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Effect of Wildfire Smoke on Asthma Emergency Department Visits and Acute Hospitalizations in Colorado

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An abstract of A thesis submitted to the Faculty of the Rollins School of Public Health of Emory University in partial fulfillment of the requirements for the degree of Master of Public Health in Environmental Health 2014

Abstract

Effect of Wildfire Smoke on Asthma Emergency Department Visits and Acute Hospitalizations in Colorado By Breanna Alman

In 2012, Colorado experienced one of its worst wildfire seasons of the past decade. The goal of this study is to explore the relationship of local PM_{2.5} levels with emergency room visits and acute hospitalizations for asthma during the Colorado wildfires of 2012 and to determine whether increased air pollution from wildfire smoke was a contributing factor. Spearman's correlation coefficient and conditional logistic regression were used to assess the relationship between PM_{2.5} and asthma visits from June 5th to July 6th 2012. The allage conditional logistic regression model showed a significant positive correlation between daily number of emergency room visits and acute hospitalizations and PM_{2.5}. Categorized PM2.5 revealed a linear increase in OR with increases in PM2.5 concentration above the referent group, $<10 \ \mu\text{g/m}^3$ (Linear Trend Test P = 0.0012); 10-20 $\mu\text{g/m}^3$ (OR 1.17 95% CI 0.877, 1.563), 20-30 μg/m³ (OR 1.559, 95% CI 1.127, 2.158), 30-40 μg/m³ (OR 1.586, 95% CI 1.047, 2.403), 40-50 μg/m³ (OR 1.937, 95% CI 1.225, 3.065), 50+ μ g/m³ (OR 2.02, 95% CI 1.254, 3.252). Increases in PM_{2.5} concentration during a wildfire period were associated with an increase in asthma visits. These results, combined with previous toxicological and epidemiological studies, provide evidence for the need for further research into the potential for adverse health effects with exposure to wildfire air pollutants. Due to climate change, in the coming decades wildfires are expected to become both more frequent and intense and thus more work is needed to ensure public health preparedness.

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I. Introduction

The 2012 Colorado Wildfires

Between March 26th and July 10th 2012, Colorado experienced one of its worst wildfire seasons of the past decade ^[1]. By the time the final fire was contained, over 600 homes had been destroyed ^[2], and more than 32,000 people had been evacuated from areas near actively burning fires ^[3]. Among the areas most affected by these fires were the heavily populated cities of Fort Collins, Colorado Springs, and Denver. During this time, Colorado also experienced an intense heat wave and drought making it easier for new fires to both start and spread ^[4],in addition to making containment considerably more difficult.

While the physical damage to homes and property is readily apparent, wildfire smoke is also hazardous to human health. Particulate matter (PM), a mixture of solid particles and liquid droplets suspended in the air, is a primary component of wildfire smoke ^[5] and has been linked with health problems, including asthma ^[5, 6].

Particulate matter is a mixture of various chemical compounds, including organic compounds, nitrates, sulfates, and various metals ^[5]. It is distinguished by particle size, with PM₁₀ characterized as particles between 2.5 and 10 micrometers in diameter, and PM_{2.5} characterized as particles that are less than 2.5 micrometers in diameter ^[5]. PM₁₀ is often referred to as "coarse" particulate matter, while PM_{2.5} is often referred to as "fine" particulate matter ^[5]. The size of the particle determines how deep the particle can travel into the respiratory tract, with smaller particles travelling further and generally having a stronger association with numerous adverse health outcomes, including asthma

exacerbation, decreased lung function, and irregular heartbeats ^[6]. Particulate matter from wildfires contains large amounts of calcium, sodium, sulfur, potassium, and magnesium ^[7]. Although there is a variable amount of PM_{2.5} in ambient urban air, wildfire smoke produces a higher exposure to these compounds than would normally occur in a non-fire situation ^[8]. Furthermore, recent toxicological studies suggest that PM_{2.5} from wildfires may have different effects, particularly in the amount of oxidative stress generated, than urban PM_{2.5} ^[9-11]. This suggests possible differences in health outcomes from PM_{2.5} from this source.

II. Literature Review

Wildfires and Asthma

Though the relationship between asthma and wildfire particulate matter has been less extensively covered than particulate matter from other sources, there is evidence to suggest that the composition of wildfire particulate matter may differ from typical urban ambient air particulate matter ^[12]. A study on the 2007 southern California wildfires found that wildfire particulate matter differs in its levels of potassium and levoglucosan, both tracers of biomass burning which were elevated 2-fold during wildfire events, as well as water-soluble organic carbon, which were also elevated ^[7]. Other studies have also found an increase in phosphorous, chloride potassium, zinc, bromine, calcium, silicone ^[13], and sulfur ^[14]. These toxicological differences may lead to differences in how particulate matter affects the body.

Toxicological literature suggests that the health outcomes may vary by particulate matter source due to a difference in cytokine and macrophage response^[9-11]. Rodent in-

vivo models using coarse wildfire PM installation have shown an increase in cytotoxicity (defined as both a decrease in viable macrophages and increase in dead macrophages in the bronchoalveolar lavage fluid) when compared to rodents exposed to equal amounts of non-wildfire coarse particulate matter ^[9]. An increase in free isoprostanes was observed within half an hour of exposure, which indicates an increase in oxidative stress ^[9].

Experiments conducted using in-vitro epithelial cells have yielded similar results ^[10]. A study in California's San Joaquin Valley compared ambient air particulate matter and wildfire particulate matter, and found that wildfire smoke increased the xenobiotic metabolism and oxidative stress response in bronchial epithelial cells more than ambient urban air pollution ^[10]. Another toxicological study that looked at macrophage death during wildfires attributed the increase in cytotoxicity to higher amounts of oxidative stress caused by wildfire particulate matter ^[15]. This difference in response indicates that there may be distinct health outcomes depending on the source of the particulate matter. Asthmatics in particular are already sensitive to particulate matter inhalation; however, wildfire smoke may prove to be an even more dangerous exposure due to the link between oxidative stress and asthma severity ^[16].

Epidemiological studies have looked at the association between respiratory admissions and ambient urban pollution; however, far fewer studies have been conducted on respiratory admissions and $PM_{2.5}$ from wildfires. The wildfire studies that have been conducted have largely found associations between wildfire particulate matter and mortality ^[17] and hospital admissions for respiratory illnesses ^[18-20].

Not all studies have shown an association between particulate matter and asthma hospitalizations. A study conducted in 1994 in Sydney, Australia called into question the relationship between acute asthma exacerbation emergency department visits and wildfire smoke; however, this study had a short duration (<10 days) and it is not clear exactly what the sample size was or whether the study was adequately powered to identify a difference if one existed ^[21]. Though cardiovascular morbidity has been linked to exposure to urban particulate matter ^[22], studies have not consistently demonstrated a link with wildfire particulate matter, and those that have tried largely achieved null results ^[18-20, 23, 24]

While various health outcomes have been linked with exposure to wildfire particulate matter, studies of respiratory illnesses have shown the most consistent results. A study conducted in southern California found positive associations between a $10 \ \mu g/m^3$ increase in 2-day moving average PM_{2.5} and respiratory admissions ^[25]. These associations were stronger after the fires than before them, suggesting that the wildfire particulate matter strengthened the relationship. Asthma admissions increased more with a $10 \ \mu g/m^3 \ PM_{2.5}$ increase than other respiratory admissions during the wildfire period (RR 1.048, 95% CI 1.021 – 1.076), including chronic obstructive pulmonary disease (COPD) and bronchitis. The largest increase was seen in those 65 years old and above (RR 1.101, 95% CI 1.030 – 1.178) and in children up to four years old (RR 1.083, 95% CI 1.021- 1.149) ^[25].

A study conducted in British Columbia yielded similar results. Researchers there found that the region of Kelowna had significant weekly increases between 46.4 - 77.7% for physician visits for respiratory diseases during the wildfires compared to the

previously established 10-year average ^[20]. Another impacted region, Kamloops, did not show this increase; however this may be partially explained by the lower overall $PM_{2.5}$ levels during the wildfire compared to Kelowna ^[20].

Though no studies to date have been able to fully separate the effect of wildfire particulate matter from urban particulate matter, studies conducted in Darwin, Australia are expected to have little urban background PM_{10} ^[26], making it an ideal place to isolate the effects of wildfire particulate matter. Results from a study conducted in Darwin, Australia during three distinct wildfire periods in 2000, 2004, and 2005, showed a positive association between $10\mu g/m^3$ increases in PM_{10} and hospital admissions. The strongest associations were found between both COPD and asthma hospital admissions. However, neither asthma (OR 1.14, 95% CI 0.90, 1.44) nor COPD (OR 1.21, 95% CI 1.0, 1.47) were significant independently; only when COPD and asthma were analyzed together did the results reach statistical significance (OR 1.19, 95% CI 1.03, 1.38).^[24] This may be partially due to a small sample size; there were only 558 hospital admissions for asthma and COPD combined, with 253 and 305 for each outcome respectively.

Similarly, in Brisbane, Australia researchers looking at PM_{10} and hospitalizations found a significant increase in hospitalizations for respiratory outcomes with higher PM_{10} levels. The researchers observed this relationship on both wildfire and non-bushfire days; however, bushfire days strengthened the relationship, suggesting that PM_{10} increases from bushfires may have a stronger effect on respiratory hospital admissions.^[27]

While many of these studies have found an association between wildfire particulate matter and an increase in respiratory illnesses, most have relied on hospital admissions rather than emergency department visits. Furthermore, many have relied on wildfire periods that were fairly short in duration, which often resulted in small sample sizes. Only one study identified looked at morbidity during wildfire periods over multiple years ^[17]. The 2012 Colorado wildfires therefore present an interesting situation: they burned continuously throughout the summer months and affected a wider geographic area across the state, thus allowing for a larger sample size than often found during typical wildfire periods, as well as creating a high variability in PM_{2.5} over time.

Given the intensity of the Colorado wildfire season of 2012, as well as the potential for stronger adverse respiratory effects from exposure to particulate matter from wildfires compared to ambient urban air, it is important to assess the health impact associated with air pollution from wildfires, particularly in sensitive populations like asthmatics.

III. Methods

Aims and Hypotheses

The goal of this study is to explore the relationship between local $PM_{2.5}$ levels and emergency room visits and acute hospitalizations during the Colorado wildfires of 2012 and to determine whether increases in air pollution from wildfire smoke contribute to emergency department visits and acute hospitalizations for asthma.

<u>Aim 1</u>:

- a. To obtain hospital admissions and emergency department visit data over a specific time period (June 5th 2012 to July 6th 2012) and then obtain PM_{2.5} data for the same time period.
- b. Determine the correlation between $PM_{2.5}$ level and emergency department visits and acute hospitalizations for asthma using the previously obtained data sets.

HYPOTHESIS: There is a positive correlation between the daily number of emergency room visits and acute hospitalizations for asthma and daily PM_{2.5} levels.

<u>Aim 2:</u>

To perform regression analysis with emergency department visits and acute hospitalizations for asthma as the dependent variable and $PM_{2.5}$ as the independent variable controlling for temperature, day of the week, and ozone.

HYPOTHESIS: The number of asthma-related emergency department visits and acute hospitalizations is positively associated with PM_{2.5} due to wildfires.

Study Design

a. Data Collection

The following data were used to assess the relationship between the exposure $(PM_{2.5} \text{ level from wildfires})$ and outcome (Emergency department visits and acute hospitalizations for asthma):

i. Hospitalization and Emergency Room Data

The Colorado Hospital Association (CHA) provided asthma hospitalization data, including patient residence, and emergency department visits from June 5th 2012 to July 6th 2012. Acute hospitalizations are defined as all hospitalizations that do not have an admit type code that is elective combined with all hospitalizations that have an admit type code that is elective that were hospitalized because they came through the emergency room. Asthma cases were identified using the International Classification of Diseases version 9 (ICD9) case group 493 for asthma. Taken together, these asthma emergency department visits and acute hospitalizations will be referred to as "asthma visits." The Colorado Department of Public Health and Environment (CDPHE) has geocoded and aggregated patient residence into 12km by 12km grids to remove identifiers. These data include information on the age, sex, date of admission, and payment method of the patient. Previous studies have concluded that hospital attendance is a valid measurement of asthma periodicity ^[28].

ii. Particulate Matter (PM 2.5) and Ozone Data

The National Center for Atmospheric Research (NCAR) provided air pollution data in the form of hourly PM_{2.5} levels between June 5th and July 6th 2012 during the wildfire period. These data were modeled using a Regional Weather Research and Forecasting Model with Chemistry (WRF-Chem) ^[29]. This model ran at a 12km by 12km spatial resolution across the Western US, and its results were used to characterize PM_{2.5} and ozone for all of Colorado. The global Model for Ozone and Related Chemical tracers (MOZART-4) ^[30] was used for the chemical boundary conditions, and NCEP/NAM was used for the metrological boundary conditions. The Wildfire emission estimates that were used to inform the model are from the NCAR Fire Inventory (FINN)^[31]. For these analyses, the daily mean and daily maximum PM_{2.5} levels for each of these 12km by 12km grids were used.

iii. Temperature Data

The North American Land Data Assimilation System (NLDS)^[32] provided mapped temperature data in 12km by 12km grids. These data will be included in the analysis when necessary, using the mean recorded temperature for the day within the specified area.

b. Data Analysis

To determine whether wildfire smoke contributes to an increase in asthma visits, two separate analyses were conducted, both using SAS 9.3 for Windows^[33].

Spearman's correlation coefficient was used to estimate the correlation between the number of asthma visits per day with $PM_{2.5}$ levels. This type of non-parametric analysis diminished the influence of potential outliers and is appropriate both when there is not expected to be a linear dose-response relationship, and when the outcome is not expected to be normally distributed. Spearman's correlation coefficient was computed using both the mean and highest daily $PM_{2.5}$ level for each of the top ten counties for the highest number of asthma visits. Random-effects meta-analysis was used to estimate the overall correlation between $PM_{2.5}$ and the daily count of asthma visits ^[34]. Conditional logistic regression was used to assess whether $PM_{2.5}$ is associated with higher daily asthma visits. Conditional logistic regression was used to control for possible confounders, including income and grid population, which are expected to vary spatially but not temporally by stratifying on the 12km by 12km grid cells. Each 12km by 12km grid including patient data and daily $PM_{2.5}$ level was matched to itself with control for temperature, day of the week, and ozone. As the analysis spanned from June 5, 2012 to July 6, 2012 this resulted in 32 observations per stratum. Concordant strata (i.e., those with zero asthma visits during the 32-day period) were dropped from the analysis. There were no missing exposure data, and no strata had less than 32 observations. This analysis relies on accurate residence geocoding; out of 1166 cases of asthma, 61 cases were unable to be geocoded, and as such were not able to be included in the analyses. These data comprise 5.2% of all cases, and it is unknown whether these cases did not provide accurate residence information, or if they were unable to be geocoded because they do not live in Colorado.

For this analysis the mean daily temperature for each 12km by 12km grid was used. To assess possible interaction with age, two additional models were created. While the initial model included all ages, the second included only patients 18 years and younger, and third included only those over 18 years of age.

c. Exposure Estimation

 $PM_{2.5}$ exposure was assigned in the Spearman's correlation coefficient analysis using the mean daily and maximum daily $PM_{2.5}$ level for the top ten counties in Colorado that had the highest amount of asthma visits. The county-level $PM_{2.5}$ concentrations were then used to characterize daily exposure for those living within the county.

For the conditional logistic regression models, grid-level exposure was estimated by spatially joining the meteorological data with the health data. This ensured that each grid had daily temperature, PM_{2.5} mean concentration, PM_{2.5} maximum concentration, ozone level, whether there was an asthma case within that grid on that day, and how many asthma cases the grid had in the thirty-two day period. The grid-level meteorological data was then used to characterize exposure for the cases within the grid. Exposures were examined as both continuous, linear dose-response variables, and as categories. All analyses were completed using zero lag exposure, one-day lag was also analyzed, but the results were not significant.

IV. Results

Demographic data on patients' asthma visits (June 5th to July 6th) are presented in Table 1. The majority of cases occurred in the highly populated areas of Denver and El Paso County, which together make up nearly half of all cases (47.19%). Females make up 58.49% of all asthma emergency department visits, and overall 45.19% of asthma patients used Medicare or Medicaid.

The results for Spearman's correlation coefficient for mean daily $PM_{2.5}$ concentration are presented in Figure 1. When considered individually, seven of the ten counties analyzed demonstrated a positive correlation ranging from 0.064 to 0.468, between $PM_{2.5}$ concentration and the number of asthma visits per day. When a meta-analysis using a random-effects model was conducted, the correlation was significant (0.177, 95% CI 0.0491, 0.298). The results remained significant when using the

maximum daily $PM_{2.5}$ concentration instead of the mean concentration, and while some precision is lost, the strength of the relationship increased (0.195, 95% CI 0.03, 0.35) (Figure 2).

In the conditional logistic regression models, ozone was analyzed with $PM_{2.5}$ in a two-pollutant model; however, it did not impact the relationship between $PM_{2.5}$ and asthma visits (Table 2). The odds ratio for the $PM_{2.5}$ model including all ages is presented in Figure 3 and Table 3. Odds ratios for same-day $PM_{2.5}$ and asthma visits were significant when $PM_{2.5}$ was above 20 µg/m³. Furthermore, there is a consistent dose-response increase in odds ratios with increasing $PM_{2.5}$ concentrations, and approximately a two-fold increase when $PM_{2.5}$ concentrations reach above 50 µg/m³ (OR 2.02, 95% CI 1.254, 3.252). When a linear trend test was performed, the results were significant (P = 0.0012).

The odds ratios for the 18 and under age group (Table 4) and the over-18 age group (Table 5) are presented in Figure 4 and Figure 5 respectively. In the 18 and under model, occurrences of asthma and wheeze were analyzed together to increase the sample size to 387 cases, of which 369 were asthma cases. The association between same-day $PM_{2.5}$ concentrations and asthma visits failed to reach significance for all $PM_{2.5}$ levels in the 18 and under model. While none of this model's $PM_{2.5}$ groups reached statistical significance, the point estimate for each group was elevated above the referent group, with the highest odds ratio observed with the highest $PM_{2.5}$ concentration of >50 µg/m³ (OR 1.777, 95% CI 0.746, 4.229). With the exception of the 30-40 µg/m³, there was an increase in odds ratio point estimates with increasing $PM_{2.5}$ concentrations.

In contrast, in the over-18 age model the odds ratios for all $PM_{2.5}$ concentration levels above 20 µg/m³ were statistically significant. The odds ratio increased to around 250% of the referent group between 40-50 µg/m³ (OR 2.427, 95% CI 1.395, 4.222). The point estimates increased with each increase in $PM_{2.5}$, with the exception of the greater than 50 µg/m³ group (OR 2.113, 95% CI 1.191, 3.748), which was slightly lower than the preceding group (OR 2.427, 95% CI 1.395, 4.222). When a linear trend test was performed, the results were significant (P= 0.0008).

Single-pollutant, 8-hour maximum ozone results for all age, and age-specific models are presented in Tables 6-8. In the all ages model, ozone was negatively correlated with asthma visits (OR 0.983, 95% CI 0.968, 0.997) (Table 6). There was no relationship in the age-specific single pollutant models (Table 7,8).

V. Discussion

Interpretation of Results

This study had two a priori hypotheses regarding the relationship between $PM_{2.5}$ and asthma visits during the 2012 Colorado wildfire season. The first postulated that there is a positive correlation between the daily number of asthma visits and daily $PM_{2.5}$ concentration at the county level. The results from the Spearman's correlation coefficient meta-analysis support this hypothesis, showing a positive correlation between the daily number of asthma visits and $PM_{2.5}$ concentration, using both the mean daily $PM_{2.5}$ (0.177, 95% CI 0.0491, 0.298) and the maximum daily $PM_{2.5}$ (0.195, 95% CI 0.03, 0.35).

The second hypothesis posited that the number of asthma visits is positively associated with $PM_{2.5}$ due to wildfires. The results from the conditional logistic regression models support this hypothesis. When all ages were included in the model, there was an

increase in the odds of asthma visits with each increase in $PM_{2.5}$ concentration, suggesting that there was an increase in asthma visits during the wildfires.

When interaction with age was assessed, the 18 and under model failed to reach significance. However, the relatively low sample size of the 18 and under group (n= 387) model may have contributed to this lack of significant findings. The increase in odds ratio point estimates with increasing $PM_{2.5}$ concentration provides evidence for a possible relationship. Further research is necessary to determine whether the relationship between asthma and $PM_{2.5}$ during wildfires varies with age.

One-day lag for $PM_{2.5}$ was also examined, with point estimates for the $PM_{2.5}$ groups ranging from 0.960 to 1.573. Only the 20-30 μ g/m³ group was significant (OR 1.573, 95% CI 1.207, 2.050). This suggests that the effects of $PM_{2.5}$ on asthma visits are likely immediate. Alternatively, there may be a small one-day lag effect that could not be detected due to a small sample size.

Overall there was a lack of association between ozone and asthma visits. Studies have demonstrated that while carbon monoxide and nitrogen monoxide are elevated during wildfires, both ozone and nitrogen dioxide remain stable ^[7, 35]. This phenomenon may be a result of reduced photochemical activity due to smoke blanket that occurs during wildfires ^[7]. This may explain the lack of association between ozone and age groups, and the significant, negative association in the all age groups model; as PM_{2.5} from wildfires increased, so did the smoke plume, and in turn ozone decreased. In general, in this data there was a weak positive association between PM_{2.5} and ozone; however, that relationship becomes negative in the highest PM_{2.5} concentration group. It

is likely that these are the levels of $PM_{2.5}$ where the particulate matter is due to wildfires, and where presumably there is also a smoke plume.

This is one of the first studies to look at concentration-response effects of PM_{2.5} over a long-lasting fire period, and one of the first to cover such a large geographic area. Furthermore, the conditional logistic regression models were able to control for the spatial variations in socio-economic status and population density at the county level that many previous studies did not take into account. These results support published research on exposure to particulate matter from wildfires and asthma ^[17-20], and taken together, suggests that there is an increased risk of asthma visits with an increase in PM_{2.5}

Potential Biases and Limitations

Both the study period and the potential for exposure misclassification presented a challenge for this study. In both the Spearman's correlation coefficient analyses and the conditional logistic regression models there were only thirty two days of exposure data, which limited the overall sample size. While the fires burned from March 26th until July 10th 2012, this study was limited to exposure data from the June 5th to July 6th period ^[1], representing approximately 30% of the total number of days when the wildfires burned, and 38.4% of all asthma cases. This limited this study's power, and was potentially an issue in the age-specific models, specifically the 18 and under model. In this model, even when wheeze cases were included with asthma, there were only 387 total cases. While this model failed to reach significance, the point estimates still suggest that there is an increase in the odds of an asthma visit with an increase in wildfire PM_{2.5} concentration.

The short study period also limited the scope of the study, and we were unable to take into account potential seasonal variations in asthma emergency department visits. However, it is unlikely that seasonal variations in asthma visits would fully explain the relationship. When examining emergency department visits from 2008 and hospitalizations from 2008-2012 each year followed a basic trend with approximately a 15 to 20% decrease in cases from June 5th to July 6th. However, 2012 did not see this general trend, instead, where there was a decrease in 2011, there were two distinct spikes in 2012, one from June 11-17th, and the other from June 25th to June 29th. While fires burned throughout the state during the summer months, the two most devastating fires, High Park and Waldo canyon, occurred on June 9th and June 23rd respectively. It is likely that these spikes in asthma emergency department visits are due in part to these fires and not seasonal trends that would naturally occur.

Within this study there is no individual-level exposure measurement. Rather, exposure was determined by patient residence within Colorado. There is a possibility of exposure misclassification in both analyses; however, it most likely influenced the correlation analyses in which PM_{2.5} concentration was averaged for the entire county, rather than a 12km by 12km grid. Within larger counties like Larimer County, which was heavily impacted by the High Park Fire, it is possible that the PM_{2.5} concentration varied widely within the county such that a person residing in a part of the county with a lower increase in PM_{2.5} was assigned a higher exposure status, potentially resulting in a modifiable areal unit problem. The potential for exposure misclassification in the conditional logistic regression models exists, but is lessened by the fact that the exposure estimate is more precise, as there is less variation of PM_{2.5} in the smaller area size. Another potential limitation of this study is selection bias. Out of the 1,166 asthma cases that occurred throughout Colorado during this time, this study was only able to use 1,105 of them, representing approximately 95% of recorded cases. The excluded cases' addresses could not be geocoded and therefore they could not be assigned an exposure level. In this situation, selection bias could result if excluded cases lived in an area of Colorado that had low PM_{2.5} concentration on the days that they visited the hospital, which would bias the results away from the null. Even so, it seems unlikely that geocoding success would be related to PM_{2.5} from the wildfires, making it non-differential.

During the Waldo Canyon fire, it was estimated that approximately 32,000 people evacuated the area ^[36]. It is possible that those who chose to evacuate had medical conditions that would make them more susceptible to an asthma attack. It is very likely that those who evacuated ended up in a different part of Colorado, and if they went to the hospital in a different area, their exposure classification would still be based on their Waldo Canyon area residence, resulting in an exposure misclassification.

VI. Conclusion

Summary

This research found that increases in $PM_{2.5}$ concentration during a wildfire period resulted in an increase in asthma visits. Furthermore, when all ages were taken together, the odds of having an ED visit or acute hospitalization increase relative to the $PM_{2.5}$ concentration.

People are exposed to wildfire particulate matter relatively infrequently compared to ambient air pollutants. However, this study, combined with previous toxicological and epidemiological studies, provide evidence for the need for further research into the potential for adverse health effects with exposure to wildfire air pollutants. This is particularly important when considering that lengthier burn seasons and more intense fire periods are projected for the future ^[37]

Recommendations for Future Research

Future research on wildfire air pollutants should focus on isolating the potential differences in health outcome differences resulting from ambient urban air pollutants versus wildfire pollutants. This study was not able to distinguish between PM_{2.5} deriving from wildfires and ambient PM_{2.5}, so it is not possible to determine whether the concentration-response for asthma would be different between the two at similar levels. Earlier studies have suggested that PM_{2.5} from wildfires may have a stronger adverse effect at the same levels ^[9], and that there is a difference in toxicological response based on particulate matter source ^[9-11].

This study did not find evidence of effect modification with age; however, this analysis may have been hindered by small sample sizes within some age categories. Given that respiratory outcomes are likely to differentially affect different age groups, it is important to study this further. Previous studies have found links between PM_{2.5} from ambient air pollution and an increase in emergency department visits for pediatric asthma ^[38, 39]. It is likely that children are especially susceptible to the adverse health effects of wildfire particulate matter, even when not as active outside, because of the potential for

the same amount of irritation causing proportionally greater airway narrowing than in adults ^[40, 41], as well as the increased oxidative stress caused by wildfire particulate matter ^[9]. Future wildfire research should focus on children as a potentially at-risk population.

Recommendations for Policy Makers

Currently, the Colorado Department of Public Health and Environment bases their public warnings about air quality on visibility, stating that if visibility is less than five miles due to smoke in your neighborhood, then the air is unhealthy ^[42]. However, this policy fails to take into account differences in health outcomes between pollutant sources, and higher oxidative stress from wildfire smoke that has been found in toxicological studies. Policy makers should focus on finding different ways to reduce exposure to air pollutants from wildfires particularly for sensitive populations, and continue to support research into the adverse effects of wildfire air pollution.

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VIII. Tables and Figures

	Variable	Frequency	%
County of Residence			
	Denver	233	20.3
	El Paso	197	17.16
	Adams	136	11.85
	Arapahoe	132	11.5
	Larimer	88	7.67
	Jefferson	85	7.4
	Pueblo	68	5.92
	Weld	56	4.88
	Douglas	26	2.26
	Boulder	23	2
Sex of Patient	Male	484	41.51
	Female	682	58.49
Age of Patient	18 and Under	369 (387 *)	34.46
	Older than 18	736	65.54
Payer Type	Private Insurance	98	8.41
	Medicare & Medicaid	527	45.19
	Self-Pay, No Charge, & Medically Indigent	261	22.38
	HMO-PPO/Managed Care/Discounted	241	20.67
	Other Government Health Insurance	37	3.17

Table 1. Demographics of asthma emergency department visits and acute hospitalizations

*Includes both asthma and wheeze cases

Table 2. Concentration-response associations for one vs. two-pollutant models with control for temperature, day of the week, and the 4th of July

Con	centration	OR Point Estimate	95% Confidence Interval	P Value
0	- 10	1		
10) - 20	1.17	(0.877, 1.563)	0.2861
20) - 30	1.559	(1.127, 2.158)	0.0074
30) - 40	1.586	(1.047, 2.403)	0.0296
40) - 50	1.937	(1.225, 3.065)	0.0047
50) +	2.02	(1.254, 3.252)	0.0038
$PM_{25} (\mu g/m^3)$) Mean with (Dzone - Two Pollutant	Model	

PM_{2.5} (µg/m³) Mean without Ozone - Single Pollutant Model

$PM_{2.5}$ (µg/m ³) Mean with Ozone - Two Pollutant Model					
Concentration	OR Point Estimate	95% Confidence Interval	P Value		
0 - 10	1				
10 - 20	1.191	(0.891, 1.592)	0.2386		
20 - 30	1.588	(1.146, 2.202)	0.0055		
30 - 40	1.598	(1.054, 2.424)	0.0272		
40 - 50	1.911	(1.209, 3.021)	0.0056		
50 +	2.002	(1.242, 3.228)	0.0044		

Figure 1. Summary of county-level Spearman's correlation coefficient using the mean daily PM_{2.5} concentration decreasing maximum daily PM_{2.5} concentration





Figure 2. Summary of county-level Spearman's correlation coefficient using the maximum daily $PM_{2.5}$ concentration by decreasing maximum daily $PM_{2.5}$ concentration

Figure 3. Concentration-response associations for PM_{2.5} and asthma visits, all ages



Linear trend test P = 0.0012

Figure 4. Concentration-response associations for $PM_{2.5}$ and asthma visits, age 18 and under



Linear Trend Test P = 0.4102

Figure 5. Concentration-response associations for $PM_{2.5}$ and asthma visits, over 18 years of age



Linear Trend Test P = 0.0008

	Variable	OR Point Estimate	95% C I	P value
Day of the Week				
	Wednesday*	1		0.3503
	Friday	0.816	(0.646, 1.031)	
	Monday	0.932	(0.73, 1.19)	
	Saturday	0.942	(0.74, 1.197)	
	Sunday	0.973	(0.764, 1.239)	
	Thursday	1.018	(0.811, 1.277)	
	Tuesday	1.053	(0.845, 1.312)	
<i>Temperature (C)**</i>				
	0-18.85*	1		
	18.85 - 24.9	1.149	(0.793, 1.665)	0.4628
	24.9 - 25.9	1.35	(0.946, 1.925)	0.098
	25.9 - 28.9	1.292	(0.914, 1.826)	0.1471
	28.9 - 30.9	1.521	(1.057, 2.19)	0.024
	30.9 +	1.501	(1.026, 2.195)	0.0363
<i>PM_{2.5} Group</i> (μg/m ³)**				
	$0 - 10^{*}$	1		
	10 - 20	1.17	(0.877, 1.563)	0.2861
	20 - 30	1.559	(1.127, 2.158)	0.0074
	30 - 40	1.586	(1.047, 2.403)	0.0296
	40 - 50	1.937	(1.225, 3.065)	0.0047
	50 +	2.02	(1.254, 3.252)	0.0038
Holiday				
	4th of July	0.895	(0.606, 1.322)	0.5788
	* Referent Gro	າມກ		

Table 3. $PM_{2.5}$ single pollutant model, all ages

* Referent Group

	Variable	OR Point Estimate	95% C I	P value
Day of the Week				
	Wednesday*	1		0.0031
	Friday	1.09	(0.7, 1.696)	
	Monday	1.042	(0.647, 1.676)	
	Saturday	1.683	(1.096, 2.584)	
	Sunday	1.626	(1.048, 2.524)	
	Thursday	1.47	(0.957, 2.257)	
	Tuesday	1.809	(1.209, 2.708)	
<i>Temperature (K)**</i>				
	0-18.85*	1		
	18.85 - 24.9	1.159	(0.599, 2.243)	0.6616
	24.9 - 25.9	1.1	(0.576, 2.101)	0.7727
	25.9 - 28.9	1.088	(0.58, 2.042)	0.7919
	28.9 - 30.9	1.074	(0.556, 2.071)	0.8324
	30.9 +	1.184	(0.608, 2.306)	0.6185
<i>PM</i> _{2.5} Group (µg/m ³)**				
	$0 - 10^{*}$	1		
	10 - 20	1.097	(0.632, 1.903)	0.7423
	20 - 30	1.503	(0.823, 2.745)	0.185
	30 - 40	1.332	(0.623, 2.847)	0.459
	40 - 50	1.211	(0.53, 2.769)	0.6499
	50 +	1.777	(0.746, 4.229)	0.194
Holiday				
	4th of July	0.899	(0.411, 1.966)	0.7896
	* Referent Gro	oun		

 Table 4. PM_{2.5} single pollutant model, 18 years old and younger

* Referent Group

	Variable	OR Point Estimate	95% C I	P value
Day of the Week				
	Wednesday*	1		0.2395
	Friday	0.726	(0.551, 0.957)	
	Monday	0.895	(0.673, 1.19)	
	Saturday	0.708	(0.527, 0.953)	
	Sunday	0.772	(0.577, 1.035)	
	Thursday	0.878	(0.671, 1.148)	
	Tuesday	0.817	(0.625, 1.068)	
Temperature (C)**				
	0-18.85*	1		
	18.85 - 24.9	1.141	(0.728, 1.79)	0.5642
	24.9 - 25.9	1.456	(0.951, 2.228)	0.0837
	25.9 - 28.9	1.392	(0.919, 2.108)	0.1188
	28.9 - 30.9	1.792	(1.156, 2.78)	0.0091
	30.9 +	1.661	(1.042, 2.649)	0.0329
<i>PM</i> _{2.5} <i>Group</i> (μg/m ³)**				
	0-10*	1		
	10 - 20	1.21	(0.862, 1.698)	0.2698
	20 - 30	1.581	(1.073, 2.33)	0.0206
	30 - 40	1.698	(1.032, 2.795)	0.0372
	40 - 50	2.427	(1.395, 4.222)	0.0017
	50 +	2.113	(1.191, 3.748)	0.0105
Holiday				
	4th of July	0.891	(0.568, 1.397)	0.6151
	* R	eferent Group		

 Table 5. PM2.5 single pollutant model, older than 18

* Referent Group

	Variable	OR Point Estimate	95% C I	P value
Day of the Week				
	Wednesday*	1		0.2947
	Friday	0.858	(0.679, 1.085)	
	Monday	0.948	(0.743, 1.211)	
	Saturday	0.938	(0.738, 1.191)	
	Sunday	1.067	(0.839, 1.358)	
	Thursday	0.977	(0.779, 1.225)	
	Tuesday	1.119	(0.896, 1.397)	
Temperature (C)**				
	0-18.85*	1		
	18.85 - 24.9	1.015	(0.71, 1.45)	0.9356
	24.9 - 25.9	1.213	(0.861, 1.709)	0.27
	25.9 - 28.9	1.25	(0.882, 1.772)	0.2102
	28.9 - 30.9	1.479	(1.016, 2.153)	0.041
	30.9 +	1.503	(1.009, 2.237)	0.0449
Ozone (8hr max)				
	Continuous	0.983	(0.968, 0.997)	0.0204
Holiday				
	4th of July	0.909	(0.617, 1.34)	0.6316

 Table 6. Ozone single pollutant models, all ages

* Referent Group

	Variable	OR Point Estimate	95% C I	P value
Day of the Week				
	Wednesday*	1		0.0021
	Friday	1.136	(0.729, 1.771)	
	Monday	1.038	(0.646, 1.668)	
	Saturday	1.627	(1.062, 2.493)	
	Sunday	1.687	(1.09, 2.611)	
	Thursday	1.369	(0.892, 2.101)	
	Tuesday	1.906	(1.271, 2.856)	
<i>Temperature</i> (C)**				
	0-18.85*	1		
	18.85 - 24.9	1.073	(0.566, 2.035)	0.8298
	24.9 - 25.9	1.113	(0.595, 2.082)	0.7379
	25.9 - 28.9	1.19	(0.632, 2.24)	0.5894
	28.9 - 30.9	1.199	(0.608, 2.364)	0.6008
	30.9 +	1.43	(0.708, 2.891)	0.319
Ozone (8hr max)				
	Continuous	0.977	(0.952, 1.002)	0.0752
Holiday				
	4th of July	0.895	(0.41, 1.953)	0.7806

Table 7. Ozone single pollutant models, 18 years old and younger

* Referent Group

	Variable	OR Point Estimate	95% C I	P value
Day of the Week				
	Wednesday*	1		0.4157
	Friday	0.767	(0.582, 1.011)	
	Monday	0.917	(0.689, 1.219)	
	Saturday	0.715	(0.532, 0.96)	
	Sunday	0.861	(0.643, 1.154)	
	Thursday	0.853	(0.653, 1.115)	
	Tuesday	0.865	(0.66, 1.134)	
Temperature (C)**				
	0-18.85*	1		
	18.85 - 24.9	0.988	(0.642, 1.52)	0.9564
	24.9 - 25.9	1.244	(0.826, 1.875)	0.2959
	25.9 - 28.9	1.27	(0.836, 1.931)	0.2627
	28.9 - 30.9	1.636	(1.042, 2.567)	0.0324
	30.9 +	1.51	(0.93, 2.452)	0.0955
Ozone (8hr max)				
	Continuous	0.986	(0.968, 1.005)	0.1415
Holiday				
	4th of July	0.915	(0.585, 1.431)	0.6974

 Table 8. Ozone single pollutant models, older than 18

* Referent Group