

1 Peat Bog Wildfire Smoke Exposure in Rural North Carolina Is Associated with Cardio-
2 Pulmonary Emergency Department Visits Assessed Through Syndromic Surveillance

3
4 Ana G. Rappold¹, Susan L. Stone¹, Wayne E. Cascio¹, Lucas M. Neas¹, Vasu J. Kilaru², Martha
5 Sue Carraway¹, James J. Szykman³, Amy Ising⁴, William E. Cleve⁵, John T. Meredith⁶, Heather
6 Vaughan-Batten,⁷ Lana Deyneka⁷, and Robert B. Devlin¹

7
8 Authors' Affiliations:

9 1. Environmental Public Health Division, National Health and Environmental Effects

10 Research Laboratory, United States Environmental Protection Agency, Research Triangle
11 Park, North Carolina

12 2. National Exposure Research Laboratory, United States Environmental Protection Agency,
13 Research Triangle Park, North Carolina

14 3. Environmental Sciences Division, National Exposure Research Laboratory, US
15 Environmental Protection Agency, c/o NASA Langley Research Center, Hampton, VA
16 23681.

17 4. Department of Emergency Medicine, School of Medicine, University of North Carolina at
18 Chapel Hill

19 5. Pitt County Memorial Hospital, Greenville, North Carolina

20
21 6. Brody School of Medicine at East Carolina University, Department of Cardiovascular
22 Sciences and the East Carolina Heart Institute, Greenville, North Carolina

23 7. NC Division of Public Health, NC Division of Health and Human Services

24 Corresponding Author:

25 Ana G. Rappold
26 MD 58B
27 109 TW Alexander Drive,
28 Research Triangle Park, NC 27711
29 (919) 843 9504 office
30 (919) 966 6367 fax
31 Rappold.ana@epa.gov

32

33 *Running Title:*

34 | Peat wildfire smoke and emergency visits

35 *Key Words:*

36 Cardio-Pulmonary Health Effects, Satellite Data, Syndromic Surveillance, Wildfire Smoke
37 Exposure

38 *Financial Interest Declaration:*

39 This work was supported by internal funding by US Environmental Protection Agency. The
40 authors declare they have no competing financial interests.

41

42 **Abbreviations:**

43 CI: Confidence Interval

44 AOD: Aerosol Optical Depth

45 AQI: Air Quality Index

46 ICD-9-CM Codes: International Statistical Classification of Diseases and Related Health
47 Problems

48 ED: Emergency Department

49 COPD: Chronic Obstructive Pulmonary Disease

50 URIs: Upper Respiratory tract Infections

51 RR: Relative Risk

52 PM2.5: Particulate matter with diameter of 2.5 micrometers and smaller

53

54
55
56
57
58
59
60
61
62
63
64
65
66
67
68
69
70
71
72
73
74
75
76
77

Abstract

Background: In June 2008 burning deposits of peat produced haze and air pollution far in excess of National Ambient Air Quality Standards, encroaching on rural communities of eastern North Carolina (NC). While the association of mortality and morbidity with exposure to urban air pollution is well established, the health effects associated with exposure to wildfire emissions are less understood.

Objective: To determine the effects of exposure on cardio-respiratory outcomes in the population affected by the fire.

Methods: A population-based study was performed using Emergency Department (ED) visits reported through the syndromic surveillance program NCDETECT. Aerosol optical depth measured by a satellite was used to determine a high-exposure window and distinguish counties most impacted by the dense smoke plume from surrounding reference counties. Poisson log-linear regression with a five day distributed lag was used to estimate changes in the cumulative relative risk (RR).

Results: In the exposed counties significant increases in cumulative RR for asthma (1.65(95% confidence interval [1.25, 2.17]), COPD (1.73[1.06, 2.83]), pneumonia and acute bronchitis (1.59[1.07, 2.34]) were observed. ED visits associated with cardiopulmonary symptoms (1.23[1.06, 1.43]) and heart failure (1.37[1.01, 1.85]) were also significantly increased.

Conclusions: Satellite data and syndromic surveillance were combined to assess the health impacts of wildfire smoke in rural counties with sparse air quality monitoring. This is the first study to demonstrate both respiratory and cardiac effects following brief exposure to peat wildfire smoke.

78 **Introduction**

79 On June 1st, 2008 a lightning strike initiated a fire in the eastern plains of North Carolina
80 (NC). Low humidity and prolonged drought contributed to the spread of the fire into the Pocosin
81 Lakes National Wildlife Refuge where it smoldered through rich deposits of peat. Peat in the
82 refuge was on average 3ft and in places up to 15ft deep. Poor oxygen supply during combustion
83 of carbon in the decomposing vegetation produced massive amounts of smoke in the area. The
84 region exposed to the hazardous levels of air pollution generated by the fire was largely rural,
85 sparsely populated, and economically disadvantaged.

86 To investigate health effects associated with this fire, we obtained daily emergency
87 department (ED) visits for cardiac and respiratory conditions for eastern NC counties reported
88 through the state-wide syndromic surveillance system. Satellite measurements of aerosol optical
89 depth (AOD) were used to determine a 3 day window of dense plume. Relative risks associated
90 with these days and five lag days were estimated for the counties most impacted by the smoke
91 plume and the surrounding, less impacted, referent counties.

92 Air pollution is described as a complex mixture of gasses and particles whose toxicity
93 depends on the type of fuel and conditions of combustion. There are more than 100
94 epidemiological studies demonstrating mortality and morbidity associated with both chronic and
95 acute exposures to air pollution focusing mostly on emissions of burning fossil fuels, from
96 automobiles, diesel engines, and coal or oil fired power plants (Integrated Science Assessment
97 for Particulate Matter 2009). During the episodes of wildfires, affected regions experience acute
98 exposures, with concentrations orders of magnitude larger than observed in urban centers.
99 However, health implications of the exposure to wildfire emissions are less well understood
100 (Naeher et al. 2007). In contrast to health studies of urban air pollution, wildfire studies are
101 constrained by the size and distribution of the exposed population, duration of the episode, and

102 the retrospective accessibility of data on exposure and health outcomes. The most
103 comprehensive studies, conducted for forest fires near large metropolitan areas, reported
104 significant increases in symptoms and exacerbations of underlying respiratory illnesses (Jeffrey
105 et al. 2005; Delfino et al. 2009; Duclos P 1990; Kunzli et al. 2006). Less conclusive results have
106 been found within smaller cohort and convenience sample-based studies and less populated
107 regions (Moore 2006; Mott 2005). The effects of exposure on cardiovascular outcomes have
108 been reported with more varied results (Delfino et al. 2009; Moore 2006; Mott 2005; Sastry
109 2002; Vedal and Dutton 2006) in part due to lack of statistical power.

110 Climate related changes and past land use practices are expected to increase the risk of
111 wildfires in upcoming decades (Quadrennial Fire Review: Final Report 2009). In addition to the
112 forests, particular concerning is the vulnerability of large deposits of peat bogs to wildfires. Peat
113 soil accounts for approximately 2% of global land cover, mostly in the Boreal regions, where it
114 has been traditionally harvested for energy. In draught conditions peat bogs are exceptionally
115 susceptible to ignition, can smolder indefinitely, and are notoriously difficult to extinguish.
116 Although less common than forest fires, they have an important impact on regional climate and
117 ecosystems. In 1997, Indonesian peat fires released the equivalent of 13-40% of the mean
118 annual global carbon emissions from fossil fuels, and contributed to the largest annual increase
119 in atmospheric CO₂ in four decades (Page 2002). Despite their large impact on the environment,
120 significantly less is known about the associated health effects. One study of Indonesian fires
121 reported more than 500 haze-related deaths, 290,000 cases of asthma exacerbations, 58,000 cases
122 of bronchitis, and 1,440,000 cases of acute respiratory infection between September and
123 November 1997 (Kunii O et al. 2002).

124 **Methods**

125 Daily counts of ED visits were obtained from the NC Disease Event Tracking and
126 Epidemiologic Collection Tool (NCDETECT 2010), a statewide, early event detection and
127 public health surveillance system which records daily ED visits from 111 of 114 civilian NC
128 EDs. We considered visits for selected cardiovascular and respiratory outcomes by adults
129 throughout the eastern portion of the state, as well as county of residence, gender, age, date of
130 admission, and discharge ICD-9-CM codes for all visits. Prior to the analysis, outcomes of
131 interest were defined through ICD-9-CM codes for asthma [493], chronic obstructive pulmonary
132 disease [491 - 492], pneumonia and acute bronchitis [481, 482, 485, 486, and 466], upper
133 respiratory tract infections (URIs) [465], heart failure [428], cardiac dysrhythmia [427],
134 myocardial infarction [410, 411.1] and symptoms involving the respiratory system and other
135 chest symptoms [786]. For respiratory related outcomes the population was stratified into two
136 age groups: 19-64, and 65 and older. The small numbers of ED visits for respiratory-related
137 events in those younger than 19 precluded analysis of this subgroup. Cardiac outcomes were
138 also stratified into two age groups: 45-64 and 65 and older. Age of 45 was used to differentiate
139 ischemic heart disease related to atherosclerosis, from coronary artery spasm seen in a younger
140 population. The Human Subjects Institutional Review Board of the University of North Carolina
141 at Chapel Hill, East Carolina University, and the Environmental Protection Agency approved the
142 study.

143 The study period was defined as the time between the onset of the wildfire through mid-
144 July when controlled flooding, increased humidity, and the first rainfall largely contained the fire
145 (June 1st through July 14th, 2008). During this period, average daily temperatures ranged from
146 69 to 86°F with overnight lows always below 75°F (See Supplemental Material, Figure 1). For
147 most of the period, winds blew from the west and smoke affected only a few sparsely populated

148 neighboring counties. However, on June 10th easterly winds directed the smoke plume inland,
149 exposing a large portion of eastern and central NC for a three day period (Figure 1A-C). On
150 June 12th, the maximum one-hour concentration of fine particulate matter (PM_{2.5}) exceeded
151 200µg/m³ at ground-based monitors located 200 kilometers from the fire. We defined June 10th,
152 11th, and 12th as a window of high exposure and estimated risks associated with the exposure
153 days and one to five lag days relative to the non-exposed days of the six week study period. The
154 relative risks for lags 0 -5 was summarized to define cumulative risk of exposure.

155 County-level exposure to the plume was classified using AOD, measured by instruments
156 aboard a Geostationary Operational Environmental Satellite. AOD is a unit-less measure with
157 scale from 0 to 2 of the atmospheric scattering and absorption of light by aerosols where larger
158 values indicate higher concentration of particles in the atmosphere and lower visibility. Half-
159 hour, 4 x 4 km resolution grid maps of AOD were averaged over the available day time hours.
160 AOD has been shown to be a good surrogate for boundary layer fine particulate matter
161 concentrations, and a predictor of the Air Quality Index, a nationally uniform index for reporting
162 daily air quality (Al-Saadi et al. 2005; AQI 2009; Engel-Cox et al. 2004; Wang and Christopher
163 2003). Typical background levels of AOD for this region were well below 0.5. Based on the
164 sharp difference between the high density plume and background, we chose an AOD of 1.25 and
165 greater as an indicator of the high density plume. Counties with a minimum of 25% of the
166 geographic area exceeding this threshold were defined as exposed to the smoke plume for each
167 day in the high exposure window (Figure 1). The satellite's operational algorithm considers high
168 AOD values created by strong reflectance from clouds as unreliable and removes them from the
169 standard data product. The dense smoke plume was, at times, classified as a cloud, resulting in

170 missing AOD values on the interior of the plume. We considered such values as right censored
171 and classified the respective grid cells as exposed to the plume.

172 The study population resided in 42 contiguous counties in eastern NC. One sparsely
173 populated county (Gates County) was significantly impacted by another wildfire and was
174 excluded from this analysis. Counties with smoke exposure on at least two days were considered
175 exposed (18 counties in Figure 1D). The remaining 23 counties, exposed one day (15 counties)
176 or less (8 counties), were used as referent counties. The populations of exposed and referent
177 counties are similar with respect to age structure, ethnicity, population density, and
178 socioeconomic status. Counties in eastern NC are more rural and agricultural with a higher
179 percentage of African-Americans, and of lower socioeconomic status than most of the remaining
180 North Carolina counties. A table with demographic characteristics of the two groups of counties
181 is included in the online supplement (See Supplemental Material, Table 1).

182 We applied a Poisson regression model to daily counts of ED visits for combined and
183 individual cardiovascular and respiratory outcomes separately with explanatory variables
184 indicating days within the three-day window of dense smoke and subsequent 5 days of lagged
185 exposure. This was done for both exposed and referent counties. The effects of exposure on
186 outcomes at lags was estimated using an unconstrained distributed lag model (Peng 2008). A
187 number of published studies (Pope et al. 2008; Braga et al. 2001), have determined that air
188 pollution produces immediate and delayed effects on morbidity and mortality and that the time to
189 adverse outcome may vary by pollutant and health outcomes. From the perspective of public
190 health, in this study, we were interested in the total burden on human health associated with the
191 wildfire episode. Inference on delay between the exposure and effect is not appropriate for this
192 study without personal exposure measurements. Here the results are summarized in terms of the

193 cumulative relative risk (cRR), i.e. cumulative risk over lags 0-5, following exposure (Peng
194 2008) according to

195
$$\left[\exp\left(\sum_{i=0}^5 \beta_i\right) \right]$$

196 where β_i is the relative risk estimate associated with the i^{th} day following the exposure. Figures 3
197 and 4 summarize percent change in cRR or excess risk according to $(\text{cRR}-1)\times 100\%$. The analysis
198 was stratified by age and sex in both exposed and reference county cohorts.
199

200 **Results**

201 Asthma related visits accounted for 44% of all respiratory codes considered, and heart
202 failure accounted for 33% of all cardiac events. Consistent with the distribution of asthma
203 prevalence by sex, the aggregate counts of asthma-related visits occurred in more women (70%)
204 than men, and in those between 18 and 64 years (85%). Cardiac events were substantially more
205 common in those individuals over 65 (67%). The number of clinical events reported for each
206 ICD-9-CM code during the study period is given in Table 1.

207 The cumulative impact during the three high exposure days and five subsequent lag days
208 resulted in significant increases in ED visits for several outcomes when compared with visits
209 during the remainder of the six week study period in the exposed counties (Figure 3). ED visits
210 for all the respiratory diagnoses were elevated in the exposed counties (cRR= 1.66, 95%
211 confidence interval [CI] 1.38 to 1.99) but not in the referent counties (1.06 [0.89, 1.25]). Among
212 the respiratory outcomes, ED visits for asthma (1.65[1.25, 2.17]), COPD (1.73[1.06, 2.83]),
213 pneumonia and acute bronchitis (1.59[1.07, 2.34]) increased significantly. Visits for URIs
214 (1.68[0.94, 3.00]) also increased but were not statistically significant. We found no changes for
215 respiratory outcomes in the referent counties.

216 Cumulative relative risk for heart failure related ED visits (1.37 [1.01, 1.85]) was
217 increased in the exposed counties, while visits for myocardial infarction and cardiac
218 dysrhythmias were not increased in exposed or referent counties. Reflecting the increase in
219 cardiac and respiratory events, ED visits associated with cardiopulmonary symptoms (ICD-9-
220 CM 786) were significantly increased (1.23[1.06, 1.43]) in the exposed counties.

221 Age and sex had varying effects on respiratory outcomes and the analysis reflected higher
222 uncertainty due to lower counts of events in these subgroups. Visits for asthma, pneumonia,

223 acute bronchitis and URI increased to a greater extent among women than men in exposed
224 counties (Figure 4A). In contrast, visits related to COPD were only elevated in men. There were
225 more ED visits for asthma, COPD, pneumonia and acute bronchitis among those younger than 65
226 in the exposed counties (Figure 4B). There were no differences in ED visits for cardiovascular
227 events stratified by age or sex, possibly because the smaller number of visits for these ICD-9-CM
228 codes diminished the power to observe effects in these subgroups.

229 **Discussion**

230 This is the first population-based health study of peat bog fire exposures utilizing a
231 syndromic surveillance system with a nearly comprehensive record of health outcomes from an
232 entire geographic region. We determined relative risk of cardio-respiratory outcomes
233 cumulative over the lag days 0 through 5 of the exposure to smoke. The study demonstrated that
234 exposure to smoke from the wildfire increased ED visits for asthma, COPD, pneumonia, acute
235 bronchitis and heart failure in a sparsely populated non-urban area. The study also demonstrates
236 the utility of syndromic surveillance in assessment of health burden during widespread
237 environmental events. In turn, such assessments should help guide development of strategies and
238 the allocation of resources for the public health response.

239 Consistent with the results from other studies, asthma related outcomes were most
240 prevalent, especially among adult women. A surprising and important observation is the
241 statistically significant association between smoke exposure and an increase in ED visits for
242 heart failure, as well as the trend towards a positive association with acute coronary syndrome
243 (myocardial infarction and unstable angina). We restricted the cardiovascular clinical endpoints
244 to acute coronary syndrome, which included myocardial infarction and unstable angina (ICD-9
245 410 and 411.1), and heart failure (ICD-9 428) and excluded hypertensive heart disease.

246 Although previous studies have shown positive associations between ambient concentrations of
247 PM_{2.5} and ED visits and hospitalization for heart failure (Wellenius et al. 2005; Wellenius et al.
248 2006; Brook et al. 2010; Dominici et al. 2007), to our knowledge this is the first study that has
249 reported ED visits for heart failure associated with wildfire exposure. However, epidemiologic
250 studies in areas with high residential wood burning suggest have suggested an increased risk of
251 cardiovascular mortality and morbidity may be in part due to wood smoke emissions (Sanhueza
252 et al. 2009; Schwartz et al. 1993). Our study demonstrated an increased percent change in
253 relative risk of 37%. In comparison to the rest of the state, counties of eastern NC are among the
254 poorest and least healthy counties, characterized by higher prevalence of hypertension, diabetes,
255 ischemic heart disease, and heart failure (NCSCHS 2009). These are clinical conditions that are
256 known to be associated with individuals more vulnerable to the health effects of ambient air
257 particle pollution and may have contributed to the large number of events in this relatively sparse
258 population (Wellenius et al. 2005; O'Neill et al. 2007; O'Neill et al. 2005).

259 Unlike the hot canopy forest fires often seen in the western portion of the country,
260 Pocosin fire was not associated with high temperatures and heat waves. Instead, a prolonged
261 drought lead to the unusually dry conditions in the region which allowed for rapid spread of fire
262 across the peat bogs following a lightning strike. Therefore, it is not surprising we did not
263 observe long-term linear trends or seasonality for the principal respiratory and cardiac diagnoses
264 during this study period as shown for visits for asthma (Figure 2). However, at least three types
265 of misclassification in exposure could have occurred in the study. First, any days with elevated
266 PM levels outside the three day window, were misclassified into periods and could have resulted
267 in a bias towards the null hypothesis. Second, some of the referent counties were exposed to
268 emissions from the fire at some point during the six week period, though not to the extent of the

269 exposed counties, also potentially resulting in bias toward the null hypothesis. Finally, a degree
270 of mis classification at the individual level may occur due to differences in exposure between
271 county groups. According to the 2000 Census information exposed counties are demographically
272 similar to, but more rural, than referent counties, which may have resulted in a higher exposure
273 (see Supplemental Material, Table 1). However no individual data was available for the
274 analysis. A well documented risk of increased morbidity and mortality due to air-pollution is
275 largely based on central site monitoring and time-series of health outcomes within major urban
276 centers. Rural and remote areas are often not studied because of sparse population and lack of
277 monitoring data. The type and availability of monitoring most often found in sparsely populated
278 areas, such as in this study, provides limited information about the geographic scope of exposure
279 times of environmental events. Air pollution monitors collect data at irregular temporal
280 resolution (e.g. every 24 hrs or every 3 days or every 6 days) and do not provide comprehensive
281 information regarding the geographic scope of the exposure, depending on monitor location,
282 wind direction, terrain (e.g. differences in elevation). Developing new methods for assessing
283 exposure will be increasingly more important as wildfires and other environmental events
284 become more frequent. Several computer models for atmospheric pollutant dispersion already
285 exist but are difficult to validate against sparse ground measurements. In this study we used
286 satellite-derived measurements of AOD to define spatial boundaries of the smoke plume. This
287 allowed us to capture the geographic extent of ED visits in the region and thereby increase
288 statistical power. Such exposure assessment however allows us to associate changes in relative
289 risk to the exposure period but not to the concentration of air pollutants.

290 Central reporting of ED visits and the high rate of case identification afforded by the
291 NCDETECT syndromic surveillance system contributed to the strength of the associations found

292 in this study. Syndromic surveillance systems were developed to detect epidemics and monitor
293 outbreaks in near real-time (Buehler et al. 2008). The program is implemented at the state level
294 and the median rate of hospital participation in the US is 35%, and ranges between 2 and 100%.
295 Most states report chief complaint data alone but a few, including NC, report diagnostic ICD-9-
296 CM codes. NC has a uniquely comprehensive program with 98% of its hospitals participating.
297 Two other studies of health effects of wildfires in California and Florida (Jeffrey et al. 2005;
298 Sorenset et al. 1999) used syndromic surveillance data and reported increases in respiratory
299 outcomes, asthma, respiratory complaints, eye irritation, and smoke inhalation for respiratory-
300 related chief complaints. The strength of evidence found in the present study further supports
301 efforts to expand automated surveillance system for near-real time delivery of information and
302 health advisories during emergencies. While ICD-9-CM codes are not as timely as chief
303 complaints, they can provide more specific measurements of health outcomes and should be
304 added into syndromic surveillance systems in other states.

305 Peat fires burn significantly more biological mass, produce massive amounts of smoke,
306 and are notoriously more difficult to extinguish than hot canopy forest fires or grassland fires
307 (Page 2002; See et al. 2007; Soja et al. 2004; Muraleedharan et al. 2000). One study of peat fire
308 particle composition reported carbonaceous particles, particularly organic carbon, NO₃⁻, and
309 SO₄(²⁻) as major components of PM_{2.5}, while the less abundant constituents included ions such
310 as NH₄⁺, NO₂⁻, Na⁺, K⁺, organic acids, and metals such as Al, Fe, and Ti (See et al., 2007).
311 Another study reported CO, CO₂, and CH₄ to be the most abundant gaseous emissions
312 (Muraleedharan et al. 2000). Although, emissions from peat fires may differ from forest fires in
313 chemical composition, it is not known if they differ in toxicity.

314 Population growth and land use alterations are the primary bases for increased wildfire
315 events worldwide. Additional stress is created by earlier snow melts, rising temperatures,
316 cumulative effects of the current drought and other climate-related changes (Quadrennial Fire
317 Review: Final Report 2009; Westerling et al. 2006). Recent peat fires around Elektrogorsk,
318 Russia exemplify the synergistic effect of these factors and their impact on health and economy
319 in the region (Reilly 2010). Unprecedented hot and dry weather in Russia last year, eased the
320 spread of fires in swamps long ago drained to harvest energy from peat. The smoke obstructed
321 ground, as well as air travel, and resulted in numerous health advisories (Williams 2010). Peat
322 bogs have been exploited for energy use in many parts of the world leaving vast areas of dried
323 wetlands and swamps particularly vulnerable to droughts. It is expected, therefore, that the risk
324 of peat fires is likely to increase in upcoming decades (Quadrennial Fire Review: Final Report
325 2009).

326 **Conclusion**

327 A consistent increase in relative risk in the exposed counties for nearly all outcome
328 categories is striking and persuasive in comparison to the referent counties and has potentially
329 significant public health implications. The precision of relative risk estimates in this study is
330 attributed to the use of comprehensive population health data from the NC public surveillance
331 program as well as the use of spatially and temporally dense satellite measurements of aerosol
332 optical depth. The region of the state most affected by the smoke was sparsely populated with
333 few available air quality monitors. Therefore, traditional exposure assessment based on
334 monitoring and hospital admissions data alone would not provide us with enough information to
335 assess the true risk. Both data sources used in this paper are readily available to state and local
336 public health officials and lend themselves to the application of traditional statistical methods. In

337 the near future, public health officials may benefit from dashboard-like visualization tools that
338 allow end users to overlay environmental data with healthcare data in order to improve event
339 characterization capabilities and response efforts. Mitigation of exposure and raising public
340 awareness would be expected to decrease the burden to the health care system, and improve the
341 wellbeing of the public.

342
343 **Disclaimer:** The research described in this article has been reviewed by the National Health and
344 Environmental Effects Research Laboratory, US Environmental Protection Agency and approved
345 for publication. Approval does not signify that the contents necessarily reflect the views and the
346 policies of the Agency nor does mention of trade names or commercial products constitute
347 endorsement or recommendation for use. The NC Public Health Data Group and NC DETECT
348 do not take responsibility for the scientific validity or accuracy of methodology, results,
349 statistical analyses, or conclusions presented.

350

351 **Acknowledgements:**

352 Clifton Barnett for pulling the ED data from the surveillance database and Shobha Kondragunta
353 NOAA/NESDIS Center for Satellite Applications and Research for obtaining GOES data.

354

355

Tables and Figure Legends

		Total	<65	≥65	Female	Male	
Exposed Counties	Respiratory Outcomes	All†	4702	3485	1217	2963	1739
		Asthma (ICD-9 493)	2081	1775	306	1463	618
		Chronic Obstructive Pulmonary Disease (ICD-9 491, 492)	647	314	333	317	330
		Pneumonia and Acute Bronchitis (ICD-9 481, 482, 485, 486, 466)	1053	607	446	575	478
		Upper Respiratory Infections (ICD-9 465)	444	189	255	202	242
	Cardiac outcomes	All‡	6078	2037	4041	3357	2721
		Myocardial Infarction (ICD-9 410, 411)	444	189	255	202	242
		Heart Failure (ICD-9 428)	1817	579	1238	1068	749
		Cardiac Dysrhythmias (ICD-9 427)	1756	538	1218	937	819
	Respiratory/Other chest Symptoms(786)		7716	5752	1964	4532	3184
Referent Counties	Respiratory Outcomes	All†	6074	4347	1727	3819	2255
		Asthma (ICD-9 493)	2199	1886	313	1591	608
		Chronic Obstructive Pulmonary Disease (ICD-9 491, 492)	1158	558	600	601	557
		Pneumonia and Acute Bronchitis (ICD-9 481, 482, 485, 486, 466)	1815	1146	669	1039	777
		Upper Respiratory Infections (ICD-9 465)	490	429	61	344	146
	Cardiac Outcomes	All‡	7999	2704	5295	4279	3720
		Myocardial Infarction (ICD-9 410, 411)	674	334	340	288	386
		Heart Failure (ICD-9 428)	2374	740	1634	1337	1037
		Cardiac Dysrhythmias (ICD-9 427)	2580	785	1795	1381	1199
	Respiratory/Other chest Symptoms(786)		10102	7801	2301	5968	4134

357

358

359

360

361

362

363

364

365

Table 1. Total counts of emergency department visits in the exposed and referent counties by outcomes, age group, and gender, between June 1 and July 14, 2008. Minimum age for respiratory and cardiac outcomes was 18 and 45 respectively.

†All Respiratory ICD-9 Codes: 465, 466, 480, 481, 482, 483, 484, 485, 486, 490, 491, 492, 493

‡All Cardiac ICD-9 Codes: 410, 411, 413, 415, 416, 417, 420, 421, 422, 423, 424, 425, 426, 427, 428, 429, 434, 435, 444, 445, 451

366

367 **Figure Legends**

368

369 Figure 1. Aerial maps showing counties impacted by the Evans Road Fire at the Pocosin Lakes

370 National Wildlife Refuge on June 10th, 11th, and 12th (Panels A, B, and C respectively) as

371 measured by satellite AOD images. Panel D shows assignment of counties as exposed or

372 referent.

373

374 Figure 2. Daily counts of asthma related ED visits in the exposed counties. Arrows represent the

375 3 days of high exposure (red) and subsequent 5 lags (grey).

376

377 Figure 3. Percent change in cumulative relative risk and 95% confidence intervals by discharge

378 diagnosis category for exposed and reference North Carolina counties during the 3 day period of

379 high exposure compared with the entire six week study period. Dark circles denote exposed

380 counties and open circles denote referent counties. The grey line indicates the null hypothesis of

381 no change in the cumulative relative risk.

382

383 Figure 4. Percent change in cumulative relative risk and 95% confidence intervals by discharge

384 diagnosis category, age, and gender for exposed and reference North Carolina counties during

385 the 3 day period of high exposure compared with the entire six week study period. Panel (a)

386 Dark and open squares denote ED visits by females in exposed and referent counties,

387 respectively. Dark and open diamonds denote ED visits by males in exposed and referent

388 counties, respectively. Panel (b) Dark and open squares denote ED visits by those under 65 in

389 exposed and referent counties, respectively. Dark and open diamonds denote ED visits for those

390 65 and older in exposed and referent counties, respectively. The grey line indicates the null

391 hypothesis of no change in the cumulative relative risk. Confidence interval extending out of the
392 figure region reaches 1816% in excess risk (19.16 cumulative Relative Risk) see Supplemental
393 Material, Table 4.

394
395
396 **References**Integrated Science Assessment for Particulate Matter. 2009. EPA/600/R-08/139F.
397 Washington, DC.
398 Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, et al. 2007. Woodsmoke
399 health effects: A review. *Inhalation Toxicology* 19(1): 67-106.
400 Jeffrey M, Johnson L, Hicks C, McClean, Ginsberg M. 2005. Leveraging Syndromic
401 Surveillance During the San Diego Wildfires, 2003 MMWR Morb Mortal Wkly Rep
402 54(Suppl(190)).
403 Delfino RJ, Brummel S, Wu J, Stern H, Ostro B, Lipsett M, et al. 2009. The relationship of
404 respiratory and cardiovascular hospital admissions to the southern California wildfires of 2003.
405 *Occup Environ Med* 66(3): 189-197.
406 Duclos P SL, Lipset M. 1990. The 1987 forest fire disaster in California: assessment of
407 emergency room visits. *Anrch Environ Health* 45: 53-58.
408 Kunzli N, Avol E, Wu J, Gauderman WJ, Rappaport E, Millstein J, et al. 2006. Health effects of
409 the 2003 Southern California wildfires on children. *Am J Respir Crit Care Med* 174(11): 1221-
410 1228.
411 Moore D, Copes R, Fisk R, *et al.* 2006. Population health effects of air quality changes due to
412 forest fires in British Columbia in 2003; estimates from physician-visit billing data. *Can J Public*
413 *Health* 5: 175-182
414
415 Mott J, Mannino DM, Alverson CJ, *et al.* 2005. Cardiorespiratory hospitalizations associated
416 with smoke exposure during the 1997, Southeast Asian forest fires. *Int J Hyg Environ Health*
417 2008: 75-85.
418 Sastry N. 2002. Forest fires, air pollution, and mortality in Southeast Asia. *Demography* 39: 1-
419 23.
420 Vedal S, Dutton SJ. 2006. Wildfire air pollution and daily mortality in a large urban area.
421 *Environ Res* 102(1): 29-35.
422 Quadrennial Fire Review: Final Report. 2009. National Interagency Fire Center
423 2010.
424 Page S, Siegert, F., Rieley, J., Boehm, H., Jaya, A., and Limin, S. 2002. The Amount of Carbon
425 Released From Peat and Forest Fires in Indonesia During 1997. *Nature* 420: 61-65.
426 Kunii O, Kanagawa S, Yajima I, *al e.* 2002. The 1997 haze disaster in Indonesia: its air quality
427 and health effects. *Arch Environ Health* 174: 16-22.
428 NCDetect. year. North Carolina Disease Event Tracking and Epidemiologic Collection Tool
429 (NC DETECT) Available: <http://www.ncdetect.org/> [accessed March 9, 2010 2010].
430 Al-Saadi J, Szykman J, Pierce RB, Kittaka C, Neil D, Chu DA, et al. 2005. Improving national
431 air quality forecasts with satellite aerosol observations. *B Am Meteorol Soc* 86(9): 1249-+.
432 AQI. 2009. Air Quality Index, A Guide to Air Quality and
433 Your Health. US EPA, Office of Air Quality Planning and Standards
434 Outreach and Information Division Research Triangle Park, NC 2009: EPA-456/F-409-002
435
436 Engel-Cox JA, Holloman CH, Coutant BW, Hoff RM. 2004. Qualitative and quantitative
437 evaluation of MODIS satellite sensor data for regional and urban scale air quality. *Atmospheric*
438 *Environment* 38(16): 2495-2509.

439 Wang J, Christopher SA. 2003. Intercomparison between satellite-derived aerosol optical
440 thickness and PM_{2.5} mass: Implications for air quality studies. *Geophys Res Lett* 30(21): -.
441 Peng D, Dominici, F. 2008. *Statistical Methods for Environmental Epidemiology with R*: Springer
442 Science + Business Media, LLC.

443 Pope CA, 3rd, Renlund DG, Kfoury AG, May HT, Horne BD. 2008. Relation of heart failure
444 hospitalization to exposure to fine particulate air pollution. *Am J Cardiol* 102(9): 1230-1234.

445 Braga AL, Zanobetti A, Schwartz J. 2001. The lag structure between particulate air pollution and
446 respiratory and cardiovascular deaths in 10 US cities. *J Occup Environ Med* 43(11): 927-933.

447 Wellenius GA, Bateson TF, Mittleman MA, Schwartz J. 2005. Particulate air pollution and the
448 rate of hospitalization for congestive heart failure among medicare beneficiaries in Pittsburgh,
449 Pennsylvania. *Am J Epidemiol* 161(11): 1030-1036.

450 Wellenius GA, Schwartz J, Mittleman MA. 2006. Particulate air pollution and hospital
451 admissions for congestive heart failure in seven United States cities. *Am J Cardiol* 97(3): 404-
452 408.

453 Brook RD, Rajagopalan S, Pope CA, 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. 2010.
454 Particulate matter air pollution and cardiovascular disease: An update to the scientific statement
455 from the American Heart Association. *Circulation* 121(21): 2331-2378.

456 Dominici F, Peng RD, Zeger SL, White RH, Samet JM. 2007. Particulate air pollution and
457 mortality in the United States: did the risks change from 1987 to 2000? *Am J Epidemiol* 166(8):
458 880-888.

459 Sanhueza PA, Torreblanca MA, Diaz-Robles LA, Schiappacasse LN, Silva MP, Astete TD.
460 2009. Particulate air pollution and health effects for cardiovascular and respiratory causes in
461 Temuco, Chile: a wood-smoke-polluted urban area. *J Air Waste Manag Assoc* 59(12): 1481-
462 1488.

463 Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ. 1993. Particulate air pollution and
464 hospital emergency room visits for asthma in Seattle. *Am Rev Respir Dis* 147(4): 826-831.

465 NCSCHS. year. North Carolina State Center of Health Statistics. Available:
466 www.schs.state.nc.us/SCHS/brfss/2009/east/topics.html [accessed May 10, 2010].

467 O'Neill MS, Veves A, Sarnat JA, Zanobetti A, Gold DR, Economides PA, et al. 2007. Air
468 pollution and inflammation in type 2 diabetes: a mechanism for susceptibility. *Occup Environ*
469 *Med* 64(6): 373-379.

470 O'Neill MS, Veves A, Zanobetti A, Sarnat JA, Gold DR, Economides PA, et al. 2005. Diabetes
471 enhances vulnerability to particulate air pollution-associated impairment in vascular reactivity
472 and endothelial function. *Circulation* 111(22): 2913-2920.

473 Buehler JW, Sonricker A, Paladin M, Soper P, Mostashari F. 2008. Syndromic Surveillance
474 Practice in the United States: Findings from a Survey of State,
475 Territorial, and Selected Local Health Departments. *Advances in Disease Surveillance* 6(3).

476 Sorensen B, Fuss M, Mulla Z, Bigler W, Wiersma S, Hopkins R. 1999. Surveillance of morbidity
477 during wildfires--Central Florida, 1998. *MMWR Morb Mortal Wkly Rep* 48(4): 78-79.

478 See S, Balasubramanian R, Rianawati E, Karthikeyan S, Streets D. 2007. Characterization and
479 Source Apportionment of Particulate Matter $\leq 2.5 \mu\text{m}$ in Sumatra, Indonesia, during a Recent
480 Peat Fire Episode. *Environ Sci Technol* 41: 3488-3494.

481 Soja AJ, Cofer WR, Shugart HH, Sukhinin AI, Stackhouse PW, McRae DJ, et al. 2004.
482 Estimating fire emissions and disparities in boreal Siberia (1998-2002). *J Geophys Res-Atmos*
483 109(D14).

484 Muraleedharan TR, Radojevic M, Waugh A, Caruana A. 2000. Emissions from the combustion
485 of peat: an experimental study. Atmospheric Environment 34(18): 3033-3035.
486 Westerling AL, Hidalgo HG, Cayan DR, Swetnam TW. 2006. Warming and earlier spring
487 increase western US forest wildfire activity. Science 313(5789): 940-943.
488 Reilly M. 2010. Russian Fires, Heat Waves, and a Drive Through Hell newsdiscoverycom Aug
489 9.
490 Williams S. year. Smog from Fires Chokes Moscow. Available:
491 <http://news.discovery.com/earth/moscow-smog-wildfires-russia.html> [accessed Aug 6,
492 2010].
493
494