highly suggest a linking role between asthma and obesity. Future studies should further elucidate the effects of obesity on the inflammatory system both inside and outside the adipose tissue.

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Wildfire smoke and respiratory symptoms in patients with chronic obstructive pulmonary disease

To the Editor:

Short-term increases in concentrations of particulate air pollution are associated with increased morbidity and mortality in patients with chronic obstructive pulmonary disease (COPD).[1-3] Although most particulate air pollution is attributable to vehicular and industrial sources, biomass combustion during wildfires has been shown to have significant effects on outdoor air quality[4] and climate as a result of generation of fine particulate matter. Although the relationship of domestic biomass fuel combustion with COPD has been described,[5] the acute effect of wildfire smoke-related air pollution on patients with established COPD has not been well studied. Recent reports suggest that wildfire smoke exposure leads to increased health care utilization by subjects with asthma,[6] raising the possibility that patients with COPD are similarly affected.

Air pollution events caused by wildfires can serve as natural experiments to test the hypothesis that short-term increases in air pollution concentrations have adverse health effects. On June 8, 2002, a large wildfire known as the Hayman fire began southwest of Denver and was not controlled until July 18, 2002, ultimately burning 137,760 acres. On at least 2 days during this period, a plume of smoke and ash was carried over the Denver metropolitan area by prevailing winds (Fig 1). We prospectively evaluated the effects of this wildfire on air pollution and respiratory symptoms in adult subjects with COPD.

All research was approved by the National Jewish Medical and Research Center Institutional Review Board, and all subjects provided written informed consent. We recruited 21 Denver residents with COPD. Subjects were identified through previous participation in a panel study of air quality and respiratory symptoms in subjects with COPD. Eligibility criteria were as follows: (1) age $\geq$40 years, (2) >10 pack-year history of tobacco use, (3) airflow limitation as manifested by a FEV_1 < 70% of predicted and FEV_1:forced vital capacity (FVC) ratio of <60%, and (4) diffusing capacity for carbon monoxide <70% of predicted.

Between June 8, 2002, and June 29, 2002, subjects completed a daily telephone interview in which they reported 12-hour change in 5 symptoms: dyspnea, cough, chest tightness, wheezing, and sputum production. Change in symptoms was recorded on a 7-point scale on which symptoms over the last 12 hours were rated relative to the subject's baseline. Baseline symptoms were assigned a score of 4, with a score of 20 reflecting baseline severity of all 5 symptoms. A 1-point increase in any 1 symptom reflected worsening to slightly greater than usual, and a 2-point increase reflected worsening to much greater than usual. Over this same time frame, measurements of particulate matter (PM) with an aerodynamic diameter <10 μm (PM_{10}, $\mu g/m^3$) and <2.5 μm ($PM_{2.5}$, $\mu g/m^3$) and carbon monoxide (CO, ppm) were obtained from an air quality monitoring station located in Denver and operated by the Air Pollution Control Division of the Colorado Department of Public Health and Environment.

Evaluation of $PM_{2.5}$ concentrations revealed that there were significantly increased concentrations of particulate matter on 2 separate days; these were termed *spike days*. Concentrations of $PM_{2.5}$, PM_{10}, and CO on spike days were averaged and compared with mean concentrations on nonspike days (Wilcoxon rank-sum test). Median symptom scores on spike days were compared with median symptom scores on nonspike days using a repeated measurements ANOVA (SAS macro GLIMMIX) that accounted for the correlated nature of individual symptom. Analyses were performed using SAS (version 8.2; SAS Institute, Cary, NC).

Mean (±SEM) age was 69.4 years ± 9.1 years, and 38.1% of subjects were female. Mean FEV_1:FVC ratio was 53% ± 21%, with a group mean FEV_1 (L) of 1.6 ± 1.1 and FVC (L) of 2.8 ± 1.1. Supplemental oxygen was used by 13 of 21 subjects (61.9%). Increased concentrations of $PM_{2.5}$, PM_{10}, and CO were observed on June 9, 2002, and June 18, 2002 (spike days 1 and 2). Concentrations of $PM_{2.5}$, PM_{10}, and CO on both spike days were

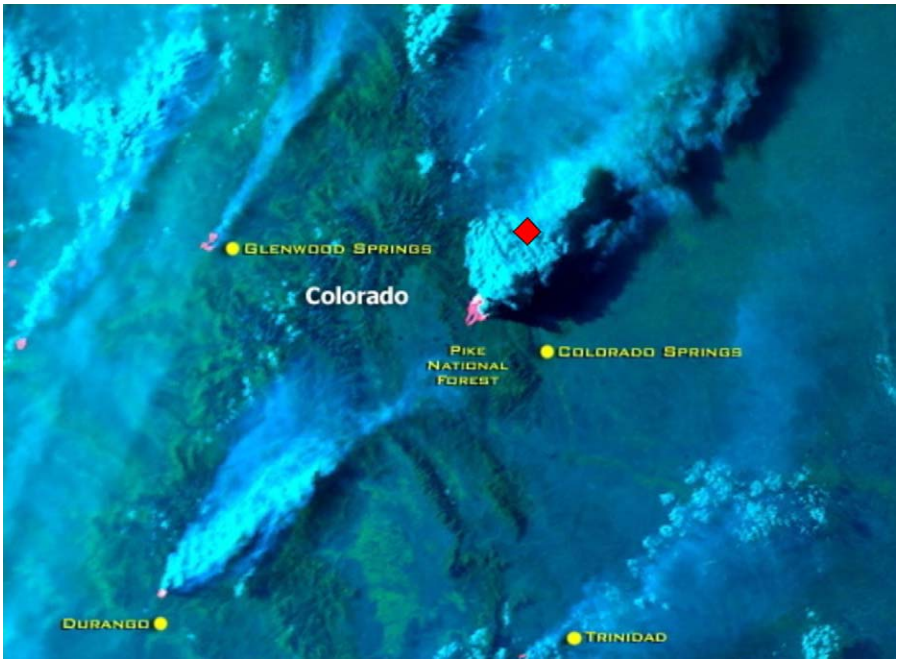

FIG 1. June 9, 2002 (spike day 1), satellite image demonstrating a large smoke plume traveling from the Hayman wildfire northeast over the Denver metropolitan area *(red diamond)*.[10]

higher than on nonspike days ($P = .03$; Table I). Symptom scores were significantly elevated on spike days versus nonspike days ($P = .0002$; Table I). Qualitative review of daily trends in air pollution and symptoms indicated that symptom scores increased on spike days and then returned to baseline levels the next day.

This study demonstrates that wildfire smoke significantly increases levels of fine particulate air pollution and that these increases are associated with increases in respiratory symptoms in patients with COPD. Subjects were not evaluated for objective measures of airflow limitation, thus limiting our ability to associate increases in symptoms with decreases in airflow. However, objective measures of airflow and symptoms are not well-correlated in COPD,[7] and the observed increase in symptoms may well have occurred in the absence of significant changes in airflow. Although the clinical significance of the numerically small observed increase in symptoms requires further validation, the observed median change in symptoms score of 1.5 suggests that subjects on average reported an increase in dyspnea, cough, chest tightness, wheezing, or sputum production that ranged between slightly more and much more than usual, and therefore that clinical characteristics of an acute exacerbation are elevated even after wildfire smoke exposures of $\leq$24 hours. Although we were unable to evaluate other short-term (ie, increased use of rescue medications) or intermediate-term (ie, increase in unscheduled visits or exacerbations) effects of this exposure more formally, our findings suggest an increased risk of adverse short-term and intermediate-term outcomes. Observed concentrations of particulate matter were much higher than those reported in other studies of air pollution and symptom reporting in COPD,[8] which may account for our ability to demonstrate an association between increased in air pollution and symptoms. However, a number of potential confounders, including tobacco smoke and time spent indoors versus outdoors (a potential marker of exposure), were not evaluated.

TABLE I. Air pollutant concentrations and mean respiratory symptom scores on spike days (June 9, 2002, and June 18, 2002) versus nonspike days.

	Spike days	Nonspike days	*P* value‡
$PM_{2.5}$ ($\mu g/m^3$)*	63.1 ± 0.1	14.0 ± 4.8	.03
PM_{10} ($\mu g/m^3$)*	89.4 ± 2.3*	39.8 ± 10.0	.03
CO (ppm)*	0.96 ± 0.07*	0.67 ± 0.18	.05
Symptom scores†	21.5 (3.0)	20.0 (1.0)	.0002

*24-Hour mean ± SD.
†Median (interquartile range).
‡For comparison of spike and nonspike days.

A strength of this quasiexperimental study is that confounding by meteorological phenomena,[9] in which the health effects of air pollution are not easily disentangled from those resulting from changes in meteorology, was likely less of a factor in influencing symptom scores. Although the contribution of temperature to air pollution is well established, in this case, the effect of prevailing winds acting as a delivery mechanism for particulate matter was likely greater than any effect of small variations in temperature.

Our findings reinforce the need for larger prospectively planned studies of the health effects of wildfire smoke and suggest that patients with COPD should be informed of the risk of increased respiratory symptoms in relation to wildfire smoke and counseled about appropriate protective and therapeutic measures.

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