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Comparison of wildfire smoke estimation methods and associations with cardiopulmonary-related hospital admissions

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Abstract

Climate forecasts predict an increase in frequency and intensity of wildfires. Associations between health outcomes and population exposure to smoke from Washington 2012 wildfires were compared using surface monitors, chemical-weather models, and a novel method blending three exposure information sources. The association between smoke particulate matter $2.5 \mu\text{m}$ in diameter ($\text{PM}_{2.5}$) and cardiopulmonary hospital admissions occurring in Washington from 1 July to 31 October 2012 was evaluated using a time-stratified case-crossover design. Hospital admissions aggregated by ZIP code were linked with population-weighted daily average concentrations of smoke $\text{PM}_{2.5}$ estimated using three distinct methods: a simulation with the Weather Research and Forecasting with Chemistry (WRF-Chem) model, a kriged interpolation of $\text{PM}_{2.5}$ measurements from surface monitors, and a geographically weighted ridge regression (GWR) that blended inputs from WRF-Chem, satellite observations of aerosol optical depth, and kriged $\text{PM}_{2.5}$. A $10 \mu\text{g}/\text{m}^3$ increase in GWR smoke $\text{PM}_{2.5}$ was associated with an 8% increased risk in asthma-related hospital admissions (odds ratio (OR): 1.076, 95% confidence interval (CI): 1.019–1.136); other smoke estimation methods yielded similar results. However, point estimates for chronic obstructive pulmonary disease (COPD) differed by smoke $\text{PM}_{2.5}$ exposure method: a $10 \mu\text{g}/\text{m}^3$ increase using GWR was significantly associated with increased risk of COPD (OR: 1.084, 95%CI: 1.026–1.145) and not significant using WRF-Chem (OR: 0.986, 95%CI: 0.931–1.045). The magnitude (OR) and uncertainty (95%CI) of associations between smoke $\text{PM}_{2.5}$ and hospital admissions were dependent on estimation method used and outcome evaluated. Choice of smoke exposure estimation method used can impact the overall conclusion of the study.

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Supporting Information: Supporting Information S1

1. Introduction

There is growing evidence that wildfires are increasing in intensity [Westerling et al., 2006; Langmann et al., 2009; Turetsky et al., 2011; Moritz et al., 2012], which is projected to have a negative impact on air quality in certain areas of the United States [Val Martin et al., 2015; Liu et al., 2016a]. This projected degradation of air quality due to wildfires presents challenges to public health. Quantification of the relationship between smoke exposure and population-level health outcomes would assist in the anticipation of and preparation for public health planning.

Previous estimates of population-level health effects from wildfire smoke exposure have been inferred from epidemiologic studies that have evaluated health effects of particulate air pollution on cardiorespiratory morbidity and mortality outcomes [Brunekreef and Holgate, 2002; Dominici et al., 2002, 2005; Pope et al., 2002, 2009; Miller et al., 2007]. Many of these studies have informed and guided epidemiologic studies of wildfire smoke and population health, where a growing body of epidemiologic evidence consistently finds exposure to wildfire smoke increases risk for adverse pulmonary outcomes [Liu et al., 2015; Reid et al., 2016]. However, unlike the ambient air pollution studies that have found a relationship with adverse cardiovascular outcomes [Miller et al., 2007], the current wildfire smoke studies have found inconsistent associations with cardiovascular outcomes [Liu et al., 2015; Reid et al., 2016].

A possible source of bias in these epidemiologic wildfire smoke studies is exposure misclassification. In their review of wildfire smoke and population health, Liu et al. suggest that improvement is needed in the assessment and quantification of smoke exposure [Liu et al., 2015]. Existing population-based studies have primarily used three types of methods when assessing the relationship between smoke exposure and health: measurements from surface air pollutant monitors, measurements from satellite sensors, or chemical-weather models. Surface particulate matter (PM) 2.5 measurements have been used most frequently [Liu et al., 2015], with satellite observations increasing in popularity as a method for assessing wildfire smoke exposure [Henderson et al., 2011; Rappold et al., 2011; Liu et al., 2015]. Most recently, chemical-weather models have been used to simulate wildfire smoke exposure; studies employing this method to estimate smoke exposure have found an increased risk for respiratory-related morbidity [Alman et al., 2016; Liu et al., 2016b] and cardiovascular-related morbidity [Haikerwal et al., 2015]. While in situ measurements from surface monitors, satellite-based measurements, and simulations with chemical-weather models all have utility for estimation of wildfire smoke exposure, each method has inherent limitations. Surface in situ monitors offer sparse spatial coverage, and there is no information between individual monitors. Sensors on satellites often produce information about smoke (or other particulates) in the entire atmospheric column, not at the ground level. Further, retrievals often fail in the presence of clouds or extremely thick smoke. In chemical-weather models, simulation of the movement and chemical processing of fire smoke is difficult [Garcia-Menendez et al., 2013; Alvarado et al., 2015; Saide et al., 2015; Baker et al., 2016; Paugam et al., 2016].

Given the limitations of each of these exposure assessment methods, we previously proposed a novel application using all three of these methods in a blended model (also referred to as data fusion) to produce a more accurate estimation of surface-level wildfire smoke concentrations in our companion paper by Lassman et al. [2017], which describes the model and its performance. We implemented these methods in Washington state in late summer 2012, a period when series of large and intense wildfires produced extensive smoke that impacted communities throughout the state. For this paper, we used this model to assess the relationship between various estimation methods for wildfire smoke exposure and cardiorespiratory-related hospital admissions in Washington state from 1 July to 31 October 2012. Our objective was to determine if the choice of exposure model resulted in significant and meaningful differences in health risk estimates for a range of health endpoints.

2. Methods

2.1. Measures of Health Outcomes

Our study population consisted of persons who were admitted to a hospital in Washington state and were recorded in the Washington State Department of Health Comprehensive Hospital Abstract Reporting System (CHARS) for the year 2012. CHARS is a hospital admission data set that contains records for both inpatient and outpatient hospital admissions. Variables for our study included a unique deidentified patient identifier, patient-level information on International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) diagnosis codes, date of admission and discharge, admission type, and reported ZIP code of residence, age, and sex. We limited the study population to admission dates between 1 July through 31 October 2012 and to reported ZIP codes of residence within Washington. We further limited our study population to admissions classified as either emergency or urgent care as these patients are most likely in need of immediate medical care due to a recent event (i.e., car accident) or exposure (i.e., smoke exposure). Finally, we limited our study population to subjects with only a single observation using the unique patient identifier to reduce potential bias and complexities of accounting for multiple adverse events.

Given the established relationship between air pollution with cardiopulmonary health outcomes [Miller et al., 2007; Brook et al., 2010], and in line with other studies evaluating the health effects of wildfire smoke [Johnston et al., 2007; Henderson et al., 2011; Rappold et al., 2011; Haikerwal et al., 2015; Liu et al., 2015, 2016b; Alman et al., 2016; Reid et al., 2016], we assessed the following primary reported ICD-9-CM code within the 2012 CHARS data set as a proxy measure for an event: respiratory (ICD-9-CM: 460–519), asthma (ICD-9-CM: 493), chronic obstructive pulmonary disease (COPD)(ICD-9-CM: 490–492, 494, and 496), pneumonia (ICD-9-CM: 480–486), acute bronchitis (ICD-9-CM: 466), cardiovascular disease (ICD-9-CM: 390–459), arrhythmia (ICD-9-CM: 427), cerebrovascular disease (ICD-9-CM: 430–438), heart failure (ICD-9-CM: 428), ischemic heart disease (ICD-9-CM: 410–413), and myocardial infarction (ICD-9-CM: 410). In addition, we also assessed fracture of radius and ulna (broken arm) (ICD-9-CM: 813) as an expected null outcome, hypothesizing that there would be no association with wildfire smoke exposure.

2.2. Time-Stratified Case-Crossover Study Design

In the time-stratified case-crossover design, each subject's exposure value on an index date (e.g., admission date) is compared to subject-specific referent periods that are matched to the index date based on the same day of the week within a time period. This method controls for time-invariant confounding variables (e.g., age and sex) and reduces bias from any time trends in the exposure and time-varying factors (e.g., day of the week and seasonal trends in exposure) [Lumley and Levy, 2000; Janes et al., 2005a, 2005b]. For each of our defined health outcomes, we identified all cases within the CHARS data that met the criteria defined above, and we created time-stratified case-crossover data sets. Referent periods were selected on the same day of the week as the index period within the entire wildfire season of 1 July to 31 October 2012. Estimations of smoke PM_{2.5} and meteorological measurements were linked to these case-crossover data sets using the reported ZIP code of residence.

2.3. Estimating Wildfire Smoke Exposure

2.3.1. Estimation of PM_{2.5} Attributed to Wildfire Smoke—Exposure assessment methods and performance statistics are described in full in Lassman et al. [2017]. Briefly, exposure data were obtained from multiple information sources from 1 July through 31 October 2012. We created a grid such that simulated surface PM_{2.5} concentrations and other estimates of surface PM_{2.5} from both satellite and in situ measures were estimated for the same spatial location. We estimated daily average surface PM_{2.5} concentrations for each grid box using three distinct estimation methods:

1. *Weather Research and Forecasting with Chemistry (WRF-Chem) chemical-weather model:* Daily PM_{2.5} concentrations were simulated using the Weather Research and Forecasting with Chemistry (WRF-Chem) model [Grell et al., 2005]. Simulations were conducted at 15 km × 15 km resolution. The Fire Inventory from National Center for Atmospheric Research v1.5 was used to estimate biomass burning emissions [Wiedinmyer et al., 2011]. Additional simulations were conducted with biomass burning emissions turned off to estimate nonwildfire smoke PM_{2.5} concentrations. Details about other simulation parameters and settings are described in Lassman et al. [2017]. Performance statistics of WRF-Chem simulations compared to observed surface PM_{2.5} were as follows: estimated slope of the fitted trend line = 0.67, $R^2 = 0.25$, mean absolute error = 11.45 µg/m³, and mean bias = 10.22 µg/m³ [Lassman et al., 2017]. In this context, the slope of the fitted trend line describes a regression of the observations onto the model-predicted values; a value of less than one implies that the model overestimates PM_{2.5} concentrations.
2. *Kriging of in situ surface monitors:* Surface monitor PM_{2.5} concentration data were obtained from the Environmental Protection Agency Air Quality System (AQS) for Washington. Additional monitors were deployed during the smoke time period by the Washington Department of Ecology; these data were integrated into the surface network. We used Gaussian process regression (i.e., ordinary kriging) [Isaaks and Srivastava, 1988] to interpolate the measured PM_{2.5} concentrations to our 15 × 15 km grid for the entire state. To reduce the potential for bias from edge effects [Jerrett et al., 2004], we obtained additional surface

monitor $PM_{2.5}$ data from the AQS for Northern Oregon, Western Idaho, and Western Montana, as well as surface monitor $PM_{2.5}$ for British Columbia, Canada (data provided by Dr. Sarah Henderson at the University of British Columbia School of Population and Public Health). There were a total of 212 surface $PM_{2.5}$ monitors in the region that were input to the kriging model. Because the surface monitors report values on varying temporal frequencies, the number of available measurements for each 24 h period varied; we use all available measurements on each day as input to the kriging approach, with a static semi-variogram. Leave-one-out cross validation was used to assess performance of the kriging approach compared to observed surface sites. The model was evaluated at each in situ measurement site that was inside the domain of interest (156 of the 212 total monitors) on every day that the site reported a value. Evaluation was not done at the boundary sites due to instabilities in kriging at these locations. The approach and justification are described in detail in Lassman et al. [2017]. Performance statistics of kriging were as follows: estimated slope of the fitted trend line = 0.70, $R^2 = 0.69$, mean absolute error = $2.09 \mu\text{g}/\text{m}^3$, and mean bias = $0.00 \mu\text{g}/\text{m}^3$ [Lassman et al., 2017]. The slope is less than one, but closer to one than the WRF-Chem estimates value, which implies improved slope compared to the WRF-Chem model.

3. *Geographically weighted regression (GWR)*: Additional data were obtained on aerosol optical depth (AOD), a measure of the extinction of solar light by all particle types within the atmospheric column, from the Moderate Resolution Imaging Spectroradiometer instrument aboard the National Aeronautics and Space Administration (NASA) Terra and Aqua polar-orbiting satellites. GWR was used to estimate the expected $PM_{2.5}$ concentrations of each grid box by combining the kriged in situ measurements, satellite aerosol optical depth (AOD), and simulated WRF-Chem estimations while accounting for multicollinearity and spatial variability [Brunsdon et al., 1998a, 1998b]. Leave-one-out cross validation was used to assess model performance of the GWR compared to observed surface sites. Performance statistics for GWR were as follows: estimated slope of the fitted trend line = 0.78, $R^2 = 0.66$, mean absolute error = $2.40 \mu\text{g}/\text{m}^3$, and mean bias = $0.37 \mu\text{g}/\text{m}^3$ [Lassman et al., 2017]. All of the exposure estimates were evaluated with an additional sensitivity analysis in the companion publication [Lassman et al., 2017].

To distinguish $PM_{2.5}$ that may be attributed to wildfire smoke from other sources for the WRF-Chem method, we took our estimated daily average surface $PM_{2.5}$ estimates for each grid box and subtracted estimates of nonsmoke $PM_{2.5}$ produced by WRF-Chem. The contributions of nonsmoke sources to total $PM_{2.5}$ in our study region were produced using WRF-Chem simulations where fire emissions were turned off. We refer to the difference in simulated $PM_{2.5}$ between simulation with and without fire emissions as WRF-Chem smoke. To estimate $PM_{2.5}$ that may be attributed to smoke for the kriging and GWR methods, we estimated background $PM_{2.5}$ using the National Oceanic and Atmospheric Administration's (NOAA) Hazard Mapping System (HMS) to identify days where wildfire smoke was not in the vicinity of a surface monitor. We then calculated the median $PM_{2.5}$ concentrations for

each surface monitor over the nonfire impacted days of the study period; these levels were then interpolated using kriging to each grid box to estimate background levels of $PM_{2.5}$ derived from nonwildfire sources. Finally, we subtracted these background levels from each method to produce estimates of $PM_{2.5}$ that may be attributed to smoke. We refer to the estimates as kriging smoke and GWR smoke. All $PM_{2.5}$ concentrations are reported in $\mu\text{g}/\text{m}^3$.

2.3.2. ZIP Code Population-Weighted Average of $PM_{2.5}$ and Census-Weighted Meteorological Measures—Each of our smoke variables was calculated as a population-weighted average $PM_{2.5}$ concentration at the ZIP-code level (to correspond to the resolution of our health outcome data) for each day within the study period. This approach weights the average of gridded $PM_{2.5}$ concentrations according to the known population density within a given ZIP code. We used available measures of temperature, humidity, wind speed, and precipitation that were census weighted to the ZIP-code level. For more details on both these population-weighted methods, please see Text S1 in the supporting information.

2.4. Analyses

To describe the temporal variability of the three smoke estimates, we plotted time series of the daily range (minimum and maximum) of ZIP code population-weighted $PM_{2.5}$ concentrations for each Washington Department of Ecology Regional Office Jurisdiction. To visualize the spatial extent of the smoke exposure, we mapped counts of days impacted by smoke for each Washington ZIP code from 1 July to 31 October 2012, for which daily GWR $PM_{2.5}$ concentration exceeding $10 \mu\text{g}/\text{m}^3$ within each ZIP code indicated a smoke day. GWR was chosen for this map as Lassman et al. found this method to be the most accurate compared to WRF-Chem or kriging [Lassman et al., 2017].

Total counts for emergency department or urgent care hospital admissions for each outcome that took place in Washington from 1 July to 31 October 2012 were calculated; proportions for each outcome-specific age and sex strata were calculated as well. We did not report age strata with less than 15 cases due to internal data protocols when using protected health information. Conditional logistic regression models with patient-specific strata were used to calculate the odds ratio (OR); the health outcome of interest was regressed on a continuous estimate of smoke $PM_{2.5}$ (derived from the three smoke estimation methods) while adjusting for temperature, relative humidity, wind speed, and precipitation. Additional analyses were performed to evaluate lagged exposure days and effect modification by age and sex. For these analyses, we only compared the WRF-Chem smoke method to GWR smoke method so that the results were easier to view and interpret, and as Lassman et al. found kriging and GWR methods were similar in performance [Lassman et al., 2017]. Lagged analyses considered the time period from 0 to 5 days prior to admission. Analyses for age as a potential modifying factor in these associations were conducted for the following age groups: <15 years (some outcomes were not analyzed as there were less than 15 cases for this age group), 15 to 65 years, and >65 years; sex was also evaluated as a potential modifying factor.

All data analyses were performed using R software [R Core Team, 2016]. Spatial-related functions used the “sp” [Pebesma and Bivand, 2005], “rgdal” [Bivand et al., 2016], and “rgeos” [Bivand and Rundel, 2016] packages. Maps were created using the “ggmap” package [Kahle and Wickham, 2013]. Plots were created using the “ggplot2” package [Wickham, 2009, p.2]. Conditional logistic regression models were implemented using the “survival” package [Terry M. Therneau, 2015]. R code used for this study is available at the project GitHub repository (https://github.com/RyanGan/washington_wildfire_2012).

All study procedures were approved by the Institutional Review Boards of Colorado State University and Washington State Institutional Review Board and Department of Health.

2.5. Data Availability

Smoke estimate and meteorological data sets can be found on the repository for our companion paper by Lassman et al. [2017] at <http://hdl.handle.net/10217/179811>. The Health Insurance Portability and Accountability Act precludes distribution of the health outcome data used in this analysis.

3. Results

3.1. Descriptive Results for Smoke and Hospital Admissions

Figure 1 shows the range of ZIP code population-weighted $PM_{2.5}$ concentrations of smoke for each estimation method by Department of Ecology Region from 1 July to 31 October 2012. In general, all time series plots showed a large increase in maximum smoke $PM_{2.5}$ starting in mid-September and extending through early-October. The central and eastern regions had the highest concentrations of smoke $PM_{2.5}$, but the northwestern and southwestern regions also had elevated concentrations of smoke $PM_{2.5}$. The maximum WRF-Chem smoke $PM_{2.5}$ concentrations were also much greater at similar time points when compared to kriged and GWR smoke $PM_{2.5}$ concentrations. On multiple occasions, WRF-Chem estimated elevated smoke concentrations for the eastern and southwestern regions that were not elevated for the kriged smoke or GWR smoke time series.

Figure 2 shows the number of days in Washington ZIP codes that were impacted by smoke from 1 July to 31 October 2012. Central and eastern Washington had the highest number of days impacted by smoke during this time, consistent geographically with the time series ranges in Figure 1 and the prevailing wind patterns in relation to the fire locations.

For all of 2012, there were a total of 730,970 hospital admission records in the CHARS data set. During the 1 July to 31 October 2012 wildfire season in Washington state, there was a total 248,647 admissions, of which 26,835 were hospital emergency departments or urgent care for the specified cardiopulmonary codes. The number of persons (cases), as well as age-specific and sex-specific strata, that needed admission to an emergency department or urgent care with a cardiopulmonary primary diagnosis in Washington state from 1 July to 31 October 2012 is presented in Table 1.

3.2. Wildfire Smoke and Hospital Admission Results

Our main results are presented in Figure 3. A 10 $\mu\text{g}/\text{m}^3$ increase in smoke $\text{PM}_{2.5}$ on the same day of admission was significantly associated with an increase in hospital admissions for all respiratory outcomes using our three smoke estimation methods: WRF-Chem smoke (OR: 1.024, 95% confidence interval (CI): 1.002–1.046), kriging smoke (OR: 1.066, 95% CI: 1.033–1.100), and GWR smoke (OR: 1.052, 95% CI: 1.025–1.080) (Figure 3). Similarly, a 10 $\mu\text{g}/\text{m}^3$ increase in smoke $\text{PM}_{2.5}$ was significantly associated with an increase in asthma hospitalizations: WRF-Chem smoke (OR: 1.100, 95% CI: 1.059–1.142), kriging smoke (OR: 1.086, 95% CI: 1.016–1.161), and GWR smoke (OR: 1.076, 95% CI: 1.019–1.136) (Figure 3). However, the three smoke-estimation methods did not produce consistent results for other health outcomes. For COPD, no significant association was observed with WRF-Chem smoke (OR: 0.986, 95% CI: 0.931–1.045), but significant associations were observed with kriging (OR: 1.106, 95% CI: 0.931–1.045), and GWR (OR: 1.084, 95% CI: 1.026–1.145) methods (Figure 3). For pneumonia, no significant association was observed with WRF-Chem smoke (OR: 1.019, 95% CI: 0.980–1.059) and GWR smoke (OR: 1.051, 9% CI: 0.999–1.105) methods, but significant associations were observed with kriging smoke estimates (OR: 1.069, 95% CI: 1.009–1.133). Conversely, a significant association was observed between the WRF-Chem smoke method and cerebrovascular disease hospital admissions (OR: 1.037, 95% CI: 1.006–1.069) that was not observed with kriging smoke (OR: 1.009, 95% CI: 0.959–1.061) or GWR smoke (OR: 1.011, 95% CI: 0.969–1.054) methods (Figure 3); no association was observed between estimates of smoke $\text{PM}_{2.5}$ and other cardiovascular outcomes or broken arm (Figure 3).

3.3. Lagged Smoke Exposure

Model results for lag effects (0–5; 0 = same day, 5 = 5 days following exposure) for associations between smoke $\text{PM}_{2.5}$ and cardiopulmonary hospital admissions are presented in Figure 4 for both WRF-Chem and GWR smoke estimation methods. GWR smoke estimates were consistently associated with increases in “all respiratory” admissions across all six lag days (0 = OR: 1.052, 95% CI: 1.025–1.080; 1 = OR: 1.043, 95% CI: 1.014–1.072; 2 = OR: 1.042, 95% CI: 1.013–1.071; 3 = OR: 1.051, 95% CI: 1.025–1.078; 4 = OR: 1.038, 95% CI: 1.011–1.065; and 5 = OR: 1.030, 95% CI: 1.003–1.058). Similar estimates made using WRF-Chem smoke were associated with respiratory admissions on four of the six lag days: 0 (OR: 1.024, 95% CI: 1.002–1.046), 1 (OR: 1.023, 95% CI: 1.002–1.044), 4 (OR: 1.026, 95% CI: 1.004–1.047), and 5 (OR: 1.022, 95% CI: 1.001–1.045) (Figure 4). For asthma-only admissions, GWR smoke was significantly associated on days 0 (OR: 1.076, 95% CI: 1.019–1.136), 2 (OR: 1.068, 95% CI: 1.026–1.113), 3 (OR: 1.080, 95% CI: 1.021–1.142), 4 (OR: 1.058, 95% CI: 1.002–1.117), and 5 (OR: 1.080, 95% CI: 1.019–1.144), where WRF-Chem smoke was associated on all lagged days (0 = OR: 1.100, 95% CI: 1.059–1.142; 1 = OR: 1.073, 95% CI: 1.035–1.113; 2 = OR: 1.061, 95% CI: 1.020–1.103; 3 = OR: 1.059, 95% CI: 1.019–1.100; 4 = OR: 1.066, 95% CI: 1.026–1.109; and 5 = OR: 1.068, 95% CI: 1.026–1.113) (Figure 4). For COPD admissions, GWR smoke was associated on lagged days 0 (OR: 1.084, 95% CI: 1.026–1.145), 1 (OR: 1.068, 95% CI: 1.008–1.132), and 4 (OR: 1.070, 95% CI: 1.010–1.134), where WRF-Chem smoke was not associated on any lagged days (Figure 4). For pneumonia admissions, GWR smoke was significantly associated on lagged day 3 (OR: 1.060, 95% CI: 1.016–1.106), where WRF-Chem smoke was not associated on

any lagged days (Figure 4). For GWR smoke a significant increased risk in arrhythmia admissions was observed on lagged days 1 (OR: 1.045, 95%CI: 1.000–1.091), 2 (OR: 1.046, 95%CI: 1.000–1.093), and 3 (OR: 1.044, 95%CI: 1.004–1.085), where for WRF-Chem smoke no association was observed (Figure 4). For the remaining outcomes, no significant associations with lagged smoke PM_{2.5} were observed, including broken arm (Figure 4).

3.4. Stratified Estimates/Effect Modification

The association between increasing smoke PM_{2.5} estimated using WRF-Chem smoke and GWR smoke for cardiopulmonary hospital admissions stratified by age categories is presented in Figure 5. We did not estimate associations for the outcomes of COPD, arrhythmia, cerebrovascular disease, heart failure, ischemic heart disease, and myocardial infarction for the <15 age category as there were under 15 cases for this study period, which we suppressed due to data protocols for using protected health information. For the outcome of all respiratory admissions, GWR smoke was associated in the <15 age category (OR: 1.069, 95%CI: 1.001–1.141) and >65 age category (OR: 1.057, 95%CI: 1.018–1.097), where WRF-Chem smoke was associated only in the <15 age category (OR: 1.057, 95%CI: 1.009–1.109) (Figure 5). For asthma admissions, GWR smoke was significantly associated with PM_{2.5} only within the >65 age category (OR: 1.173, 95%CI: 1.003–1.370), where WRF-Chem smoke was associated for all three age categories (<15 = OR: 1.113, 95%CI: 1.048–1.182; 15–65 = OR: 1.095, 95%CI: 1.031–1.163; and >65 = OR: 1.094, 95%CI: 1.007–1.188) (Figure 5). For COPD, the association with smoke PM_{2.5} was significant only within the age category >65 (OR: 1.061, 95%CI: 1.029–1.160) (Figure 5).

The association between increasing smoke PM_{2.5} estimated using WRF-Chem smoke and GWR smoke and cardiopulmonary hospital admissions for sex strata is presented in Figure 6. For the outcome of all respiratory admissions, the association with GWR smoke was similar between males (OR: 1.055, 95%CI: 1.018–1.094) and females (OR: 1.048, 95%CI: 1.008–1.090), where WRF-Chem smoke was not associated in either sex strata (Figure 6). For asthma admissions, GWR smoke was not associated in either sex strata, while WRF-Chem smoke was associated and was similar between males (OR: 1.113, 95%CI: 1.052–1.179) and females (OR: 1.090, 95%CI: 1.036–1.46) (Figure 6). For COPD admissions, associations with GWR smoke were observed in the male strata (OR: 1.102, 95%CI: 1.026–1.182) but not the female strata (OR: 1.056, 95%CI: 0.966–1.155); WRF-Chem smoke was not associated with COPD admissions in either sex strata (Figure 6). For acute bronchitis admissions, associations with GWR smoke were observed in the male strata (OR: 1.340, 95%CI: 1.061–1.692) but not the female strata (OR: 0.714, 95%CI: 0.475–1.073); WRF-Chem smoke was not associated with acute bronchitis admissions in either sex strata (Figure 6). For cardiovascular disease admissions, associations in males were observed for WRF-Chem smoke (OR: 1.023, 95%CI: 1.000–1.046) and GWR smoke (OR: 1.029, 95%CI: 1.000–1.059) was associated; no such associations were observed in females (Figure 6). WRF-Chem smoke was associated with cerebrovascular disease admissions in males (OR: 1.046, 95%CI: 1.004–1.090) (Figure 6).

4. Discussion

Our results indicate significant positive associations between increased PM_{2.5} concentrations attributable to wildfire smoke and risk of hospital admissions for asthma, and COPD, and the all respiratory outcome category. Our findings were robust for asthma and all respiratory outcomes for all three methods of estimating smoke exposure; these findings are consistent with other reports of adverse respiratory outcomes following wildfire smoke exposure, regardless of the smoke exposure estimation method used [Johnston et al., 2007; Henderson et al., 2011]. Likewise, no associations were found between broken arm hospital admissions and the three methods of estimating smoke exposure, as expected. However, differing results were observed for COPD admissions, where we did not see an association with WRF-Chem smoke method but saw an increased risk using the kriging smoke and GWR smoke method. As for cardiovascular outcomes, we did not find any significant associations with wildfire smoke exposure, with the following exception: we observed a significant association between smoke estimated using the WRF-Chem smoke method and cerebrovascular disease that was not observed for the other smoke estimation methods, suggesting a spurious finding.

One possibility that may explain our differing results could be our methods for attempting to parse out PM_{2.5} from wildfire smoke versus other sources. For WRF-Chem smoke, we attempted to estimate background PM_{2.5} by running the simulation without the fire emissions option. However, for the kriging and GWR smoke methods, we estimated background PM_{2.5} concentrations by kriging the surface monitoring data on non-smoky days as identified by the NOAA HMS. We believe our attempt to parse out PM_{2.5} due to wildfire smoke versus other sources did not overly influence our results as we treated smoke as a continuous variable in our conditional logistic regression models, where the estimated OR was likely driven by the higher PM_{2.5} values due to wildfire smoke. Other studies have attempted to distinguish smoke PM_{2.5} by creating a binary smoke variable, such as when there were >2 days above varying PM_{2.5} thresholds [Liu et al., 2016b], or through time period restrictions known to be impacted by smoke [Alman et al., 2016]. Our methods allowed us to estimate smoke PM_{2.5} over a longer time period, thereby increasing our outcome sample size. Our methods also retained the information of continuous values while distinguishing smoke versus nonsmoke contributions to PM_{2.5}, rather than creating binary smoke exposures that can result in a loss of information regarding any concentration-response relationship.

Exposure misclassification may also explain the differences in odds ratios associated with our three exposure methods. In our complement paper, WRF-Chem simulations were less precise and less accurate at estimating surface measurements of PM_{2.5} (estimated slope of fitted trend line = 0.67, $R^2 = 0.25$, mean absolute error = 11.45 $\mu\text{g}/\text{m}^3$, and mean bias = 0.22 $\mu\text{g}/\text{m}^3$) compared to both kriging (estimated slope of fitted trend line = 0.70, $R^2 = 0.69$, mean absolute error = 2.09 $\mu\text{g}/\text{m}^3$, and mean bias = 0.00 $\mu\text{g}/\text{m}^3$) and GWR estimates (estimated slope of fitted trend line = 0.78, $R^2 = 0.66$, mean absolute error = 2.40 $\mu\text{g}/\text{m}^3$, and mean bias = 0.37 $\mu\text{g}/\text{m}^3$) [Lassman et al., 2017]. Our time series results (Figure 1) also indicated that WRF-Chem smoke tended to overestimate surface-level concentrations as compared to both kriging smoke and GWR smoke methods. WRF-Chem smoke also

indicated smoke exposure in late July for eastern Washington that was not corroborated by the kriging smoke or GWR smoke methods (Figure 1). Both the tendency to estimate higher concentrations of PM_{2.5} and estimating possible smoke exposure when there was none is a strong indication that WRF-Chem estimates lead to exposure misclassification to a greater degree than the other two methods.

The kriging and GWR methods were also subject to exposure misclassification, but to a lesser degree as they provided better predictions of observed results at surface monitors [Lassman et al., 2017]. These two methods also produced relatively similar consistent associations with the various cardiopulmonary outcomes studied herein. This is likely because the kriging estimates of PM_{2.5} performed well in Washington state during the 2012 wildfire season, as additional in situ monitors were deployed by Washington Department of Ecology to provide better spatial coverage of PM_{2.5} concentrations over this period [Lassman et al., 2017]. Because of the extensive spatial coverage of in situ monitors, the kriging and GWR estimates produced essentially the same results as the kriging estimates carried the most weight in the GWR models that used information from the kriging in situ estimates, WRF-Chem model estimates, and satellite AOD [Lassman et al., 2017].

Other studies of wildfire smoke exposure and cardiopulmonary health outcomes have also reported conflicting results [Liu et al., 2015; Reid et al., 2016], and these inconsistencies may be the result of varying methods of exposure assessment. For instance, in an evaluation of counts of hospitalizations in North Carolina, USA, Rappold et al. assigned county-level smoke exposure using satellite aerosol optical depth and found no association when evaluating all cardiovascular hospitalizations [Rappold et al., 2011]. However, when specified to heart failure Rappold et al. found a significant increase in hospitalizations associated with smoke exposure [Rappold et al., 2011]. Likewise, in a time-stratified case-crossover study using a ground-based monitors Johnston et al. found no association between increases (in PM₁₀) and cardiovascular hospitalizations during a smoke event [Johnston et al., 2007]. However, in the same study, they reported that increases in PM₁₀ concentrations 3 days prior significantly increased indigenous persons' risk for ischemic heart disease (IHD) hospitalizations but same-day increases in PM₁₀ significantly decreased risk for hospitalizations in nonindigenous persons [Johnston et al., 2007]. In a more recent time-stratified case-crossover study using a chemical-weather model, Haikerwal et al. found increases in both out-of-hospital cardiac arrests and IHD hospitalizations in persons < 65 years of age with increasing concentrations of PM_{2.5} [Haikerwal et al., 2015]. When stratified by sex, Haikerwal et al. found that only males had an increased risk of out-of-hospital cardiac arrests and only females had an increased risk in IHD [Haikerwal et al., 2015]. In a study that used surface monitors, an atmospheric dispersion model, and satellites, varying effects were found for cardiovascular physician visits, where persons age 40 to 50 were more likely to have an admission for increases in concentrations of PM₁₀ assessed using the in situ and dispersion model, but not by satellite [Henderson et al., 2011]. Like other studies, exposure misclassification and conflicting results in our study were not limited to cardiovascular outcomes, speaking to the larger problem of exposure misclassification in general.

An additional source of exposure misclassification is our use of reported ZIP code of residence to assign smoke exposure. This type of exposure misclassification is inherent in air pollution epidemiology based on retrospective admissions or billing data, in which ecologic-level exposure (i.e., reported ZIP code of residence) is used as a proxy for individual-level exposure (i.e., personal PM monitor). However, it is unlikely that this type of exposure misclassification was the main driving factor behind our results, as the case-crossover design compared within-subject variability of exposure.

Exposure misclassification may persist when relying on reported ZIP code of residence as our finest spatial resolution. We were unable to obtain finer resolution of residential location such as address, which is often the case when using hospitalization records. We addressed this issue by population-weighting the average daily PM_{2.5} in each ZIP code to reduced the impact of exposure misclassification. This method likely had the most benefit in the spatially larger ZIP codes in rural areas with sparse populations, which were also most impacted by wildfire smoke. Although this method of population-weighting the average exposure estimate within a spatial boundary likely reduces exposure misclassification, future work based on reported residential address will likely provide the most informative and unbiased estimate of the true association between wildfire smoke and cardiopulmonary outcomes. Further refinements may also consider the location of the admitting hospital in exposure assignment, as most patients are likely admitted to the closest hospital when seeking emergency or urgent medical care.

With regard to effect modification, there were some differences in associations with smoke exposure and asthma hospital admissions depending on age strata. We observed a significant association between asthma and GWR smoke for >65 categories. Our results are consistent with other literature that suggests that older persons may be more susceptible to adverse health outcomes due to inhaled exposures [Gouveia, 2000; Pope et al., 2002; Kan et al., 2008]. However, for the same outcome, WRF-Chem smoke was associated across all age categories. Furthermore, some of our null results could be the result of small sample sizes and not necessarily due to an age effect. We also found some evidence of effect modification by sex, particularly for smoke exposure on the outcome of cardiovascular disease in males as both WRF-Chem smoke and GWR smoke produced similar estimates. However, we suggest a cautious interpretation for our stratified results, as they were not always consistent between smoke estimation methods. Furthermore, there were trends of an increased risk of a broken arm admission in females for both smoke estimation methods that raise some concerns of the validity of our effect modification results. Given that both estimation methods showed this increased risk, it suggests that some other underlying factors may explain these results, such as the selection of our referent days. Likewise, our evaluation of lagged periods of smoke exposure produces some differing results depending on smoke estimation method used. The most interesting pattern observed for the lag periods was with arrhythmia, where the GWR smoke method suggested that perhaps there was an increased risk of hospitalization a couple days after exposure to smoke, while the WRF-Chem smoke method showed null associations. However, like our assessments of age and sex modification we advise a cautious interpretation of these results. Further evaluation of lagged effects and effect modification by age and sex on the relationship between smoke exposure and

cardiopulmonary morbidity in multiple studies with larger sample sizes will be necessary to draw any definitive conclusions.

The use of hospital admission claim data has several limitations, including misclassification of outcomes. In the case of our work, ICD-9-CM diagnosis codes were used as the primary way to identify proxy measures of the health outcome of interest. However, ICD-9 codes were originally designed for billing purposes. Therefore, their use as accurate proxy measures for health outcomes depends on many factors at the patient, provider, and health care system level [O'Malley et al., 2005]. Our decision to limit our case definition to only to the primary ICD-9 code reduced our sample size of each cardiopulmonary condition but should also have reduced our misclassification of cases [O'Malley et al., 2005]. Additionally, we also limited our case definition to include only emergency department or urgent care admissions to eliminate patients who sought elective care, which likely resulted in an underestimation of the true relationship between wildfire smoke and adverse cardiopulmonary events. Despite these limitations, the strength of administrative claim data in the study of environmental exposures that affect sparsely populated area over a short window of time (i.e., wildfire smoke) is that these data provide enough events for sufficiently powered analysis.

Another limitation of using administrative data is the lack of information on potential confounding factors. However, our use of a time-stratified case-crossover design controlled for subject-specific confounding factors that do not change over short periods of time or at all (e.g., smoking, comorbidities, sex, and age); thus, our cases serve as their own control. Further, the time-stratified design has benefits relative to other case-crossover referent selection strategies in that time-varying confounding effects such as day of the week or seasonal trends in $PM_{2.5}$ are also accounted for in the design [Janes et al., 2005a, 2005b]. In general, case-crossover studies are most appropriate when used to evaluate associations between acute exposures (e.g., wildfire smoke) and acute outcomes (e.g., asthma), and their application with conditions that do not resolve (e.g., heart failure or mortality) may not be appropriate.

In addition, based on design, case-crossover studies create unobserved referent observations, assuming that the case did not have an event on those additional dates. The time-stratified case-crossover design reduces bias from both time-varying and time-invariant confounding compared to some other referent selection strategies such as symmetric-bidirectional [Lumley and Levy, 2000; Janes et al., 2005a, 2005b]. However, the time period for which the referent periods are to represent must still be selected in a way that balances two main considerations: (1) minimizing bias from time-varying confounding (i.e., seasonal effects), which can be achieved by selecting referent periods closer in time to the index date [Janes et al., 2005b], and (2) considering if the number of referent periods is adequate to capture the heterogeneity of the exposure while maintaining a sufficiently powered sample size [Janes et al., 2005b]. These two considerations played an important role in our selection of referent periods, as smoke $PM_{2.5}$ concentrations varied greatly in time where extremely high concentrations were observed for a week or more. In an effort to average out these extreme periods so that they are more representative of normal $PM_{2.5}$ concentrations, we used a referent period selection strategy that included each same-day period as the index date over

the entire wildfire season from 1 July to 31 October. This strategy also provided sufficient statistical power.

Another limitation when using case-crossover designs is accounting for patients that may have multiple admissions within the referent time period. In this analysis, we excluded patients with multiple admissions (indicated by the patient identifier variable) for the same outcome over the wildfire season. In a case-crossover analysis that uses conditional logistic regression, patients with multiple admissions violate regression model assumptions that there is only one event for each case [Janes et al., 2005a]. However, we may have also eliminated those with the highest likelihood of morbidity, indicated by multiple emergency or urgent encounters with the health care system. Future work will address how to incorporate multiple observations per patient in the case-crossover design.

The case-crossover design is best suited for studying the relationship between a short-term exposure and acute outcome that resolves, which explains why our study and others consistently observe associations with asthma or other acute events. However, given our varying associations between smoke and cardiovascular outcomes in our study and others, it remains unclear whether smoke has any short-term effect on cardiovascular hospital admissions. The effects of smoke on cardiovascular disease may produce long-term effects. However, time-stratified case-crossover or time series designs are not well suited for this application.

To date, evaluation of the health effects due to wildfire smoke that use a continuous concentration of PM_{2.5} has generally assumed a linear relationship. The relationship between ambient anthropogenic levels of air pollution PM and adverse health outcomes generally exhibits a linear exposure concentration-response relationship, where risk for adverse health events increases linearly as concentrations of PM increase [Daniels et al., 2000; Dominici et al., 2002, 2005; Pope et al., 2002, 2009; Samoli et al., 2004]. This linear relationship has been assumed in current studies on wildfire smoke, including herein. However, there is evidence that suggests a nonlinear relationship between adverse health outcomes and inhaled particulate matter, where the concentration-response is relatively steep at low levels of PM but begins to level out at higher levels [Pope et al., 2009]. This nonlinear response could be relevant for wildfire smoke, as concentrations, while not as high as inhaled cigarette smoke, are still much higher than background levels of PM. Future work should determine the shape of the concentration-response function for cardiopulmonary outcomes, and if the response differs depending on the pollution source (wildfire smoke versus anthropogenic).

We found a significant relationship between exposure to wildfire smoke PM_{2.5} and an increase in the risk for hospital admission for the pulmonary outcomes asthma, COPD, and pneumonia. Our results also suggest that a blended estimate of smoke using information from surface monitors, satellites, and chemical-weather models is a robust approach in quantifying population exposure to wildfire smoke that will be useful in reducing exposure misclassification for epidemiologic studies. From a public health perspective, our results contribute further evidence linking wildfire smoke to adverse health outcomes to the existing literature. Wildfires place a significant burden on states from a disaster preparedness

standpoint where the initial wildfire can be destructive to property and dangerous to human life. However, from a broader public health standpoint, there is also potential for population-level exposure that could lead to increased strain on the health care system due to the resulting smoke. Our work and the work of others can help guide the planning and preparation of health care systems and public health agencies during future wildfire events.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Key Points

- Geographically weighted regression combines measures of wildfire smoke from many sources that can be used in epidemiologic studies
- Wildfire smoke estimated using geographically weighted regression was associated with increased risk for respiratory outcomes
- Geographically weighted regression is a useful approach that can reduce exposure misclassification in epidemiologic studies

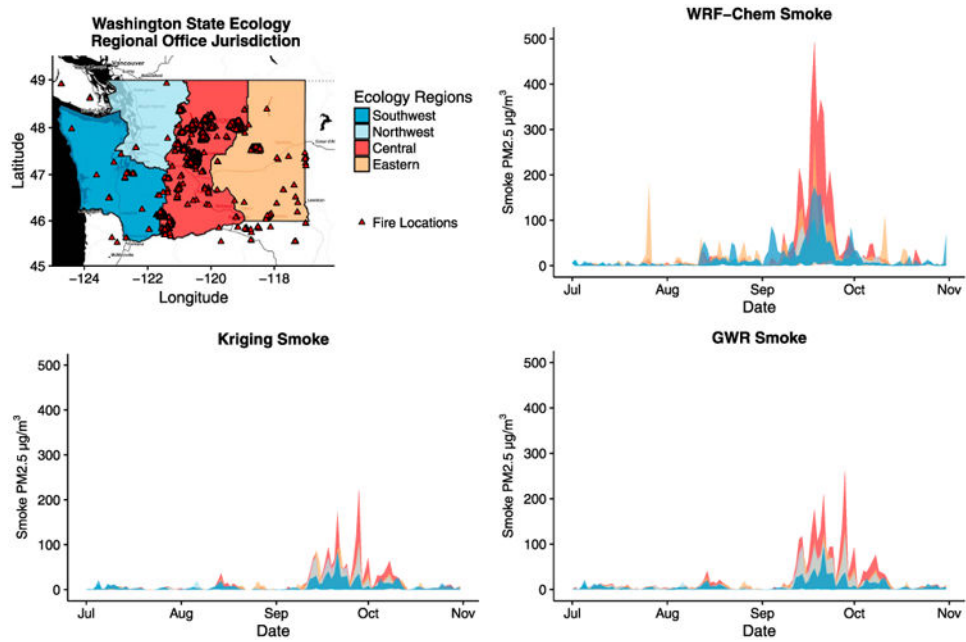


Figure 1. Time series of the range (minimum to maximum) of ZIP code population-weighted PM_{2.5} concentrations of wildfire smoke from 1 July to 31 October 2012 for WRF-Chem smoke, kriging smoke, and geographically weighted ridge regression (GWR) smoke by ecology region.

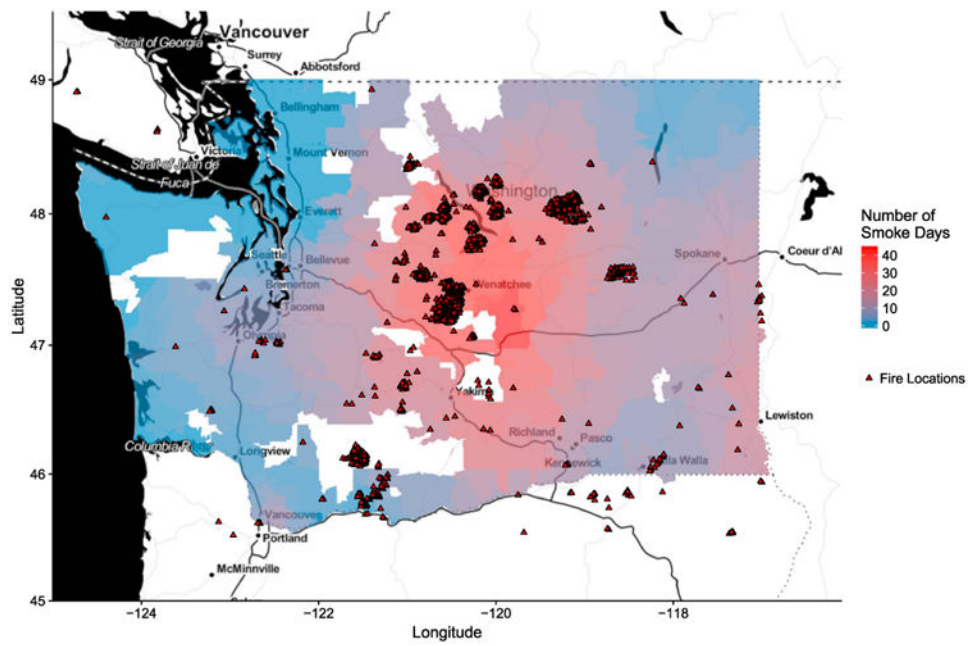


Figure 2. Counts of days in each ZIP code impacted by smoke in Washington from 1 July to 31 October 2012. A smoke day is any day where GWR smoke population-weighted $PM_{2.5}$ was $>10 \mu\text{g}/\text{m}^3$. Smoke counts were not estimated for some areas on the map (indicated in white) as there were no events reported at these ZIP codes.

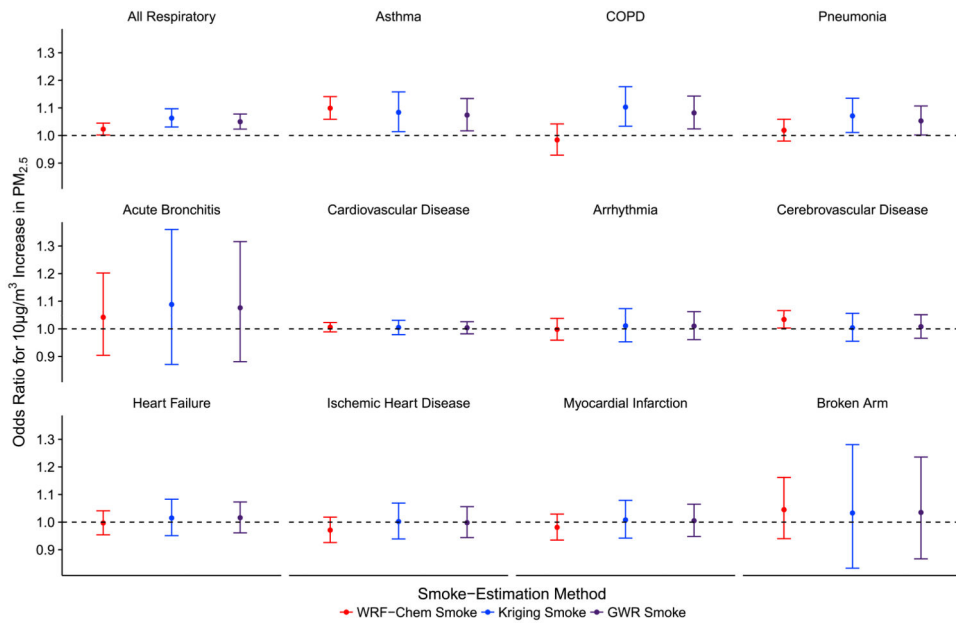


Figure 3. Association between a 10 $\mu\text{g}/\text{m}^3$ increase in smoke $\text{PM}_{2.5}$ (using three estimation methods) and risk for a cardiopulmonary emergency department or urgent care hospital admission, adjusting for temperature, relative humidity, wind speed, and precipitation.

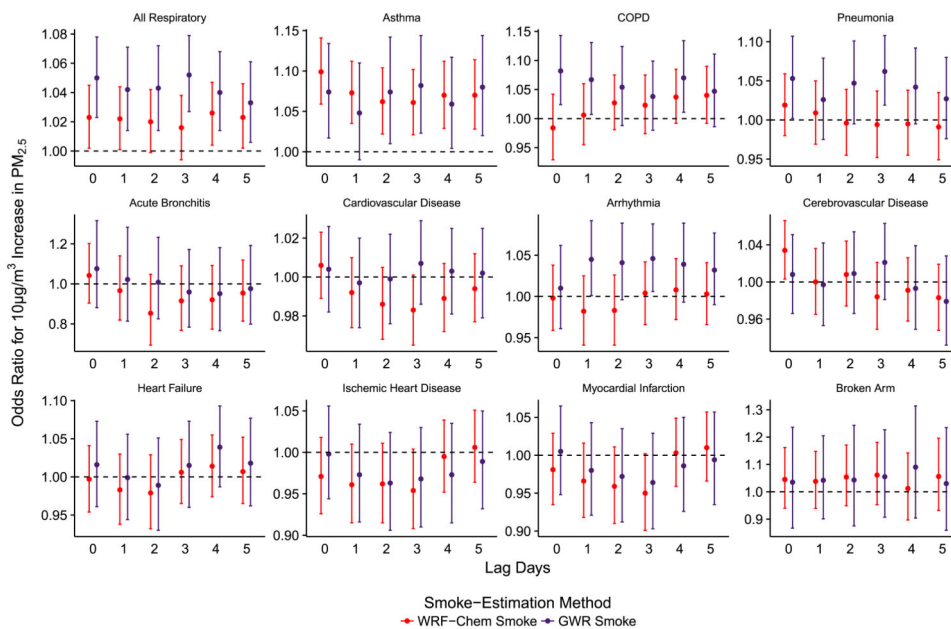


Figure 4. Lag analysis (0–5 days) for associations between a 10 $\mu\text{g}/\text{m}^3$ increase in smoke $\text{PM}_{2.5}$ (using either WRF-Chem smoke or GWR smoke) and risk for a cardiopulmonary emergency department or urgent care hospital admissions.

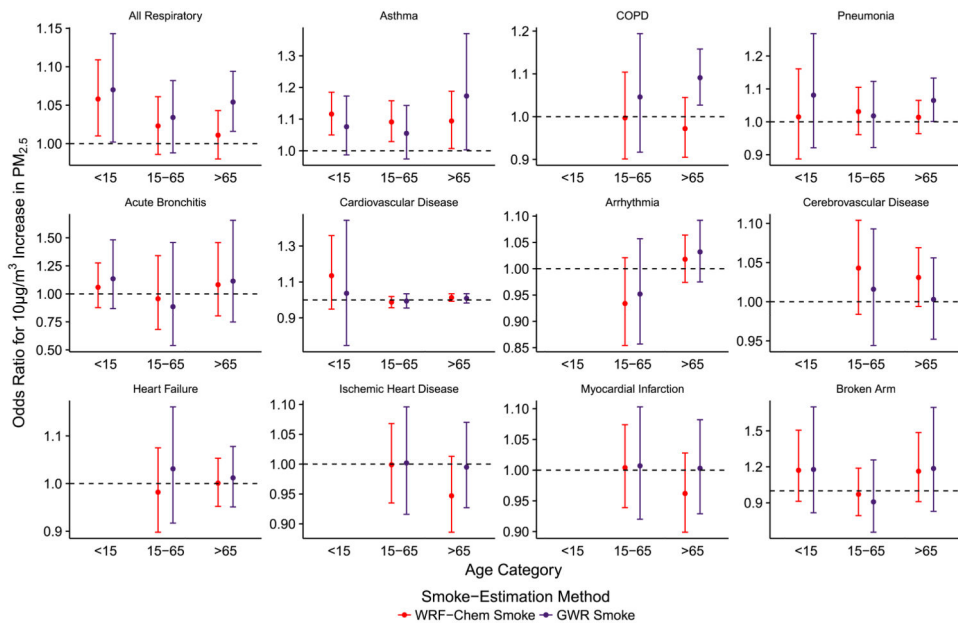


Figure 5. Age-stratified association between a 10 µg/m³ increase in WRF-Chem smoke and GWR smoke PM_{2.5} and risk for a cardiopulmonary emergency department or urgent care hospital admission, adjusting for temperature, relative humidity, wind speed, and precipitation.

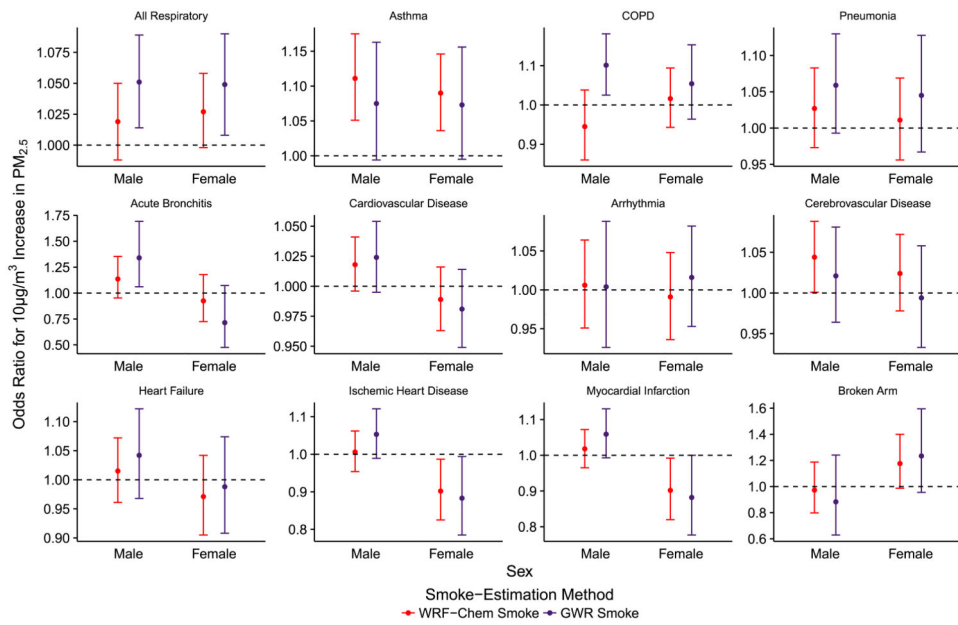


Figure 6. Sex-stratified association between a 10 $\mu\text{g}/\text{m}^3$ increase in WRF-Chem smoke and GWR smoke $\text{PM}_{2.5}$ and risk for a cardiopulmonary emergency department or urgent care hospital admission, adjusting for temperature, relative humidity, wind speed, and precipitation.

Table 1
Number of Cases for Each Primary Diagnosis of Cardiopulmonary Outcomes in Washington State From 1 July to 31 October 2012^a

Health Outcome	Cases (n)	Age Category			Sex	
		<15 (%)	15 to 65 (%)	>65 (%)	Female (%)	Male (%)
All respiratory	9,657	13.5	36.6	49.9	51.9	48.1
Asthma	1,456	44.7	41.5	13.8	55.5	44.5
COPD	1,700	--	39.5	60.5	57.3	42.7
Pneumonia	3,165	7.5	33.3	59.2	51.7	48.3
Acute bronchitis	289	48.4	25.6	26.0	50.2	49.6
Cardiovascular disease	17,178	0.4	36.4	63.2	47.9	52.1
Arrhythmia	3,238	--	30.7	69.3	50.4	49.6
Cerebrovascular disease	4,208	--	30.5	69.5	52.5	47.5
Heart failure	2,975	--	26.2	73.8	48.8	51.2
Ischemic heart disease	2,920	--	41.4	58.6	38.7	61.3
Myocardial infarction	2,689	--	41.3	58.7	37.3	62.7
Broken arm	318	15.7	52.8	31.4	45.9	54.1

^a Additional columns contain the row percent by age and sex categories for the outcome. Some cell sizes for the age category <15 had less than 15 persons. Per data protocol, these values were suppressed and indicated by "--"; the percentage was added to one of the other age categories so that these values could not be inferred.