# The association between wildfire emitted PM2.5 and hospital admissions in the greater Seattle area

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## Abstract

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Short-term exposure to wildfire particulate matter with aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$) has been associated with increases in respiratory and cardiovascular hospital admissions. Inconsistent results among studies suggest that there may be regional differences due to chemical variability in wildfire-generated $PM_{2.5}$. The present study looked at how $PM_{2.5}$ concentrations were associated with respiratory, cardiovascular and cerebrovascular hospital admissions in the greater Seattle area (GSA). The study period covered 11 years from 2007-2017 throughout the fire season (June 1– October 1) and specific days with wildfire smoke. Daily $PM_{2.5}$ measurements were obtained from a tapered element oscillating microbalance (TEOM) $PM_{2.5}$ monitor located in Seattle-Beacon Hill to estimate exposure concentrations throughout the GSA.

Generalized linear models were created using 3rd degree B-splines to estimate the relative risk (effect estimates) of daily hospital admissions for respiratory, cardiovascular and ischemic stroke diagnoses for all ages and for older adults (≥ 65). All-cause respiratory hospital admissions increased by 5.0% (95% Confidence Interval (CI) 1.3% - 8.9%) for all ages and ischemic stroke admissions increased by 4.4% (0.9% - 8.0%) for older adults (≥ 65) for every 10 $\mu g/m^3$ increase in $PM_{2.5}$ during the fire season. Adults aged ≥ 65 showed no increases for all-cause respiratory or all-cause cardiovascular diagnoses during the fire season. Cardiovascular diagnoses and ischemic stroke showed null associations during wildfire smoke days. Respiratory morbidity was pronounced for wildfire smoke days, with significant increases in hospital admissions for children ages 0-19. Whereas many similar studies have looked at wildfire smoke episodes that frequently exceeded the U.S. Environmental Protection Agency's (EPA) federal $PM_{2.5}$ regulatory standards, few have looked at lower concentrations. This study found that hospital admissions for respiratory diagnoses in the GSA significantly increased during periods of wildfire smoke, even though the EPA's $PM_{2.5}$ standards were infrequently exceeded. These results suggest that increased exposure to wildfire smoke could result in lower quality of life, reduced labor productivity, and increases in health costs in the GSA.

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***Dedicado a mi mamá y mi papá, porque sin ellos, nada de esto hubiera sido posible.***

***Dedicated to my mom and dad, without whom none of this would have been possible.***

**The association between wildfire emitted PM2.5 and hospital admissions in the greater Seattle area**

## Introduction

Wildfire smoke is pervasive, extending beyond the perimeter of the fire, reaching both rural and urban human populations where it can be inhaled and lead to increased risk of morbidity and mortality. With climate change, there may be an extension of the fire season, changes in wildfire behavior (Abatzoglou & Kolden, 2013) and changes in synoptic scale patterns in the Western U.S. that influence air quality and smoke transport (Brewer & Mass, 2016). These changing wildfire and synoptic patterns could increase the risk of wildfire smoke into the Greater Seattle Area (GSA). One of the components of wildfire smoke that is of great interest is particulate matter of 2.5 μm in diameter or less ($PM_{2.5}$). Particulate matter consists of fine solid particles or liquid droplets suspended in the atmosphere that vary in chemical and physical composition. Smaller particles, such as $PM_{2.5}$, can remain in the atmosphere for longer periods of time and travel longer distances before settling. Due to its small size, $PM_{2.5}$ has the ability to travel deep into the lungs and reach the alveoli sacs where it can potentially enter the bloodstream or trigger inflammatory and oxidative stress (Brook et al., 2010). Because of its potential for harm, $PM_{2.5}$ is one of the six criteria pollutants regulated by the U.S. Environmental Protection Agency (EPA) (U.S. EPA, 2004). Epidemiological studies done on $PM_{2.5}$ have consistently shown that there are increases in respiratory, cardiovascular, and cerebrovascular morbidity.

Nonetheless, findings on wildfire $PM_{2.5}$ have continued to yield mixed results. While the association between wildfire $PM_{2.5}$ and respiratory morbidity has been well established (Reid et

al., 2016), results are often mixed for cardiovascular and cerebrovascular morbidity (Reid et al., 2016). Some studies have noted increases in cardiovascular morbidity (Delfino et al., 2009; Rappold et al., 2011; Johnston et al., 2014; Haikerwal et al., 2015; Wettstein et al., 2018), while others have found null associations (Henderson et al., 2011; Martin et al., 2013; Reid et al., 2016; Hutchinson et al., 2018). This study looks at the association between $PM_{2.5}$ concentrations throughout the fire season in the GSA and its influence on cardiorespiratory hospital admissions over an eleven-year time span. The $PM_{2.5}$ monitor located in Seattle-Beacon Hill was chosen because of its proximity to the largest population segment. Generalized linear models were created to estimate the relative risk (RR) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ and to estimate the RR for wildfire smoke days compared to non-wildfire smoke days. The GSA is an area of interest due to its growing population and proximity to wildfire prone areas such as Oregon, western Canada, eastern Washington, and California. $PM_{2.5}$ concentrations during the fire season of the GSA were typically low except for days with wildfire smoke or fireworks, indicating that short-term exposure during wildfire smoke days are an issue of concern. Wildfire specific $PM_{2.5}$ emissions are expected to increase in the Western U.S. with climate change (Liu et al., 2016), making this an important public health issue for the GSA. Increases in wildfire $PM_{2.5}$ emissions could lead to reductions in quality of life, labor productivity, and tourism as well as increases in morbidity and mortality.

## Methods

### *Study Setting*

The GSA includes King, Pierce and Snohomish County (Figure 1). The GSA has grown from a population of 3.31 million in 2007 to 3.87 million residents in 2017 (U.S. Census Bureau,

2017), making it an important area of study as its population continues to grow. The GSA is located within Washington State, near the west coast of the United States where wildfires and wildfire smoke are frequent. The GSA receives wildfire smoke from California, Eastern Washington, Oregon, British Columbia and sometimes from places as far as Siberia (NESDIS, 2018). Furthermore, climate change may be making conditions more favorable for wildfires and wildfire smoke as precipitation, temperature, and synoptic patterns change in the Western U.S. (Abatzoglou & Kolden, 2013; Brewer & Mass, 2016).

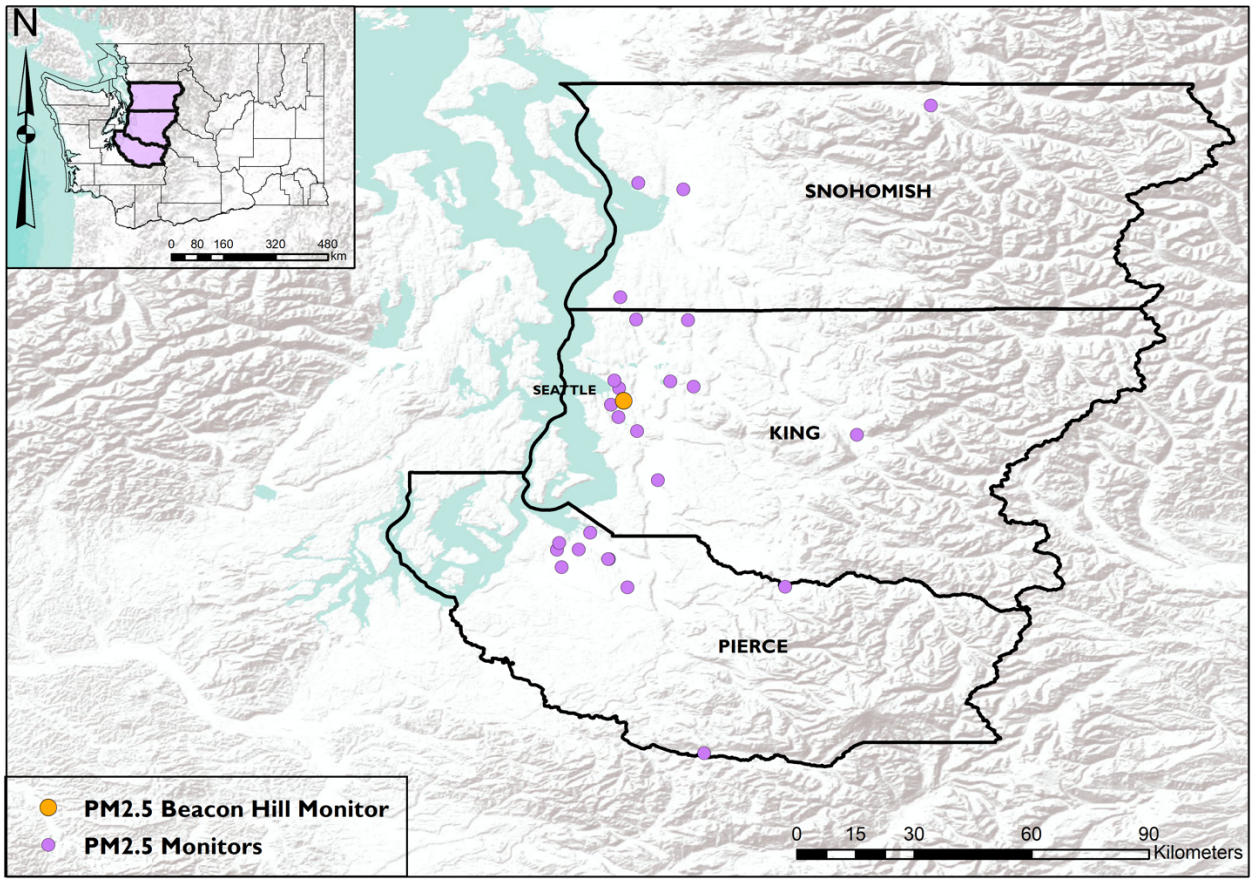


Figure 1. Greater Seattle Area with $PM_{2.5}$ monitors active during the eleven-year study period 2007-2017 from June 1st – Oct 1st

*Air Quality Assessment*

Daily average measurements of $PM_{2.5}$ were obtained from the U.S. EPA's AirNow data website for the time period 2007-2017 (June 1 – Oct 1) (U.S. EPA, 2018). Ambient $PM_{2.5}$ concentrations in Washington State are collected using a combination of tapered element oscillating microbalances (TEOM), beta attenuation monitors (BAM), and nephelometers. TEOM and BAM monitors follow the federal equivalent method (FEM) guidelines for $PM_{2.5}$ monitoring, while nephelometers are correlated with site-specific FEM monitor data (Washington State Department of Ecology, 2018). This study looked at a combination of 26 TEOM and BAM monitors and nephelometers to determine wildfire smoke days. Only one $PM_{2.5}$ monitor located in King County, Beacon Hill was chosen for the fire season time series analysis.

*Meteorological Data*

Meteorological data were obtained from the National Centers for Environmental Information for Seattle-Tacoma International Airport located in King County (NCEI, 2018). Daily average temperature was calculated from hourly measurements and daily average vapor pressure deficit was calculated from hourly relative humidity and hourly temperature measurements.

*Wildfire Smoke Affected Days*

Only one of the 26 $PM_{2.5}$ monitors in the GSA needed to exceed 30 $\mu g/m^3$ for the 24-hour average to be designated as a wildfire smoke day. If one county is affected in the GSA, it is likely that the adjacent counties are affected as well. While this threshold is lower than the EPA's $PM_{2.5}$ standard of 35 $\mu g/m^3$ for their 24-hour average, the World Health Organization

(WHO) suggests that there is no observed safe concentration of particulate matter (WHO, 2018). Therefore, this study chooses a lower threshold to detect any possible deleterious effects of wildfire smoke on human populations in the GSA while distinguishing from non-smoke days. The current study also chose a 30 $\mu g/m^3$ threshold because every day above this threshold is either a wildfire smoke day or firework-afflicted day. To ensure that the days that exceeded 30 $\mu g/m^3$ were wildfire smoke days, Terra/MODIS-corrected reflectance satellite imagery and fire and thermal anomalies (beginning 2012) were analyzed for the days where $PM_{2.5}$ was $\geq 30$ $\mu g/m^3$ from 2007-2017 (Jun 1 – Oct 1). Archived satellite smoke text product from the National Environmental Satellite, Data and Information Service (NESDIS) was used to corroborate the presence of wildfire smoke (NESDIS, 2018). NESDIS provides global text descriptions of aerosols based on satellite data and provides daily updates. July 4$^{th}$ was excluded from being classified as a wildfire smoke day due to the presence of firework-generated $PM_{2.5}$ on those days. As part of a secondary analysis, there was a 20 $\mu g/m^3$ threshold for wildfire smoke days to see whether there were any noticeable differences in hospitalizations between a lower threshold for wildfire smoke days and the entire fire season.

*Hospital Admissions*

The hospital data used in this study were obtained from the Comprehensive Hospital Abstract Reporting System (CHARS) through the Washington State Department of Health. Approval was obtained from the Washington State Department of Health. Daily counts of hospital admissions were obtained from 2007-2017 during June 1 – October 1 to reflect the fire season in the Pacific Northwest. Only hospital admissions classified as emergency or urgent were considered. The *International Classification of Diseases*, 9$^{th}$ Revision (ICD-9) from June 1,

2007- September 30, 2015 and the 10th Revision (ICD-10) from Oct 1, 2015- Oct 1, 2017 codes were used to determine cardiovascular, cerebrovascular, and respiratory diseases. ICD-9 and ICD-10 diagnosis codes used in this study are listed in Table 1.

Three case groups were chosen for this study. The first case group looked at whether increases in $PM_{2.5}$ during the fire season would be associated with increases in respiratory, cardiovascular, and ischemic stroke diagnoses in the GSA. The second case group looked at people ≥ 65 years of age since they may be more sensitive to increases in particulate matter during the fire season due to pre-existing conditions and physiological changes with age (Sacks et al., 2011). The third case looked at hospital admissions during wildfire smoke affected days versus the fire season (entire study period covering all sources of $PM_{2.5}$) to see if wildfire smoke days were associated with a higher relative risk. As part of a secondary analysis, all-cause respiratory, all-cause cardiovascular, and ischemic stroke were stratified by age and sex.

Table 1. List of ICD-9 and ICD-10 codes

| Diagnosis | ICD-9 Codes | ICD-10 Codes |
|---|---|---|
| Cardiovascular | | |
| Hypertension | 401-405 | I10 -I15 |
| Myocardial Infarction | 410 | I21-I23 |
| Ischemic Heart Disease | 410-411, 413 | I20-I24 |
| Dysrhythmia and conduction disorder | 426-427 | I44-I49 |
| Heart Failure | 428 | I50 |
| All-cause cardiovascular | 401-405, 410-411, 413, 426-428 | I10-I15, I20-I24, I44-I50 |
| Cerebrovascular | | |
| Ischemic Stroke | 434 | I63 |
| Respiratory | | |
| Asthma | 493 | J45-J46 |
| COPD | 491-492, 496 | J41-J43, J44 |
| Pneumonia | 480-486 | J12-J18 |
| Non-cardiac chest pain/ respiratory symptom | 786 | R04-R07, R09 |
| All-cause respiratory | 480–486, 491– 493, 786 | J12-J18, J41-J46, R04-R07, R09 |

*Statistical Analysis*

For the Greater Seattle Area, a generalized linear model with cubic B-splines was used to estimate the influence of $PM_{2.5}$ concentrations on the rate of hospital admissions for cardiovascular, cerebrovascular and respiratory conditions during the fire season period and for smoke affected days. The following model was used to estimate the rate of hospital admissions for every 10 μg/m$^3$ increase in $PM_{2.5}$ concentrations:

$$H_t = quasipoisson(\mu_t, \sigma^2)$$

$$f(\mu_t) = PM_{t=lag0\text{-}7} + T_{t=lag0\text{-}7} + VPD_{t=lag0\text{-}7} + TimeSpline_{(knots=44)} + DOW_t$$

And the following model was used to estimate the rate of hospital admissions for smoky days versus non-smoky days:

$$g(\mu_t) = Smoky_{t=lag0\text{-}7} + T_{t=lag0\text{-}7} + VPD_{t=lag0\text{-}7} + TimeSpline_{(knots=44)} + DOW_t$$

Where $H_t$ = observed hospital counts with a quasipoisson distribution, $\mu_t$ = hospital count population mean at time t and $\sigma^2$ = hospital count variance. The terms $f(\mu_t)$ and $g(\mu_t)$ represent the expected hospital count outcomes at time t. $PM_{lag0\text{-}7}$ is $PM_{2.5}$ concentrations lagged from 0-7 days, $T_{t=lag0\text{-}7}$ = mean temperature at day *t* lagged from 0-7 days, $VPD_{t=lag0\text{-}7}$ = mean vapor pressure deficit at day *t* lagged from 0-7 days, factor variable $DOW_t$ = day of week on day *t* (federal holidays are treated as Sundays). $T_t$, $VPD_t$ and $DOW_t$ are treated as potential confounding variables. This is to ensure that increases in hospital admission counts are not incorrectly attributed to changes in $PM_{2.5}$ that may have resulted from changes in $T_t$, $VPD_t$ and $DOW_t$. $Smoky_{lag0\text{-}7}$ = indicator variable where 0 = non-smoky day and 1 = smoky day lagged from 0-7 days. $TimeSpline_{(knots=44)}$ are 3rd degree B-splines fitted with 44 knots over the 11-year time span. The knots represent breaks in the hospital data and capture long-term and seasonal trends. 44 knots were chosen to represent each month in the study and capture monthly hospital count patterns.

Constrained distributed lag models were chosen to counter autocorrelation and increase precision of the effect estimates. The constrained models were chosen based on effect estimates that were modeled one at a time. Lags from 0-7 days were tested and results were considered statistically significant at the 95% confidence level. A sensitivity analysis was done on the statistically significant results from the constrained model by varying the number of knots from 22-88. More information about the statistical analysis and the code used in the present study can be found in Bhaskaran et al. (2013). All analyses were completed on the R statistical environment version 3.5.1.

## Results

*Air Quality Assessment*

Only nine monitors were active for most of the 11-year study period (< 15% data missing). Table 2 shows the summary statistics for monitors located in King, Pierce and Snohomish County over the entire study period. Mean and median $PM_{2.5}$ concentrations are all under 10 $\mu g/m^3$. The monitor located in Seattle-Beacon Hill was chosen to represent the entire GSA due to the low amount of missing data (1.4% missing data) and its proximity to a large population center. The Seattle-Beacon Hill monitor typically had lower concentrations than most of the other monitors that were continuously present throughout the GSA (Figure 2).

Table 2. Summary statistics for $PM_{2.5}$ monitors with less than 15% data missing during the study period from 2007-2017 (June 1st – Oct 1st)

| County | Location | Mean ($\mu g/m^3$) | Median ($\mu g/m^3$) | Range ($\mu g/m^3$) |
|---|---|---|---|---|
| King | Seattle-Beacon Hill | 6.7 | 5.9 | 1.3 - 56.8 |
| King | Seattle-South Park | 8.0 | 7.2 | 1.3 - 69.2 |
| King | Seattle-Duwamish | 8.3 | 7.5 | 0.9 - 57.6 |
| King | North Bend-North Bend Way | 6.5 | 5.1 | 0.5 - 151.7 |
| Pierce | Tacoma-L St | 6.4 | 5.3 | 0.9 - 66.6 |
| Pierce | Tacoma-Alexander Ave | 6.9 | 6.0 | 1.3 - 76.0 |
| Pierce | Puyallup-128th St | 6.1 | 4.6 | 0.5 - 90.9 |
| Snohomish | Darrington-Fir St | 4.7 | 3.4 | 0 - 70.3 |
| Snohomish | Marysville-7th Ave | 6.8 | 5.9 | 0.9 - 64.8 |

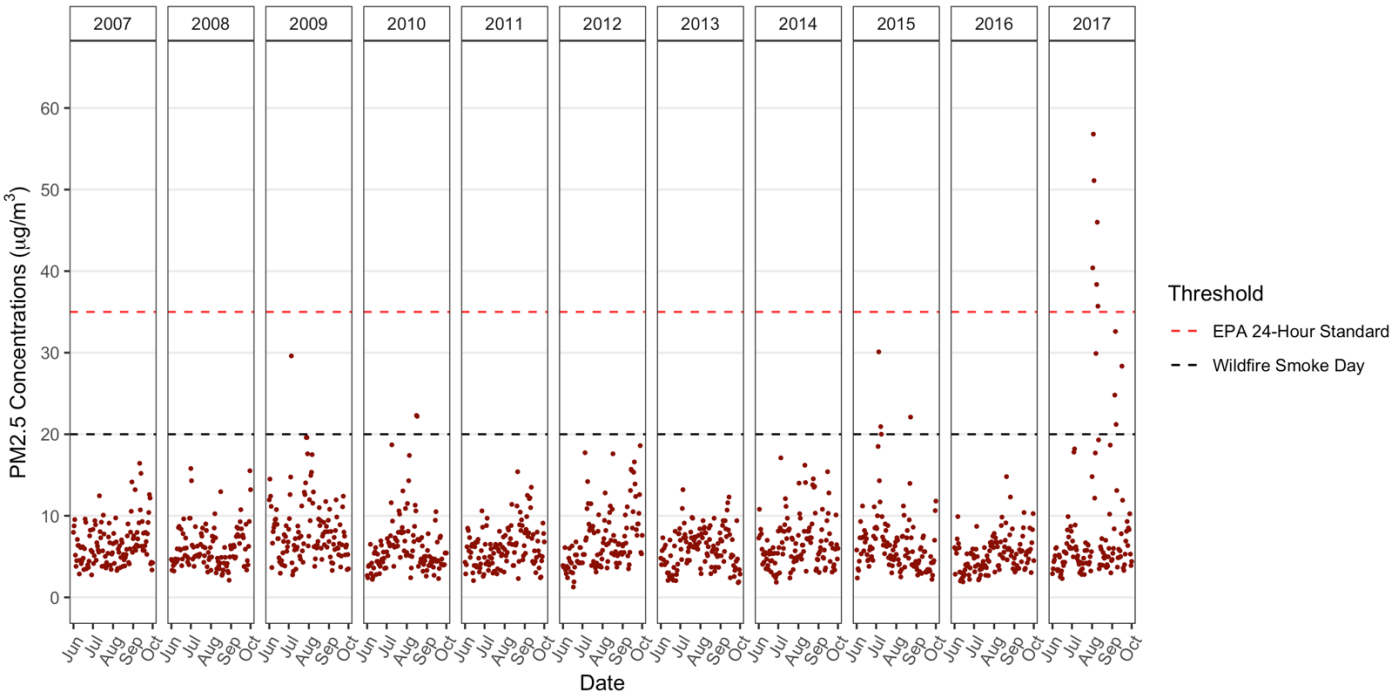


Figure 2. Time series of daily $PM_{2.5}$ concentrations in µg/m$^3$ from the Seattle-Beacon Hill monitor from 2007-2017 (June 1 – Oct 1) with 20 µg/m$^3$ threshold for wildfire smoke days and 35 µg/m$^3$ 24-hour EPA federal regulatory $PM_{2.5}$ standard for sensitive populations

*Wildfire Smoke Days*

Only 26 $PM_{2.5}$ monitors were intermittently active throughout the entire study period. Out of the 35 days, only 24 exceeded the 30 µg/m$^3$ threshold, were not July 4$^{th}$, and were verified to be wildfire smoke days. Days with concentrations over 30 µg/m$^3$ that were not influenced by wildfires were likely caused by fireworks, since all the non-wildfire smoke days over 30 µg/m$^3$ were between July 4$^{th}$ – 5$^{th}$. Most of the wildfire smoke days (13) were in 2017, 5 were in 2015, 4 in 2012, and 2 in 2009. When the threshold was lowered to 20 µg/m$^3$, 52 out of 80 days were verified to be wildfire smoke days. More than half of the remaining non-wildfire smoke days (18 out of the 28 days) were caused by fireworks since they fell within the July 4$^{th}$ – 5$^{th}$ time period and the other 10 were likely caused by other sources of urban $PM_{2.5}$. The low number of days that were ≥ 20 µg/m$^3$ and were caused by urban sources of $PM_{2.5}$ shows that wildfire smoke days

play a larger role in elevating $PM_{2.5}$ concentrations in the GSA. At the lower cutoff, 17 of the wildfire smoke days were in 2017, with 11 in 2012, 8 in 2015, 8 in 2009, 3 in 2010, 2 in 2008 and 2014, and 1 in 2007. A table of all wildfire smoke days is provided in Appendix A.

*Hospital Admission Results*

After sorting by ICD codes of interest, there were a total of 111,721 daily unscheduled hospital admissions in the GSA over the eleven-year study period. This study assessed whether the relative risk (RR) increased for every 10 $\mu g/m^3$ increase in all sources of $PM_{2.5}$ for respiratory diagnoses, cardiovascular diagnoses, and ischemic stroke for all ages and those 65 and older during the fire season. This study also evaluated whether wildfire smoke days with $PM_{2.5}$ concentrations $\geq 20$ $\mu g/m^3$ and $\geq 30$ $\mu g/m^3$ had a greater relative risk (RR) than all sources of $PM_{2.5}$ during the fire season. Appendix B contains graphs with cubic B-splines fit through all-cause respiratory, all-cause cardiovascular, and ischemic stroke for all ages and Appendix C contains the same diagnoses, but for adults ≥65 years of age. Due to inconclusive results for wildfire smoke days at $\geq 30$ $\mu g/m^3$, wildfire smoke days will explicitly refer to the cutoff $\geq 20$ $\mu g/m^3$, unless otherwise stated. The next two sections will be divided into results for the fire season and results for wildfire smoke days $\geq 20$ $\mu g/m^3$.

*Fire Season Results*

The fire season encompassed the entire study period from 2007-2017 (June 1 – October 1) and all sources of $PM_{2.5}$. All-cause diagnoses for all-ages and for those ≥ 65 years of age were not significantly associated with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ for the fire season (Appendix D). All-cause respiratory diagnoses increased by 5.0% (95% CI 1.3% - 8.9%) for all ages two days

after exposure. There was a cumulative (net) increase of 6.2% (0.4% - 12.2%) 0-7 days after exposure during the fire season (Figure 3a). The sensitivity analysis for all-cause respiratory diagnoses at lag day 2 is constant as the degrees of freedom (knots) change (Figure 4a).

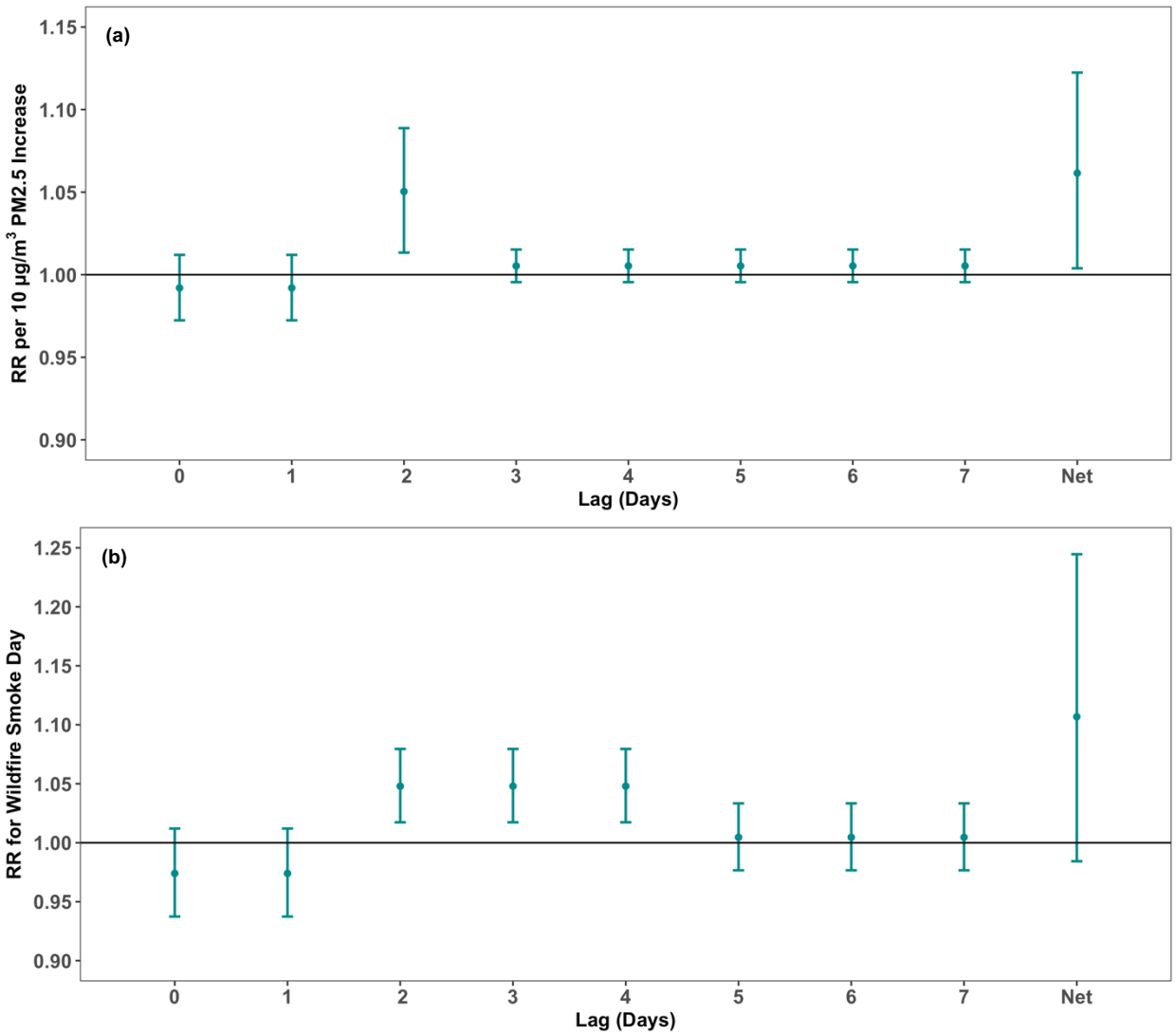


Figure 3. All-cause respiratory; relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days (b). Lag (Days) indicates days after exposure Note change in y-axis scale

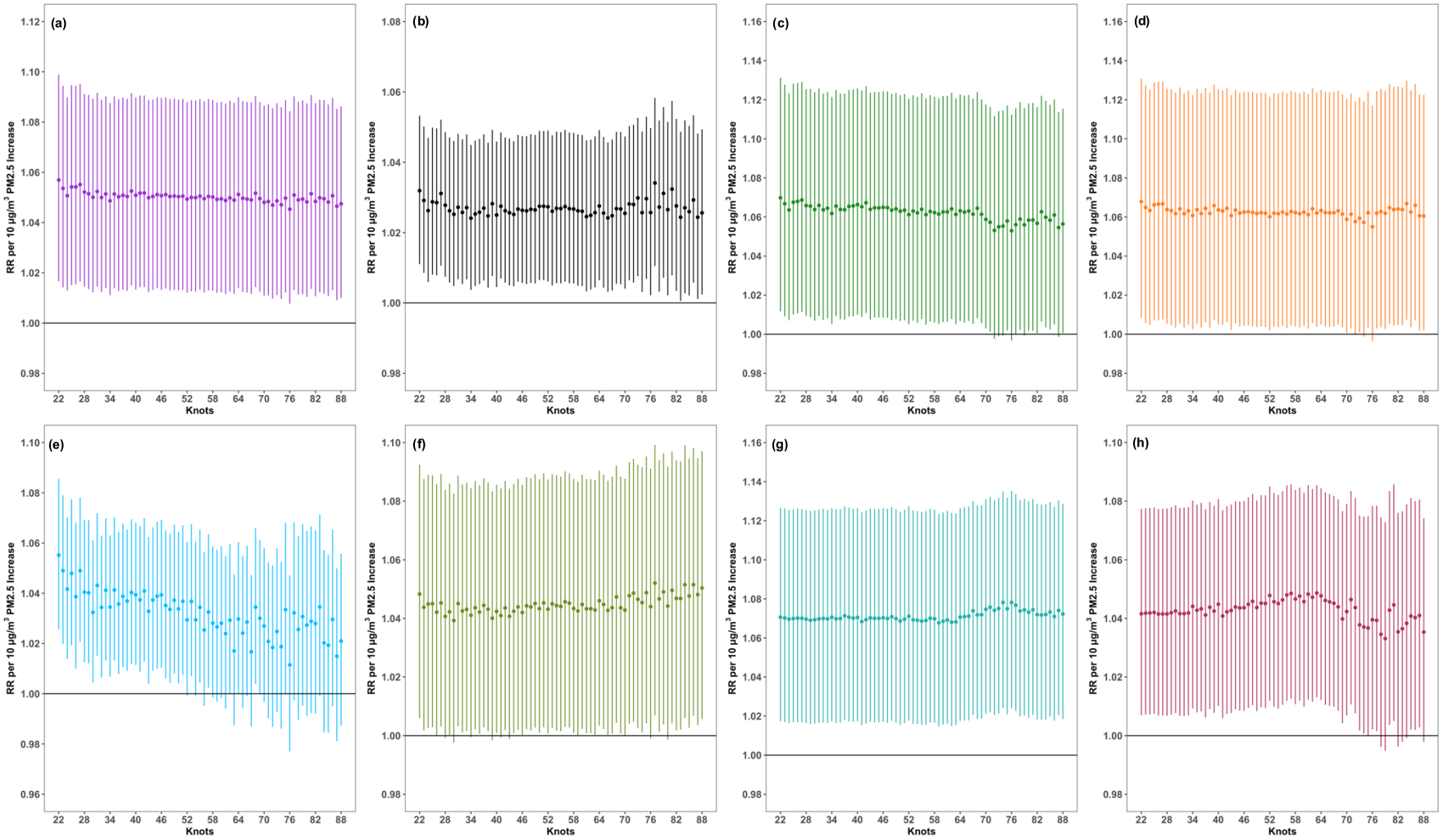


Figure 4. Sensitivity analysis for every 10 µ g/m$^3$ increase in $PM_{2.5}$ concentrations during the fire season. Degrees of freedom (knots) range from 22-88. (a) All-cause respiratory for all ages for lags 2. (b) All-cause respiratory for females lags 2-4. (c) All-cause respiratory for males lag 2. (d) All-cause respiratory for ages 20-64 lag 2. (e) Asthma diagnoses lags 1-5. (f) Non-cardiac chest pain/other respiratory symptoms lags 1-2. (g) All-cause cardiovascular for ages 20-64 lag 1. (h) Ischemic stroke for ages 65 and up lags 3-4. Note change in y-axis scale

After stratifying by sex, all-cause respiratory hospitalizations increased for both females and males, but at different lags. Males showed an increase in hospital admissions of 6.5% (0.9% - 12.4%) two days after exposure, however females showed an increase of 2.5% (0.5% - 4.6%) two to four days after exposure (Figure 5a). Sensitivity analysis of the effect estimates for all-cause respiratory diagnoses for males and females is constant (Figure 4b-c).

The most vulnerable age group for all-cause respiratory diagnoses were younger adults, ages 20-64. Hospital admissions for adults (20-64) increased by 6.4% (0.6% - 12.5%) two days after exposure (Figure 6a). Sensitivity analysis of the effect estimates for the age group 20-64 are consistent as the degrees of freedom vary (Figure 4d). All-cause respiratory hospital admissions for older adults ≥ 65 years were not significantly associated with increases in $PM_{2.5}$ concentrations (Figure 6a).

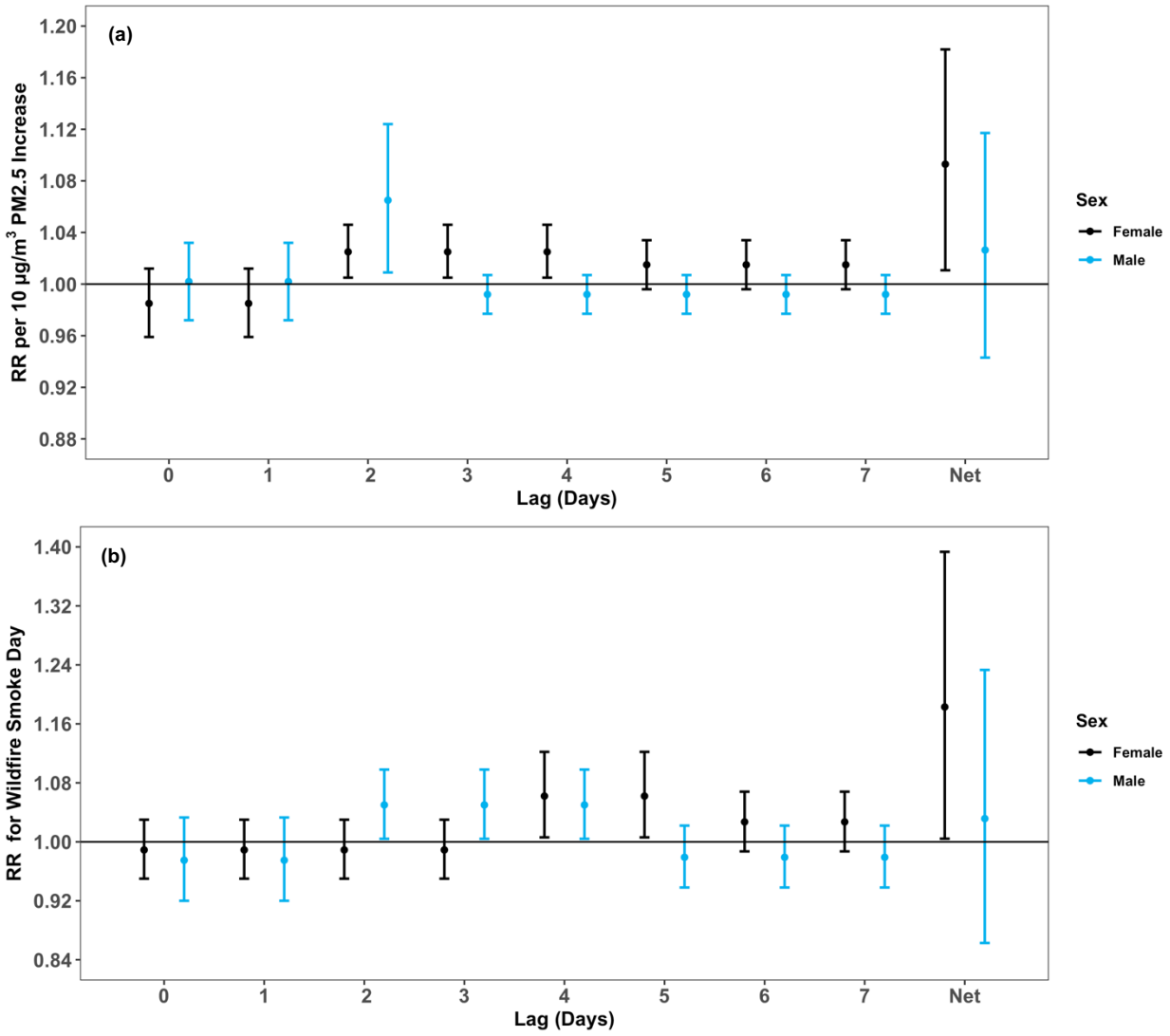


Figure 5. All-cause respiratory for males and females; relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days (b). Lag (Days) indicates days after exposure. Note change in y-axis scale

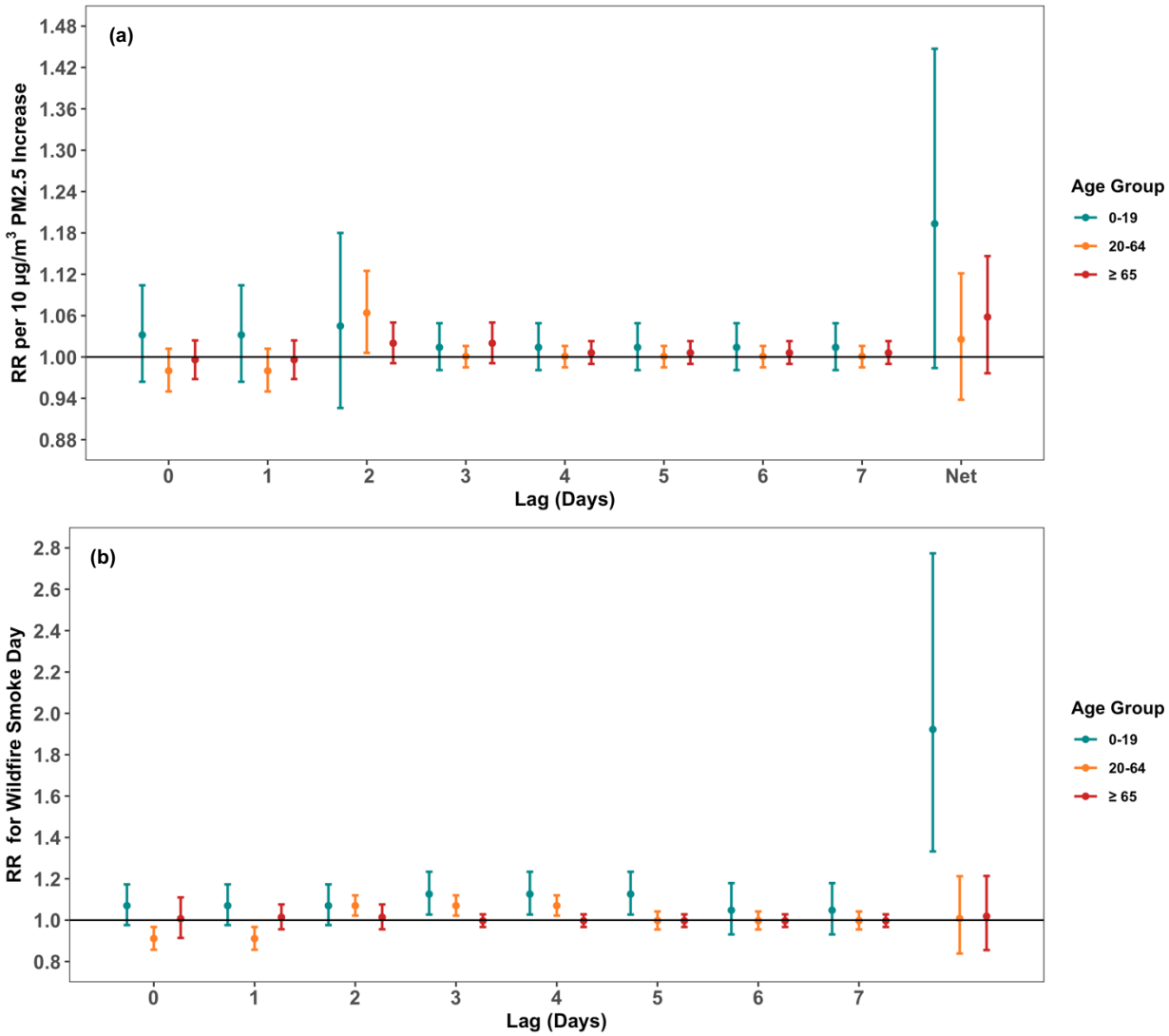


Figure 6. All-cause respiratory (by age group); relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days (b). Lag (Days) indicates days after exposure. Note change in y-axis scale

Hospital admissions for asthma increased by 3.7% (0.9% - 6.6%) one to five days after exposure, with a net increase of 20.3% (3.0% - 40.5%) (Figure 7a). The sensitivity analysis results are stable until 52 degrees of freedom (knots), at which point the confidence intervals widen and overfitting occurs (Figure 4e).

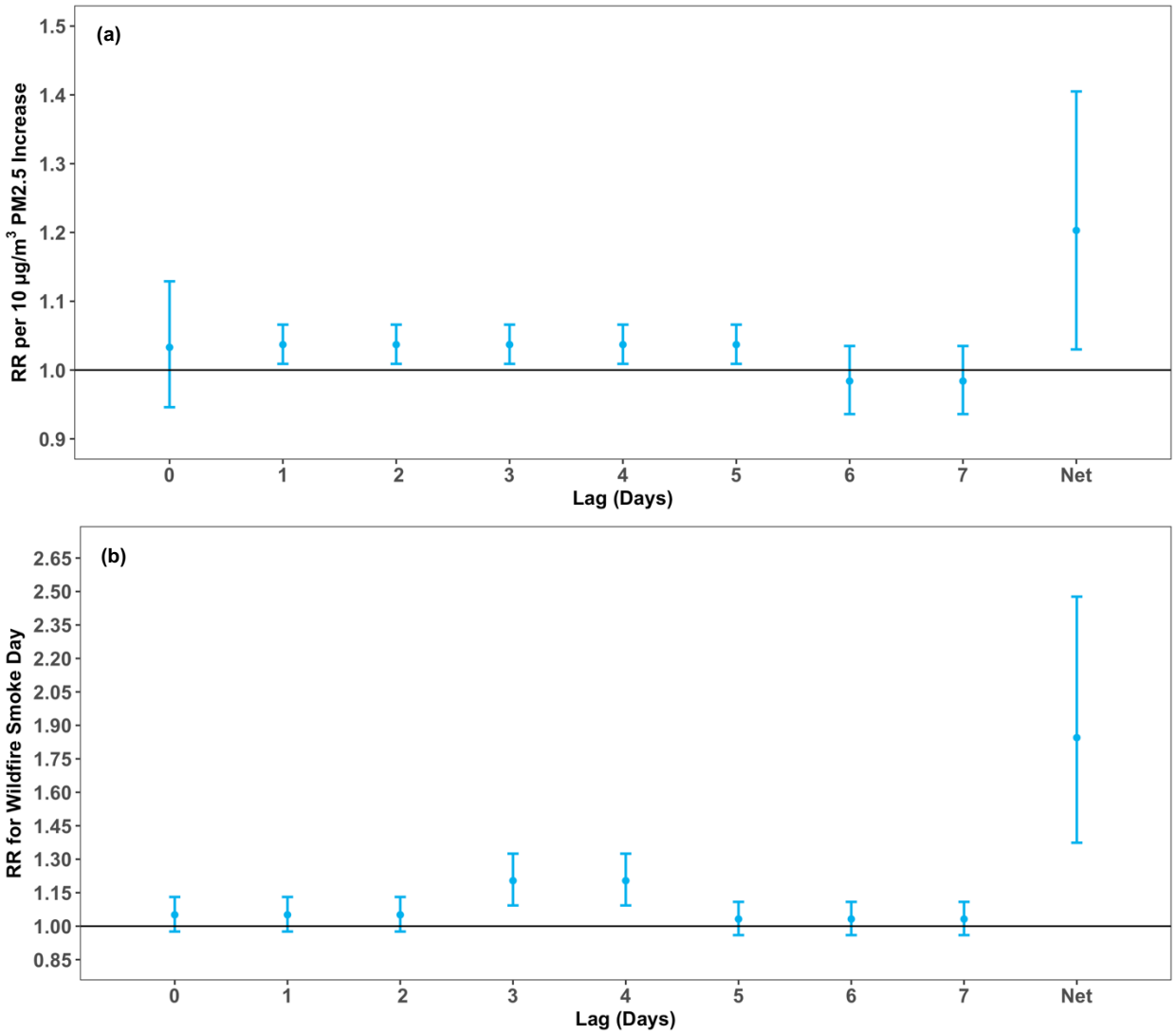


Figure 7. Asthma; relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days (b). Lag (Days) indicates days after exposure. Note change in y-axis scale

Non-cardiac chest pain or other respiratory symptoms also increased by 4.2% (0.1%-8.6%) one to two days after increased $PM_{2.5}$ exposure, with a net increase of 13.5% (1.0% - 27.4%) (Figure 9a). Sensitivity analysis of the effect estimates for lags 1 and 2 show little variation as the knots vary (Figure 4f). Other respiratory diagnoses of interest (COPD and pneumonia) were not statistically significant throughout the fire season.

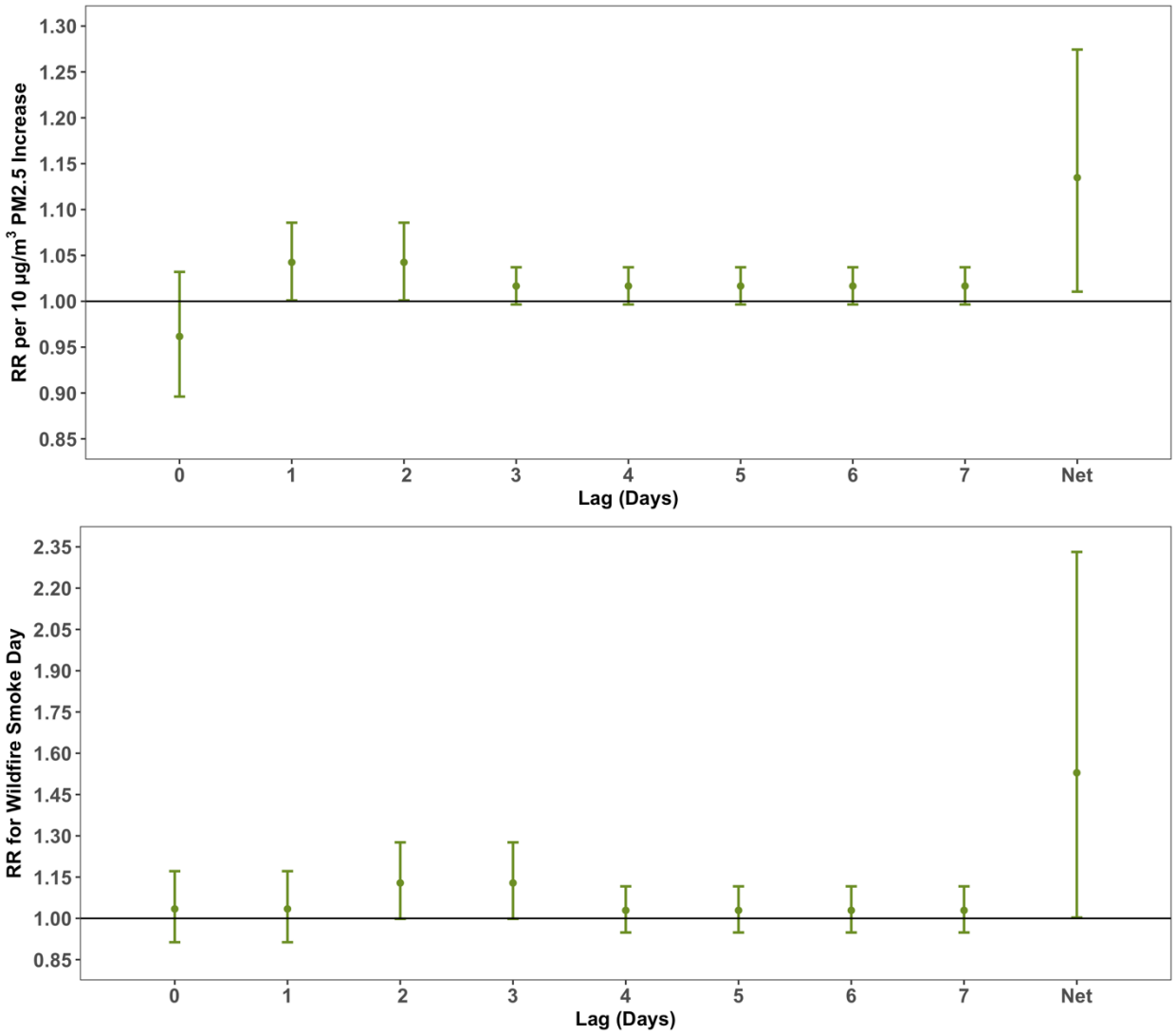


Figure 8. Non-cardiac chest pain or other respiratory symptom for all ages; relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days at 30 µg/m$^3$ threshold (b). Lag (Days) indicates days after exposure. Note change in y-axis scale

All-cause cardiovascular diagnoses were not significantly associated with increases in $PM_{2.5}$ during the fire season, except for younger adults 20-64 years of age (Figure 9a). Hospitalizations in this group increased by 7.0% (1.7% - 12.6%) one day after exposure. The effect estimates for age group 20-64 are consistent as the number of knots change (Figure 4g). Due to low counts for age group 0-19, this age group was not tested. Cardiovascular hospital admissions were null for those aged 65 and older. The association between $PM_{2.5}$ and ischemic

stroke admissions was null for all-ages, with an increase in hospital admissions for older adults ≥ 65 years of age three and four days after exposure (4.4%, 95% CI: 0.9% - 8.0%) (Figure 10a). The effect estimates for ischemic stroke approach the null as the number of knots increase in the sensitivity analysis (Figure 4h). Table 3 shows a summary of the results for the entire fire season that were considered statistically significant, with specific estimates in Appendix E.

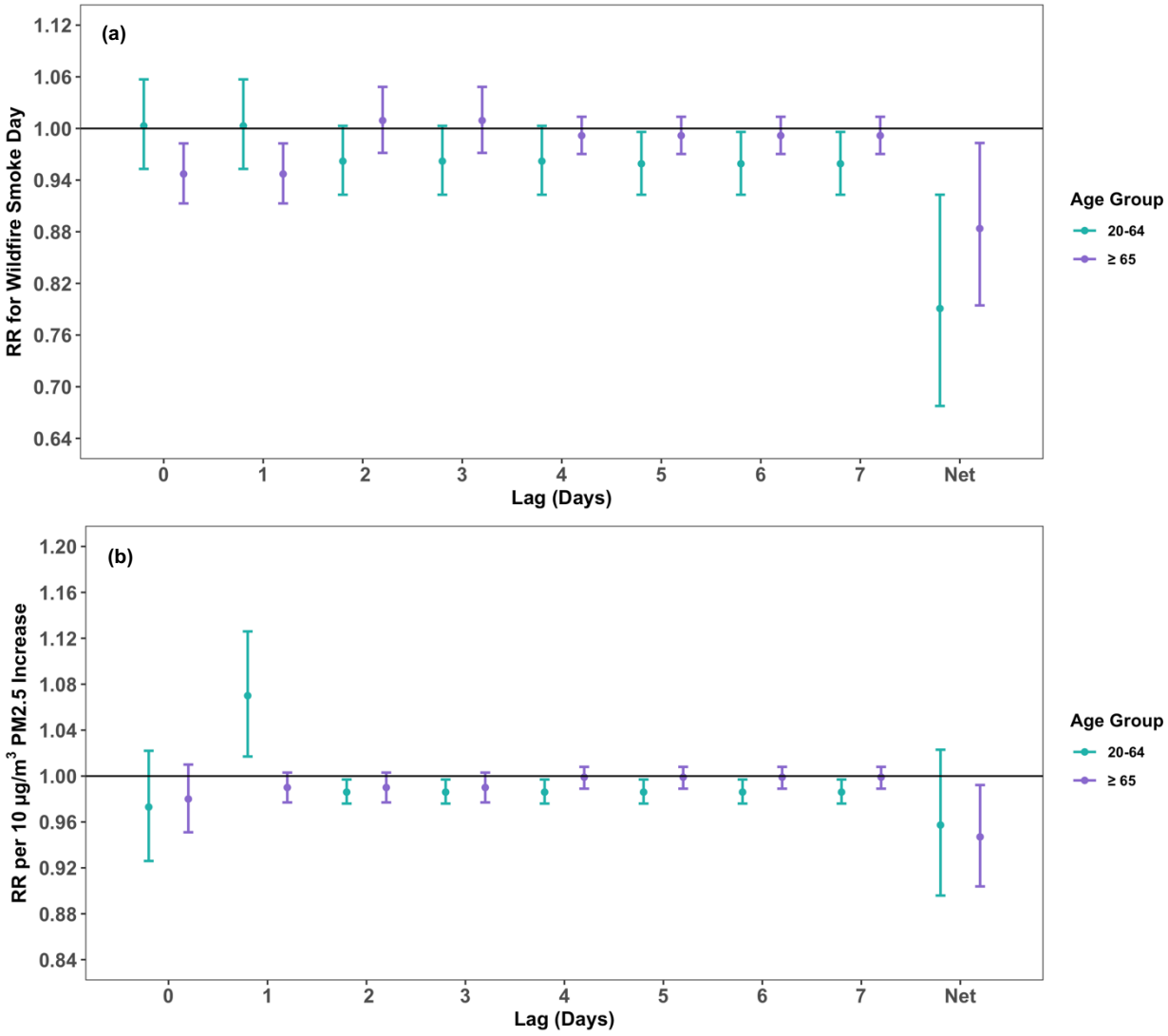


Figure 9. All-cause cardiovascular by age; relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days (b). Lag (Days) indicates days after exposure. Note change in y-axis scale

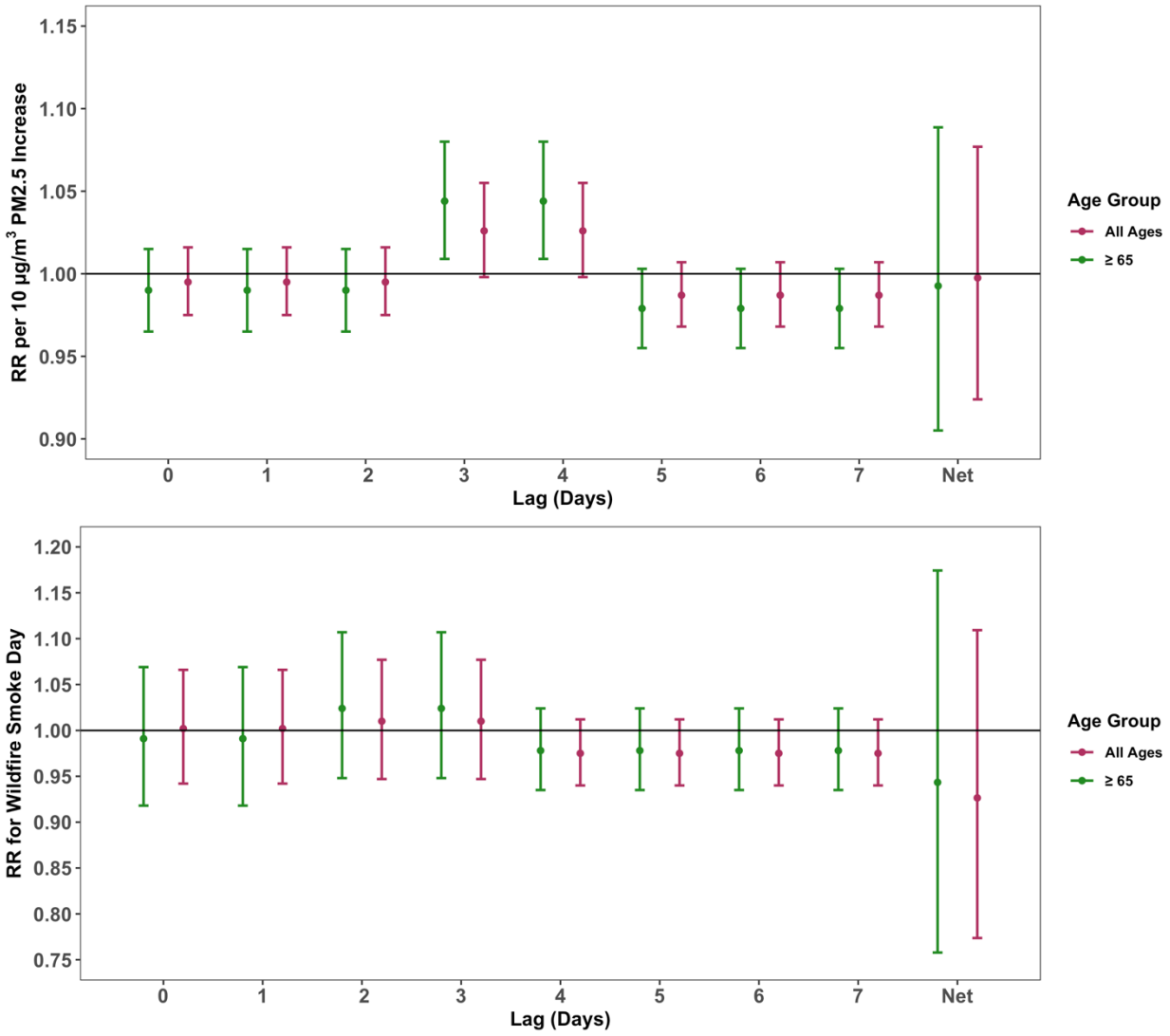


Figure 10. Ischemic stroke by age; relative risk and 95% confidence interval for every 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season (a) and relative risk for wildfire smoke days versus non-wildfire smoke days (b). Lag (Days) indicates days after exposure. Note change in y-axis scale

Table 3. Statistically significant results at the 95% confidence level for every 10 μg/m$^3$ increase in $PM_{2.5}$ concentrations during the fire season. Further details can be found in Appendix E.

| Hospital admission diagnosis | Lag 0 | Lag 1 | Lag 2 | Lag 3 | Lag 4 | Lag 5 | Lag 6 | Lag 7 | Net |
|---|---|---|---|---|---|---|---|---|---|
| All-Respiratory | | | | | | | | | |
| All ages | | | **x** | | | | | | **x** |
| Female | | | **x** | **x** | **x** | | | | **x** |
| Male | | | **x** | | | | | | |
| Ages 0-19 | | | | | | | | | |
| Ages 20-64 | | | **x** | | | | | | |
| Ages ≥ 65 | | | | | | | | | |
| Asthma | | **x** | **x** | **x** | **x** | **x** | | | **x** |
| COPD | | | | | | | | | |
| Pneumonia | | | | | | | | | |
| Other non-cardiac chest pain or respiratory symptom | | **x** | **x** | | | | | | **x** |
| All-Cardiovascular | | | | | | | | | |
| All ages | | | | | | | | | |
| Female | | | | | | | | | |
| Male | | | | | | | | | |
| Ages 0-19 | | | | | | | | | |
| Ages 20-64 | | **x** | | | | | | | |
| Ages ≥ 65 | | | | | | | | | |
| Hypertension | | | | | | | | | |
| Myocardial infarction | | | | | | | | | |
| Ischemic heart disease | | | | | | | | | |
| Dysrhythmia and conduction disorder | | | | | | | | | |
| Heart failure | | | | | | | | | |
| Cerebrovascular | | | | | | | | | |
| Ischemic stroke | | | | | | | | | |
| All ages | | | | | | | | | |
| Ages ≥ 65 | | | | **x** | **x** | | | | |

*Wildfire Smoke Day Results*

The present study also evaluated whether days with wildfire smoke had a higher relative risk than the entire fire season in the GSA. To test this, an indicator variable was assigned to days with wildfire smoke. Due to the low number of wildfire smoke days at the 30 μg/m$^3$ threshold, there were no conclusive results. Only one result was statistically significant, which was non-cardiac chest pain or other respiratory symptoms with a net increase from lags 0-7 of 52.9% (0.3%-133.2%) (Figure 8b). At the 20 μg/m$^3$ cutoff, non-cardiac chest pain or other respiratory symptoms was no longer significant.

All-cause diagnoses were not significant for all ages or for those ≥ 65 years of age for wildfire smoke days at both the 20 and 30 μg/m$^3$ threshold (Appendix D). After lowering the threshold to 20 μg/m$^3$, there was a 4.8% (1.7% - 7.9%) increase in all-cause respiratory outcomes two to four days after exposure (Figure 3b). All-cause respiratory hospital admissions were significantly associated with wildfire smoke days for age group 0-19 (12.6%, 95% CI 2.7% - 23.4%). All-cause respiratory diagnoses increased for all ages and younger adults (20-64), but not for older adults (≥ 65 years of age). Male and female hospital admissions also increased during wildfire smoke days (Figure 5b). Asthma admissions were also significantly associated with wildfire smoke days. All-cause cardiovascular diagnoses were no longer statistically significant for the age group 20-64. All-cause cardiovascular admissions were not statistically significant for all ages or for those ≥ 65 years of age either. Ischemic stroke was not statistically significant for all ages or for those ≥ 65 years of age (Figure 10b). Sensitivity analysis of wildfire smoke effect estimates resulted in little variation until about 65-70 knots, at which point confidence intervals widened (Figure 11). A summary of the statistically significant results for wildfire smoke days can be found in Table 4 for the 20 μg/m$^3$ threshold with further details in Appendix F and Appendix G for 30 μg/m$^3$.

To summarize, hospital admissions for respiratory diagnoses increased with every 10 μg/m$^3$ increase in $PM_{2.5}$ during the fire season. For every 10 μg/m$^3$ increase in $PM_{2.5}$, there was a rise in non-cardiac chest pain or other respiratory symptoms during the fire season. Hospital admissions increased for age group 20-64 for all-cause respiratory and all-cause cardiovascular diagnoses. Hospital admissions did not significantly increase for those 65 and over, except for ischemic stroke during the fire season. Respiratory morbidity increased for both male and females, with differences in the days they sought care after exposure.

Wildfire smoke days resulted in extended increases for respiratory diagnoses but were not associated with rises in cardiovascular or cerebrovascular diagnoses. Wildfire smoke days also resulted in increases for all-cause respiratory diagnoses in the age group 20-64, males, and females with differences in the days they sought care. Respiratory hospital admissions for the age group 0-19 was the only significant result during wildfire smoke days that was not also significant in the fire season. Wildfire smoke days were associated with increases in respiratory morbidity in the GSA, with slightly greater relative risks than the fire season.

Table 4. Statistically significant results at the 95% confidence level for wildfire smoke days ≥ 20 μg/m3. Further details can be found in Appendix F.

| Wildfire Smoke 20 μg/m$^3$ | Lag 0 | Lag 1 | Lag 2 | Lag 3 | Lag 4 | Lag 5 | Lag 6 | Lag 7 | Net |
|---|---|---|---|---|---|---|---|---|---|
| All-Respiratory | | | | | | | | | |
| All ages | | | **x** | **x** | **x** | | | | |
| Female | | | | **x** | **x** | | | | **x** |
| Male | | | **x** | **x** | **x** | | | | |
| Ages 0-19 | | | | **x** | **x** | **x** | | | **x** |
| Ages 20-64 | | | **x** | **x** | **x** | | | | |
| Ages ≥ 65 | | | | | | | | | |
| Asthma | | | | **x** | **x** | | | | **x** |
| COPD | | | | | | | | | |
| Pneumonia | | | | | | | | | |
| Other non-cardiac chest pain or respiratory symptom | | | | | | | | | |
| All-Cardiovascular | | | | | | | | | |
| All ages | | | | | | | | | |
| Female | | | | | | | | | |
| Male | | | | | | | | | |
| Ages 0-19 | | | | | | | | | |
| Ages 20-64 | | | | | | | | | |
| Ages ≥ 65 | | | | | | | | | |
| Hypertension | | | | | | | | | |
| Myocardial infarction | | | | | | | | | |
| Ischemic heart disease | | | | | | | | | |
| Dysrhythmia and conduction disorder | | | | | | | | | |
| Heart failure | | | | | | | | | |
| Cerebrovascular | | | | | | | | | |
| Ischemic stroke | | | | | | | | | |
| All ages | | | | | | | | | |
| Ages ≥ 65 | | | | | | | | | |

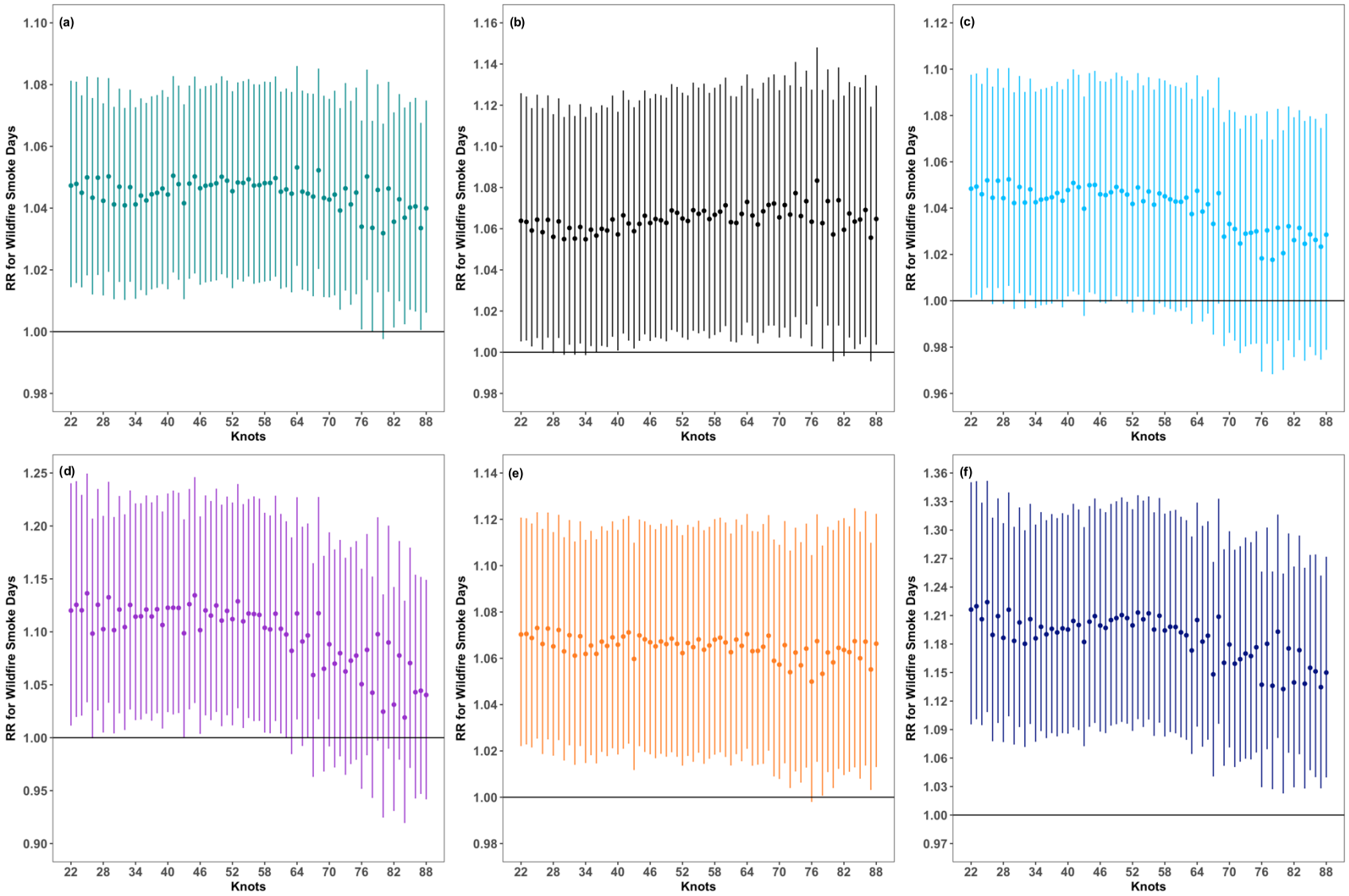


Figure 11. Sensitivity analysis for wildfire smoke days with cutoff 20 μ g/m$^3$. Degrees of freedom (knots) range from 22-88. (a) All-cause respiratory for all ages for lags 2-4. (b) All-cause respiratory for females lags 3-4. (c) All-cause respiratory for males lags 2-4. (d) All-cause respiratory for ages 0-19 lags 3-5. (e) All-cause respiratory for ages 20-64 lags 2-4. (f) Asthma diagnoses lags 3-4. Note change in y-axis scale

## Discussion

The current study evaluated how increases in all sources of $PM_{2.5}$ concentrations were associated with hospital admissions during the fire season in the GSA and during wildfire smoke days where concentrations were equal to or exceeded 20 $\mu g/m^3$ and 30 $\mu g/m^3$ for the 24-hour average. There were no conclusive results for wildfire smoke days at the 30 $\mu g/m^3$ cutoff due to the low number of wildfire smoke days. The only result that was significant was the net effect from lags 0-7 for non-cardiac chest pain or other respiratory symptoms. Any outcomes presented for wildfire smoke days going forward will be referring to days that were greater than or equal to 20 $\mu g/m^3$. The fire season effect estimates were based off the Beacon Hill $PM_{2.5}$ monitor, which consistently had lower $PM_{2.5}$ concentrations than other monitors in the GSA. The Beacon Hill monitor was used due to the completeness of the data during the study period and its proximity to the largest population sector of the GSA.

### *Respiratory Diagnoses*

All-cause respiratory diagnoses were significantly associated with increases in $PM_{2.5}$ during the fire season. This is in accordance with other studies that have looked at increases in $PM_{2.5}$ concentrations and respiratory morbidity (Dominici et al., 2006; Sheppard et al., 1999; Norris et al., 1999). More specifically, femaeles sought increased hospital care for all-cause respiratory diagnoses two to four days after exposure. In comparison, males only sought care two days after exposure during the fire season. The extended period of increased hospital admissions for females suggest that females may be more susceptible to acute increases in ambient $PM_{2.5}$ concentrations. All-cause respiratory hospital admissions increased two days after exposure to increases in $PM_{2.5}$ during the fire season with differential outcomes based on sex.

During wildfire smoke days, there was a prolonged increase in all-cause respiratory hospital admissions two to four days after exposure. This was consistent with other studies that have looked at the association between wildfire $PM_{2.5}$ and respiratory morbidity (Delfino et al., 2009; Johnston et al., 2014; Reid et al., 2016; Wettstein et al., 2018). Males had an extended increase compared to females on wildfire smoke days (Figure 5). However, when comparing respiratory hospital admissions for males during the fire season to wildfire smoke days, males exhibited a lengthier response in hospital care than females. Wildfire smoke days resulted in increased hospital admissions for all-cause respiratory diagnoses with different outcomes based on sex.

Reasons for the disparities in respiratory outcomes may be due to physiological traits such as lung structure, hormones, immunology, or sociocultural and environmental determinants (Becklake & Kauffman, 1999). Other studies have also found contrasts between males and females. Delfino et al. (2009) noticed that female asthma hospitalizations were greater than men between ages 5-64, Haikerwal et al. (2016) found greater increases in female emergency department visits for asthma than males, and Bell et al. (2015) found that females 65 and over had higher rates of hospitalization for respiratory tract infections than men. The results from the present study and several others (Delfino et al., 2009; Haikerwal et al., 2016; Bell et al., 2015) suggest that males and females exhibit differential outcomes to increases in acute $PM_{2.5}$ exposure.

Increases in $PM_{2.5}$ during the fire season and wildfire smoke days resulted in increased respiratory hospital admissions for young adults (20-64), but not for those ≥ 65 years of age (Figure 6). During the fire season, there were increases in hospital admissions two days after a 10 $\mu g/m^3$ increase in $PM_{2.5}$ for young adults (20-64). On the other hand, wildfire smoke days

resulted in extended increases two to four days after exposure for young adults. Adults ≥ 65 years of age were not significantly associated with increased respiratory hospital admissions and $PM_{2.5}$.

The current study expected to see a relationship between $PM_{2.5}$ and older adults (≥ 65) because of their susceptibility to increases in particulate matter concentrations due to physiological changes with age and pre-existing respiratory or cardiovascular conditions (Sacks et al., 2011). Despite this, the results from this study are in line with other studies such as Johnston et al. (2014), Henderson et al. (2011), and Reid et al. (2016) who found that all-cause respiratory morbidity was greater for younger adults than older adults ≥ 65 years of age. In contrast, Sheppard et al. (1999) found no association between $PM_{2.5}$ and nonelderly asthma hospital visits in Seattle during summer and fall periods. Moreover, the age group 20-64 did have a median age of 55 and was highly skewed towards older ages. Thus, this age group is more representative of adults ages 55-64. Other reasons for increases in the 20-64 age group could also be that younger adults may spend more time doing outdoor activities and thus be more susceptible than older adults.

Children ages 0-19 showed significant increases in respiratory hospitalizations only during wildfire smoke days. The age group (0-19) was the most vulnerable age group for wildfire smoke days, with a cumulative increase in all-cause respiratory diagnoses from lags 0-7 of 92.2% and increases in hospitalizations three to five days after exposure (Figure 6b). Children are known to be a susceptible group to increases in $PM_{2.5}$ due to the amount of time spent outdoors and the relative exposure of PM per unit body weight (Sacks et al., 2011, U.S. EPA 2004). Kunzli et al. (2006) and Vicedo-Cabrera et al. (2016) have documented the negative respiratory health effects on children in response to wildfire smoke. Their studies found that

wildfire smoke was positively associated with itchy/watery eyes and sore throat symptoms in both asthmatic and non-asthmatic children. Previous studies such as Norris et al. (1999), found that emergency department visits for asthma in Seattle were significantly associated with increases in $PM_{2.5}$ one day after exposure. Delfino et al. (2009) also found significant increases in all-cause respiratory hospitalizations in children after a wildfire smoke episode in southern California. Delfino et al.'s (2009) study found delays between exposure to wildfire smoke and hospital admissions, similar to the present study. The reasons for the delay in hospital care may be due to slow clearance of particulate matter from the lungs and its cumulative effects (U.S. EPA, 2004) or economic constraints, disruption of daily activities, fear of hospitals or delaying hospitalization until symptoms worsen (Janson & Becker, 1998). Furthermore, short-term exposure to wildfire smoke at younger ages may be associated with long-term changes in immune response. A recent study found that primates that were exposed to short-term wildfire smoke from the northern California fires of 2008 during their infancy had altered lung function and a reduction in proinflammatory cytokines not present in their older primate counterparts (Black et al., 2017). The present study further corroborates other studies that have seen associations between respiratory morbidity in children and $PM_{2.5}$ (Norris et al., 1999; Delfino et al., 2009; Kunzli et al., 2006; Vicedo-Cabrera et al., 2016).

Asthma was another respiratory disease that was significantly associated with increases in $PM_{2.5}$ concentrations during the fire season (Figure 7a) and during wildfire smoke days (Figure 7b). Increases in $PM_{2.5}$ during the fire season were associated with increased hospitalizations one to five days after exposure. The cumulative effect from lags 0-7 showed a 20.4% increase throughout the fire season. This result is consistent with other studies that have looked at the association between $PM_{2.5}$ and asthma morbidity in Seattle (Norris et al., 1999;

Sheppard et al., 1999). In addition, asthma admissions also showed increases during wildfire smoke days three to four days after exposure (Figure 7b). When comparing the net effect from lags 0-7 during wildfire smoke days, asthma increased by 84.5% compared to 20.4% for the fire season. These results are consistent with many other studies that have looked at wildfire PM2.5 and asthma morbidity (Elliott et al., 2013; Johnston et al., 2014; Wettstein et al., 2018; Delfino et al., 2009; Reid et al., 2016; Martin et al., 2013; Rappold et al., 2011; Haikerwal et al., 2016). The greater prevalence of asthma hospitalizations during wildfire smoke days suggest that wildfire smoke exposure may be more hazardous to people with asthma than urban $PM_{2.5}$.

Other respiratory diseases of interest were non-cardiac chest pain or other respiratory symptoms, COPD, and pneumonia. The present study found that non-cardiac chest pain or other respiratory symptoms were significantly associated with increases in $PM_{2.5}$ during the fire season and was marginally significant during wildfire smoke days with a 30 $\mu g/m^3$ cutoff (Figure 8). Non-cardiac chest pain or other respiratory symptoms were not significantly associated with wildfire smoke days at the 20 $\mu g/m^3$ threshold. Wettstein et al. (2018) found that non-cardiac chest pain and other respiratory symptoms were significantly associated with emergency department visits during episodes of medium density wildfire smoke. The mixed results for non-cardiac chest pain or other respiratory symptoms in the current study may be due to low diagnosis counts or the low number of wildfire smoke days during the 2007-2017 study period in the GSA.

COPD was not considered statistically significant at any point during the study period even though it has been strongly associated with wildfire smoke in many other studies (Reid et al., 2016). A few studies such as Kollanus et al. 2016 and Hutchinson et al. 2018, did not find significant increases in hospital admissions for COPD. Another reason for why there was no

statistically significant association between $PM_{2.5}$ and COPD may be due to the specific population being studied. Dominici et al. (2006) found that hospital admissions for COPD were not significantly associated with $PM_{2.5}$ in the Northwest U.S. Additionally, pneumonia was not significantly associated with increases in $PM_{2.5}$ concentrations during the fire season or during wildfire smoke days, similar to Johnston et al.'s (2014) study.

Overall, the sensitivity analysis for respiratory effect estimates were consistent through the fire season (4a-4f). The lack of variability in effect estimates for the fire season suggests that the results are not sensitive to the change in the number of knots. The only exception was asthma during the fire season (Figure 4e). Around 50 knots, the effect estimate begins to exhibit a downward trend and a widening of confidence intervals, suggesting that the temporal trend has been overfit. Consistency in respiratory effect estimates indicates fairly robust results that minimally vary with changes in the degrees of freedom.

Respiratory effect estimates during wildfire smoke days show a bit more variation (Figure 11a-f). Males (Figure 11c) and children 0-19 (Figure 11d) for all-cause respiratory diagnoses and asthma diagnoses (Figure 11f) begin to show increased variation at around 60 knots. The downward trend towards the null and lengthier confidence intervals suggest that the temporal trend has been overfit. These three subgroups show greater variability than other subgroups, with male and female estimates teetering on significance based on the number of knots. Despite this, the number of knots chosen for the study suggest that the time trend has been sufficiently captured for most respiratory subgroups.

*Cardiovascular Diagnoses*

Increases in $PM_{2.5}$ during the fire season were positively associated with increases in all-cause cardiovascular hospital admissions, but only for young adults (20-64) (Figure 9). Older adults (≥ 65 years) are usually considered the most vulnerable groups (Sacks et al., 2011), but there was no association between cardiovascular morbidity and the elderly. The results from this study may be more reflective of middle-aged adults in the 55-64 age range as the median age for the 20-64 age group was 55 and skewed towards older ages. Younger adults may also be more active during periods of increased $PM_{2.5}$ concentrations than older adults. Individual cardiovascular diagnoses were not statistically significant. The lack of significance could be due to low diagnosis counts or consistently low $PM_{2.5}$ concentrations in the GSA. This result was consistently robust as the degrees of freedom varied (Figure 4g). Part of the reason for the robust result is also due to the insensitivity of cardiovascular diseases to changes in time. Even though the GSA has low yearly $PM_{2.5}$ concentrations, a 10 $\mu g/m^3$ increase was associated with an increased relative risk for all-cause cardiovascular diagnoses for young adults (20-64) one day after increased exposure.

In contrast, wildfire smoke days were not associated with cardiovascular hospital admissions in the present study. The relationship between cardiovascular morbidity and ambient $PM_{2.5}$ has been well established (Brook et al., 2010), however the relationship between wildfire $PM_{2.5}$ and cardiovascular morbidity is mixed (Reid et al., 2016. Some studies have found associations between cardiovascular morbidity and wildfire $PM_{2.5}$ (Delfino et al., 2009; Rappold et al., 2011; Johnston et al., 2014; Haikerwal et al., 2015; Wettstein et al., 2018), while others have found a null association similar to the present study (Henderson et al., 2011; Martin et al., 2013; Reid et al., 2016; Hutchinson et al., 2018). Reasons for the varying results across studies looking at wildfire $PM_{2.5}$ may be due to differences in the chemical composition of wildfire

emitted particulate matter. Even so, large gaps still exist regarding the toxicology of wildfire $PM_{2.5}$ and its influence on the cardiovascular system. The contradicting results between the fire season and wildfire smoke days suggest that cardiovascular hospital admissions may be more attributable to urban sources of $PM_{2.5}$ rather than wildfire smoke in the GSA.

*Ischemic Stroke*

Ischemic stroke admissions significantly increased in older adults (≥ 65) during the fire season, but not during wildfire smoke days (Figure 10). The discrepancy may be because urban $PM_{2.5}$ plays a larger role in triggering ischemic stroke than wildfire $PM_{2.5}$. Urban sources of particulate matter have been associated with ischemic stroke in those 65 and older (Kettunen et al., 2007, Wellenius et al., 2005), with a general consensus that ambient particulate matter is associated with increases in ischemic stroke morbidity (Shah et al., 2015). Others that have looked at the association between wildfire $PM_{2.5}$ and cerebrovascular causes have typically yielded mixed results. Morgan et al. (2010) and Johnston et al. (2014) found null associations, while Delfino et al. (2009) and Wettstein et al. (2018) found significant associations between wildfire smoke and increased risk of ischemic stroke. The current study suggests that ischemic stroke for older adults (≥ 65) in the GSA may be more attributable to urban $PM_{2.5}$ during the fire season than wildfire smoke days. Figure 4h shows stable effect estimates with changes in temporal trend fits, suggesting that this is a robust result.

Ischemic stroke diagnoses increased three to four days after exposure to increased $PM_{2.5}$ concentrations in the GSA for older adults (≥ 65), which is consistent with another study done in Copenhagen. Andersen et al. (2010) looked at the association between ultrafine particulate matter (particles of aerodynamic diameter ≤ 0.1μm in diameter) and hospital admissions for

stroke and found that the majority of the patients that went in later had milder strokes. This suggests that increases in $PM_{2.5}$ could result in mild strokes rather than severe ones in the GSA. Other reasons for the delay in hospitalizations after increased $PM_{2.5}$ exposure could be due to prior hospital experiences or the perception of burden on family members (Mackintosh et al., 2012). However, the pathophysiological processes between $PM_{2.5}$ and stroke are still not well understood, and more research still needs to be done (Lee, Miller & Shah, 2018).

*Summary*

Respiratory morbidity was associated with increases in $PM_{2.5}$ throughout the entire fire season, with prolonged increases during wildfire smoke days. Reasons for these extended increases may be because of elevated concentrations during wildfire smoke days rather than the toxicity of wildfire particulate matter (Jalava et al., 2006). Although, some studies have found that wildfire particulate matter (PM) may be more toxic than ambient PM (Wegesser et al., 2009). Wegesser et al. (2009) found that on an equal-weight basis wildfire PM decreased the number of macrophages in mice compared to particulate matter from ambient air when PM was inserted via lung lavage. The weakness of Wegesser et al.'s (2009) study is that they intratracheally instilled particulate matter in mice, which may not accurately reflect how wildfire particulate matter is deposited in the lungs. Differences in the size and hygroscopicity of particles can influence PM deposition in the lungs (Löndahl et al., 2008). Furthermore, size, hygroscopicity, and toxicity of wildfire aerosols may also vary based on what vegetation is burning (Liu & Peng, 2018) and the distance that the aerosols travel (Jalava et al., 2006). Long-range transport of aerosols in wildfire smoke results in photodegradation and settling out of larger particles (Jalava et al., 2006) that influence wildfire smoke composition. More research is

needed to assess the differences in toxicology and chemical composition of wildfire-generated $PM_{2.5}$ and how that differentially impacts health. This could help elucidate the variability in health outcomes between urban and wildfire $PM_{2.5}$, as well as the variability within wildfire $PM_{2.5}$ studies themselves.

Wildfire smoke studies that have looked at cardiovascular and cerebrovascular morbidity have had mixed results. The current study found no association between wildfire smoke days and cardiovascular and cerebrovascular hospital admissions. This may have been because of low hospital counts and the low number of wildfire smoke days. However, this study suggests that urban sources of $PM_{2.5}$ may be more detrimental than wildfire smoke for cardiovascular and cerebrovascular morbidity in the GSA.

*Limitations*

There were several limitations in this study; the biggest caveat being that only one monitor was used to represent the entire greater Seattle area. $PM_{2.5}$ is variable and spatially heterogenous, therefore this study may be underestimating areas that may have had higher $PM_{2.5}$ concentrations. Low $PM_{2.5}$ concentrations in the GSA may have also been another reason for why there were no statistically significant results for certain hospital diagnoses. Another weakness in this study was the low daily counts for certain diagnoses and the small number of wildfire smoke days during the study period. This resulted in wider confidence intervals and made it difficult to discern if hospitalizations significantly increased during times of wildfire smoke. The constrained model approach helped ascertain significant associations between $PM_{2.5}$ and hospitalizations, however it is also criticized for being too data-driven (Bhaskaran et al., 2013). This study was unable to stratify by race due to the small number of daily hospital counts

and because CHARS did not begin recording race until 2008. Despite the shortcomings in this study, the negative health effects associated with increased $PM_{2.5}$ concentrations are important public health issues to address.

Due to the varying sources of wildfire smoke that appear in the GSA, future studies should look at how fresh and aged wildfire plumes may differentially affect health. Future research should also look at the health impacts of increased $PM_{2.5}$ concentrations on communities of low socioeconomic status (SES) and people of color. People that have a lower SES and are non-white tend to be more vulnerable to the deleterious effects of $PM_{2.5}$ (Sacks et al., 2011). Due to possible increases in wildfire smoke frequency with climate change, future research should try to evaluate how varying sources of wildfire smoke and differences in SES and race lead to disparate health outcomes in the GSA.

**Conclusion**

Increases in respiratory hospital admissions were significantly associated with increases in $PM_{2.5}$ concentrations throughout the entire fire season with extended increases in respiratory hospitalizations during wildfire smoke days in the greater Seattle area (GSA). During the fire season, all-cause respiratory diagnoses significantly increased two days after exposure, whereas wildfire smoke days resulted in increases two to four days after exposure. Similar results were obtained for all-cause respiratory diagnoses in the age group 20-64. The only result that was statistically significant during wildfire smoke days that was not considered significant during the fire season was all-cause respiratory diagnoses for the age group 0-19. This suggests that children may be particularly susceptible to the harmful effects of wildfire emitted $PM_{2.5}$ rather

than urban $PM_{2.5}$. Wildfire smoke days consistently showed drawn-out increases in respiratory hospital admissions compared to increases in $PM_{2.5}$ during the fire season.

$PM_{2.5}$ during the fire season was associated with increases in cardiovascular and ischemic stroke diagnoses that were not present during wildfire smoke days. The lack of significance during wildfire smoke days suggests that cardiovascular diagnoses and ischemic stroke may triggered by urban sources of $PM_{2.5}$. Part of this reasoning may be due to the differences in particle chemistry between urban and wildfire sources of $PM_{2.5}$. Another reason could be because wildfire smoke days in the GSA rarely exceeded federal $PM_{2.5}$ standards.

Despite the infrequency of wildfire smoke events exceeding federal $PM_{2.5}$ regulatory standards during the 2007-2017 time period, smoke events may become more severe and frequent in the GSA with climate change. This makes wildfire smoke a growing issue of public health concern. Longer fire seasons, changes in wildfire behavior (Abatzoglou & Kolden, 2013), a greater probability of larger fires (Podschwit et al., 2018), and changes in synoptic patterns that influence smoke transport and air quality in the Western U.S. (Brewer & Mass, 2016) may make wildfire smoke more frequent and severe in the GSA. Public health announcements should make people aware of incoming wildfire smoke in the area, even if it is not expected to exceed EPA's current 24-hour $PM_{2.5}$ standard. The present study suggests that children are particularly susceptible to the negative health effects of wildfire smoke. Future studies should evaluate how changes in wildfire smoke patterns with climate change could influence public health outcomes in the GSA.

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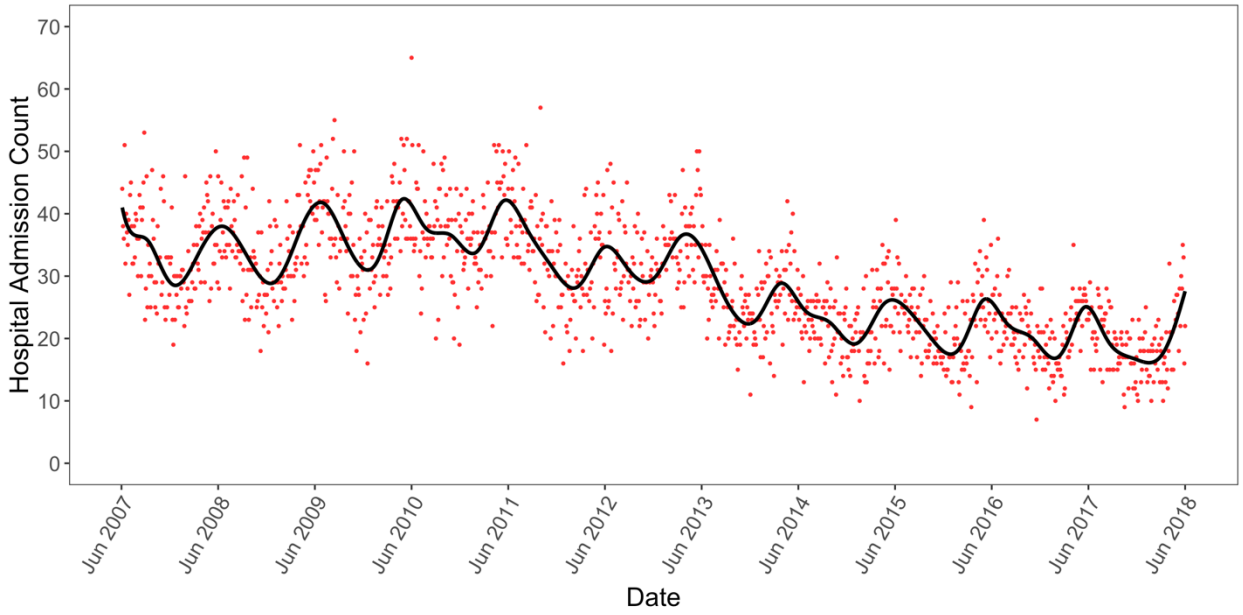
## Appendix A: Wildfire smoke days

List of smoky dates where 1 = smoky and 0 = non-smoky day

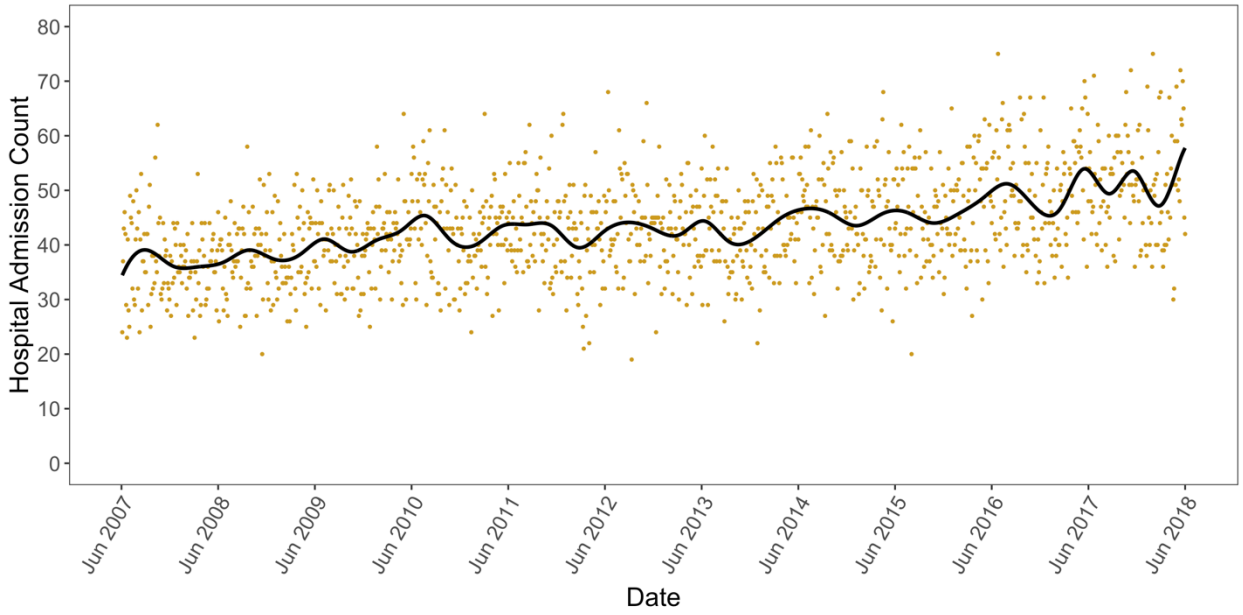
| Wildfire Smoke 20 μg/m$^3$ | | Wildfire Smoke 30 μg/m$^3$ | |
|---|---|---|---|
| Date | Smoky | Date | Smoky |
| 7/4/07 | 0 | 7/5/07 | 0 |
| 7/5/07 | 0 | 7/4/09 | 0 |
| 7/11/07 | 0 | 7/5/09 | 0 |
| 9/13/07 | 1 | 7/29/09 | 1 |
| 7/1/08 | 1 | 7/30/09 | 1 |
| 7/2/08 | 1 | 7/5/11 | 0 |
| 7/4/08 | 0 | 7/4/12 | 0 |
| 7/5/08 | 0 | 7/5/12 | 1 |
| 10/1/08 | 0 | 7/8/12 | 1 |
| 6/1/09 | 0 | 9/13/12 | 1 |
| 6/2/09 | 0 | 9/18/12 | 1 |
| 6/4/09 | 0 | 7/4/13 | 0 |
| 7/4/09 | 0 | 7/4/14 | 0 |
| 7/5/09 | 0 | 7/5/14 | 0 |
| 7/25/09 | 0 | 7/4/15 | 0 |
| 7/28/09 | 1 | 7/5/15 | 1 |
| 7/29/09 | 1 | 7/9/15 | 1 |
| 7/30/09 | 1 | 8/22/15 | 1 |
| 8/4/09 | 1 | 8/23/15 | 1 |
| 8/5/09 | 1 | 8/27/15 | 1 |
| 8/6/09 | 1 | 7/4/16 | 0 |
| 9/12/09 | 0 | 7/5/17 | 0 |
| 9/22/09 | 1 | 8/2/17 | 1 |
| 9/23/09 | 1 | 8/3/17 | 1 |
| 8/5/10 | 1 | 8/4/17 | 1 |
| 8/16/10 | 1 | 8/7/17 | 1 |
| 8/17/10 | 1 | 8/8/17 | 1 |
| 7/4/11 | 0 | 8/9/17 | 1 |
| 7/5/11 | 0 | 8/10/17 | 1 |
| 9/30/11 | 0 | 8/11/17 | 1 |
| 7/4/12 | 0 | 8/29/17 | 1 |
| 7/5/12 | 1 | 9/5/17 | 1 |
| 7/8/12 | 1 | 9/6/17 | 1 |
| 8/17/12 | 1 | 9/7/17 | 1 |
| 9/13/12 | 1 | 9/16/17 | 1 |
| 9/14/12 | 1 | | |
| 9/15/12 | 1 | | |
| 9/17/12 | 1 | | |
| 9/18/12 | 1 | | |
| 9/19/12 | 1 | | |

| | |
|---|---|
| 9/21/12 | 1 |
| 9/28/12 | 1 |
| 7/4/13 | 0 |
| 7/5/13 | 0 |
| 9/14/13 | 0 |
| 7/4/14 | 0 |
| 7/5/14 | 0 |
| 8/11/14 | 1 |
| 8/12/14 | 1 |
| 7/4/15 | 0 |
| 7/5/15 | 1 |
| 7/8/15 | 1 |
| 7/9/15 | 1 |
| 8/12/15 | 1 |
| 8/22/15 | 1 |
| 8/23/15 | 1 |
| 8/26/15 | 1 |
| 8/27/15 | 1 |
| 7/4/16 | 0 |
| 6/17/17 | 0 |
| 7/4/17 | 0 |
| 7/5/17 | 0 |
| 8/1/17 | 1 |
| 8/2/17 | 1 |
| 8/3/17 | 1 |
| 8/4/17 | 1 |
| 8/5/17 | 1 |
| 8/6/17 | 1 |
| 8/7/17 | 1 |
| 8/8/17 | 1 |
| 8/9/17 | 1 |
| 8/10/17 | 1 |
| 8/11/17 | 1 |
| 8/29/17 | 1 |
| 9/5/17 | 1 |
| 9/6/17 | 1 |
| 9/7/17 | 1 |
| 9/16/17 | 1 |
| 9/17/17 | 1 |

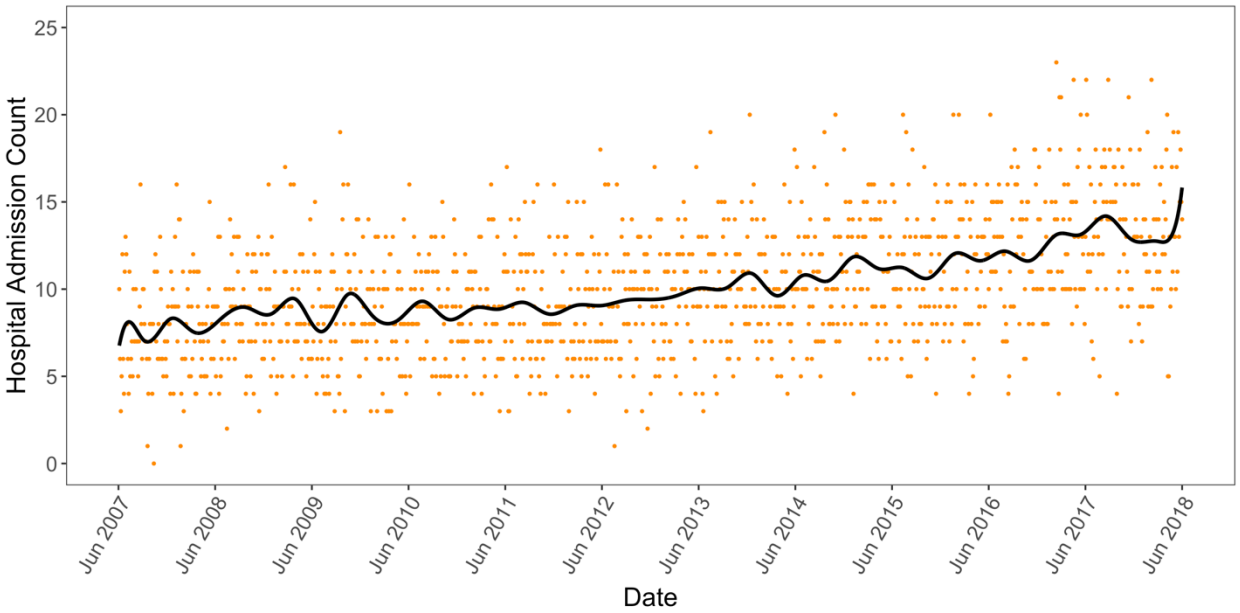
## Appendix B: B-Splines fit through all-cause respiratory, cardiovascular and cerebrovascular for all ages




B.1 – B-Spline with 44 knots fit through daily all-cause respiratory hospital admissions

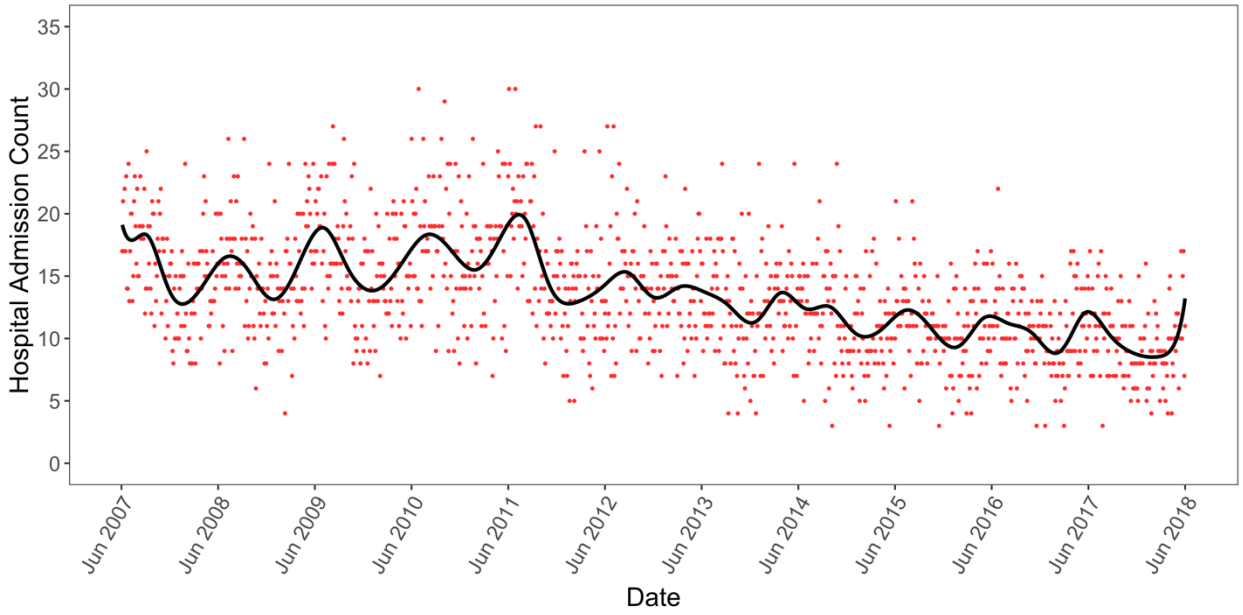



B.2 – B-Spline with 44 knots fit through daily all-cause cardiovascular hospital admissions

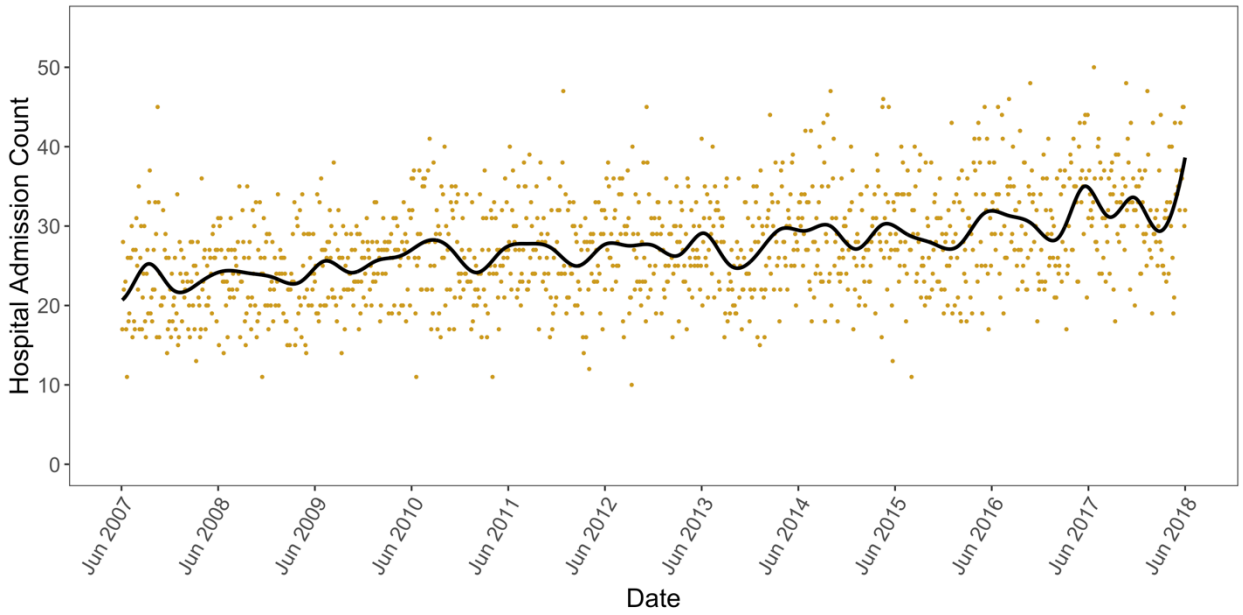



B.3 – B-Spline with 44 knots fit through daily ischemic stroke hospital admissions

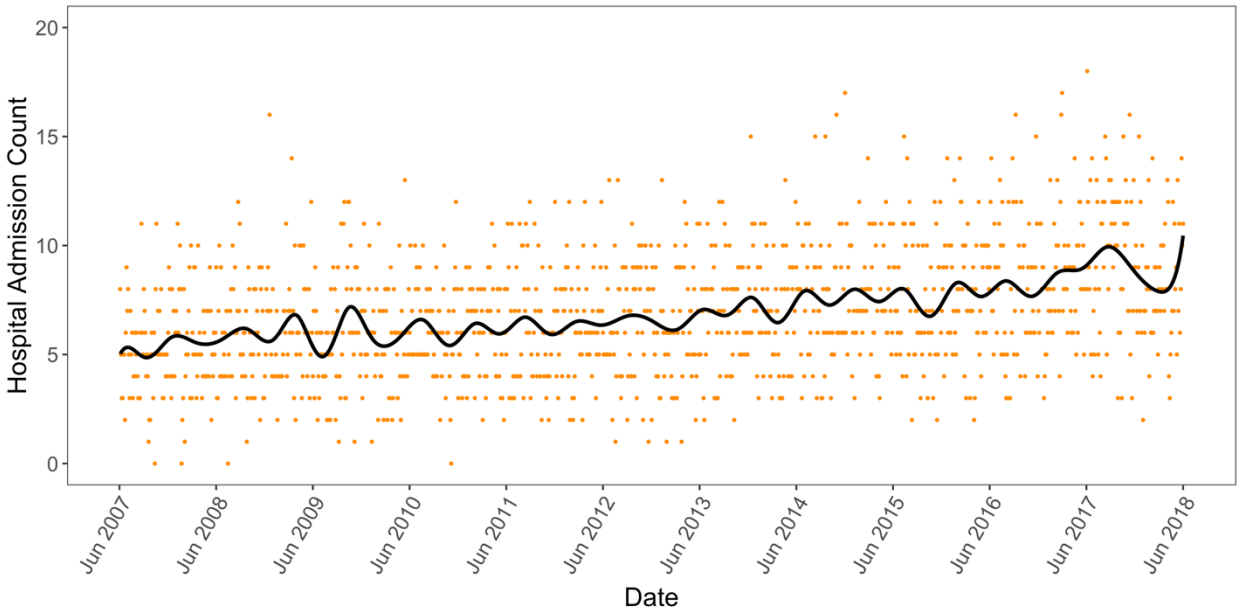
## Appendix C: B-Splines fit through all-cause respiratory, cardiovascular and cerebrovascular for older adults ≥ 65




C.1 – B-Spline with 44 knots fit through daily all-cause respiratory hospital admissions

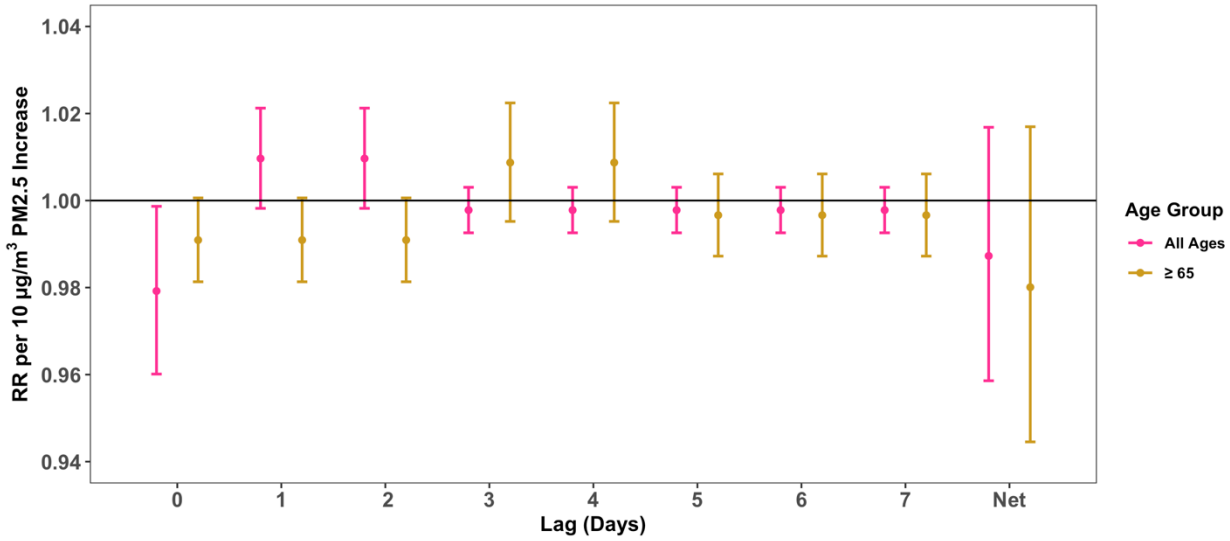



C.2 – B-Spline with 44 knots fit through daily all-cause cardiovascular hospital admissions

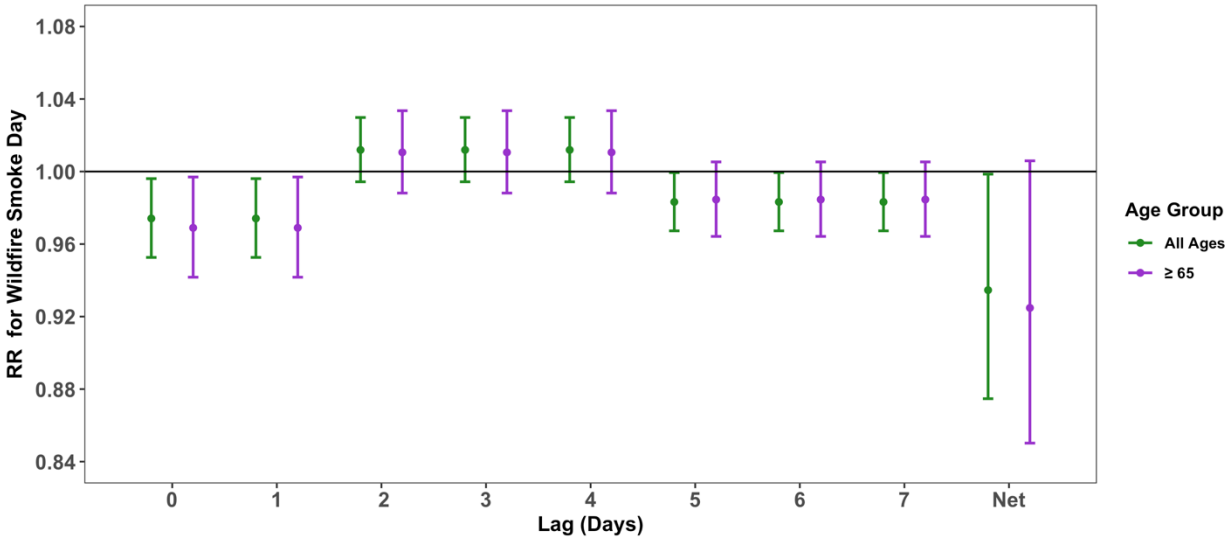



C. 3 – B-Spline with 44 knots fit through daily ischemic stroke hospital admissions

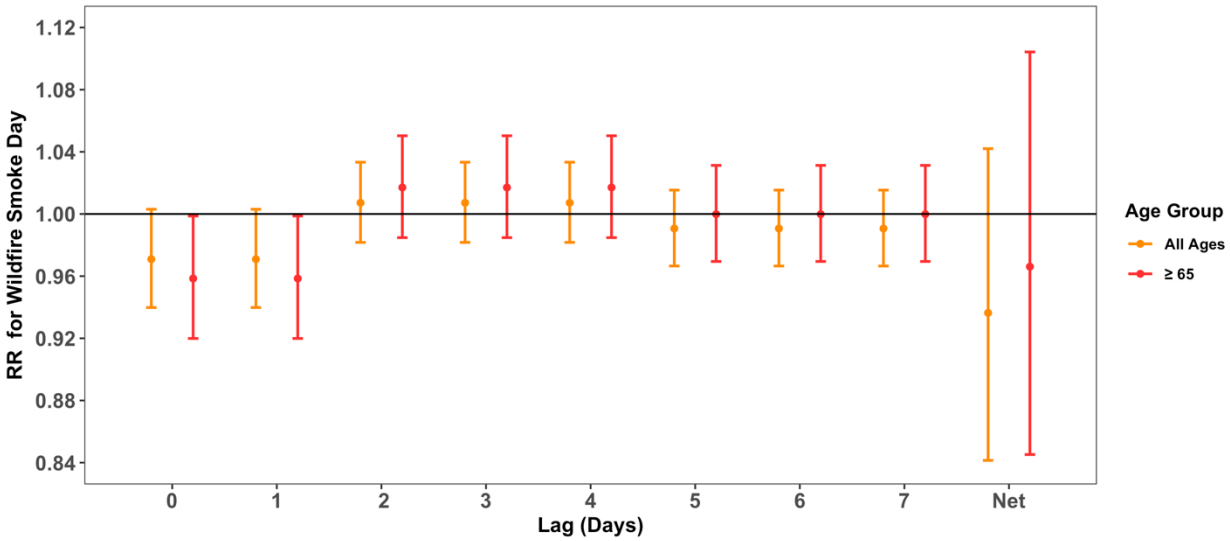
## Appendix D: All-cause diagnoses of interest for fire season and wildfire smoke days




D.1 Relative risk and 95% confidence interval for all-cause diagnoses of interest for all ages and for older adults ≥ 65 for every ≥ 10 µg/m$^3$ increase in all sources of $PM_{2.5}$ during the fire season




D.2 Relative risk and 95% confidence interval for all-cause diagnoses of interest for all ages and for older adults ≥ 65 for wildfire smoke days ≥ 20 µg/m$^3$




D.3 Relative risk and 95% confidence interval for all-cause diagnoses of interest for all ages and for older adults ≥ 65 for wildfire smoke days ≥ 30 μg/m$^3$

# Appendix E: Relative risk and 95% confidence intervals for every 10 μg/m$^3$ increase in PM2.5 concentrations during the fire season

Table E. Relative risk 95% confidence intervals for every 10 μ g/m3 increase in PM2.5 concentrations with bolded numbers indicating statistically significant increases.

| Hospital admission diagnosis | Lag 0 | Lag 1 | Lag 2 | Lag 3 | Lag 4 | Lag 5 | Lag 6 | Lag 7 | Net |
|---|---|---|---|---|---|---|---|---|---|
| All-Respiratory | | | | | | | | | |
| All ages | 0.992 (0.972-1.012) | 0.992 (0.972-1.012) | **1.050 (1.013-1.089)** | 1.005 (0.995-1.015) | 1.005 (0.995-1.015) | 1.005 (0.995-1.015) | 1.005 (0.995-1.015) | 1.005 (0.995-1.015) | **1.062 (1.004-1.122)** |
| Female | 0.985 (0.959-1.012) | 0.985 (0.959-1.012) | **1.025 (1.005-1.046)** | **1.025 (1.005-1.046)** | **1.025 (1.005-1.046)** | 1.015 (0.996-1.034) | 1.015 (0.996-1.034) | 1.015 (0.996-1.034) | **1.093 (1.011-1.182)** |
| Male | 1.002 (0.972-1.032) | 1.002 (0.972-1.032) | **1.065 (1.009-1.124)** | 0.992 (0.977-1.007) | 0.992 (0.977-1.007) | 0.992 (0.977-1.007) | 0.992 (0.977-1.007) | 0.992 (0.977-1.007) | 1.026 (0.943-1.117) |
| Ages 0-19 | 1.032 (0.964-1.104) | 1.032 (0.964-1.104) | 1.045 (0.926-1.180) | 1.014 (0.981-1.049) | 1.014 (0.981-1.049) | 1.014 (0.981-1.049) | 1.014 (0.981-1.049) | 1.014 (0.981-1.049) | 1.193 (0.984-1.447) |
| Ages 20-64 | 0.980 (0.950-1.012) | 0.980 (0.950-1.012) | **1.064 (1.006-1.125)** | 1.001 (0.985-1.016) | 1.001 (0.985-1.016) | 1.001 (0.985-1.016) | 1.001 (0.985-1.016) | 1.001 (0.985-1.016) | 1.026 (0.938-1.121) |
| Ages ≥ 65 | 0.996 (0.968-1.024) | 0.996 (0.968-1.024) | 1.020 (0.991-1.050) | 1.020 (0.991-1.050) | 1.006 (0.990-1.023) | 1.006 (0.990-1.023) | 1.006 (0.990-1.023) | 1.006 (0.990-1.023) | 1.058 (0.976-1.146) |
| Asthma | 1.033 (0.946-1.129) | **1.037 (1.009-1.066)** | **1.037 (1.009-1.066)** | **1.037 (1.009-1.066)** | **1.037 (1.009-1.066)** | **1.037 (1.009-1.066)** | 0.984 (0.936-1.035) | 0.984 (0.936-1.035) | **1.203 (1.030-1.405)** |
| COPD | 1.001 (0.937-1.070) | 1.021 (0.993-1.050) | 1.021 (0.993-1.050) | 1.021 (0.993-1.050) | 0.988 (0.967-1.009) | 0.988 (0.967-1.009) | 0.988 (0.967-1.009) | 0.988 (0.967-1.009) | 1.015 (0.915-1.126) |
| Pneumonia | 0.963 (0.931-0.995) | 0.963 (0.931-0.995) | 1.049 (0.988-1.114) | 1.001 (0.985-1.018) | 1.001 (0.985-1.018) | 1.001 (0.985-1.018) | 1.001 (0.985-1.018) | 1.001 (0.985-1.018) | 0.979 (0.890-1.076) |
| Other non-cardiac chest pain or respiratory symptom | 0.962 (0.896-1.032) | **1.042 (1.001-1.086)** | **1.042 (1.001-1.086)** | 1.017 (0.997-1.037) | 1.017 (0.997-1.037) | 1.017 (0.997-1.037) | 1.017 (0.997-1.037) | 1.017 (0.997-1.037) | **1.135 (1.010-1.274)** |
| All-Cardiovascular | | | | | | | | | |
| All ages | 0.977 (0.950-1.005) | 1.019 (0.990-1.050) | 0.992 (0.986-0.999) | 0.992 (0.986-0.999) | 0.992 (0.986-0.999) | 0.992 (0.986-0.999) | 0.992 (0.986-0.999) | 0.992 (0.986-0.999) | 0.952 (0.916-0.989) |
| Female | 1.000 (0.981-1.020) | 1.000 (0.981-1.020) | 0.994 (0.979-1.009) | 0.994 (0.979-1.009) | 0.994 (0.979-1.009) | 0.994 (0.980-1.008) | 0.994 (0.980-1.008) | 0.994 (0.980-1.008) | 0.965 (0.913-1.020) |
| Male | 0.996 (0.978-1.015) | 0.996 (0.978-1.015) | 0.988 (0.968-1.008) | 0.988 (0.968-1.008) | 0.994 (0.984-1.005) | 0.994 (0.984-1.005) | 0.994 (0.984-1.005) | 0.994 (0.984-1.005) | 0.947 (0.899-0.997) |
| Ages 0-19 | Insufficient counts | | | | | | | | |
| Ages 20-64 | 0.973 (0.926-1.022) | **1.070 (1.017-1.126)** | 0.986 (0.976-0.997) | 0.986 (0.976-0.997) | 0.986 (0.976-0.997) | 0.986 (0.976-0.997) | 0.986 (0.976-0.997) | 0.986 (0.976-0.997) | 0.957 (0.896-1.023) |
| Ages ≥ 65 | 0.980 (0.951-1.010) | 0.990 (0.977-1.003) | 0.990 (0.977-1.003) | 0.990 (0.977-1.003) | 0.999 (0.989-1.008) | 0.999 (0.989-1.008) | 0.999 (0.989-1.008) | 0.999 (0.989-1.008) | 0.947 (0.904-0.992) |
| Hypertension | 0.991 (0.967-1.015) | 0.991 (0.967-1.015) | 0.991 (0.967-1.015) | 1.014 (0.982-1.047) | 1.014 (0.982-1.047) | 1.000 (0.979-1.023) | 1.000 (0.979-1.023) | 1.000 (0.979-1.023) | 1.001 (0.919-1.090) |
| Myocardial infarction | 1.009 (0.993-1.026) | 1.009 (0.993-1.026) | 1.009 (0.993-1.026) | 1.009 (0.993-1.026) | 0.983 (0.934-1.035) | 0.978 (0.958-0.998) | 0.978 (0.958-0.998) | 0.978 (0.958-0.998) | 0.954 (0.885-1.029) |
| Ischemic heart disease | 1.009 (0.993-1.026) | 1.009 (0.993-1.026) | 1.009 (0.993-1.026) | 1.009 (0.993-1.026) | 0.977 (0.928-1.028) | 0.978 (0.959-0.998) | 0.978 (0.959-0.998) | 0.978 (0.959-0.998) | 0.948 (0.879-1.022) |
| Dysrhythmia and conduction disorder | 1.007 (0.988-1.027) | 1.007 (0.988-1.027) | 1.007 (0.988-1.027) | 0.928 (0.883-0.975) | 1.006 (0.991-1.021) | 1.006 (0.991-1.021) | 1.006 (0.991-1.021) | 1.006 (0.991-1.021) | 0.969 (0.903-1.041) |
| Heart failure | 0.996 (0.966-1.026) | 0.996 (0.966-1.026) | 0.973 (0.954-0.992) | 0.973 (0.954-0.992) | 0.973 (0.954-0.992) | 0.973 (0.954-0.992) | 1.011 (0.981-1.042) | 1.011 (0.981-1.042) | 0.907 (0.829-0.992) |
| Cerebrovascular | | | | | | | | | |
| Ischemic stroke | | | | | | | | | |
| All ages | 0.995 (0.975-1.016) | 0.995 (0.975-1.016) | 0.995 (0.975-1.016) | 1.026 (0.998-1.055) | 1.026 (0.998-1.055) | 0.987 (0.968-1.007) | 0.987 (0.968-1.007) | 0.987 (0.968-1.007) | 0.998 (0.924-1.077) |
| Ages ≥ 65 | 0.990 (0.965-1.015) | 0.990 (0.965-1.015) | 0.990 (0.965-1.015) | **1.044 (1.009-1.080)** | **1.044 (1.009-1.080)** | 0.979 (0.955-1.003) | 0.979 (0.955-1.003) | 0.979 (0.955-1.003) | 0.993 (0.905-1.089) |

## Appendix F. Relative risk and 95% confidence intervals for wildfire smoke days ≥ 20 μg/m3 vs. non-wildfire smoke days

Table F. Relative risk 95% confidence intervals for wildfire smoke days ≥ 20 μ g/m$^3$ vs. non-wildfire smoke days with bolded numbers indicating statistically significant increases

| Wildfire Smoke 20 μg/m$^3$ | Lag 0 | Lag 1 | Lag 2 | Lag 3 | Lag 4 | Lag 5 | Lag 6 | Lag 7 | Net |
|---|---|---|---|---|---|---|---|---|---|
| All-Respiratory | | | | | | | | | |
| All ages | 0.974 (0.937-1.012) | 0.974 (0.937-1.012) | **1.048 (1.017-1.079)** | **1.048 (1.017-1.079)** | **1.048 (1.017-1.079)** | 1.005 (0.977-1.033) | 1.005 (0.977-1.033) | 1.005 (0.977-1.033) | 1.107 (0.984-1.245) |
| Female | 0.989 (0.950-1.030) | 0.989 (0.950-1.030) | 0.989 (0.950-1.030) | **1.062 (1.006-1.122)** | **1.062 (1.006-1.122)** | 1.027 (0.987-1.068) | 1.027 (0.987-1.068) | 1.027 (0.987-1.068) | **1.183 (1.004-1.393)** |
| Male | 0.975 (0.920-1.033) | 0.975 (0.920-1.033) | **1.050 (1.004-1.098)** | **1.050 (1.004-1.098)** | **1.050 (1.004-1.098)** | 0.979 (0.938-1.022) | 0.979 (0.938-1.022) | 0.979 (0.938-1.022) | 1.031 (0.863-1.233) |
| Ages 0-19 | 1.070 (0.976-1.173) | 1.070 (0.976-1.173) | 1.070 (0.976-1.173) | **1.126 (1.027-1.234)** | **1.126 (1.027-1.234)** | **1.126 (1.027-1.234)** | 1.048 (0.931-1.179) | 1.048 (0.931-1.179) | **1.922 (1.332-2.774)** |
| Ages 20-64 | 0.911 (0.857-0.967) | 0.911 (0.857-0.967) | **1.070 (1.022-1.120)** | **1.070 (1.022-1.120)** | **1.070 (1.022-1.120)** | 0.998 (0.955-1.042) | 0.998 (0.955-1.042) | 0.998 (0.955-1.042) | 1.008 (0.839-1.212) |
| Ages ≥ 65 | 1.007 (0.914-1.110) | 1.014 (0.956-1.076) | 1.014 (0.956-1.076) | 0.997 (0.967-1.028) | 0.997 (0.967-1.028) | 0.997 (0.967-1.028) | 0.997 (0.967-1.028) | 0.997 (0.967-1.028) | 1.019 (0.855-1.214) |
| Asthma | 1.051 (0.976-1.131) | 1.051 (0.976-1.131) | 1.051 (0.976-1.131) | **1.204 (1.093-1.325)** | **1.204 (1.093-1.325)** | 1.032 (0.960-1.109) | 1.032 (0.960-1.109) | 1.032 (0.960-1.109) | **1.845 (1.374-2.477)** |
| COPD | 0.905 (0.788-1.038) | 1.026 (0.964-1.092) | 1.026 (0.964-1.092) | 1.026 (0.964-1.092) | 0.980 (0.935-1.028) | 0.980 (0.935-1.028) | 0.980 (0.935-1.028) | 0.980 (0.935-1.028) | 0.902 (0.712-1.143) |
| Pneumonia | 0.901 (0.843-0.962) | 0.901 (0.843-0.962) | 1.032 (0.981-1.085) | 1.032 (0.981-1.085) | 1.032 (0.981-1.085) | 0.987 (0.941-1.036) | 0.987 (0.941-1.036) | 0.987 (0.941-1.036) | 0.859 (0.701-1.052) |
| Other non-cardiac chest pain or respiratory symptom | 1.003 (0.931-1.081) | 1.003 (0.931-1.081) | 1.106 (0.975-1.256) | 1.019 (0.979-1.060) | 1.019 (0.979-1.060) | 1.019 (0.979-1.060) | 1.019 (0.979-1.060) | 1.019 (0.979-1.060) | 1.221 (0.971-1.536) |
| All-Cardiovascular | | | | | | | | | |
| All ages | 0.970 (0.948-0.992) | 0.970 (0.948-0.992) | 0.970 (0.948-0.992) | 1.003 (0.972-1.035) | 1.003 (0.972-1.035) | 0.975 (0.954-0.997) | 0.975 (0.954-0.997) | 0.975 (0.954-0.997) | 0.851 (0.779-0.929) |
| Female | 0.967 (0.926-1.009) | 0.967 (0.926-1.009) | 1.003 (0.970-1.038) | 1.003 (0.970-1.038) | 1.003 (0.970-1.038) | 0.978 (0.948-1.009) | 0.978 (0.948-1.009) | 0.978 (0.948-1.009) | 0.883 (0.778-1.003) |
| Male | 0.979 (0.913-1.050) | 0.968 (0.937-1.000) | 0.968 (0.937-1.000) | 0.968 (0.937-1.000) | 0.982 (0.958-1.007) | 0.982 (0.958-1.007) | 0.982 (0.958-1.007) | 0.982 (0.958-1.007) | 0.827 (0.733-0.933) |
| Ages 0-19 | Insufficient counts | | | | | | | | |
| Ages 20-64 | 1.003 (0.953-1.057) | 1.003 (0.953-1.057) | 0.962 (0.923-1.003) | 0.962 (0.923-1.003) | 0.962 (0.923-1.003) | 0.959 (0.923-0.996) | 0.959 (0.923-0.996) | 0.959 (0.923-0.996) | 0.791 (0.678-0.923) |
| Ages ≥ 65 | 0.947 (0.913-0.983) | 0.947 (0.913-0.983) | 1.009 (0.972-1.048) | 1.009 (0.972-1.048) | 0.992 (0.970-1.014) | 0.992 (0.970-1.014) | 0.992 (0.970-1.014) | 0.992 (0.970-1.014) | 0.884 (0.794-0.983) |
| Hypertension | 0.989 (0.911-1.074) | 0.989 (0.911-1.074) | 1.025 (0.961-1.092) | 1.025 (0.961-1.092) | 1.025 (0.961-1.092) | 0.996 (0.943-1.052) | 0.996 (0.943-1.052) | 0.996 (0.943-1.052) | 1.040 (0.835-1.294) |
| Myocardial infarction | 0.954 (0.898-1.013) | 0.954 (0.898-1.013) | 1.046 (0.984-1.112) | 1.046 (0.984-1.112) | 0.948 (0.914-0.983) | 0.948 (0.914-0.983) | 0.948 (0.914-0.983) | 0.948 (0.914-0.983) | 0.803 (0.673-0.958) |
| Ischemic heart disease | 0.954 (0.898-1.013) | 0.954 (0.898-1.013) | 1.046 (0.985-1.112) | 1.046 (0.985-1.112) | 0.947 (0.913-0.982) | 0.947 (0.913-0.982) | 0.947 (0.913-0.982) | 0.947 (0.913-0.982) | 0.801 (0.672-0.955) |
| Dysrhythmia and conduction disorder | 1.010 (0.921-1.108) | 0.952 (0.912-0.994) | 0.952 (0.912-0.994) | 0.952 (0.912-0.994) | 1.008 (0.975-1.041) | 1.008 (0.975-1.041) | 1.008 (0.975-1.041) | 1.008 (0.975-1.041) | 0.899 (0.764-1.057) |
| Heart failure | 0.955 (0.912-1.000) | 0.955 (0.912-1.000) | 0.955 (0.912-1.000) | 0.967 (0.921-1.015) | 0.967 (0.921-1.015) | 0.967 (0.921-1.015) | 0.972 (0.916-1.032) | 0.972 (0.916-1.032) | 0.744 (0.616-0.899) |
| Cerebrovascular | | | | | | | | | |
| Ischemic stroke | | | | | | | | | |
| All ages | 1.002 (0.942-1.066) | 1.002 (0.942-1.066) | 1.010 (0.947-1.077) | 1.010 (0.947-1.077) | 0.975 (0.940-1.012) | 0.975 (0.940-1.012) | 0.975 (0.940-1.012) | 0.975 (0.940-1.012) | 0.926 (0.774-1.109) |
| Ages ≥ 65 | 0.991 (0.918-1.069) | 0.991 (0.918-1.069) | 1.024 (0.948-1.107) | 1.024 (0.948-1.107) | 0.978 (0.935-1.024) | 0.978 (0.935-1.024) | 0.978 (0.935-1.024) | 0.978 (0.935-1.024) | 0.943 (0.758-1.174) |

## Appendix G: Relative risk and 95% confidence intervals for wildfire smoke days ≥ 30 μg/m$^3$ vs. non-wildfire smoke days

| Hospital Admission Diagnosis | Lag 0 | Lag 1 | Lag 2 | Lag 3 | Lag 4 | Lag 5 | Lag 6 | Lag 7 | Net |
|---|---|---|---|---|---|---|---|---|---|
| All-Respiratory | | | | | | | | | |
| All ages | 1.012 (0.965-1.061) | 1.012 (0.965-1.061) | 1.012 (0.965-1.061) | 1.023 (0.962-1.089) | 1.023 (0.962-1.089) | 1.005 (0.959-1.054) | 1.005 (0.959-1.054) | 1.005 (0.959-1.054) | 1.102 (0.897-1.354) |
| Female | 0.995 (0.930-1.065) | 0.995 (0.930-1.065) | 0.995 (0.930-1.065) | 1.060 (0.973-1.154) | 1.060 (0.973-1.154) | 1.034 (0.968-1.105) | 1.034 (0.968-1.105) | 1.034 (0.968-1.105) | 1.224 (0.918-1.634) |
| Male | 1.029 (0.959-1.105) | 1.029 (0.959-1.105) | 1.029 (0.959-1.105) | 0.987 (0.918-1.062) | 0.987 (0.918-1.062) | 0.987 (0.918-1.062) | 0.964 (0.878-1.058) | 0.964 (0.878-1.058) | 0.976 (0.714-1.335) |
| Ages 0-19 | 1.291 (0.949-1.758) | 0.964 (0.815-1.141) | 0.964 (0.815-1.141) | 0.964 (0.815-1.141) | 1.134 (0.995-1.293) | 1.134 (0.995-1.293) | 1.134 (0.995-1.293) | 1.134 (0.995-1.293) | 1.914 (0.937-3.913) |
| Ages 20-64 | 1.039 (0.975-1.107) | 1.039 (0.975-1.107) | 1.039 (0.975-1.107) | 1.039 (0.975-1.107) | 0.961 (0.871-1.060) | 0.961 (0.871-1.060) | 0.996 (0.904-1.096) | 0.996 (0.904-1.096) | 1.066 (0.769-1.478) |
| Ages ≥ 65 | 0.994 (0.928-1.066) | 0.994 (0.928-1.066) | 0.994 (0.928-1.066) | 1.018 (0.930-1.114) | 1.018 (0.930-1.114) | 0.996 (0.930-1.067) | 0.996 (0.930-1.067) | 0.996 (0.930-1.067) | 1.006 (0.748-1.354) |
| Asthma | 1.237 (0.974-1.570) | 0.947 (0.800-1.121) | 0.947 (0.800-1.121) | 1.086 (0.990-1.190) | 1.086 (0.990-1.190) | 1.086 (0.990-1.190) | 1.086 (0.990-1.190) | 1.086 (0.990-1.190) | 1.673 (0.956-2.929) |
| COPD | 1.042 (0.930-1.167) | 1.042 (0.930-1.167) | 1.027 (0.914-1.155) | 1.027 (0.914-1.155) | 0.949 (0.881-1.023) | 0.949 (0.881-1.023) | 0.949 (0.881-1.023) | 0.949 (0.881-1.023) | 0.932 (0.637-1.362) |
| Pneumonia | 0.956 (0.891-1.026) | 0.956 (0.891-1.026) | 0.956 (0.891-1.026) | 0.956 (0.891-1.026) | 1.011 (0.933-1.095) | 1.011 (0.933-1.095) | 1.011 (0.933-1.095) | 0.943 (0.797-1.115) | 0.813 (0.571-1.159) |
| Other non-cardiac chest pain/respiratory | 1.034 (0.913-1.171) | 1.034 (0.913-1.171) | 1.129 (0.998-1.276) | 1.129 (0.998-1.276) | 1.029 (0.949-1.116) | 1.029 (0.949-1.116) | 1.029 (0.949-1.116) | 1.029 (0.949-1.116) | **1.529 (1.003-2.332)** |
| All-Cardiovascular | | | | | | | | | |
| All ages | 0.941 (0.902-0.983) | 0.941 (0.902-0.983) | 1.025 (0.955-1.099) | 0.988 (0.966-1.012) | 0.988 (0.966-1.012) | 0.988 (0.966-1.012) | 0.988 (0.966-1.012) | 0.988 (0.966-1.012) | 0.857 (0.747-0.983) |
| Female | 0.946 (0.889-1.006) | 0.946 (0.889-1.006) | 1.002 (0.954-1.051) | 1.002 (0.954-1.051) | 1.002 (0.954-1.051) | 0.975 (0.931-1.021) | 0.975 (0.931-1.021) | 0.975 (0.931-1.021) | 0.834 (0.682-1.020) |
| Male | 0.945 (0.891-1.001) | 0.945 (0.891-1.001) | 0.986 (0.929-1.046) | 0.986 (0.929-1.046) | 1.002 (0.967-1.039) | 1.002 (0.967-1.039) | 1.002 (0.967-1.039) | 1.002 (0.967-1.039) | 0.875 (0.725-1.055) |
| Ages 0-19 | Insufficient Counts | | | | | | | | |
| Ages 20-64 | 0.945 (0.878-1.019) | 0.945 (0.878-1.019) | 0.984 (0.928-1.043) | 0.984 (0.928-1.043) | 0.984 (0.928-1.043) | 0.958 (0.906-1.013) | 0.958 (0.906-1.013) | 0.958 (0.906-1.013) | 0.748 (0.587-0.953) |
| Ages ≥ 65 | 0.944 (0.897-0.995) | 0.944 (0.897-0.995) | 1.012 (0.972-1.054) | 1.012 (0.972-1.054) | 1.012 (0.972-1.054) | 1.000 (0.963-1.040) | 1.000 (0.963-1.040) | 1.000 (0.963-1.040) | 0.926 (0.783-1.095) |
| Hypertension | 0.945 (0.856-1.043) | 0.945 (0.856-1.043) | 1.026 (0.951-1.107) | 1.026 (0.951-1.107) | 1.026 (0.951-1.107) | 1.006 (0.940-1.077) | 1.006 (0.940-1.077) | 1.006 (0.940-1.077) | 0.981 (0.715-1.345) |
| Myocardial infarction | 0.948 (0.871-1.033) | 0.948 (0.871-1.033) | 1.065 (0.980-1.158) | 1.065 (0.980-1.158) | 0.953 (0.904-1.006) | 0.953 (0.904-1.006) | 0.953 (0.904-1.006) | 0.953 (0.904-1.006) | 0.843 (0.641-1.109) |
| Ischemic heart disease | 0.941 (0.864-1.024) | 0.941 (0.864-1.024) | 1.060 (0.975-1.152) | 1.060 (0.975-1.152) | 0.953 (0.903-1.005) | 0.953 (0.903-1.005) | 0.953 (0.903-1.005) | 0.953 (0.903-1.005) | 0.819 (0.623-1.075) |
| Dysrhythmia and conduction disorder | 1.048 (0.924-1.188) | 0.992 (0.941-1.046) | 0.992 (0.941-1.046) | 0.992 (0.941-1.046) | 0.992 (0.941-1.046) | 0.995 (0.937-1.057) | 0.995 (0.937-1.057) | 0.995 (0.937-1.057) | 1.001 (0.773-1.296) |
| Heart failure | 1.014 (0.872-1.179) | 0.883 (0.814-0.958) | 0.883 (0.814-0.958) | 0.883 (0.814-0.958) | 0.981 (0.920-1.047) | 0.981 (0.920-1.047) | 0.981 (0.920-1.047) | 0.981 (0.920-1.047) | 0.647 (0.465-0.900) |
| Cerebrovascular Ischemic stroke | | | | | | | | | |
| All ages | 0.981 (0.901-1.068) | 0.981 (0.901-1.068) | 1.027 (0.961-1.098) | 1.027 (0.961-1.098) | 1.027 (0.961-1.098) | 0.980 (0.919-1.045) | 0.980 (0.919-1.045) | 0.980 (0.919-1.045) | 0.982 (0.745-1.294) |
| Ages ≥ 65 | 0.951 (0.856-1.057) | 0.951 (0.856-1.057) | 1.055 (0.973-1.144) | 1.055 (0.973-1.144) | 1.055 (0.973-1.144) | 0.997 (0.922-1.078) | 0.997 (0.922-1.078) | 0.997 (0.922-1.078) | 1.052 (0.752-1.474) |