Associations between Health Effects and Particulate Matter and Black Carbon in Subjects with Respiratory Disease

Karen L. Jansen,1 Timothy V. Larson,1 Jane Q. Koenig,1 Therese F. Mar,1 Carrie Fields,1 Jim Stewart,1 and Morton Lippmann2

1University of Washington, Seattle, Washington, USA; 2New York University School of Medicine, Tuxedo, New York, USA

We measured fractional exhaled nitric oxide (FENO), spirometry, blood pressure, oxygen saturation of the blood (SaO2), and pulse rate in 16 older subjects with asthma or chronic obstructive pulmonary disease (COPD) in Seattle, Washington. Data were collected daily for 12 days. We simultaneously collected PM10 and PM2.5 (particulate matter ≤ 10 µm or ≤ 2.5 µm, respectively) filter samples at a central outdoor site, as well as outside and inside the subjects’ homes. Personal PM10 filter samples were also collected. All filters were analyzed for mass and light absorbance. We analyzed within-subject associations between health outcomes and air pollution metrics using a linear mixed-effects model with random intercept, controlling for age, ambient relative humidity, and ambient temperature. For the 7 subjects with asthma, a 10 µg/m³ increase in 24-hour average outdoor PM10 and PM2.5 was associated with a 5.9 [95% confidence interval (CI), 2.9–8.9] and 4.2 ppb (95% CI, 1.3–7.1) increase in FENO, respectively. A 1 µg/m³ increase in outdoor, indoor, and personal black carbon (BC) was associated with increases in FENO of 2.3 ppb (95% CI, 1.1–3.6), 4.0 ppb (95% CI, 2.0–5.9), and 1.2 ppb (95% CI, 0.2–2.2), respectively. No significant association was found between PM or BC measures and changes in spirometry, blood pressure, pulse rate, or SaO2 in these subjects. Results from this study indicate that FENO may be a more sensitive marker of PM exposure than traditional health outcomes and that particle-associated BC is useful for examining associations between primary combustion constituents of PM and health outcomes. Key words: asthma, black carbon, chronic obstructive pulmonary disease, fractional exhaled nitric oxide, panel study, particulate matter. "Environ Health Perspect 113:1741–1746 (2005). doi:10.1289/ehp.8153 available via http://dx.doi.org/ [Online 25 August 2005]"

Interest in particulate matter (PM) air pollution has been driven by epidemiologic studies reporting adverse cardiac and respiratory health effects [Bascom et al. 1996; Dockery 2001; U.S. Environmental Protection Agency (EPA) 2004]. To further investigate the basis for these epidemiologic findings, it is important to assess individual exposures to PM and their related health effects. Panel studies that include indoor, outdoor, personal, and fixed-site PM monitoring can provide an important link between the effects observed in a population and the effects at the individual subject level.

Panel studies often report gravimetric measures of PM. However, current research is focusing on the constituents of PM (Brunekreef et al. 2005). Elemental carbon (EC) is one component of PM that has been associated with respiratory health effects in children. In a 10-year study of 1,759 children, Gauderman et al. (2004) found a strong association between reduced annual growth in forced expiratory volume in 1 sec (FEV1) in children and exposure to EC, nitrogen dioxide, and acid vapor. EC, measured on quartz filters by thermal desorption, is strongly associated with, but not identical to, “black carbon” (BC), as measured by diffuse transmittance through or reflectance from a Teflon filter. In a recent study, Kim et al. (2004) reported that concentrations of traffic-related pollutants (PM, BC, total nitrogen oxides, and NO3) were associated with respiratory symptoms in children.

EC and BC have also been associated with cardiovascular health effects. In a study of defibrillator discharge interventions among 100 adult patients, Peters et al. (2000) found that patients with ≥ 10 interventions experienced increased arrhythmias in association with short-term variations in BC, NO2, carbon monoxide, and fine particulate mass (PM2.5). In a study of 269 elderly Boston, Massachusetts, residents equipped with Holter monitors, an elevated BC level was associated with a −0.1 mm ST-segment depression; this BC level predicted increased risk of ST-segment depression among those with at least one episode of that level of ST-segment depression (Gold et al. 2005).

Furthermore, in elderly subjects in Boston, BC increases were associated with a decrease in flow-mediated vascular reactivity (−12.6%; O’Neill et al. 2005). These studies implicate particles whose predominant source is traffic as a risk factor for adverse health effects.

Accumulated data suggest that PM exposure may lead to pulmonary inflammation (Gong et al. 2003; Li et al. 1996; Salvi et al. 1999). Chronic inflammation is a hallmark of lung diseases such as asthma and chronic obstructive pulmonary disease (COPD) (Gan et al. 2004) and may be aggravated in susceptible groups by PM pollution. A noninvasive method of estimating airway inflammation among sensitive groups is fractional exhaled nitric oxide (FENO). Over the last decade, FENO has been shown to be reproducible, inexpensive, and easy to measure serially. FENO concentrations are also highly correlated with other markers of airway inflammation, such as sputum eosinophils and bronchial hyperresponsiveness in subjects with asthma (Jones et al. 2002). Studies have reported positive associations between FENO and ambient PM2.5 exposures to air pollutants in community-based studies (Adamkiewicz et al. 2004; Koenig et al. 2003).

Spirometry has historically been used as a method of measuring health effects of exposure to PM air pollution. Numerous panel studies have examined the effects of short-term ambient PM exposure on daily lung function [FEV1, forced vital capacity (FVC), and peak expiratory flow rate (PEF)] (U.S. EPA 2004). Subjects with asthma tended to show small PEF decrements for increases in PM10 and PM2.5 concentrations, as seen in several studies (Gielen et al. 1997; Pekkanen et al. 1997; Peters et al. 1997; Romieu et al. 1996).

Another measure of respiratory health, oxygen saturation of the arterial blood (SaO2), has been collected in panel studies. In a study of 90 elderly subjects, Pope et al. (1999a) found that SaO2 decreased in association with PM10 in the Utah Valley; however, the association was not statistically significant and may have been confounded by atmospheric conditions. We thank our subjects for their enthusiastic participation. We also thank D. Lennington and R. Murashige for technical assistance and the Washington State Department of Ecology for atmospheric data.

Address correspondence to J. Q. Koenig, Department of Environmental and Occupational Health Sciences, School of Public Health and Community Medicine, University of Washington, Box 357234, Seattle, WA 98195 USA. Telephone: (206) 543-2026. Fax: (206) 685-3990. E-mail: jkoenig@u.washington.edu

We thank our subjects for their enthusiastic participation. We also thank D. Lennington and R. Murashige for technical assistance and the Washington State Department of Ecology for atmospheric data.

This research was supported by grant R 827355 from the U.S. Environmental Protection Agency (EPA), grant PO ES 07033 from the National Institutes of Health, and a subcontract from New York University under U.S. EPA Cooperative Agreement CR 827164.

This study has not been subjected to the U.S. EPA’s required peer and policy review. It does not necessarily reflect the views of the U.S. EPA, and no official endorsement should be inferred.

The authors declare they have no competing financial interests. Received 29 March 2005; accepted 25 August 2005.

Environmental Health Perspectives • VOLUME 113 | NUMBER 12 | December 2005 1741
pressure (Pope et al. 1999b). Linn et al. (1999) found no association of SaO2 and PM2.5 in a panel study of 30 subjects in Los Angeles, but DeMeo et al. (2004) found a reduction in oxygen saturation associated with PM2.5 in a 12-week repeated-measures study of 28 elderly Boston residents.

Changes in cardiac measures such as blood pressure and pulse rate, which are possible risk factors for cardiovascular morbidity and mortality, have been the focus of several PM panel studies. A study in Germany showed a consistent significant increase in blood pressure in adults in association with increased concentrations of total suspended particulates (TSP) at a central site (Ibadal-Mulli et al. 2001). Other studies also have shown increases in blood pressure with PM (Linn et al. 1999; Mar et al., 2005). Pope et al. (1999a) reported an association between PM10 and pulse rate; a 10 µg/m3 increase in the previous 1–5 day average PM10 was associated with an average increase of 0.8 beats per minute. Peters et al. (1999) found increases in pulse rate during an air pollution episode in Europe in January 1985. However, Mar et al. (2005) found decreases in heart rate associated with indoor and outdoor PM2.5 and PM10.

Therefore, based on the literature, there is some suggestion of associations between PM and changes in FeNO, spirometry, SaO2, blood pressure, and pulse rate. To determine whether changes in these health endpoints were associated with residential and personal PM and BC exposures, we conducted a panel study in Seattle, Washington, of 16 older subjects with COPD and/or asthma. This research was part of a multicity panel study designed to evaluate geographical differences in PM and cardiopulmonary health effects due to PM exposure. The study was conducted in New York City and Seattle. Seattle was chosen because it is known to have elevated wood smoke levels in winter. Our primary hypothesis was that airway inflammation in individuals with asthma and/or COPD would be associated with PM air pollution and BC, a measure shown to represent elemental carbon.

Materials and Methods

PM exposures and health effects were measured in this panel study of susceptible subjects in Seattle during the winter of 2002–2003. The study included 16 individuals with physician-diagnosed asthma, COPD, or asthma and COPD. Those individuals diagnosed with both asthma and COPD were grouped under COPD. A seventeenth subject (#2) did not participate in the full study period and was not included in the analyses. The health outcomes measured during the study were FeNO, spirometry, exhaled breath condensate, pulse oximetry, heart rate, blood pressure, symptoms, and medication use. Exhaled breath condensate and symptoms are not reported here. We collected PM2.5 and PM10 Harvard Impactor (HI; Air Diagnostics and Engineering, Inc., Naples, ME) 24-hr filter samples simultaneously at a central outdoor site, as well as outside and inside the subject’s home. Marple Personal Environmental Monitors for PM10 (MPREM10; MSP Corporation, Shoreview, MN) were worn to record personal exposure. We subsequently analyzed the filters for mass, light absorbance to estimate BC, and trace elemental compositions via X-ray fluorescence. Only mass and BC are reported here.

Study subjects. The participants were recruited from a community in north Seattle, ranging from 60–86 years of age, and were nonsmokers living alone or with other non-smokers. Each subject in the panel was asked to participate for a 12-day monitoring session. Approximately 75% of the subjects were prescribed inhaled corticosteroid therapy, and two were prescribed a leukotriene receptor antagonist (montelukast). Both of these anti-inflammation medications have been shown to prevent increases in FeNO in atopic subjects with asthma (Jones et al. 2002; Piccini et al. 2002). The remaining subjects were prescribed only inhaled albuterol as needed. Subjects filled out a questionnaire to describe their medical, residential, and occupational history before enrollment in the study. A second questionnaire was administered daily during the study period to record typical physical activity, time spent outdoors, home behavior, travel, and daily medication use. All subjects read and signed a consent form approved by the University of Washington Human Subjects Office.

Offline FeNO. Exhaled breath was collected according to American Thoracic Society recommendations for offline measurement (Slutsky et al. 1999); however, we collected only one sample per subject visit during the late morning of each day. Previous replicate measures with the same collection devices showed good agreement. The sample was collected daily in the subjects’ homes for up to 12 consecutive days. We collected exhaled breath before taking lung function measurements because deep inspirations may affect NO concentration (Deykin et al. 1998), and subjects were asked not to eat 1 hr before collection. The subjects were instructed to inhale to nearly total lung capacity and exhale through an offline collection device (Model 280i; Sievers Ionics, Boulder, CO). The subjects repeated this inhalation–exhalation cycle twice, and the third breath was collected into a nonreactive, self-sealing Mylar-like balloon. Subjects maintained a constant flow rate (0.35 mL/sec), inhaled NO-free air during the entire procedure, and exhaled with sufficient pressure (13 cm H2O) to close the epiglottis and prevent contamination of the airway NO sample by nasal NO. We collected samples at the same time of day (late morning) at their residences. NO was measured within 24 hr of collection using a chemiluminescent nitrogen oxide (NOx) monitor (model 280i; Sievers Ionics). Multiple NO concentrations from Mylar-like bags varied by < 2 ppb over a 24-hr period, consistent with that found by Jobis et al. (1999). The monitor was calibrated daily using zero air and 45 ppm NO.

Lung function and SaO2. Spirometry was performed according to American Thoracic Society recommendations (Crapo et al. 1995). The subjects performed the spirometry maneuvers during the technician visit. We measured FEV1, FVC, FEV1/FVC, PEF, and MEF (mid-expiratory flow). We recorded maximum forced expiratory maneuvers using diaphragm spirometers (SMI III Spirometer; Spirometrics Inc., Gray, ME). Subjects performed the maneuvers while sitting. Each subject was asked to perform three satisfactory blows, defined as FVC and FEV1, agreeing within 5% and a forced expiratory time exceeding 6 sec. No more than five blows were attempted. Height, weight, age, sex, and ethnicity were determined from subject’s questionnaire responses. Spirometers were kept at the subject’s home and calibrated just before the test session using 3-L calibration syringes (Ohio Medical Products; Airco, Inc., Madison, WI). The use of respiratory medication was recorded daily. Three times daily (morning, mid-day, and evening) the subjects sat at rest and placed the sensor of a pulse oximeter (Nellcor Model N-20P; Nellcor, Pleasanton, CA) on the left index finger. Date, SaO2, and pulse rate were recorded.

Cardiac measurements. Blood pressure was recorded, using the left arm while at rest, during the technician visits. The blood pressure cuffs (AND UA-767; A&D Medical, Milpitas, CA) were calibrated before and after the study period. Any cardiac medications used were recorded daily.

PM mass monitoring. We collected 24-hr PM2.5 and PM10 measurements during each 12-day session inside and outside the subjects’ residences and at a central agency site (Lynnwood) using HIs. Radiance Research (Seattle, WA) nephelometers provided continuous data on fine particles, comparable to PM1 (Liu et al. 2002). The indoor and outdoor PM concentrations were measured with single-stage inertial HIs and 37-mm Teflon filters for PM10 and PM2.5. One HI2.5–HI10 pair was located inside each home in the main activity room and connected to a pump (SP 280, Air Diagnostics Inc.). Another HI2.5–HI10 pair was located outside each home and connected to a pump (SP 280). The on and off flow rates were calibrated and recorded daily with a rotameter (150-nm Tube 604; Cole-Parmer...
Instrument Co., Vernon Hills, IL). All HI sampling periods were for 24 hr (approximately 1100 hr to 1100 hr) at a flow rate of 10 L/min. Our research group has previously evaluated the performance of continuous PM monitors ( nephelometers) and HI s used in the context of a panel study (Liu et al. 2003).

Simultaneous data also were collected with a MPEM_{10} during the study period (24 hr for 12 consecutive session days). The MPEM_{10} was connected to a personal pump (400S: BGI, Inc., Waltham, MA) with a mass flow controller operated at 4 L/min. Each subject carried an MPEM_{10} in the breathing zone for 24 hr, except while sleeping or showering. The monitor was attached to the shoulder strap of either a backpack or a fanny pack that contained the air pump. When the monitor was not worn, it was placed on an elevation of 3–5 ft (e.g., on a table) close to the subjects. Field technicians visited the subjects daily to calibrate the pumps with a rotameter and to record on and off flow rates and change samplers.

We weighed the filters before and after sample collection for particle mass concentration. All filter weights were measured in either duplicate or triplicate using an electronic ultramicrobalance (UMT2: Mettler Toledo, Greifensee, Switzerland). The filters were calibrated for at least 24 hr before weighing. We performed both equilibration and weighing inside a controlled environmental chamber with constant relative humidity (34.7°C ± 2.5%) and temperature (22.4°C ± 1.9%) (Allen et al. 2001). Standard protocols included the use of field blanks, filter-lot blanks, laboratory blanks, and externally certified standard weights for all gravimetric analyses for quality assurance and quality control purposes.

Relative humidity, outdoor temperature, NO, and NO_{2} concentrations were monitored continuously at the Beacon Hill central site by the Washington State Department of Ecology.

**Black carbon measurements.** We estimated BC, a measure shown to represent EC from motor vehicles and woodstoves in Seattle (Larson et al. 2004), using an integrated plate reader (Lin et al. 1973). It is generally agreed that the major contributor to light absorption by airborne particles is BC, and levels of BC can easily be measured by this nondestructive optical technique. The method derives absorption from the change in light transmission through a Teflon filter on which particles have been collected. We analyzed the filters from the HIs for BC (wavelength of 525 nm) after the mass measurements. The integrated plate reader was re-zeroed with a blank filter between measurements. The light absorption coefficient, $b_{\text{ap}}$, was computed using the amount of light transmitted through this exposed filter, the amount transmitted through the same filter before sampling, and the volume of air that passed through the filter. We used a previously derived association between $b_{\text{ap}}$ and EC in Seattle to quantify the BC concentrations (Larson et al. 2004).

**Statistical analysis.** We hypothesized that increases in PM_{2.5} and BC are associated with increases in FENO. We analyzed within-subject, within-session associations between FENO and air pollution metrics using a linear mixed effects model with random intercept, controlling for age, relative humidity, and temperature. Subjects were stratified by health status in the FENO, spirometry, and SaO_{2} analyses. We put use of cardiac medications into the model as an interaction term for the blood pressure and pulse rate analyses. The model included terms for within-subject, within-session (12-day monitoring period) effects; within-subject, between-session effects; the confounding variable of temperature; and relative humidity. Our primary interest was the within-subject, within-session effects of PM_{2.5} and BC on FENO levels. Our numerous exploratory analyses, the within-subject, within-session effects of PM_{2.5}, PM_{10}, and BC on spirometry, SaO_{2}, blood pressure, and pulse rate required use of the Bonferroni test for multiple comparisons. The Bonferroni test indicated a value of $p < 0.0001$ was significant. Therefore, for these analyses we chose $p < 0.0001$ as our criteria for statistical significance. We used STATA software (Stata Corp., College Station, TX). The model used was as follows:

$$\text{FE NO} = B_{0} + B_{1}(X_{\text{adj}} - \bar{X}) + B_{2}X_{i} + B_{3}\text{med} + B_{4}\text{med} + \text{X}_{\text{adj}} - \bar{X} + B_{6}\text{temp},$$

where $X_{\text{adj}}$ is the PM_{2.5} reading for individual $i$ on day $d$; $\bar{X}$ is the mean PM_{2.5} reading for a subject; and $\text{med}$ is an indicator for medication use (constant for each subject).

**Results**

**Subject characteristics.** Characteristics of the 16 subjects are given in Table 1. On average, the subjects spent 88% of their time indoors at home, 3% of their time in transit, and 9% of their time indoors away from home. Four subjects reported having received both a doctor’s diagnosis of asthma and of COPD.

**Airborne concentration measurements.** The measured concentrations and interquartile ranges of PM_{10}, PM_{2.5}, and BC are presented in Table 2 for all the subjects, for the 7 subjects with asthma alone, and for the 9 subjects with COPD. At the fixed-site monitor, the overall 24-hr average PM_{2.5} was 14.0 µg/m³, the 24-hr minimum was 1.5 µg/m³, and the 24-hr maximum was 44 µg/m³. At the same site the overall 24-hr average PM_{10} was 18.0 µg/m³, the 24-hr minimum was 2.5 µg/m³, and the 24-hr mean of PM_{10} was 12.0 µg/m³. The overall 24-hr average PM_{10} was 18.0 µg/m³, the 24-hr minimum was 2.5 µg/m³, and the 24-hr mean of PM_{10} was 12.0 µg/m³.
maximum was 51 µg/m³. The overall 24-hr average BC was 7.2 µg/m³, the 24-hr minimum was below detection limits, and the 24-hr maximum was 2.6 µg/m³.

**Exhaled NO.** A total of 179 midday breath samples were collected during the 12-day monitoring periods. Average FE\_NO\_levels are shown in Table 3. The mean FE\_NO\_levels were higher for those with COPD (25.4 ppb) than for those with asthma (19.2 ppb) or COPD and asthma (16.5 ppb).

In those subjects with asthma, a 10 µg/m³ increase in outdoor PM\_2.5 and PM\_10\_relate to each subject session average, was associated with a 4.2 ppb increase in FE\_NO\_95% CI, 1.3–7.1; \( p = 0.004 \)) and 5.9 ppb (95% CI, 2.9–8.9; \( p = 0.000 \)) increase in FE\_NO, respectively. There was no association between FE\_NO\_and the 24-hr measures of indoor PM\_2.5 or PM\_10. A 1 µg/m³ increase in outdoor, indoor, and personal BC, relative to each subject session average, was associated with a 2.3 ppb increase in FE\_NO\_95% CI, 1.08–3.57; \( p = 0.000 \)), a 4.0 ppb increase in FE\_NO\_95% CI, 2.02–5.91; \( p = 0.000 \)), and a 1.2 ppb increase in FE\_NO\_95% CI, 0.17–2.22; \( p = 0.02 \)), respectively (Table 3). No significant association was found between PM or BC and changes in FE\_NO in subjects with COPD. The effect levels and confidence intervals are given in Table 3.

**SaO\_2, blood pressure, and pulse rate.** No associations were observed between air pollution and SaO\_2, blood pressure, or pulse rate in this study.

**Discussion**

This study showed an association between FE\_NO\_in elderly subjects with asthma and indoor and outdoor BC. Increases in FE\_NO\_also were associated with outdoor PM\_10 and PM\_2.5 in these same subjects. Results of this study are consistent with our earlier study of children with asthma who were not on corticosteroid therapy (Koenig et al. 2003). That study showed an increase of approximately 4 ppb FE\_NO\_associated with a 10 µg/m³ increase in indoor, outdoor, personal, and central site PM\_2.5 in Seattle. Finding a similar magnitude of response in the two different groups (children and elderly with asthma) strengthens the importance of this finding. Results of the present study also are consistent with other earlier studies in Seattle showing that hospitalizations for asthma (Sheppard et al. 1999) as well as increases in asthma symptoms and increased use of rescue medications (Yu et al. 2000; Slaughter et al. 2004) are associated with fine particles in Seattle.

Our data suggest that exposure to PM\_10 may play an important role in asthma exacerbation. This significant association between FE\_NO\_and PM\_10 was not surprising, especially for subjects with asthma that have narrowed airways, as the thoracic coarse particles deposit preferentially in the larger bronchial airways and these airways may be the ones with the greatest inflammation potential (U.S. EPA 2004). The observed association is supported by studies that have linked PM\_10 to pulmonary inflammation in animal models (Li et al. 1996) and the induction of inductive nitric oxide synthase in human bronchial epithelial cells (Martin et al. 1997).

Other studies (Steerenberg et al. 1999; Tunniffille et al. 2003; van Amstderam et al. 1999) have also reported positive associations between FE\_NO\_and ambient exposures to air pollutants in community-based studies. Adarkiewicz et al. (2004) reported that an increase in the 24-hr average PM\_2.5 concentration of 17.7 µg/m³ was associated with a 1.45 ppb increase in FE\_NO\_in elderly subjects with asthma and COPD in a panel study in Steubenville, Ohio. Fischer et al. (2002) reported a 1-day and 2-day lag association between FE\_NO\_and PM\_10, black smoke, and NO. In contrast, no increase in FE\_NO\_was seen in adult subjects with asthma after exposure to concentrated coarse particles (Gong et al. 2003) or ultrafine particles (Pietropaoli et al. 2004). Several controlled ozone exposure studies have assessed FE\_NO\_in atopic subjects with asthma (Newson et al. 2000; Nightingale et al. 1999) and healthy subjects (Olin et al. 2001), but none has found an association.

We found that FE\_NO\_was associated with PM air pollution in study participants with asthma but not those with COPD. It is interesting that five of the seven subjects with asthma were using inhaled corticosteroids, which has been associated with mitigation of eNO in air pollution studies (Koenig et al. 2003) and clinical settings (Deykin et al. 1998). This finding contrasts with that of a study of elderly subjects by Adarkiewicz et al. (2004) that found a PM\_2.5 response in subjects with COPD but not asthma, although there was some overlap in the study population and medications were not recorded. In our study, levels of FE\_NO\_on average, were higher in COPD than asthma subjects. Exhaled NO in stable COPD has been found to be lower than in nonsmoking asthmatics (Kharitonov et al. 1995), but patients with unstable COPD have higher NO levels than ex-smokers with COPD (Maziak et al. 1998).

BC may more closely identify the sources of PM than standard measures of mass concentration. The contribution of BC to total PM varies geographically and temporally due to the distribution of the combustion sources that produce BC. Although BC is a major component of diesel exhaust, it is also a major component of particles produced by burning vegetation (Conny and Slater 2002; Hobbs et al. 2003; Mayol-Braceno et al. 2002; Fosfai et al. 2004). Recent source apportionment studies in Seattle found that burning vegetation and mobile sources are major contributors to PM\_2.5 (Maykut et al. 2003) and that burning vegetation is the dominant contributor to variations in the day-to-day BC in the winter (Larson et al. 2004). Burning vegetation, and to a lesser extent, mobile sources, may therefore be responsible for the observed increases in FE\_NO\_associated with BC.

It is somewhat surprising that we did not find an association between standard spirometry measures and association with PM\_2.5, PM\_10, and BC. An earlier study completed in Seattle during the wood-burning season (Koenig et al. 1993) showed that spirometry, specifically FVC and FEV\_1, decreased in association with increases in particulate matter air pollution in children with asthma. Another study, in Vancouver, British Columbia Canada, showed a slight but not statistically significant decrease in daily FEV\_1 change in subjects with COPD was associated with increase in PM\_2.5 (Brauer et al. 2001). In three separate longitudinal diary studies, decreases in PEF were shown to be associated with increased levels of PM\_2.5 (Schwartz and Neas 2000).

Our exploratory hypotheses were that increases in PM\_2.5 and BC are associated with decreases in spirometry (FEV\_1, MEF) and SaO\_2 and with increases in blood pressure and pulse rate. In our study, no significant associations were seen between these health measures and PM\_2.5, PM\_10, or BC (indoor, outdoor, personal). Some studies have found that PM\_10 and PM\_2.5 both appear to affect lung function.

**Table 3. Associations between FE\_NO\_ (ppb) and 24-hr average PM\_2.5 and PM\_10 (µg/m³) in subjects with asthma and COPD.**

<table>
<thead>
<tr>
<th>Pollution Location</th>
<th>Asthma (n = 7)</th>
<th>COPD (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( B )</td>
<td>( p)-Value</td>
</tr>
<tr>
<td>PM_2.5 Indoor</td>
<td>3.89</td>
<td>0.10</td>
</tr>
<tr>
<td>PM_10 Indoor</td>
<td>4.23</td>
<td>0.004*</td>
</tr>
<tr>
<td>PM_2.5 Outdoor</td>
<td>3.81</td>
<td>0.11</td>
</tr>
<tr>
<td>PM_10 Outdoor</td>
<td>5.87</td>
<td>0.000*</td>
</tr>
<tr>
<td>PM_2.5 Personal</td>
<td>0.66</td>
<td>0.29</td>
</tr>
<tr>
<td>PM_10 Personal</td>
<td>3.97</td>
<td>0.000*</td>
</tr>
<tr>
<td>PM_2.5 Outdoor</td>
<td>2.32</td>
<td>0.000*</td>
</tr>
<tr>
<td>PM_10 Outdoor</td>
<td>1.20</td>
<td>0.02*</td>
</tr>
</tbody>
</table>

Values for PM\_2.5 and PM\_10 are given as change per 10 µg/m³; values for BC = are given as change per 1 µg/m³. *Statistically significant.
in asthmatics (U.S. EPA 2004); however, many of the studies experienced higher mean PM concentrations (in the range of 50 µg/m³) than were experienced by subjects in this study. The lack of significant associations between SaO₂ and PM has also been observed in other studies (Linn et al. 1999; Mar et al. 2005). In contrast to studies that have reported increases in blood pressure (Ibald-Mulli et al. 2001; Linn et al. 1999, Mar et al. 2005) and pulse rate (Peters et al. 1999; Pope et al. 1999a) with exposure to PM. Our study results are consistent with those of a larger panel study in Seattle (Mar et al. 2005), but that study did see minor decreases in pulse rate in healthy subjects. Yet another study did find some changes in ectopic beats in subjects with asthma (Jackson et al. 1997, 1999). Linn et al. 1999; Mar et al. 2005) and pulse oximetry in elderly subjects. Epidemiology 16:681–686.

In conclusion, these data implicate combustion-derived PM as a risk factor for respiratory and cardiovascular health effects of particulate air pollution. Environ Health Perspect 111:909–918.


