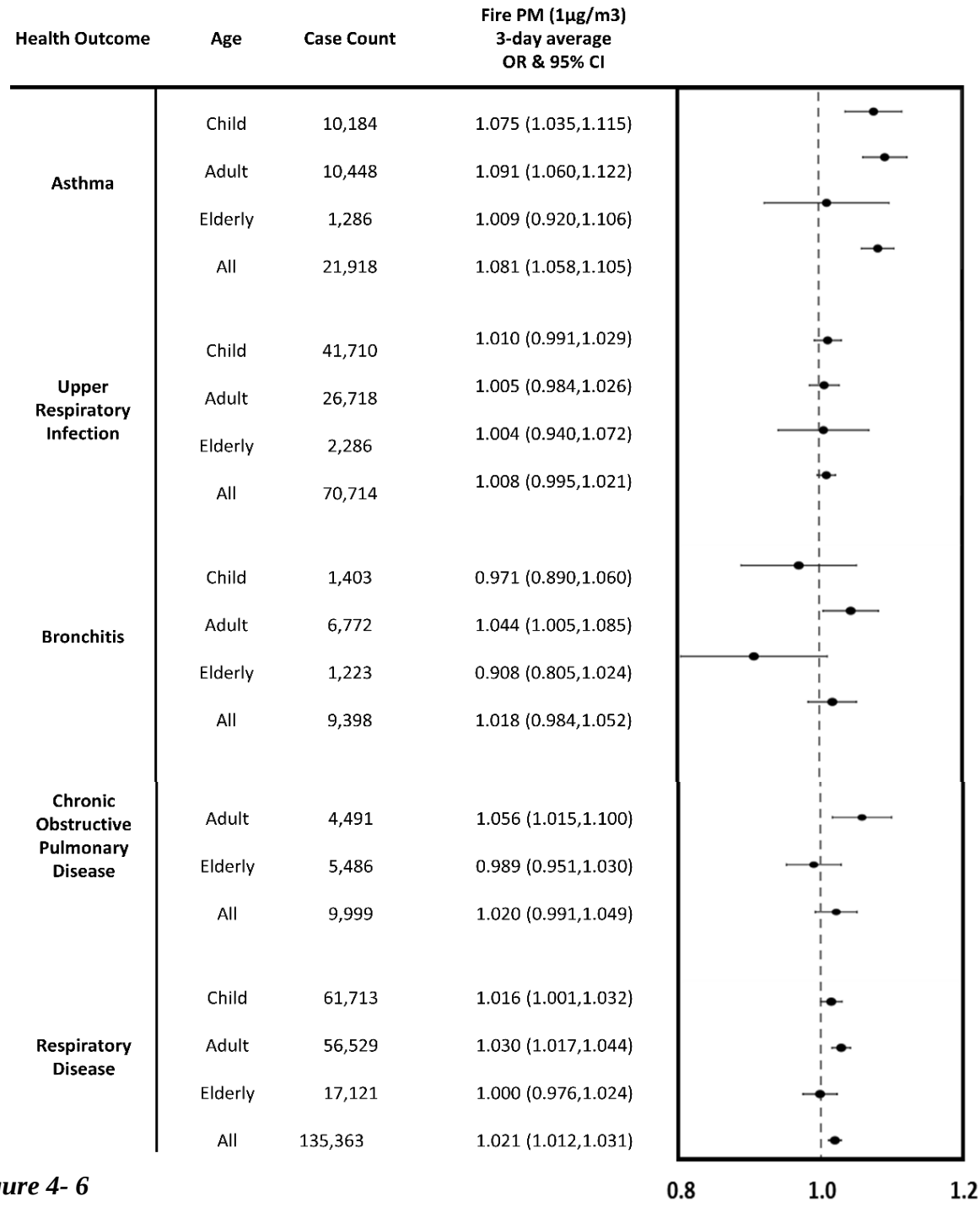

Figure 4- 6

Wildfire Smoke $PM_{2.5}$ Exposure and Respiratory Outcomes. *Odds ratios for both total and age-stratified respiratory outcomes per 1 µg/m^3 increase in wildfire smoke $PM_{2.5}$ exposure, arranged by outcome and age group.*

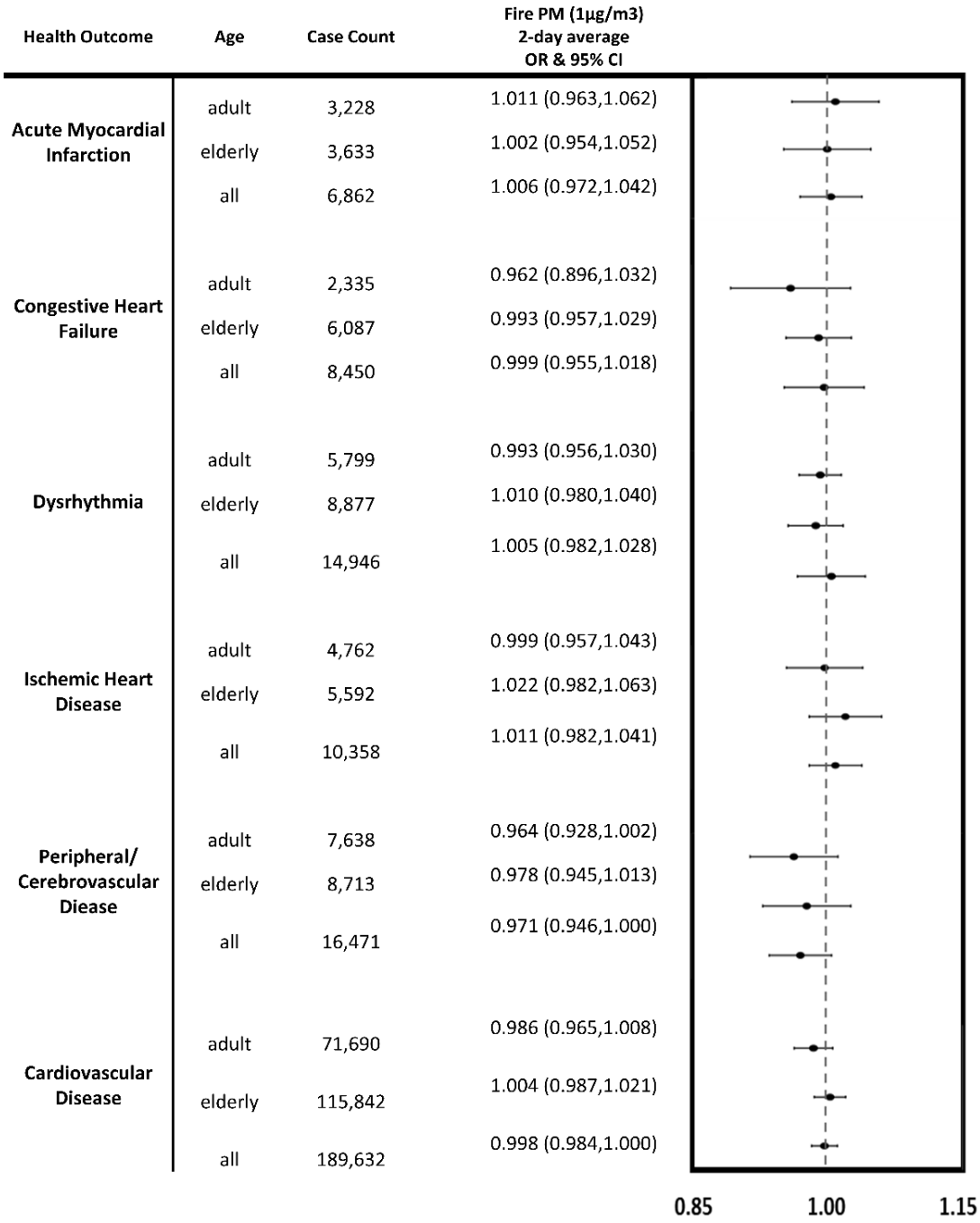

Figure 4- 7

Wildfire Smoke $PM_{2.5}$ Exposure and Cardiovascular Outcomes.

Odds ratios for both total and age-stratified cardiovascular outcomes per 1 µg/m^3 increase in wildfire smoke $PM_{2.5}$ exposure, arranged by outcome and age group.

4.4.3 Stratified analysis

To investigate potential effect modification of the relationship between exposures and respiratory outcomes, we conducted stratified analyses based on sex and age. While most sex-stratified total $PM_{2.5}$ results were null, an association was seen in females for bronchitis (OR 1.007, 95% CI (1.001, 1.013), see Supplemental Table 9), however, no significant results were observed for cardiovascular outcomes and both 2-day total and smoke $PM_{2.5}$, (Supplemental Tables 10 and 11). Associations for both female and male asthma cases and 3-day average smoke $PM_{2.5}$ were significant, with higher odds shown in female cases (OR 1.096, 95% CI (1.064, 1.128)) than in male cases (OR 1.063, 95% CI (1.029, 1.098)). Female bronchitis cases (OR 1.054, 95% CI (1.010, 1.101)) and female total respiratory cases (OR 1.027, 95% CI (1.015, 1.040)) were also positively associated with smoke $PM_{2.5}$. Additional sex-stratified, 3-day average smoke $PM_{2.5}$ results can be found in Supplemental Table 12.

Additionally, some outcomes exhibited differences when stratified on age. After age-stratification, there were no patterns found linking respiratory outcomes and total $PM_{2.5}$ with any specific age group (Supplemental Table 13). Regarding smoke $PM_{2.5}$, Figure 4-6 also depicts the ORs and associated confidence intervals for each of the respiratory outcomes by age group. In children ages 0 to 18 years, significant positive associations were seen for asthma (OR 1.075, 95% CI (1.035, 1.116)). Adults aged 19 to 64 years of age exhibited positive associations for asthma (OR 1.091, 95% CI (1.060, 1.122)), bronchitis (OR 1.044, 95% CI (1.005, 1.085)), COPD (OR 1.056, 95% CI (1.015, 1.100)), and combined respiratory disease (OR 1.030, 95% CI (1.017, 1.044)) (see also Supplemental Table 14). For individuals 65 and older, there were no significant positive associations seen for respiratory outcomes. We found no positive associations for age-stratified total or smoke $PM_{2.5}$ and any of the cardiovascular outcomes (See Figure 4-7 and

Supplemental Tables 15 and 16). Additional results for stratification analyses using a 7-day averaging window for respiratory outcomes and a 3-day averaging window for cardiovascular outcomes can be found in Supplemental Tables 17-24. Of note, associations for both childhood and adult asthma, adult COPD, and adult combined respiratory disease events were positively associated with 7-day average smoke $PM_{2.5}$ (see Supplemental Table 17).

4.5 Discussion

4.5.1 Summary and impact of results

In this study, we estimated associations between various health outcomes and acute exposure to non-smoke $PM_{2.5}$ and smoke $PM_{2.5}$ in the state of Colorado over a four-year period (2011-2014). The design of this study is centered on smoke $PM_{2.5}$ contributions to health outcomes. This work builds on our previous work by improving exposure data metrics and expanding from a 1-month pilot study.[56] The exposure data considers both spatial and temporal variability by including the use of satellite data to enhance the exposure estimates on an improved spatial scale of 4km x 4km. Another unique aspect of our exposure assessment is that we were able to separate smoke $PM_{2.5}$ from non-smoke sources and estimate risks attributable to wildfire smoke distinct from those due to $PM_{2.5}$ exposures from other sources.

As we hypothesized, many of the respiratory disease outcomes increased during periods of wildfire activity. For respiratory outcomes, we estimated an increase (OR=1.036 (95% CI: 1.022, 1.050%)) in ED/hospitalizations per 1$\mu g/m^3$ increase in fire smoke $PM_{2.5}$ exposure. The magnitude of the association was largest for asthma (OR=1.081 (95% CI: 1.058, 1.105)). Additionally, we observed heterogeneity in the association estimates when stratifying by age group. Positive

associations were observed for asthma events, where ED/hospitalizations increased significantly in children (OR=1.075 (95% CI: 1.035, 1.116)) and in adults (OR=1.091, (95% CI: 1.060, 1.122)) whereas the association estimate was lower in magnitude and was less precise for older adults (OR=1.009 (95% CI: 0.920, 1.106)). Similarly, an increase was seen for combined respiratory diseases with increases in ED/hospitalizations and adults (OR=1.030 (95% CI: 1.017, 1.044)). Specifically, in the adult group, increases were also shown for both bronchitis (OR=1.044 (95% CI: 1.005, 1.085)) and COPD (OR=1.056 (95% CI: 1.015, 1.100)). As opposed to other studies, there was no association shown for respiratory diseases when stratified for the older adult age group.

Unlike respiratory outcomes, we did not see a strong link between smoke $PM_{2.5}$ and cardiovascular outcomes. Results for combined cardiovascular disease yielded null results (OR=0.998 (95% CI: 0.984, 1.011)). Similar results were shown for both the adult and older adult age groups. This is not wholly surprising given differing results in current literature regarding the links between cardiovascular outcomes and wildfire events. There are fewer examples of cardiovascular associations with wildfire smoke exposure compared to respiratory outcomes. Additionally, associations with cardiovascular outcomes tended to be substantially lower in magnitude than for the respiratory outcomes. These differences are consistent with published studies on both types of outcomes.[21,57-63] For example, in Deflorio-Barker et al. (2019), most cardiovascular outcomes were not significant with fire day $PM_{2.5}$ using lag0-2. They also found similar results for smoke day all-cause cardiovascular outcomes were very similar to non-smoke days (OR 1.06 for smoke days vs OR 1.07 for non-smoke days).[61]

4.5.2 Comparison with current literature

Our high-resolution epidemiological study furthers the current knowledge in the field by incorporating random forest modeling methods combining information from MAIAC AOD, CMAQ simulations, and ground measurements to elucidate the portion of $PM_{2.5}$ present in the air due to wildfire smoke. Previous work has enhanced the spatial coverage and resolution of total $PM_{2.5}$ estimates during wildfire events—with the key distinction that this study focuses on the separation of smoke $PM_{2.5}$ from other sources. In most work, researchers compared smoke and non-smoke days using a variety of methods.[20,64] For example, satellite measurements are increasingly used to augment the spatially sparse ground monitoring for PM. However, this remains a relatively new approach to capturing the smoke PM concentrations. A study by Liu et al. looked at the entire Western US at the county-level using combined satellite and ground data.[41] They defined a fire indicator variable, or "smoke wave," which includes periods of at least two days of high pollution from wildfire smoke. Using this method, Liu et al. found associations between wildfire smoke exposure and various respiratory illnesses, but no associations with cardiovascular outcomes. Reid et al. (2015) used a machine learning approach to integrate multiple data sources including smoke indicators such as the distance to the nearest fire cluster and a smoke intensity calculation. The use of more advanced methods for predicting $PM_{2.5}$ exposure enhanced the exposure estimations, however, the $PM_{2.5}$ concentrations were not separated into smoke and non-smoke concentrations.[65]

Other work has utilized methods combining wildfire emissions and smoke plume modeling. For example, Hutchinson et al. examined similar epidemiological questions using exposure data derived from a model that combined the Wildland Fire Emissions Information System and the Hybrid Single-Particle Lagrangian Integrated Trajectories.[39] Their study found

increases in respiratory events with null cardiovascular results. However, the methods denoted fire-specific emissions due to fire location and progression from modeled progression maps and may not capture exposures as well as the use of chemical transport models. Ultimately, while our results carry similar interpretations to both studies, subtle dissimilarities may be seen as we utilize different air quality evaluation products and higher-resolution meteorological and epidemiological data to better-define the local exposures for each event.

The asthma association found in our study is substantially larger than those shown in previous publications. In addition to Reid et al. (2016), other studies found significant associations between smoke PM and health outcomes. Delfino et al. reported significant associations of OR=1.043 between asthma and 2-day moving average smoke exposure for 10 $\mu g/m^3$ increase in total $PM_{2.5}$ concentration (Delfino et al., 2009). In a more recent study, Reid et al. also found a significant association for asthma and previous 2-day moving average smoke exposure, with an OR of 1.050 during fire events for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ (Reid et al., 2019). Factoring in the domain-wide average smoke $PM_{2.5}$ ratio for the study period (~28% for days with >1% smoke PM), our result per 1 $\mu g/m^3$ roughly translates to 1.08 per 4 $\mu g/m^3$ of total $PM_{2.5}$. This converted result is more aligned with previously reported values, and the larger effect estimate is likely due to improved exposure assessment. It is also important to remember that our methods are unlike the majority of previous literature. Namely, the general approach in previous studies is to model smoke exposure using smoke day indicators. Our approach differed in that we sought to isolate the actual concentration of $PM_{2.5}$ directly from smoke. We originally hypothesized that there may be a difference in toxicity of smoke $PM_{2.5}$ compared to non-smoke $PM_{2.5}$. When compared with other literature, our findings suggest that smoke $PM_{2.5}$ may actually be more damaging to human health. Aside from asthma outcomes, the majority of the health associations in this study fall in line with

those found in previous literature. For example, Deflorio-Barker et al. 2019 also demonstrated stronger associations with respiratory outcomes than those with cardiovascular disease; with asthma exhibiting the largest OR of 1.06.[61]

While we did not investigate physiological mechanisms, these results may be explained by the toxicity of smoke $PM_{2.5}$. Since different chemical compositions of $PM_{2.5}$ may affect the body differently, it has been suggested that toxicological differences may play a role in how wildfire smoke PM affects the human anatomy and physiology. Multiple toxicological studies have shown differences in the composition and effects of wildfire smoke compared to ambient air.[24, 66-69] It has been shown that the small particles found in wildfire smoke may be responsible for stimulation of mechanisms that lead to increased oxidative stress at the cellular level. Wegesser et al. (2009) observed significant changes in macrophage and neutrophil counts in mouse lung samples exposed to wildfire smoke PM compared to ambient air. An additional study by the same group, expanded on these findings to show that substances such as polycyclic aromatic hydrocarbons (PAH) can be present in much higher concentrations in smoke versus levels detected in ambient air.[68] Franzi et al. (2011) looked specifically at the inflammatory responses due to wildfire smoke PM exposure. PM from wildfire smoke exhibited approximately five times more toxicity to lung macrophages than non-smoke exposure. This study also showed significant changes in reactive oxygen species and subsequent oxidative stress, leading to higher cell degeneration and potential apoptosis. Similarly, Kim et al. (2018) found significant increases in mouse lung neutrophils after exposure and that levels of lung toxicity were significantly associated with fuel type .[24]

4.5.3 Strengths and limitations

Despite the strengths of our study, some limitations remain. While we sought to enhance the exposure estimates for individual cases, some exposure misclassification is still possible given the assumption that the location of a person's address is a good representation of their short-term exposures to smoke PM. An additional limitation exists due to the use of modeled exposure data. However, as stated previously and despite this uncertainty, the model accurately captures the temporal and spatial trends of $PM_{2.5}$ measured by ground monitors and, thus give an accurate representation of overall trends. Additionally, several health events were left out of the analysis due to issues with address geocoding or non-Colorado residency. However, the exclusions were relatively small with only 9% of cases not used in the final analyses. Additionally, our analyses lacked the ability to differentiate chemical compositions of $PM_{2.5}$. Thus, we cannot link toxicological effects to our exposure metrics. Finally, the selection of averaging window size, though based on current literature, may also introduce error into the analysis.

Notwithstanding these limitations, our methods lend insight into important challenges that remain in the wildfire smoke exposure and health effects literature. The use of higher resolution enhanced exposure data provides a new approach to assigning exposure to individual events. Using multiple data products, our method aids in distinguishing wildfire smoke $PM_{2.5}$ from background $PM_{2.5}$. Unlike ground monitors that provide spatially sparse measurements, the exposure model used here provides daily concentrations for each 4km x 4km grid cell in our epidemiological study.

4.6 Study conclusions and future direction

Supported by high-resolution $PM_{2.5}$ exposure estimates, we found significant associations between wildfire smoke and acute respiratory outcomes in Colorado, despite an absence of

association with total $PM_{2.5}$ concentrations. Our findings point to potential toxic differences between smoke and non-smoke $PM_{2.5}$ exposure; suggesting that $PM_{2.5}$ from wildfire smoke could pose a significant threat to public health. This is especially true given the expected climate change-related impacts on wildfire incidence. It is, therefore, important to derive more accurate concentration-response relationships specific to wildfire smoke in order to develop a better understanding of future potential health risks based on increased wildfire activity. Taken together, the current analysis can inform public health agencies and healthcare systems regarding the potential future burden of wildfire smoke $PM_{2.5}$ exposure within the context of climate change. This information may be a key element in evaluating and enhancing current preparations aimed at wildfire-event response readiness.

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5. FUTURE HEALTH IMPACTS DUE TO SMOKE-RELATED $PM_{2.5}$ EXPOSURE

[Manuscript 3]

Excess Respiratory Events Due to Future Increase in Wildfire in the Western US: A Health Impact Analysis

Jennifer D. Stowell, Lin Wang, Qingyang Zhu, Eri Saikawa, Howard H. Chang, Joshua Fu, Cheng-En Yang, Qingzhao Zhu, Yang Liu and Matthew J. Strickland

5.1 Abstract

Recent years have brought increased wildfire activity to the western US. A significant concern regarding wildfires is the exposure to smoke and the fine particulates ($PM_{2.5}$) that found in the plumes. Using modeled fire season data (May-August) from present years (2003-2010) and modeled data from future years (2050-2059), climate and emissions models were run once including fire sources and once without. The difference between the results of these two models estimates the contribution of wildfire to total $PM_{2.5}$. Using the present and future wildfire smoke $PM_{2.5}$, we calculated the difference between the two models to estimate the future increase of smoke $PM_{2.5}$. All exposure data was aggregated to county level. The mean increase in average daily smoke $PM_{2.5}$ increase ranged from 0.05 to 9.5 $\mu g/m^3$. Highest exposures occurred in northern Idaho, Nevada and the Oregon coast. Additionally, we calculated the mean daily percent of smoke $PM_{2.5}$ as a ratio of total $PM_{2.5}$. Results ranged up to 85% for the gridded data (41.5% after aggregation). Using the ICLUS A2 population scenario, we estimate the western US states could experience additional daily ED visits at a rate of 19.2 visits per 10,000 people. Finally, we assessed the potential cost increase due to these visits. Based on the 2017 average ED visit cost of $1,059, the region could experience over $200 million in additional health care costs during a single fire season. The results of these analyses point to the importance of understanding areas that may bear

heavier burdens during future wildfire seasons. This information may serve as a key tool in policy and emergency response planning.

5.2 Introduction

Recent years have exhibited increased wildfire events, especially in the Western US. This heightened activity is generally characterized by increases in severity, frequency, size, and longer fire seasons.[1,2] Smoke plumes from these additional fires can affect multiple wide areas, with plumes extending across county, state, and country lines. It has been suggested that climate change may explain some of these fire anomalies. Certain conditions increase the likelihood of a wildfire occurrence. These include higher temperatures, high winds, low soil moisture or drought, and urban development of forested areas.[3-5] Most often, fires are started either by lightning or human error, and the chance of a fire event is heightened and complicated by continued favorable fire conditions due to changes in weather and long term climate shifts. Despite controversies in the political sphere, scientists continue to provide evidence for human-accelerated environmental changes that can result in increases for both extreme weather events and natural disasters such as fire. Therefore, it is critical that work continues to expand our understanding of the relationship between climate change, wildfire activity, and human health.

Climate and chemical transport models are used to project future conditions and include a myriad of parameters. Continued research seeks to enhance these models, giving predictions and projections more accuracy.[1,6] This is especially important in forecasting to improve early warning and emergency health care services. In the case of wildfire activity, projecting areas where fires will have the biggest impact can be important when considering planning and policies surrounding public health practices and response. It is, therefore, imperative that advances in sophisticated climate models continue to receive both recognition and funding to improve predictions of future climate and emissions policies.

One of the most significant threats to health from fire smoke exposure comes from the fine particulate matter less than 2.5 μm in aerodynamic diameter, or $PM_{2.5}$. It has been suggested that the particulate constituents of wildfire smoke may actually be more toxic than ambient $PM_{2.5}$, or urban air pollution.[7,8] This could be due to the higher concentrations of harmful substances in smoke-related $PM_{2.5}$ such as organic compounds like formaldehyde and methanol. Since little is known regarding the toxicity of particular types of $PM_{2.5}$ substances and their presence in wildfire smoke, it is important to find ways to differentiate between ambient $PM_{2.5}$ and smoke $PM_{2.5}$ to enhance our understanding of the potential mechanisms affecting health outcomes.

Previous work surrounding the effects of wildfire on human health have established certain associations between smoke exposure and multiple health outcomes. However, much of this research relies on the characterization of smoke as total $PM_{2.5}$ on smoke days compared to non-smoke days.[9-13] Additionally, some previous research has used chemical transport models and unique fire contributions to model past fire episodes and predict the exposure levels in the surrounding areas. For example, Koman et al. (2019) uses the CMAQ runs with and without wildfire emissions sources to create a gridded smoke $PM_{2.5}$ surface for the state of California.[14] They used a few approaches, including smoke waves to estimate the affected population. However, Koman et al. did not analyze any health outcomes. While these efforts have been informative, our understanding of the potential toxicity of smoke $PM_{2.5}$ needs to be enhanced. This requires continued efforts to separate smoke $PM_{2.5}$ concentrations from ambient $PM_{2.5}$.

The objectives of this body of work include investigating applications of novel climate and wildfire modeling to future health predictions. This study seeks to address possible increases in smoke $PM_{2.5}$ by using a modeling framework designed to separate fire contributions from all other sources of PM. Our approach includes estimating both present and future smoke $PM_{2.5}$ and

calculating the increase in total particulates due smoke $PM_{2.5}$. Using multiple present and future year (2003-2010 vs 2050-2059) meteorology and chemical transport models, we intend to separate smoke-related $PM_{2.5}$ and calculate the increased public health burden due to future increased wildfire activity.

5.3 Data & Methods

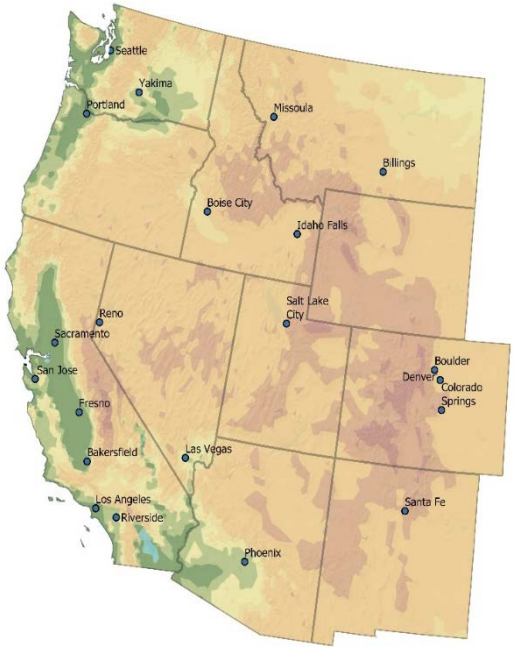

Figure 5- 1

Western United States study domain.

5.3.1 Study Domain

The study domain is shown in Figure 5-1 with multiple major cities. This region includes the states of Washington, Oregon, California, Nevada, Arizona, Idaho Montana, Wyoming, Utah, Colorado, and New Mexico. The Western US is made up of coastal lowlands, desert plains, and

mountainous regions with low-lying valleys. Much of the West receives less rainfall than states on the East Coast and arid conditions prevail in many areas. Perhaps the most striking characteristics of the region are the large mountain ranges that run from north to south (Rocky Mountain and Sierra Nevada Ranges). The topography and terrain of the area greatly affect the climate and weather patterns and patterns of development and urbanization.

5.3.2 Exposure Modeling Framework

In efforts to approach this analysis in using novel methods, we utilized unique datasets derived from chemical transport model configurations. To assess the increase in $PM_{2.5}$ from wildfire smoke, we chose to analyze data for the present scenario from the years 2003-2010 and the future dataset on the 2050-2059 IIASA (International Institute for Applied Systems Analysis) RCP8.5 configuration. Our approach is similar to Gao et al. 2012 and 2013, where meteorology was extracted using the Community Earth System Model v1.04 (CESM) to dynamically downscale meteorology from the Weather Research and Forecasting Model v3.2.1 (WRF). The CESM has four major components, one each for the atmosphere, land, ocean, and sea ice. The initial and boundary conditions for the Community Multiscale Air Quality Modeling (CMAQv5.2) included CAM4-Chem downscaling (Community Atmosphere Model with Chemistry). One main difference in our study compared with the Gao et al. studies is our use of different regional emission inputs for CMAQ present year simulations. These updated inputs came to us directly from collaborators at the EPA. Additional emissions input came from the Fire Inventory from NCAR (FINN v1.5),and the Global Fire Emissions Database (GFED4.1s) which are both daily fire emissions products designed as inputs for atmospheric chemistry modeling.

Each climate/emissions model were run twice for both the present data and future data. These model runs consisted of one including all $PM_{2.5}$ sources and one without the fire emissions

or no-fire simulations. For the present year no-fire simulations, we used the difference between the CMAQ inputs from the EPA and the first layer of the FINN fire model. For the future year no-fire scenario, the fire emission signal was removed so that the data captured would represent all ambient $PM_{2.5}$.

5.3.3 Isolation of Smoke $PM_{2.5}$ and Calculation of Increase for 2050-2059

Using the two model runs (total and no-fire $PM_{2.5}$) from both the future and present data, we were able to isolate the contribution of smoke $PM_{2.5}$ for the impact assessment. The overall framework is illustrated in Figure 5-2. Step 1 involves running a present total model and present no-fire model as well as a future total model and its accompanying no-fire model. To isolate the $PM_{2.5}$ from smoke, we subtracted the no-fire model outputs from the outputs of the total $PM_{2.5}$ runs. The difference of the total and no-fire scenarios results in a quantification of the smoke $PM_{2.5}$ contribution for each period. After calculating this parameter for both periods, we took the difference between the future and present scenarios to estimate the future increase in smoke $PM_{2.5}$ that we might expect under RCP8.5 conditions in the 2050s. With this parameter calculated, we matched the smoke $PM_{2.5}$ increase to the CMAQ 12km grid. To spatially align our exposure data with our health data, we then aggregated the smoke $PM_{2.5}$ data to the county level.

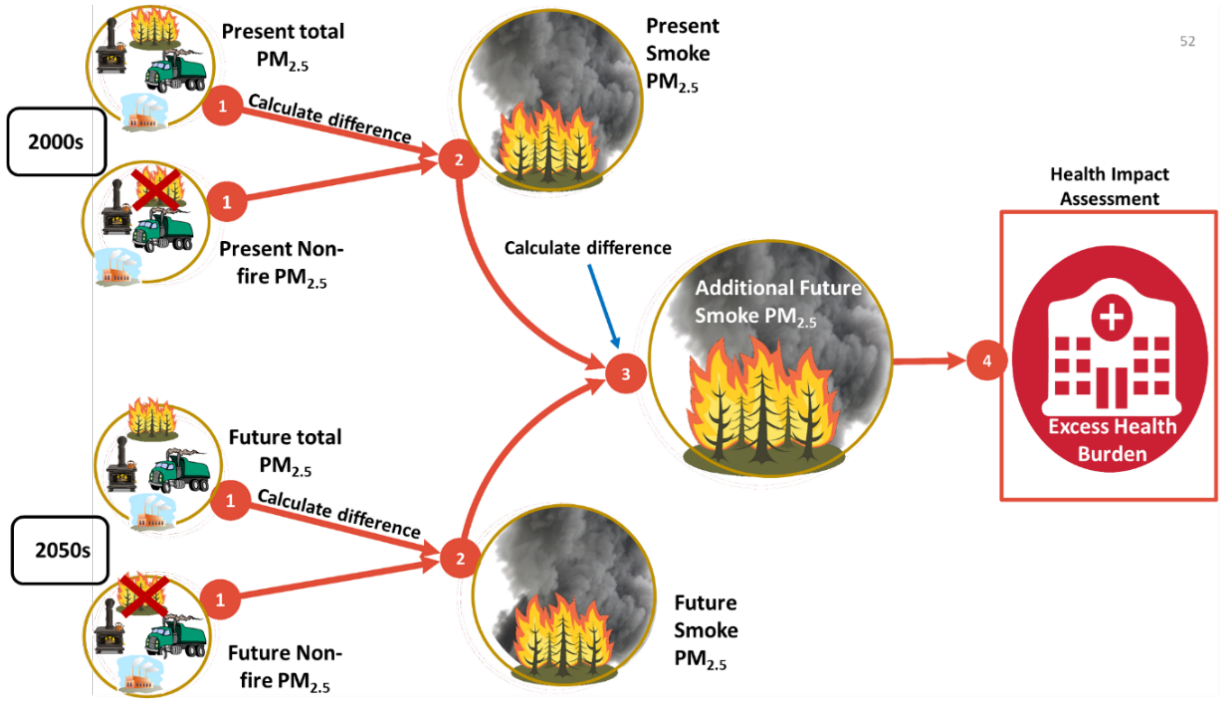

Figure 5- 2

Calculating Increases in $PM_{2.5}$ from Wildfire Smoke to use in a Health Impact Analysis. *Flow chart outlining the approach to isolate increases in smoke $PM_{2.5}$. Step 1 involves compiling two present models and two future models including one total $PM_{2.5}$ model and one non-fire $PM_{2.5}$ model. Step 2 calculates the difference between total and non-fire $PM_{2.5}$ for the 2000s and the 2050s. Step 3 calculates the difference between future and present smoke-related $PM_{2.5}$ to isolate increases in future smoke $PM_{2.5}$. Step 4 involves a health impact assessment to determine the associations between respiratory health outcomes and smoke $PM_{2.5}$ exposure.*

5.3.4 Epidemiological Metrics & Health Impact Assessment

The health metrics used in this analysis came from previously published work on the association of health outcomes with wildfire activity in Colorado. The base population from that

study encompassed all persons in the state. Records were extracted from all public and private hospitals during the fire seasons (May thru August) of 2011-2013. The odds ratios (OR) for multiple cardiorespiratory outcomes were calculated using a case-crossover framework and logistic regression. From these ORs we were able to calculate state-specific concentration response functions (CRFs) for multiple outcomes. Additionally, we were able to calculate a statewide average incidence rate for each of the outcomes. Making certain assumptions, we applied these metrics to the Western US.

In addition to the CRFs and incidence measures taken from our previous work, future population projections are required to calculate the future burden of wildfire smoke exposure. In order to calculate baseline incidence of future respiratory ED visits, we used the average daily incidence from the Colorado study. For this analysis, we utilized the Integrated Climate and Land Use Scenarios (ICLUS) population projections. ICLUS projections use the global Special Report on Emissions Scenarios or SRS to estimate future population on county-level under certain potential future conditions. In this base estimation, we use the ICLUS A2 population, characterized by high fertility rates, low economic growth and little international migration. The choice of this population projection matches closely with our projected exposure scenario, RCP 8.5.

We estimated changes in ED visits for the years 2050-2059 at the county level using the following equation:

$$\Delta ED = \Delta smokePM * \ln(OR) * EDIncidence * popA2 \qquad [1]$$

where *ΔED* represents the future change in respiratory ED visits due to 1 µg/m3 increase in smoke $PM_{2.5}$; *ΔsmokePM* is the calculated average increase in smoke $PM_{2.5}$ from the difference

calculations used above; *ln(OR)* is the natural log of the odds ratio per 1 µg/m^3 increase in smoke $PM_{2.5}$ we calculated from our previous Colorado wildfire study; *EDIncidence* is the average daily incidence of respiratory ED visits, again take from the Colorado analyses; and *popA2* is the projected county population according to the ICLUS A2 projection.

In addition to this base burden calculation, we selected multiple approaches to address and display the results on grid, county and state levels. Using the future ICLUS A2 population projections, we adjusted the numbers using population normalization as well and attempted a crude estimate of the costs associated with increased ED in the 2050s. We compared the population-normalized events with straight counts per county and state. Additionally, we calculated the potentially monetary burden placed on the healthcare system. We chose to use the average cost of a 2017 ED visit (for any reason) as published by the non-profit Health Care Cost Institute. They estimated that the average cost of an ED visit had grown 146% from 2008 levels or, from $431 per visit in 2008 to $1,059 per visit in 2017.

5.4 Results

5.4.1 Isolation of Smoke $PM_{2.5}$ and Calculation of Increase for 2050-2059

We estimated the smoke $PM_{2.5}$ and calculated the difference between 2050s levels and 2000s levels. Figure 5-3A shows the distribution of the present smoke $PM_{2.5}$ for the study domain on the CMAQ 12km grid. Figure 5-3B shows the distribution of future smoke $PM_{2.5}$ for the study domain, and Figure 5-3C represents the difference or increase in smoke $PM_{2.5}$.The mean increase ranged from 0.05 µg/m3 to 9.5 µg/m3. This represents the mean from 123 fire season days. Thus, the spatial distribution of the wildfire $PM_{2.5}$ is likely higher on some days compared with others. Nevertheless, this figure helps us to visualize the areas with more prominent average exposure. As

shown, there appears to be higher potential exposures in Northern Idaho and Southwest Montana, the Oregon coast, and much of the state of Nevada. In order to use this data with the selected population projections, it was necessary to aggregate the data to county level.

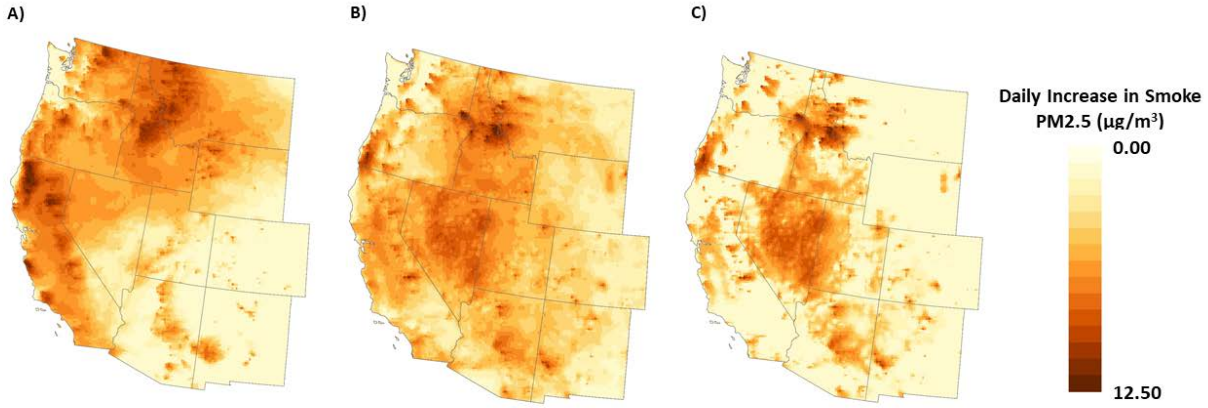

Figure 5- 3

Gridded and county-level daily increases in fire $PM_{2.5}$. *Figure 5-3A depicts the present daily mean smoke $PM_{2.5}$ for the 2000s on a 12km CMAQ grid. Figure 5-3B shows the future predicted daily mean smoke $PM_{2.5}$ for the 2050s, and 5-3C shows the increase in smoke $PM_{2.5}$ (i.e., difference between 3B and 3A).*

We also examined the smoke $PM_{2.5}$ concentrations as a ratio of the total $PM_{2.5}$ concentration. Figure 5-4A plots the gridded daily smoke $PM_{2.5}$ as a percentage of the total for the base years (2000s). As shown, on any given day, smoke $PM_{2.5}$ could represent up to 85.0% of the total $PM_{2.5}$ exposure. Figure 5-4B shows the gridded daily smoke $PM_{2.5}$ for the 2050s. After aggregation to county level, the range of percentages is limited to a maximum of 41.5% due to the loss of resolution.

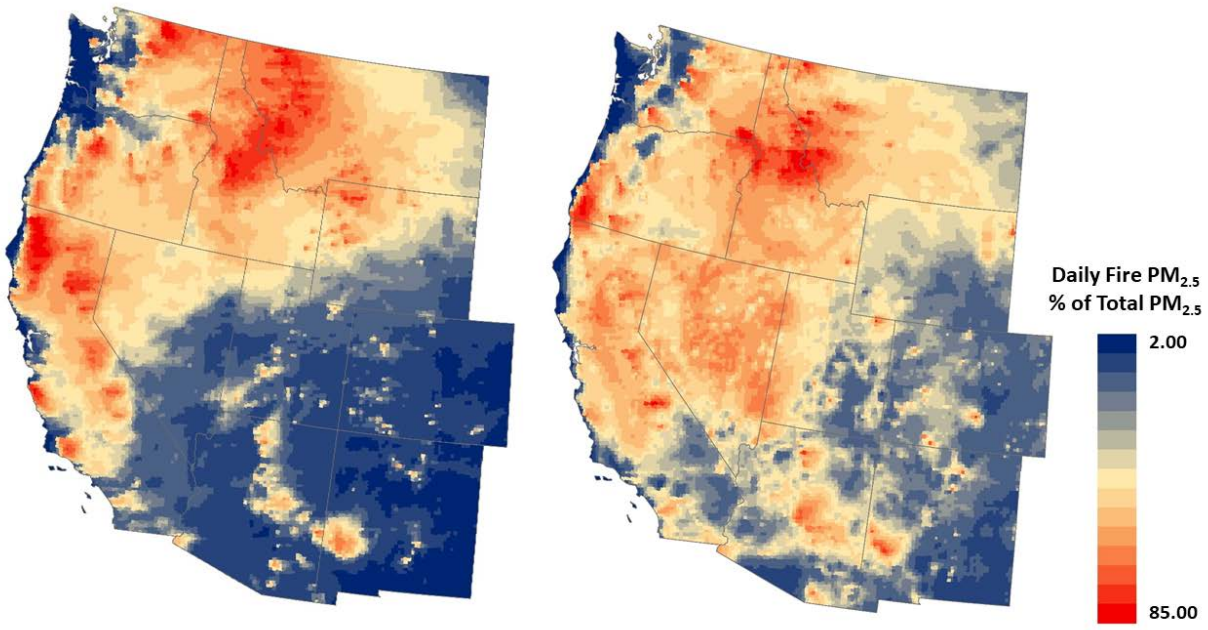

Figure 5- 4

Gridded and county-level daily increases in fire $PM_{2.5}$ as a percentage of total $PM_{2.5}$. *Figure 5-4A depicts the gridded present daily smoke $PM_{2.5}$ as a percentage of total $PM_{2.5}$ on a 12km CMAQ grid. Figure 5-4B plots the gridded future daily smoke $PM_{2.5}$ as a percentage of total $PM_{2.5}$ on a 12km CMAQ grid.*

5.4.2 Epidemiological Metrics & Health Impact Assessment

Another way to assess this data is to normalize the results by county population. Normalizing data by county population makes between county comparisons more direct and meaningful. As shown in Figure 5-5, normalizing changes some of the burden distribution, though the general overall trends remain. Some key variances are evident in central California, central Washington and Arizona, with less pronounced burden after accounting for the size of

population. However, even with normalizing, several of the burden “hotspots” remain such as Northern Idaho and the coast of Oregon.

5.4.3 Statewide Potential Increased Burden

We also chose to evaluate the data by each state to give additional context to potential burden evaluations. In Table 1, we see both population change and the potential increase in wildfire events. The states with the highest increase in ED per 10,000 persons appear to be Idaho, Montana, Nevada, Oregon, and Utah. However, it is important to look at the individual population growth for each state because some of this increase is likely due to population growth. Our analyses showed increases in population for most of the states. Two states, Montana and Wyoming, are expected to lose population under the ICLUS A2 scenario. Therefore, while the states mentioned show higher rates of increase, it is also important to look at changes in population. For example, Idaho has the highest rate of increase in total events, with a lower positive increase in population. However, the state of Montana has a lower rate of increase but loses population over the same period. Hence, emphasis on states with low population growth and relatively high rates of increase in ED events may be crucial to any action or policy plan decisions.

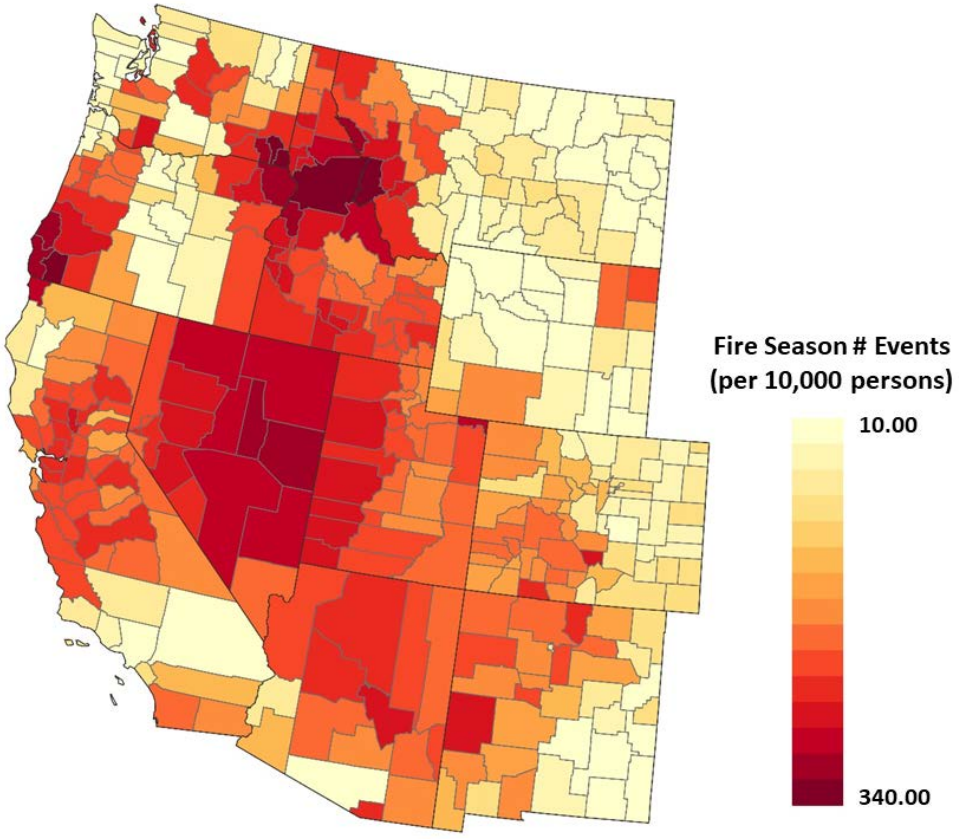

Figure 5- 5

Total Fire Season Increase in Respiratory Events. *Plotted mean total # of events in a given fire season normalized by population.*

Table 5- 1

Statewide increases in smoke $PM_{2.5}$ ED visits compared to increases in population.

State/Region	Pop 2000 (# persons)	ICLUS A2 Population 2055 (# persons)	Increase in Population (% change)	Total Events	Total Events per 10k persons
Western US	60,582,730	107,782,959	78%	207,135	19.2
Arizona	5,130,632	9,741,197	89%	19,015	19.5
California	33,094,915	58,508,475	77%	107,043	18.3
Colorado	4,301,261	10,301,536	140%	13,203	12.8
Idaho	1,293,953	1,799,131	39%	7,171	39.9
Montana	902,195	741,485	-18%	2,121	28.6
Nevada	1,998,257	5,562,262	178%	14,596	26.2
New Mexico	1,819,046	3,145,300	73%	5,464	17.4
Oregon	3,421,399	3,973,599	16%	9,957	25.0
Utah	2,233,169	4,713,124	111%	13,554	28.8
Washington	5,894,121	9,018,389	53%	14,678	16.3
Wyoming	493,782	278,461	-46%	333	12.0

5.4.3 Potential Increases in Health Care Costs

Along with increases in events and higher levels of $PM_{2.5}$ from smoke, it is important to understand the potential impact on counties and states when it comes to the actual cost due to any increase. In Figure 5-6, the average annual increase in health care from these potential respiratory ED visits can be seen on the county level. These numbers again are normalized by population and represented as increase in cost due per 10,000 persons. The distribution of the cost per county in

Figure 5-6 is very closely related to Figure 5-5 because it is a monetary calculation based on the annual fire season increase in ED visits. Thus, the areas of Idaho, Nevada, and Oregon continue to stand out as areas that may experience the largest monetary burden compared to other counties.

In Table 2, the county numbers (shown in thousands of dollars) are aggregated to the state level to see the potential impact that statewide systems may experience. Looking at some of the identified hotspots, the burden on some states is higher when compared with the relative size of the state and potential resources. For instance, Idaho, with a much smaller population and total annual cost than a state like California, has a greater burden in cost per 10,000 persons than the most populated state. Based on the results shown in this table, it appears that the same states and counties that experience higher increases in additional events may carry a disproportionate burden when it comes to the actual cost increase associated with these events.

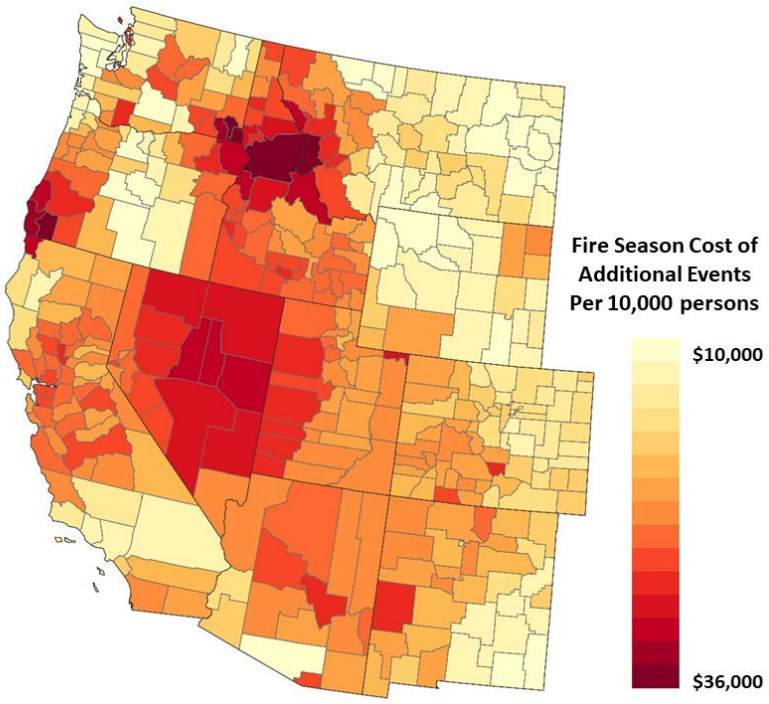

Figure 5- 6

Predicted fire season total cost increase due to smoke $PM_{2.5}$ exposure. *Average total fire season cost of increases in smoke $PM_{2.5}$ (in thousands of dollars) normalized by population.*

Table 5- 2

Predicted statewide fire season total cost increase due to smoke $PM_{2.5}$ exposure. *Increase in average fire season cost for increases in smoke $PM_{2.5}$ in total state burden and burden normalized by population.*

State/Region	Total Cost	Cost per 10k persons
Western US	$219,355,965	$20,351
Arizona	$20,136,885	$20,672
California	$113,358,537	$19,375
Colorado	$13,981,977	$13,573
Idaho	$7,594,089	$42,210
Montana	$2,246,139	$30,292
Nevada	$15,457,164	$27,789
New Mexico	$5,786,376	$18,397
Oregon	$10,544,463	$26,536
Utah	$14,353,686	$30,455
Washington	$15,544,002	$17,236
Wyoming	$352,647	$12,664

5.5 Discussion

5.5.1 Summary and impact of results

This research has sought to address some of the gaps in our understanding of health effects associated with smoke $PM_{2.5}$ exposure. Using a complex modeling scheme, we were able to separate smoke $PM_{2.5}$ from ambient levels for a present period (2003-2010) and a future period (2050-2059). By calculating the difference between future and present levels, we were able to estimate the increase in smoke $PM_{2.5}$ that we might expect under RCP8.5 in the 2050s. Additionally, we adopted region-specific health metrics from our Colorado study to tailor our approach to typical Western US conditions. This included the use of both incidence rates and OR values for fire season respiratory ED visits estimated in our previous study.

As we hypothesized, many areas in the study domain experienced substantial increases in smoke-related $PM_{2.5}$ based on comparisons between future and present results. For increased exposure to smoke $PM_{2.5}$ across the region, we found an average season increase of 244 ED respiratory visits for every 10,000 persons. Increase in expected ED visits varied by county and state, with some consistent “hotspots” seen in northern Idaho, Nevada, and the coast of Oregon. Additionally, we compared these results normalized by population. While some of the spatial distribution of burden resulted, the same identified areas remained the highest in additional expected ED visits.

It is true that some of this increase could be due to natural population growth. Thus, it was imperative that we compared the future expected population increase with the rate of fire season respiratory events. Using this strategy, Wyoming and Montana both exhibited significant loss to population, but retained some of the highest increases in ED visits. This is a useful comparison

since the burden will be felt by a smaller population—both in size of health effect and potential strain put on community and governmental entities.

Given our hypothesis that fire smoke $PM_{2.5}$ may be more toxic than ambient $PM_{2.5}$ concentrations, and, based on the results from our previous work, we also analyzed the results by the percent contribution of smoke $PM_{2.5}$ to ambient $PM_{2.5}$. After aggregation to county, the average daily smoke contribution ranged from 1.0-41.5% across all counties. While there was some loss to resolution as a result of aggregation, the overall pattern is informative and may be a useful tool in planning for future health impacts.

This additional strain is also apparent in a crude glance at potential increases in monetary burden attributed to the additional ED visits related to fire smoke $PM_{2.5}$. Using the 2017 estimated cost of an average ED visit, $1,059, we calculated the monetary burden by county. Similar patterns were seen with, again, Idaho, Nevada and Oregon experiencing some of the highest monetary impacts. The total fire-season cost for the region $219,355,965 or $20,351 per every 10,000 persons.

5.5.2 Comparison with current literature

It is difficult to make direct comparisons between our study and previous literature since our methods do not closely resemble the different methods used in other published work. This is mostly due to the objective of directly attributing respiratory health outcomes to smoke-specific $PM_{2.5}$ concentrations. For example, in Liu et al. (2016), estimates of future wildfire-specific $PM_{2.5}$ were derived from GEOS-Chem model simulations based on a future IPCC scenario, A1B.[15] This scenario assumes moderate growth in global emissions. Using this framework, authors projected future fire $PM_{2.5}$ levels on a 0.5 x 0.67 degree grid, aggregated to county level. In addition to the calculations of wildfire-specific $PM_{2.5}$, the study used “smoke wave” events within their modeling

scheme. They define smoke waves as at least two consecutive days with smoke $PM_{2.5}$ higher than the 98th quartile of all fire-specific $PM_{2.5}$. Then, using population projections from the US Census, they calculated the future population for each county. Similar to our work, they calculated the change in number of respiratory admissions by taking the difference between future and present conditions. However, these were only calculated for the elderly population, or 65+.

While the Liu et al. approach is promising, we made different choices for our projections based on the results of our previous Colorado smoke-specific $PM_{2.5}$ analysis.[16] In that study, we did not find significant associations for respiratory admissions among the elderly populations for 3-day moving average smoke $PM_{2.5}$ exposure. Additionally, we did not constrain our methods to any type of exposure categorization (such as smoke waves). Instead, we chose to estimate the overall and age-stratified burden for any amount of smoke-specific $PM_{2.5}$ exposure using the CMAQ modeling framework. Finally, another significant difference in our analysis is normalization by population to better understand the future health burden using between-county comparisons.

5.5.3 Strengths & limitations

Our analysis includes some limitations, most of which surround the uncertainty of predicting future fire activity and resulting health effects. Some uncertainty comes from the exposure modeling procedures that were used to create both present and future gridded datasets. Due to the nature of modeling, especially predictive, there could be some exposure misclassification. Additionally, the metrics used for predicting the number of future ED visits came from a state-specific study in Colorado and may not be entirely appropriate to extend to the whole Western US domain. There is also chance for bias introduced by the ICLUS population inputs and

the RCP 8.5 inputs. Since each of these are predictions based on assumed conditions, either could introduce some error in our model configuration.

However, some limitations are to be expected when using new approaches to modeling environmental exposures and health effects. To our knowledge, this is the first study to use the CMAQ fire vs. no fire capabilities to project the difference between present and future impact of wildfires. Also, the inputs we used at some stages in our model are improved inputs directly from the EPA and may not be available for other studies. As mentioned, the health metrics for incidence and risk were taken from the Colorado study. This could be viewed as a strength given that conditions in the Western US are very different than areas in other wildfire studies.

5.6 Study Conclusions and future directions

The results of this work suggest that substantial increases in health burden may be seen by the mid-21st century. Using novel modeling methods and region-specific health metrics, we calculated future increases in adverse respiratory health outcomes. From this analysis, we identified counties in the Western US that may shoulder more health events and substantial associated costs. Using a population-normalized approach, we calculated the increase in burden due to wildfire-specific $PM_{2.5}$ by county. Using these methods, several counties exhibited disparate effects. Additionally, it was important to consider potential population growth for each county and state. After analysis, some other areas (such as Montana & Wyoming) were identified as shouldering some of the heaviest health and economic costs despite expected overall loss in population. Moving forward, it will be important to look at several permutations of our modeling scheme as well as incorporate other methods for future fire-related $PM_{2.5}$.

5.7 References

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6. CONCLUSIONS

Air quality is gaining ground as one of the major concerns surrounding climate change and subsequent adaptation. Each year, an approximate 7 million deaths are linked to poor air quality, as estimated by the WHO. Additionally, the WHO estimates that 91% of the world's population is exposed to air quality deemed unhealthy. Many studies have been conducted attempting to outline and quantify the impact of climate change on human exposures to worsening air quality. The overall goal of this body of work was to provide insight into key aspects of the epidemiology surrounding this growing health concern. Specifically, this research examined the excess mortality for increased exposures to O_3 using multiple climate and emissions scenarios (Aim 1), characterized the impact of wildfire events on human health (Aim 2), and forecasted the future morbidity of smoke exposure due to changes in climate and emissions (Aim 3).

6.1 Contribution of Aim 1

Ozone is an important chemical found in earth's atmosphere. Depending on the location in the atmosphere, O_3 can have either helpful or detrimental impacts on the earth system. Aim 1 focused on the mortality impact of O_3 in the troposphere and projected the number of excess deaths from O_3 exposure that might be expected in the 2050s. In Aim 1, we utilized multiple emissions and population scenarios to identify the differences in O_3-related excess deaths. Through a hybrid modeling approach, we blended statistical and dynamical downscaling methods. This allowed for us to ascertain that changes in emissions may be the source of the main incongruities between high and low emission scenarios.

The results from this study point to the differing contributions to O_3 from climate change and emissions policies. Under both RCP4.5 and RCP8.5, we found that largest impact is likely to

be from changes in current emissions policies going forward. Using our hybrid dynamical downscaling method, we were able to separate projected future concentrations of O_3 due to the separate "sources" of emissions and climate. We do know that climate change alone could cause adverse health outcomes and mitigation strategies will serve to alleviate some of that impact. However, it appears that substantial benefits may come from limiting our future emissions. This is clear in the comparison that we made between the RCP scenarios. It is important to note that, in each of the RCPs, O_3 precursors are expected to decrease. However, our results using RCP8.5 suggest an increase in future O_3 concentrations. This could partly be due to large increases in global methane that have been estimated for the RCP8.5 scenario.

In this national-scale mortality study, we investigated the differences in excess deaths due to the overarching source of the O_3 concentrations (i.e., climate change or changes in precursor emissions). This study provides evidence that, while the impacts of both climate change and emissions policies play a part, the health burden of the exposure will vary greatly depending on the changes made to current policy and practices. Ultimately, these results can be used to inform future policy decisions and recommendations as the results show large decreases in excess deaths from O_3 under stricter policy scenarios.

We concluded that, while climate change alone may cause some adverse health effects due to poorer air quality, substantial and more immediate health benefits may be achieved by emission mitigation of O_3 precursors regardless of changing climate conditions (especially under higher emission scenarios). The results of this investigation demonstrate potential significant rises in O_3 mortality due to differences in future emissions policies. Thus, as we demonstrated, climate change induced O_3 increase in the US could be substantially offset by changes to worldwide emissions policies moving forward.

6.2 Contribution of Aim 2

Finding ways to demonstrate current health burden is a vital part of understanding potential future impacts of poor air quality. The purpose of Aim 2 was to address the extent of smoke $PM_{2.5}$ exposure under current conditions. Our study domain consisted of the state of Colorado and our health data represents all ED visits or hospitalizations from both private and public hospitals during the fire seasons of 2011-2014. Using high-resolution exposure modeling, we sought to isolate the contribution of smoke $PM_{2.5}$ to ambient levels. This was made possible using satellite data, to enhance our exposure estimates on a 4km scale. Accounting for both spatial and temporal variability, we successfully separated smoke $PM_{2.5}$ from other sources to better understand and quantify wildfire-specific exposures and their resulting health effects.

As we originally hypothesized, multiple respiratory outcomes were associated with increased smoke exposure. The results from our study estimated higher risk of respiratory ED events that are higher than those previously published in the literature. This was especially true for asthma cases where an OR of 1.081 was estimated. The interpretation of this measure is that an individual is at an 8% higher risk for and ED visit or hospitalization due to asthma on due to each increase in 1 $\mu g/m^3$ smoke $PM_{2.5}$ exposure. We also found significant increases in overall respiratory events, with some significant associations that are dependent on age stratification. For example, we found that the risk of a bronchitis event is statistically significant for adults and that we might expect a 4% increase in risk due to each unit of increase in smoke $PM_{2.5}$.

The findings from this study point to potential toxicological differences between smoke and ambient $PM_{2.5}$. We found associations with multiple respiratory outcomes for the fire seasons (May-Aug) during 2011-2014. Our results suggest that future smoke exposure could pose a significant threat to public health. This analysis can inform public health and healthcare systems

regarding the potential future burden of wildfire smoke $PM_{2.5}$ exposure within the context of climate change.

The design of this Aim 2 focused on smoke $PM_{2.5}$ contributions to multiple cardiorespiratory outcomes. We observed estimated increases in many of the respiratory outcomes during periods of wildfire activity. We also observed heterogeneity in the associations when stratifying by age. Associations were seen in asthma events and overall respiratory events. The effect was enhanced in children and adults presenting with asthma. Older adults did not exhibit a significant increase in respiratory outcomes. In the adult group, we observed increases in both bronchitis and COPD. Unlike some available literature, smoke $PM_{2.5}$ was not significantly associated with any observed cardiovascular outcome.

Our findings point to potential toxic differences between smoke and non-smoke $PM_{2.5}$ exposure; suggesting that $PM_{2.5}$ from wildfire smoke could pose a significant threat to public health. This has been suggested in both in vitro and in vivo studies with human cells and animal studies. We did not differentiate by specific compounds in smoke $PM_{2.5}$, but there seems to be a potential difference in exposure to fire vs. ambient $PM_{2.5}$. This is especially true given the expected climate change-related impacts on wildfire incidence, with exposure and risk increasing with future wildfire activity.

It is, therefore, important to derive more accurate concentration-response relationships (CRFs) specific to wildfire smoke in order to develop a better understanding of future potential health risks based on increased wildfire activity. Additionally, calculating CRFs for specific locations or regions may enhance our understanding of future wildfire $PM_{2.5}$ exposure and the variation there might be from place to place. Thus, the calculation of CRFs from the results of this study tailor the potential impact to the state of Colorado and could be extrapolated to the surrounding region.

Taken together, the results of this analysis can inform public health agencies and healthcare systems regarding the potential burden of wildfire smoke $PM_{2.5}$ exposure within the context of future climate change. This information may be a key element in evaluating and enhancing current preparations aimed at wildfire-event response readiness.

6.3 Contribution of Aim 3

Using novel methods to predict future smoke related $PM_{2.5}$, Aim 3 sought to describe the future impact of increasing wildfire on human health. Similar to Aim 2, we used complicated models to separate the future smoke $PM_{2.5}$ contributions to ambient or total $PM_{2.5}$. This was accomplished by calculating the differences in smoke $PM_{2.5}$ concentrations between a present period (2003-2010) and a future estimation (2050-2059). This analysis was designed as a "base case" scenario to help us understand the future burden using multi-year averages to estimate predicted impact. This involved converting the results from Aim 2 into updated concentration response functions (CRF) based on the exposure-disease relationship shown in the previous aim. We also calculated the annual incidence of respiratory ED events using data from Aim 2. This application was expanded to include the entire Western US and provided respiratory ED visit projections for the 2050s.

Multiple "hotspots" for fire smoke $PM_{2.5}$ and increases in ED visits, including Idaho, Nevada and the coast of Oregon. However, there was a large loss of resolution in our exposure data due to county-level aggregation. The differences can be seen when placing gridded and aggregated maps side by side as done in Chapter 5. We approached this analysis from several angles. First, we were able to calculate the daily mean increase in smoke $PM_{2.5}$. This was then analyzed using a ratio of smoke $PM_{2.5}$ to ambient concentrations. In this comparison, even with

loss of detail from aggregation, some counties averaged more than 40% smoke $PM_{2.5}$ on any given day.

We also mapped the average increase in respiratory ED events for a single future fire season. This was done, again, using both the gridded data and the aggregated data. Not surprisingly much of the variation in the data was lost due to aggregation. Again, the same areas of Idaho, Nevada and Oregon stand out as areas with more future impact. However, we were not able to do a direct comparison between counties until we normalized by population. After accounting for the population in each county, we calculated rates of future events per 10,000 people. The same hotspots emerged, but with some variations across the domain due to normalization.

Next, we calculated the average percent increase in events for a fire season and compared that to a calculation of the rate of increase in population. It is true that some of our estimation is due to population growth. However, it became clear that there are states that could be not proportional to the other statues due to low increase or negative changes in population. The best example of this is Montana, where they have the second highest rate of increase in events but lose approximately 18% of their population. Taking this into consideration, policy and emergency response plans may need to be adjusted to meet the future burden.

Finally, we estimated a substantial burden from contemporary average ED costs using the future visits. In this analysis, we looked at the average ED costs from 2017 which were estimated to be $1,059. We then applied this mean cost to future wildfire season respiratory ED events due to smoke $PM_{2.5}$ exposure. The costs varied from county to county, with some experiencing larger hardships. This is important to incorporate in any future response planning—especially since this estimation likely underestimates the future cost due to our use of contemporary inflation.

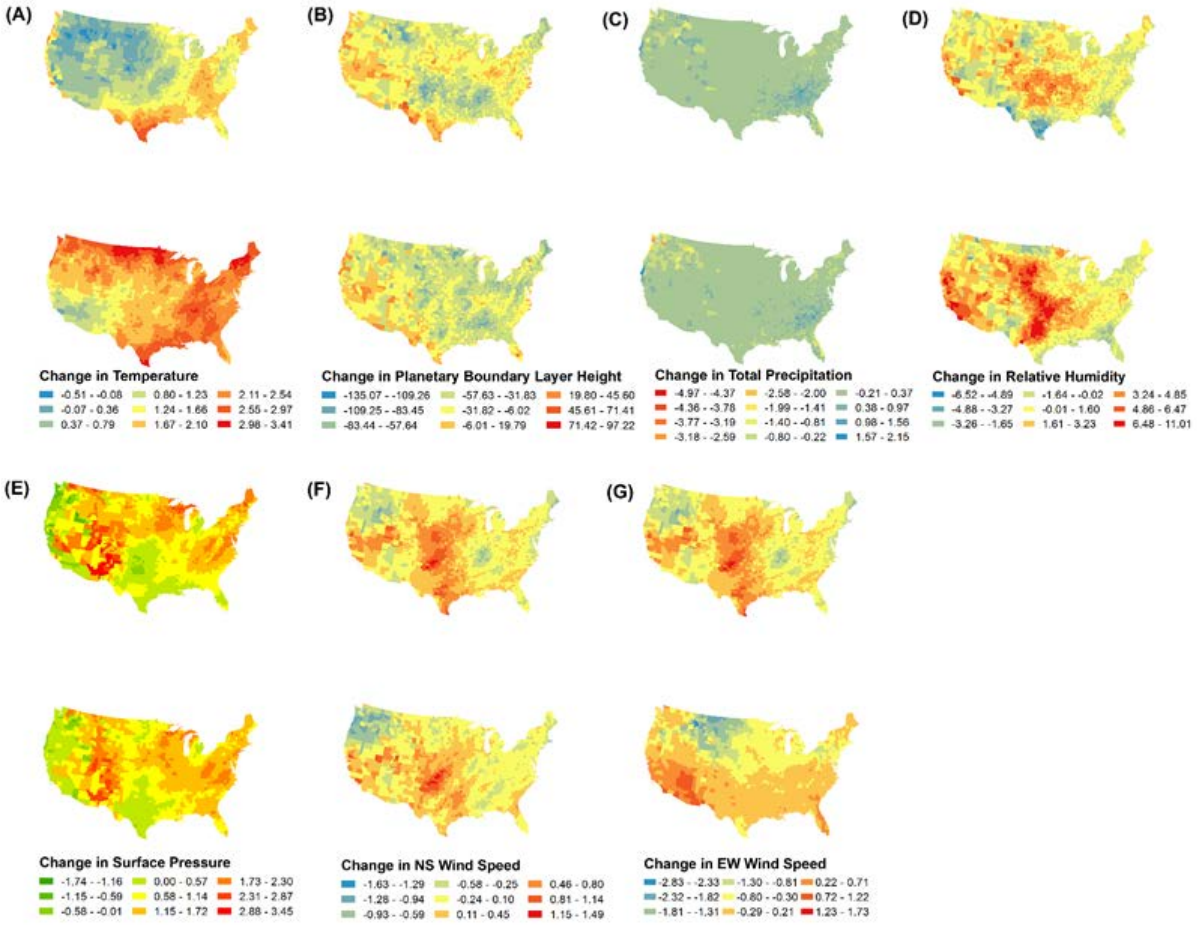
Therefore, this estimation does not take into consideration any sort of inflation or overall increase cost of provided services.

6.4 Summary & future directions

This body of work aimed to investigate methods used to understand the effect that climate change can have on air quality and human health. We approached this issue in a variety of ways and on a multiple spatial scales, including a national assessment, a regional assessment and a statewide assessment. Building upon each aim, we improved our methods of separating exposures from natural ambient conditions. This culminated in our estimation of the future "base case" health effects due to increased wildfire smoke $PM_{2.5}$ exposure. Moving forward, this analysis can be expanded to include multiple permutations of future and present results. Additionally, we plan to increase the accuracy of our cost projections by accounting for changes in inflation and cost of healthcare. Finally, we plan to incorporate the pieces necessary to contribute to a policy paper outlining the potential threat of future wildfire activity and considerations for revising current warning and response strategies.

7. APPENDICES

i. Chapter 3 Supplemental



Supplemental Figure 3- 1

Differences between 2000s and 2050s in (A) temperature, (B) planetary boundary layer, (C) precipitation, (D) relative humidity, (E) surface pressure, and (F and G) meridional and zonal wind speeds. Top row reflects RCP4.5 and bottom row reflects RCP8.5.

Parameter	Normal Distribution	Correlation Test	Average Correlation Coefficient
Temperature	Y	Pearson	0.963
Pressure	N	Spearman	0.990
Planetary Boundary Layer Height	Y	Pearson	0.412
Relative Humidity	N	Spearman	0.924
Precipitation	N	Spearman	0.559
Wind Speeds at 10 hPa	Y	Pearson	0.800
Wind Speeds at 500 hPa	N	Spearman	0.947
V Wind Vector	N	Spearman	0.862
U Wind Vector	Y	Pearson	0.770

Supplemental Table 3-1.

Correlation of NARR climate parameters to WRF modeled climate parameters for use in statistical modeling.

RCP8.5	CLIMATE CHANGE				EMISSIONS				COMBINED CLIMATE AND EMISSIONS			
REGION	**ICLUS A2**	**ICLUS A1**	**ICLUS B2**	**ICLUS B1**	**ICLUS A2**	**ICLUS A1**	**ICLUS B2**	**ICLUS B1**	**ICLUS A2**	**ICLUS A1**	**ICLUS B2**	**ICLUS B1**
National	47 (SE=525)	48 (SE=433)	63 (SE=505)	80 (SE=458)	2167 (SE=1386)	1940 (SE=1139)	1853 (SE=1319)	1897 (SE=1233)	2217 (SE=900)	2012 (SE=802)	1992 (SE=876)	2029 (SE=874)
Northeast	330 (SE=17)	310 (SE=22)	305 (SE=19)	318 (SE=27)	365 (SE=43)	365 (SE=57)	338 (SE=48)	373 (SE=68)	678 (SE=59)	653 (SE=79)	623 (SE=66)	671 (SE=95)
Southeast	-100 (SE=17)	-99 (SE=19)	-88 (SE=16)	-89 (SE=20)	405 (SE=35)	403 (SE=39)	312 (SE=34)	344 (SE=42)	298 (SE=19)	283 (SE=20)	236 (SE=17)	253 (SE=21)
East North Central	-35 (SE=1)	-31 (SE=1)	-34 (SE=1)	-33 (SE=1)	161 (SE=3)	137 (SE=2)	152 (SE=3)	145 (SE=2)	126 (SE=3)	106 (SE=2)	118 (SE=3)	112 (SE=2)
Central	87 (SE=4)	71 (SE=3)	98 (SE=4)	93 (SE=4)	161 (SE=12)	132 (SE=10)	141 (SE=12)	130 (SE=12)	239 (SE=15)	205 (SE=12)	239 (SE=17)	224 (SE=15)
West North Central	5 (SE=1)	4 (SE=1)	6 (SE=1)	5 (SE=1)	9 (SE=1)	7 (SE=1)	10 (SE=1)	9 (SE=1)	13 (SE=1)	11 (SE=1)	15 (SE=1)	14 (SE=1)
South	-13 (SE=3)	-13 (SE=3)	-16 (SE=2)	-15 (SE=2)	169 (SE=6)	149 (SE=5)	146 (SE=5)	144 (SE=5)	152 (SE=6)	135 (SE=5)	131 (SE=6)	130 (SE=5)
Southwest	18 (SE=7)	18 (SE=6)	13 (SE=5)	13 (SE=4)	98 (SE=15)	83 (SE=13)	83 (SE=13)	82 (SE=12)	114 (SE=13)	102 (SE=11)	99 (SE=10)	94 (SE=9)
Northwest	-5 (SE=2)	-3 (SE=2)	-4 (SE=2)	-1 (SE=3)	122 (SE=8)	114 (SE=9)	115 (SE=8)	116 (SE=9)	112 (SE=9)	107 (SE=10)	108 (SE=8)	115 (SE=10)
West	-170 (SE=182)	-144 (SE=143)	-130 (SE=169)	-138 (SE=153)	678 (SE=468)	551 (SE=367)	557 (SE=440)	553 (SE=395)	475 (SE=287)	404 (SE=228)	417 (SE=273)	409 (SE=242)

Supplemental Table 3-2.

RCP4.5	CLIMATE CHANGE				EMISSIONS				COMBINED CLIMATE AND EMISSIONS			
REGION	**ICLUS A2**	**ICLUS A1**	**ICLUS B2**	**ICLUS B1**	**ICLUS A2**	**ICLUS A1**	**ICLUS B2**	**ICLUS B1**	**ICLUS A2**	**ICLUS A1**	**ICLUS B2**	**ICLUS B1**
National	72 (SE=456)	120 (SE=369)	105 (SE=431)	110 (SE=400)	-41 (SE=1037)	-20 (SE=839)	-70 (SE=967)	31 (SE=882)	50 (SE=615)	68 (SE=526)	27 (SE=586)	84 (SE=591)
Northeast	204 (SE=12)	195 (SE=15)	186 (SE=13)	193 (SE=19)	-2 (SE=28)	30 (SE=37)	-1 (SE=31)	37 (SE=45)	208 (SE=40)	220 (SE=52)	183 (SE=44)	232 (SE=63)
Southeast	-47 (SE=10)	-50 (SE=11)	-41 (SE=9)	-48 (SE=11)	-186 (SE=14)	-145 (SE=16)	-149 (SE=14)	-139 (SE=16)	-237 (SE=5)	-199 (SE=5)	-198 (SE=4)	-181 (SE=5)
East North Central	-18 (SE=1)	-15 (SE=1)	-16 (SE=1)	-16 (SE=1)	22 (SE=1)	19 (SE=1)	10 (SE=1)	11 (SE=1)	4 (SE=1)	4 (SE=1)	-7 (SE=1)	-6 (SE=1)
Central	76 (SE=4)	65 (SE=3)	85 (SE=4)	80 (SE=4)	-42 (SE=7)	-37 (SE=6)	-77 (SE=8)	-72 (SE=7)	30 (SE=11)	27 (SE=9)	9 (SE=12)	15 (SE=11)
West North Central	5 (SE=1)	4 (SE=1)	5 (SE=1)	5 (SE=1)	-20 (SE=1)	-16 (SE=1)	-24 (SE=1)	-22 (SE=1)	-15 (SE=1)	-12 (SE=1)	-19 (SE=1)	-17 (SE=1)
South	-11 (SE=3)	-13 (SE=2)	-13 (SE=3)	-15 (SE=3)	-80 (SE=3)	-66 (SE=3)	-83 (SE=3)	-78 (SE=3)	-91 (SE=4)	-78 (SE=3)	-98 (SE=4)	-92 (SE=3)
Southwest	20 (SE=7)	18 (SE=6)	13 (SE=5)	13 (SE=4)	-71 (SE=7)	-59 (SE=6)	-54 (SE=6)	-48 (SE=5)	-52 (SE=3)	-40 (SE=2)	-40 (SE=2)	-35 (SE=2)
Northwest	-12 (SE=2)	-11 (SE=1)	-11 (SE=2)	-10 (SE=1)	46 (SE=6)	44 (SE=6)	37 (SE=6)	42 (SE=7)	34 (SE=5)	34 (SE=6)	27 (SE=5)	33 (SE=6)
West	-143 (SE=154)	-73 (SE=121)	-103 (SE=145)	-91 (SE=131)	292 (SE=355)	209 (SE=281)	270 (SE=328)	299 (SE=294)	165 (SE=199)	119 (SE=155)	156 (SE=183)	137 (SE=167)

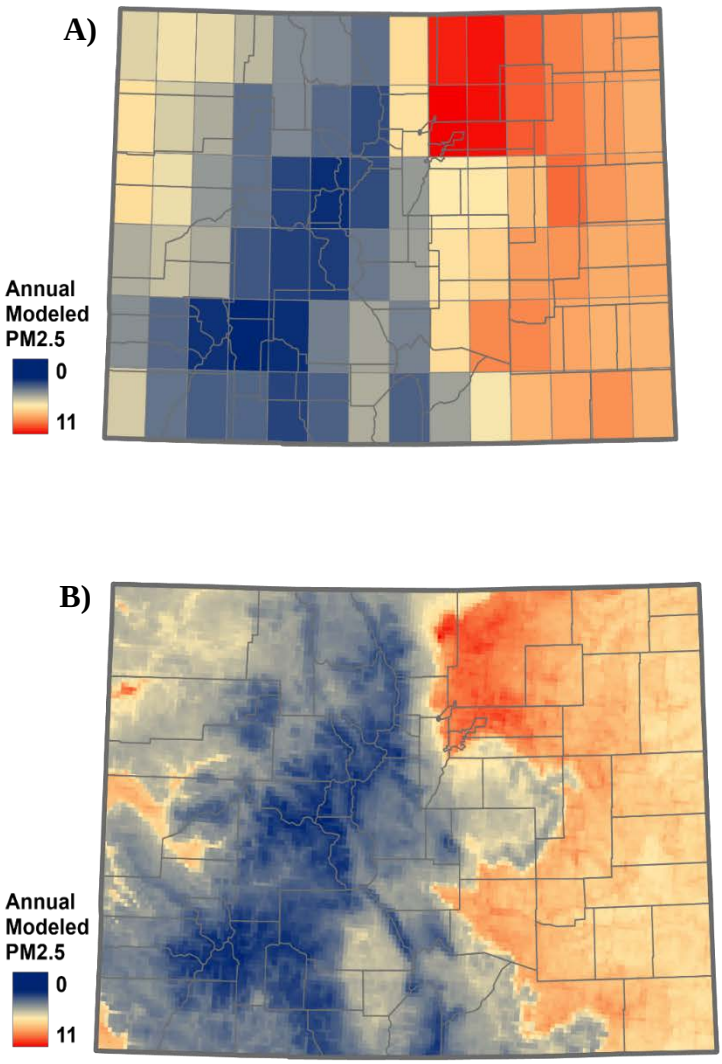
Supplemental Table 3.2-3.3. *Projected excess deaths using ICLUS population A1, A2, B1 and B2 scenarios attributable to climate change only, anthropogenic emissions only, and combined effects of both climate change and emissions for 2050s from baseline 2000s by US climatic region. Supplemental Table 2 shows results for RCP8.5 and Supplemental Table 3 shows results for RCP4.5. (SE: standard error)*

	Representative Concentration Pathway		
Source	**Population**	***RCP4.5***	***RCP8.5***
Combined Climate Change and Emissions	**A2**	230 (SE=31.54)	486 (SE=44.13)
	A1	160 (SE=23.97)	398 (SE=35.24)
	B2	201 (SE=28.11)	428 (SE=41.50)
	B1	147 (SE=25.95)	406 (SE=37.51)
Climate Change Only	**A2**	110 (SE=21.63)	180 (SE=23.92)
	A1	89 (SE=17.06)	122 (SE=18.56)
	B2	115 (SE=20.34)	161 (SE=22.36)
	B1	99 (SE=18.40)	136 (SE=19.86)
Emissions Only	**A2**	86 (SE=8.98)	314 (SE=21.21)
	A1	66 (SE=7.12)	252 (SE=16.37)
	B2	79 (SE=7.33)	292 (SE=19.89)
	B1	70 (SE=7.47)	262 (SE=17.45)

Supplemental Table 3-3.

Projected excess deaths using ICLUS population A1, A2, B1 and B2 scenarios attributable to climate change only, anthropogenic emissions only, and combined effects of both climate change and emissions for 2050s from baseline 2000s for California.

ii. Chapter 4 Supplemental



Supplemental Figure 4- 1

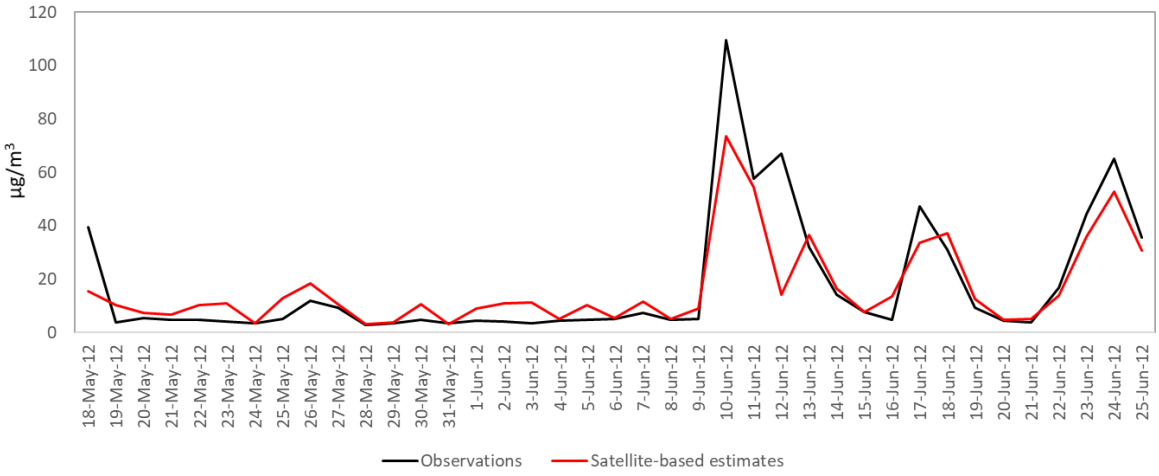
Comparing 2012 $PM_{2.5}$ annual average concentrations for A) coarse grid scale (0.50x0.67 degrees) and B) 4km study grid. As shown, the coarse resolution CTM results could not resolve the $PM_{2.5}$ concentration gradients related to the rapid terrain change.

$PM_{2.5}$ Exposure Modeling

The $PM_{2.5}$ estimates used in this study are based on our previous work (Geng et al., 2018), with the incorporation of some additional measurements. The input dataset, which includes ground $PM_{2.5}$ observations, MAIAC AOD, smoke mask, meteorological fields, land-use variables and CMAQ simulations, are adopted directly from Geng et al. (2018). More details about the data can be found in Geng et al. (2018). To better reflect the $PM_{2.5}$ enhancements during fire events, we also utilize $PM_{2.5}$ measurements during May-June, 2012 from the National Park Service made at the Atmospheric Science Department of Colorado State University, which is near the edge of the 2012 High Park Fire (Val Martin et al., 2013; Benedict et al., 2017).

Two models are involved to provide a full spatial and temporal coverage of $PM_{2.5}$ estimates. The first is a random forest model that incorporates MAIAC AOD, smoke mask, meteorological fields, and as land-use variables (i.e. the AOD model). The second is a statistical downscaler that calibrates the CMAQ $PM_{2.5}$ simulations, as described in Geng et al. (2018). For grid cells that have missing values in the AOD downscaler due to the missing of MAIAC AOD, we use the estimated values from the CMAQ downscaler to fill in the gaps.

The out-of-bag R-squared of the AOD model is 0.92, and the 10-fold cross-validated (CV) R-squared of the CMAQ downscaler is 0.52. Overall, the CV R-squared is 0.81. The estimated $PM_{2.5}$ data capture the elevated $PM_{2.5}$ concentrations during fire events, as shown in an example in Supplemental Figure 4-2.



Supplemental Figure 4- 2

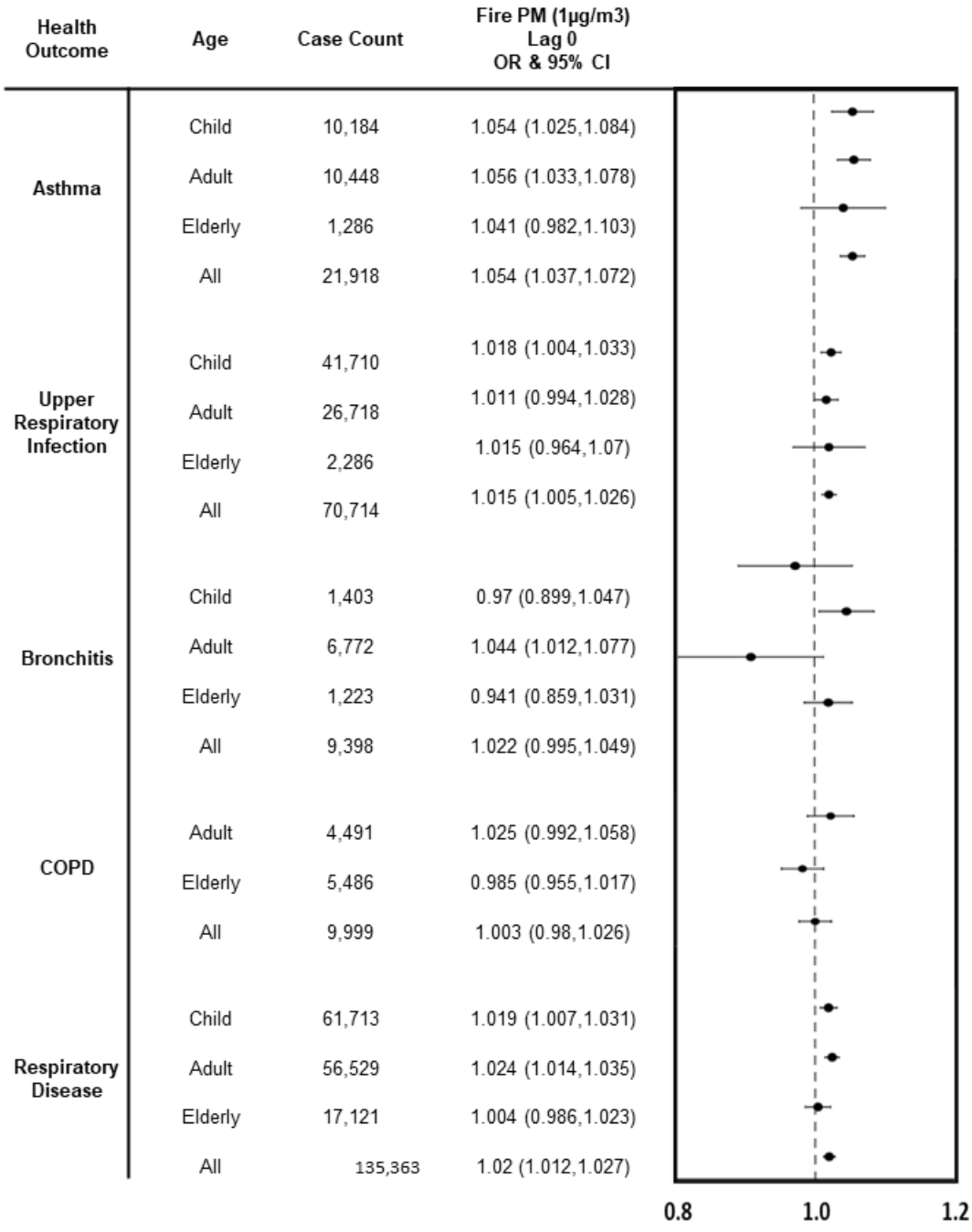
Time series of $PM_{2.5}$ observations and our estimates during High Park Fire in a near monitor

References:

Benedict, K.B., Prenni, A.J., Carrico, C.M., Sullivan, A.P., Schichtel, B.A., Collett, J.L., 2017. Enhanced concentrations of reactive nitrogen species in wildfire smoke. Atmospheric Environment 148, 8-15

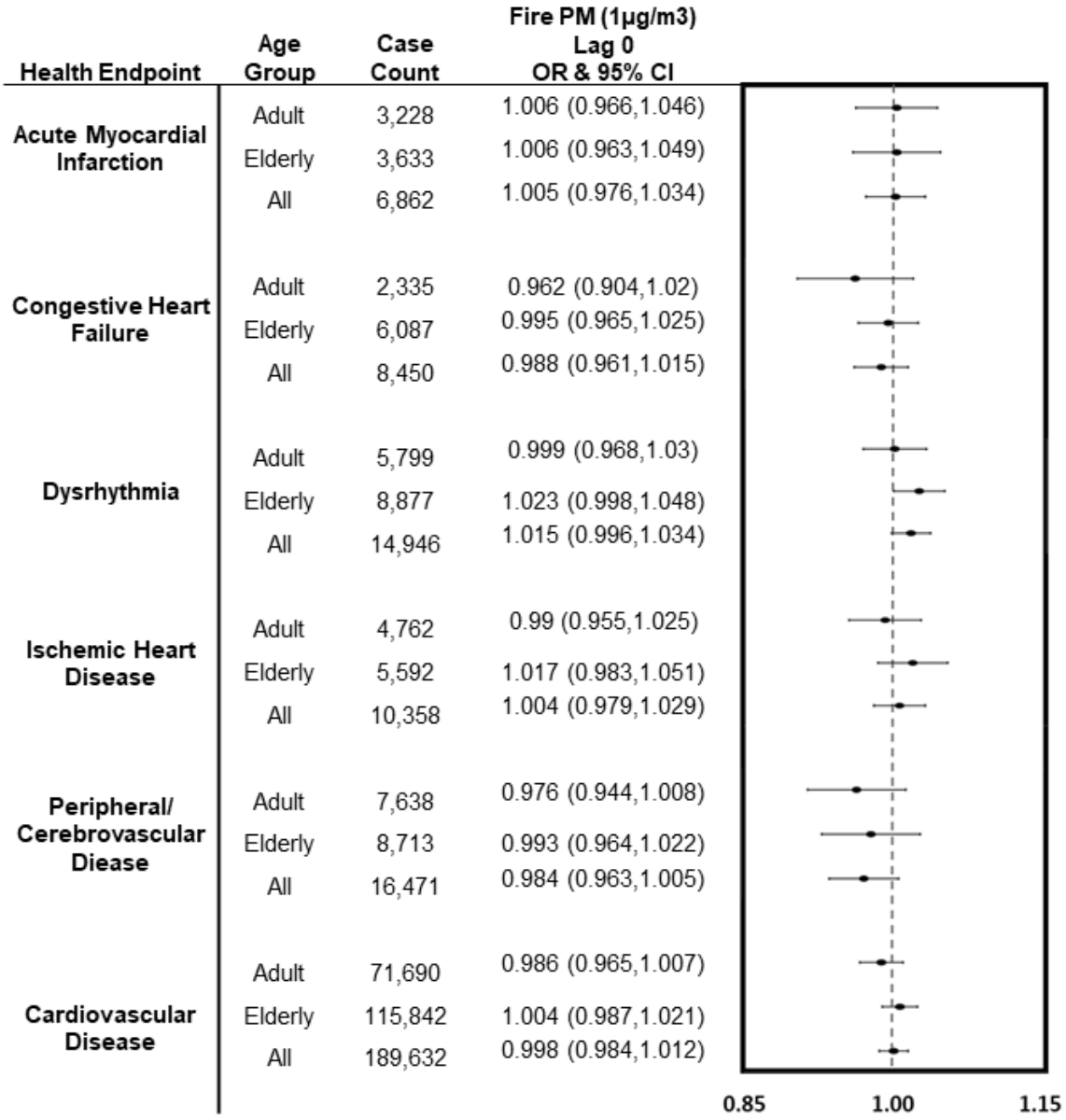
Geng, G., Murray, N.L., Tong, D., Fu, J.S., Hu, X., Lee, P., et al., 2018. Satellite-Based Daily $PM_{2.5}$ Estimates During Fire Seasons in Colorado. Journal of Geophysical Research: Atmospheres 123 (15), 8159-8171

Val Martin, M., Heald, C.L., Ford, B., Prenni, A.J., Wiedinmyer, C., 2013. A decadal satellite analysis of the origins and impacts of smoke in Colorado. Atmos Chem Phys 13 (15), 7429-7439



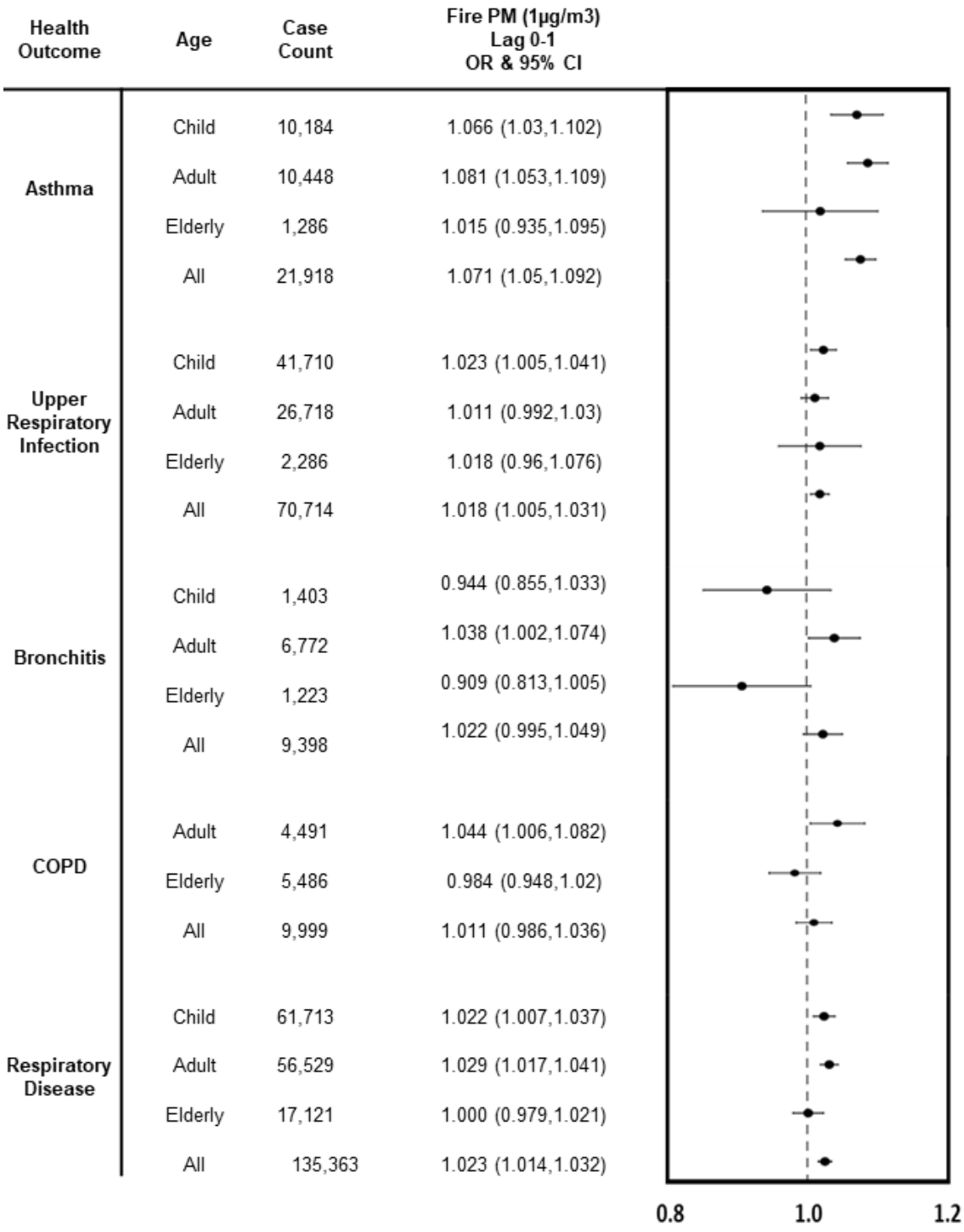
Supplemental Figure 4- 3

OR Respiratory Results for Lag 0 Exposures.



Supplemental Figure 4- 4

OR Cardiovascular Results for Lag 0 Exposures.



Supplemental Figure 4- 5

OR Respiratory Results for Lag 0-1 Exposures.

Supplemental Table 4- 1

Overall Odd Ratios for Respiratory Outcomes due to 3-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM ($1\mu g/m^3$) 3-day average OR (95% CI)
Respiratory		
Asthma	21,918	1.081(1.058, 1.105)*
Upper respiratory infection	70,714	1.008(0.995, 1.022)
Bronchitis	9,398	1.018(0.984, 1.052)
Chronic obstructive pulmonary	9,999	1.020(0.991, 1.049)
Respiratory disease	135,363	1.021(1.012, 1.031)*

Supplemental Table 4- 2

Overall Odd Ratios for Cardiovascular Outcomes due to 2-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM (1µg/m^3) 2-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	1.011(0.982, 1.041)
Acute Myocardial Infarction	6,862	1.006(0.972, 1.042)
Dysrhythmia	14,946	1.005(0.982, 1.028)
Congestive Heart Failure	8,450	0.986(0.955, 1.018)
Peripheral/Cerebrovascular Disease	16,471	0.971(0.946, 0.996)
Cardiovascular Disease	50,225	0.998(0.984, 1.011)

Supplemental Table 4- 3

Overall Odd Ratios for Respiratory Outcomes due to 3-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 3-day average OR (95% CI)
Respiratory		
Asthma	21,918	0.998(0.996, 1.001)
Upper respiratory infection	70,714	0.998(0.996, 0.999)
Bronchitis	9,398	1.003(0.998, 1.007)
Chronic obstructive pulmonary	9,999	0.998(0.994, 1.002)
Respiratory disease	135,363	0.999(0.998, 1.000)

Supplemental Table 4- 4

Overall Odd Ratios for Cardiovascular Outcomes due to 2-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 2-day average OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	0.998(0.995, 1.002)
Acute Myocardial Infarction	6,862	1.002(0.998, 1.007)
Dysrhythmia	14,946	0.997(0.994, 1.000)
Congestive Heart Failure	8,450	0.994(0.990, 0.998)
Peripheral/Cerebrovascular Disease	16,471	0.995(0.992, 0.997)
Cardiovascular Disease	189,632	0.994(0.993, 0.996)

Supplemental Table 4- 5

Overall Odd Ratios for Respiratory Outcomes due to 7-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM (1µg/m^3) 7-day average OR (95% CI)
Respiratory		
Asthma	21,918	1.081 (1.051,1.112)*
Upper respiratory infection	70,714	0.987 (0.970,1.004)
Bronchitis	9,398	1.029 (0.986,1.074)
Chronic obstructive pulmonary	9,999	1.022 (0.987,1.059)
Respiratory disease	135,363	1.007 (0.995,1.019)

Supplemental Table 4- 6

Overall Odd Ratios for Respiratory Outcomes due to 7-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 7-day average OR (95% CI)
Respiratory		
Asthma	21,918	1.001 (0.997,1.004)
Upper respiratory infection	70,714	0.998 (0.996,1.000)
Bronchitis	9,398	1.004 (0.998,1.009)
Chronic obstructive pulmonary	9,999	0.996 (0.991,1.001)
Respiratory disease	135,363	0.999 (0.998,1.000)

Supplemental Table 4- 7

Overall Odd Ratios for Cardiovascular Outcomes due to 3-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM (1µg/m^3) 3-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	1.011 (0.980,1.043)
Acute Myocardial Infarction	6,862	1.000 (0.963,1.038)
Dysrhythmia	14,946	0.995 (0.970,1.021)
Congestive Heart Failure	8,450	0.979 (0.944,1.015)
Peripheral/Cerebrovascular Disease	16,471	0.963 (0.937,0.991)
Cardiovascular Disease	50,225	0.991 (0.976,1.005)

Supplemental Table 4- 8

Overall Odd Ratios for Cardiovascular Outcomes due to 3-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 3-day average OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	0.998 (0.994,1.001)
Acute Myocardial Infarction	6,862	1.001 (0.996,1.006)
Dysrhythmia	14,946	0.996 (0.993,1.000)
Congestive Heart Failure	8,450	0.995 (0.990,1.000)
Peripheral/Cerebrovascular Disease	16,471	0.994 (0.991,0.997)
Cardiovascular Disease	189,632	0.993 (0.992,0.995)

Supplemental Table 4- 9

Sex Stratified Odd Ratios for Respiratory Outcomes due to 3-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 3-day average OR (95% CI)
Respiratory		
Asthma		
Male	10,647	0.998 (0.994,1.002)
Female	11,271	0.999 (0.995, 1.002)
Upper respiratory infection	70,714	
Male	32,013	0.998 (0.995,1.000)
Female	38,701	0.998 (0.996,1.000)
Bronchitis	9,398	
Male	3,675	0.997 (0.990,1.005)
Female	5,723	1.007 (1.001,1.013)*
Chronic obstructive pulmonary	9,999	
Male	4,699	0.997 (0.991,1.003)
Female	5,300	0.999 (0.994,1.005)
Respiratory disease	135,363	
Male	62,889	0.998 (0.997,1.000)
Female	72,474	0.999 (0.998,1.001)

Supplemental Table 4- 10

Sex Stratified Odd Ratios for Cardiovascular Outcomes due to 2-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total PM (1μg/m^3) 2-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	
Male	6,893	0.998 (0.994,1.003)
Female	3,465	0.998 (0.992,1.004)
Acute Myocardial Infarction	6,862	
Male	4,592	1.002 (0.997,1.008)
Female	2,270	1.002 (0.995,1.010)
Dysrhythmia	14,946	
Male	7,615	0.997 (0.993,1.001)
Female	7,331	0.997 (0.992,1.001)
Congestive Heart Failure	8,450	
Male	4,262	0.996 (0.991,1.002)
Female	4,188	0.992 (0.986,0.998)
Peripheral/Cerebrovascular Disease	16,471	
Male	7,816	0.994 (0.990,0.998)
Female	8,655	0.995 (0.991,0.999)
Cardiovascular Disease	50,225	
Male	26,586	0.995 (0.993,0.997)
Female	23,639	0.994 (0.991,0.996)

Supplemental Table 4- 11

Sex Stratified Odd Ratios for Cardiovascular Outcomes due to 2-day Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM ($1\mu g/m^3$) 2-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	
Male	6,893	0.997 (0.961,1.033)
Female	3,465	1.038 (0.989,1.090)
Acute Myocardial Infarction	6,862	
Male	4,592	1.004 (0.963,1.048)
Female	2,270	1.010 (0.952,1.072)
Dysrhythmia	14,946	
Male	7,615	1.011 (0.981,1.043)
Female	7,331	0.997 (0.964,1.032)
Congestive Heart Failure	8,450	
Male	4,262	0.991 (0.946,1.039)
Female	4,188	0.982 (0.939,1.026)
Peripheral/Cerebrovascular Disease	16,471	
Male	7,816	0.958 (0.922,0.996)
Female	8,655	0.981 (0.948,1.016)
Cardiovascular Disease	50,225	
Male	26,586	0.997 (0.979,1.016)
Female	23,639	0.997 (0.978,1.017)

Supplemental Table 4- 12

Sex Stratified Odd Ratios for Respiratory Outcomes due to 3-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke $PM_{2.5}$ ($3\mu g/m^3$) 3-day average OR (95% CI)
Respiratory		
Asthma		
Male	10,647	1.063 (1.029,1.098)*
Female	11,271	1.096 (1.064,1.128)*
Upper respiratory infection		
Male	32,013	1.000 (0.980,1.021)
Female	38,701	1.015 (0.997,1.034)
Bronchitis		
Male	3,675	0.970 (0.920,1.023)
Female	5,723	1.054 (1.010,1.101)*
Chronic obstructive pulmonary		
Male	4,699	1.019 (0.978,1.061)
Female	5,300	1.020 (0.981,1.061)
Respiratory disease		
Male	62,889	1.014 (1.000,1.028)
Female	72,474	1.027 (1.015,1.040)*

Supplemental Table 4- 13

Age Stratified Odd Ratios for Respiratory Outcomes due to 3-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ (3µg/m^3) 3-day average OR (95% CI)
Respiratory		
Asthma		
Children (0-18)	10,184	0.998 (0.994,1.002)
Adults (19-64)	10,448	0.999 (0.995, 1.003)
Older Adult (65+)	1,286	1.000 (0.988, 1.012)
Upper respiratory infection		
Children (0-18)	41,710	0.999 (0.997,1.001)
Adults (19-64)	26,718	0.995 (0.993,0.999)
Older Adult (65+)	2,286	0.998 (0.990,1.007)
Bronchitis		
Children (0-18)	1,403	1.008 (0.996,1.020)
Adults (19-64)	6,772	1.003 (0.997,1.008)
Older Adult (65+)	1,223	0.999 (0.986,1.011)
Chronic obstructive pulmonary		
Children (0-18)	22	--
Adults (19-64)	4,491	1.000 (0.994,1.006)
Older Adult (65+)	5,486	0.997 (0.991,1.002)
Respiratory disease		
Children (0-18)	61,713	1.001 (0.999,1.002)
Adults (19-64)	56,529	0.998 (0.996,1.000)
Older Adult (65+)	17,121	0.996 (0.993,0.999)

Supplemental Table 4- 14

Age Stratified Odd Ratios for Respiratory Outcomes due to 3-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM (1µg/m^3) 3-day average OR (95% CI)
Respiratory		
Asthma		
Children (0-18)	10,184	1.075 (1.035,1.116)*
Adults (19-64)	10,448	1.091 (1.060, 1.122)*
Older Adult (65+)	1,286	1.009 (0.920, 1.106)
Upper respiratory infection		
Children (0-18)	41,710	1.010 (0.991,1.029)
Adults (19-64)	26,718	1.005 (0.984,1.026)
Older Adult (65+)	2,286	1.004 (0.940,1.072)
Bronchitis		
Children (0-18)	1,403	0.971 (0.890,1.060)
Adults (19-64)	6,772	1.044 (1.005,1.085)*
Older Adult (65+)	1,223	0.908 (0.805,1.024)
Chronic obstructive pulmonary		
Children (0-18)	22	--
Adults (19-64)	4,491	1.056 (1.015,1.100)*
Older Adult (65+)	5,486	0.989 (0.951,1.030)
Respiratory disease		
Children (0-18)	61,713	1.016 (1.000,1.032)
Adults (19-64)	56,529	1.030 (1.017,1.044)*
Older Adult (65+)	17,121	1.000 (0.976,1.024)

Supplemental Table 4- 15

Age Stratified Odd Ratios for Cardiovascular Outcomes due to 2-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 2-day average OR (95% CI)
Cardiovascular		
Ischemic Heart Disease		
Adults (19-64)	4,762	1.000 (0.995,1.005)
Elderly (65+)	5,592	0.997 (0.992,1.002)
Acute Myocardial Infarction		
Adults (19-64)	3,228	1.005 (0.999,1.011)
Elderly (65+)	3,633	1.000 (0.994,1.006)
Dysrhythmia		
Adults (19-64)	5,799	0.999 (0.994,1.004)
Elderly (65+)	8,877	0.996 (0.992,1.000)
Congestive Heart Failure		
Adults (19-64)	2,335	0.991 (0.983,0.999)
Elderly (65+)	6,087	0.996 (0.991,1.000)
Peripheral/Cerebrovascular Disease		
Adults (19-64)	7,638	0.994 (0.990,0.998)
Elderly (65+)	8,713	0.995 (0.991,0.999)
Cardiovascular Disease		
Adults (19-64)	71,690	0.995 (0.992,0.998)
Elderly (65+)	115,842	0.994 (0.992,0.996)

Supplemental Table 4- 16

Age Stratified Odd Ratios for Cardiovascular Outcomes due to 2-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM ($1\mu g/m^3$) 2-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease		
Adults (19-64)	4,762	0.999 (0.957, 1.043)
Elderly (65+)	5,592	1.022 (0.982, 1.063)
Acute Myocardial Infarction		
Adults (19-64)	3,228	1.011 (0.963,1.062)
Elderly (65+)	3,633	1.002 (0.954,1.052)
Dysrhythmia		
Adults (19-64)	5,799	0.993(0.956,1.030)
Elderly (65+)	8,877	1.010 (0.980,1.040)
Congestive Heart Failure		
Adults (19-64)	2,335	0.962 (0.896,1.032)
Elderly (65+)	6,087	0.993 (0.957,1.029)
Peripheral/Cerebrovascular Disease		
Adults (19-64)	7,638	0.964 (0.928,1.002)
Elderly (65+)	8,713	0.978 (0.945,1.013)
Cardiovascular Disease		
Adults (19-64)	20,534	0.986 (0.965,1.008)
Elderly (65+)	29,269	1.004 (0.987,1.021)

Supplemental Table 4- 17

Age Stratified Odd Ratios for Respiratory Outcomes due to 7-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM (1µg/m^3) 7-day average OR (95% CI)
Respiratory		
Asthma		
Children (0-18)	10,184	1.053 (1.001,1.108)*
Adults (19-64)	10,448	1.097 (1.059, 1.138)*
Older Adult (65+)	1,286	1.023 (0.912, 1.148)
Upper respiratory infection		
Children (0-18)	41,710	0.980 (0.957,1.005)
Adults (19-64)	26,718	0.995 (0.970,1.021)
Older Adult (65+)	2,286	0.961 (0.879,1.049)
Bronchitis		
Children (0-18)	1,403	1.017 (0.922,1.122)
Adults (19-64)	6,772	1.046 (0.994,1.101)
Older Adult (65+)	1,223	0.935 (0.809,1.080)
Chronic obstructive pulmonary		
Children (0-18)	22	--
Adults (19-64)	4,491	1.059 (1.005,1.115)*
Older Adult (65+)	5,486	0.994 (0.946,1.045)
Respiratory disease		
Children (0-18)	61,713	0.990 (0.970,1.010)
Adults (19-64)	56,529	1.019 (1.002,1.036)*
Older Adult (65+)	17,121	0.999 (0.970,1.029)

Supplemental Table 4- 18

Age Stratified Odd Ratios for Respiratory Outcomes due to 7-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 7-day average OR (95% CI)
Respiratory		
Asthma		
Children (0-18)	10,184	1.000 (0.994,1.005)
Adults (19-64)	10,448	1.002 (0.997, 1.007)
Older Adult (65+)	1,286	0.997 (0.983, 1.012)
Upper respiratory infection	70,714	
Children (0-18)	41,710	0.999 (0.996,1.001)
Adults (19-64)	26,718	0.997 (0.994,1.000)
Older Adult (65+)	2,286	1.003 (0.993,1.014)
Bronchitis	9,398	
Children (0-18)	1,403	1.006 (0.991,1.022)
Adults (19-64)	6,772	1.003 (0.996,1.010)
Older Adult (65+)	1,223	1.004 (0.989,1.020)
Chronic obstructive pulmonary	9,999	
Children (0-18)	22	--
Adults (19-64)	4,491	0.998 (0.991,1.006)
Older Adult (65+)	5,486	0.994 (0.988,1.001)
Respiratory disease	135,363	
Children (0-18)	61,713	1.000 (0.998,1.002)
Adults (19-64)	56,529	0.999 (0.997,1.001)
Older Adult (65+)	17,121	0.995 (0.991,1.000)

Supplemental Table 4- 19

Sex Stratified Odd Ratios for Respiratory Outcomes.

Health Endpoint	Case Count	Smoke $PM_{2.5}$ ($3\mu g/m^3$) 7-day average OR (95% CI)
Respiratory		
Asthma		
Male	10,647	1.071 (1.027,1.117)*
Female	11,271	1.089 (1.048,1.131)*
Upper respiratory infection	70,714	
Male	32,013	0.973 (0.948,0.999)
Female	38,701	0.998 (0.975,1.021)
Bronchitis	9,398	
Male	3,675	0.994 (0.930,1.063)
Female	5,723	1.055 (0.998,1.116)
Chronic obstructive pulmonary	9,999	
Male	4,699	1.018 (0.967,1.071)
Female	5,300	1.026 (0.977,1.078)
Respiratory disease	135,363	
Male	62,889	1.004 (0.987,1.022)
Female	72,474	1.010 (0.994,1.026)

Supplemental Table 4- 20

Sex Stratified Odd Ratios for Respiratory Outcomes .

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 7-day average OR (95% CI)
Respiratory		
Asthma		
Male	10,647	1.000 (0.995,1.005)
Female	11,271	1.001 (0.997,1.006)
Upper respiratory infection		
Male	32,013	0.999 (0.996,1.001)
Female	38,701	0.998 (0.995,1.000)
Bronchitis		
Male	3,675	1.000 (0.991,1.009)
Female	5,723	1.006 (0.999,1.014)
Chronic obstructive pulmonary		
Male	4,699	0.994 (0.987,1.001)
Female	5,300	0.998 (0.991,1.004)
Respiratory disease		
Male	62,889	0.999 (0.997,1.001)
Female	72,474	0.999 (0.997,1.001)

Supplemental Table 4- 21

Age Stratified Odd Ratios for Cardiovascular Outcomes due to 3-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM (1µg/m^3) 3-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	
Adults (19-64)	4,762	1.011 (0.966, 1.059)
Elderly (65+)	5,592	1.010 (0.968, 1.055)
Acute Myocardial Infarction	6,862	
Adults (19-64)	3,228	1.016 (0.964,1.071)
Elderly (65+)	3,633	0.985 (0.935,1.037)
Dysrhythmia	14,946	
Adults (19-64)	5,799	0.986(0.946,1.027)
Elderly (65+)	8,877	0.997 (0.965,1.031)
Congestive Heart Failure	8,450	
Adults (19-64)	2,335	0.974 (0.906,1.048)
Elderly (65+)	6,087	0.980 (0.940,1.022)
Peripheral/Cerebrovascular Disease	16,471	
Adults (19-64)	7,638	0.952 (0.912,0.993)
Elderly (65+)	8,713	0.973 (0.937,1.011)
Cardiovascular Disease	50,225	
Adults (19-64)	20,534	0.984 (0.961,1.007)
Elderly (65+)	29,269	0.994 (0.975,1.013)

Supplemental Table 4- 22

Age Stratified Odd Ratios for Cardiovascular Outcomes due to 3-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total $PM_{2.5}$ ($3\mu g/m^3$) 3-day average OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	
Adults (19-64)	4,762	1.000 (0.995, 1.006)
Elderly (65+)	5,592	0.995 (0.990, 1.000)
Acute Myocardial Infarction	6,862	
Adults (19-64)	3,228	1.005 (0.998,1.012)
Elderly (65+)	3,633	0.998 (0.991,1.005)
Dysrhythmia	14,946	
Adults (19-64)	5,799	0.997 (0.992,1.003)
Elderly (65+)	8,877	0.995 (0.991,0.999)
Congestive Heart Failure	8,450	
Adults (19-64)	2,335	0.993 (0.985,1.002)
Elderly (65+)	6,087	0.995 (0.990,1.001)
Peripheral/Cerebrovascular Disease	16,471	
Adults (19-64)	7,638	0.992 (0.988,0.997)
Elderly (65+)	8,713	0.995 (0.991,0.999)
Cardiovascular Disease	189,632	
Adults (19-64)	71,690	0.994 (0.991,0.997)
Elderly (65+)	115,842	0.993 (0.991,0.995)

Supplemental Table 4- 23

Sex Stratified Odd Ratios for Cardiovascular Outcomes due to 3-day average Smoke $PM_{2.5}$.

Health Endpoint	Case Count	Smoke PM ($1\mu g/m^3$) 3-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	
Male	6,893	0.999 (0.960,1.038)
Female	3,465	1.034 (0.981,1.090)
Acute Myocardial Infarction	6,862	
Male	4,592	0.998 (0.953,1.044)
Female	2,270	1.003 (0.941,1.069)
Dysrhythmia	14,946	
Male	7,615	1.002 (0.968,1.038)
Female	7,331	0.987 (0.951,1.025)
Congestive Heart Failure	8,450	
Male	4,262	1.000 (0.950,1.054)
Female	4,188	0.961 (0.913,1.011)
Peripheral/Cerebrovascular Disease	16,471	
Male	7,816	0.956 (0.917,0.966)
Female	8,655	0.970 (0.933,1.008)
Cardiovascular Disease	50,225	
Male	26,586	0.995 (0.975,1.015)
Female	23,639	0.986 (0.965,1.007)

Supplemental Table 4- 24

Sex Stratified Odd Ratios for Cardiovascular Outcomes due to 3-day average Total $PM_{2.5}$.

Health Endpoint	Case Count	Total PM (1µg/m^3) 3-day OR (95% CI)
Cardiovascular		
Ischemic Heart Disease	10,358	
Male	6,893	0.998 (0.994,1.003)
Female	3,465	0.996 (0.989,1.003)
Acute Myocardial Infarction	6,862	
Male	4,592	1.002 (0.996,1.008)
Female	2,270	1.000 (0.991,1.008)
Dysrhythmia	14,946	
Male	7,615	0.996 (0.992,1.001)
Female	7,331	0.995 (0.991,1.000)
Congestive Heart Failure	8,450	
Male	4,262	0.996 (0.990,1.002)
Female	4,188	0.994 (0.987,1.000)
Peripheral/Cerebrovascular Disease	16,471	
Male	7,816	0.993 (0.989,0.998)
Female	8,655	0.994 (0.990,0.999)
Cardiovascular Disease	50,225	
Male	26,586	0.994 (0.992,0.996)
Female	23,639	0.993 (0.990,0.995)