Smoke, Dust & Haze

EPA NW Research Center for Particulate Air Pollution and Health Newsletter

Spring 2004

Which is more toxic? PM or gaseous pollutants?

New study seeks answer and evaluates HEPA filter effectiveness

Puget Sound Clean Air Agency

Seattle skyline at increasing levels of particulate matter.

This winter, investigators from the Northwest Center for Particulate Air Pollution and Health (NW PM Center) began a new study addressing microscale intervention of air quality on health. Their goal is to help differentiate adverse health effects associated with particulate matter (PM) from those associated with gaseous air pollutants.

Two research needs are to identify mechanisms of action of particulate matter and to identify more exactly whether it is particulate matter or gaseous pollutants that is the more toxic component of air pollution.

The study is entitled "The effect of $PM_{2.5}$ on exhaled nitric oxide: An intervention field study." Investigators include Jane Q. Koenig, PhD; Timothy V. Larson, PhD; Christopher Simpson, PhD; Karen Jansen, MS; and Eva Dale, graduate student.

They are examining whether intermittent use of a high-efficiency particle arrestance (HEPA) filter, which reduces particulate matter exposure, will be associated with lower exhaled nitric oxide (eNO) values in subjects with respiratory disease.

This study may demonstrate whether HEPA filters protect asthmatic subjects from air pollution-induced airway inflammation. HEPA filters remove an average of 99.97% of small (0.3 micrometer) airborne particles passing through the device, but do not filter out gases. Thus, the study also may help interpret the role of fine particles versus outdoor gaseous pollutants in the production of airway NO.

Exhaled nitric oxide measurements offer opportunities to carry out more mechanistic studies in the field. PM exposure is associated with increased eNO levels in children with asthma (Koenig et al, 2003) and adults with respiratory disease (Adamkiewicz et al, 2004). Unless an electrostatic filter is used, more than three-fourths of ambient $PM_{2.5}$ infiltrates into homes (Allen et al, 2003). An association between $PM_{2.5}$ and increased eNO lung values does not necessarily prove that $PM_{2.5}$ is the responsible agent. The responsible agent may be a pollutant that is highly correlated with $PM_{2.5}$.

This is one of the first studies to look at microscale interventions of air quality on health. Previous studies of changes in community air quality have shown associations between reduced fuel consumption and death (Clancy et al, 2002), decreases in the number of acute care asthma events in children associated with transportation reduction strategies in Atlanta during the Olympic games (Friedman et al, 2001), and decreases in respiratory symptoms in reunified Germany (Heinrich et al, 2002).

Devon DeLapp

Karen Jansen

Specific Aims

- Test the HEPA filter in a diesel facility to determine its effectiveness in removing PM and its ineffectiveness in removing gases
- measure daily eNO levels in subjects for month-long sessions, with filters being added to or removed from the HEPA device every four days
- measure $PM_{2.5}$ inside and outside subjects' homes to assess effect of HEPA filters on indoor $PM_{2.5}$
- further evaluate the role $PM_{2.5}$ plays in association with changes in airway eNO

Background

High-Efficiency Particle Arrestance (HEPA) filters

Portable air filter effectiveness is determined by the device's efficiency and the volume of air handled. While there is no universally accepted method for categorizing device effectiveness, some investigators express air filter effectiveness in terms of clean air delivery rate (CADR). Based on the CADR system, consumers may use room size to choose the recommended filtration device. Using a higher- than-recommended CADR number should result in faster room air filtration. While HEPA filters are used primarily in an occupational setting, they are also available in compact form for residential use.

Exhaled nitric oxide

Exhaled nitric oxide (eNO) has become an interesting assessment tool for documenting changes in airway inflammatory status (Kharitonov and Barnes, 2000). Collection of exhaled air is simple, noninvasive, and relatively inexpensive. Endogenous NO is produced when NO synthase (NOS) catalyzes a reaction converting L-arginine to L-citrulline and NO. Several types of NOS are known to exist endogenously, with inducible NOS (iNOS) appearing to play an important role in lower airway inflammation (Gaston et al, 1994).

Many types of cytokines active in airway inflammation, including interferon-gamma, tumor necrosis factor alpha, and interleukin1-beta, are induced when airway inflammation occurs. These cytokines induce iNOS to produce NO (Yates, 2001). Exhaled nitric oxide is generated at the same time as several very reactive endogenous reactive nitrogen species (RNS) likely to cause inflammation.

Although several health endpoints can be used to assess respiratory effects of PM, eNO is considered to be a reproducible, noninvasive indicator of airway inflammation (Kharitonov and Barnes, 2000). It has also been shown that healthy subjects respond with increased levels of exhaled endogenous NO on days with high levels of ambient air pollution (Van Amsterdam et al, 1999).

Susceptibility factors*:* Subjects with asthma and chronic obstructive pulmonary disease (COPD) have elevated levels of eNO when compared with healthy subjects. These increased amounts of eNO vary with disease activity and in response to anti-inflammatory therapy (Jones et al, 2001). While all populations have shown increased levels of eNO when exposed to air pollution including PM, asthmatic subjects and those with COPD have much higher baseline levels of eNO than healthy subjects. These sensitive populations also have been shown to have increased levels of eNO in association with exposure to $PM_{2.5}$. In the clinical setting, eNO is suggested as a tool for diagnosing asthma (Jones et al, 2001).

Study design

The study was conducted during the winter heating season when PM air pollution is highest in Seattle. The winter heating season is from October through February. This season also is associated with frequent periods of air stagnation.

Subject selection

During the 2003 and 2004 heating seasons, we recruited five adult subjects for one month each. To improve the power of the study, we hoped to recruit subjects who showed increased eNO associated with $PM_{2.5}$ exposure in previous studies or who had conditions such as asthma or COPD which made them more likely to be responders. We hoped to exclude people who were on corticosteroids or other medi-cation that will potentially alter airway inflammation and eNO levels. In this study, four of the five subjects were using corticosteroids.

HEPA filtration

A compact HEPA filter device was placed in homes for month-long sessions. The HEPA filter was added to or removed from the filtration device every four days. The filter/no-filter status was blinded to the subject. The HEPA filter was placed in the room where the subject would spend the most time before the eNO sample was obtained. We tried to include enough sessions to have adequate statistical power; session order was determined at random.

PM exposure evaluation

$PM_{2.5}$ data were collected inside and directly outside the home using a light-scattering, particle sensing device called a nephelometer. The device provides hourly data and requires minimal maintenance with an 18-day capacity of data logging. Data were downloaded from the device weekly. An additional nephelometer was placed in one of the subject's other rooms, likely a bedroom, to show the correlation of $PM_{2.5}$ between rooms in the home. The range of sensitivity to the size of particles can be adjusted on the nephelometer. In general, the nephelometer is best at identifying particles between 1 and 2.5 micrometers in diameter. A recent study in Seattle showed that gravimetric measures of $PM_{2.5}$ and nephelometric measures are the same inside and outside of residences (Liu et al, 2002).

Exhaled nitric oxide collection

Three replicate eNO samples were collected in modified Mylar balloons three times daily at the subject's home and analyzed in the lab, in accordance with American Thoracic Society standards (1999), for a total of 216 samples for each subject. Breath samples were collected into Mylar bags, which have been shown to be nonreactive. Sievers eNO collection devices were used to scrub out any ambient NO from the inhaled air. The devices also direct airflow as flow rate is associated with eNO concentration. Subjects cannot consume food or water at least one hour before sample collection. Subjects were coached through collection by a technician.

Exhaled nitric oxide measurement

The bags were returned to the lab and analyzed within 24 hours using the Sievers 2801 chemiluminescent analyzer. This is the standard method for offline, as well as online, analysis of eNO samples. Before each analysis, the Sievers NO analyzer was calibrated to zero ppb NO air and 45 ppm NO air.

Devon DeLapp

Eva Dale

Daily diary

Subjects kept a time-activity log of their activities including physical activity; time spent outdoors; the estimated number of hours in the room containing the HEPA filter; window openings; gas stove or heater use; time around tobacco smoke, wood smoke, or burning food; medication use; and symptoms. Each day, the technician verbally reviewed the previous 24-hour written entry with the subjects to improve accuracy.

Data analysis

Outcome data consisted of three measures of $PM_{2.5}$—one continuous monitor inside the home, one outside the home, one in the comparison room—and the daily eNO samples. Presence or absence of a HEPA filter was factored in. The model included terms for within-subject effects, within-session effects, and within-session effect of $PM_{2.5}$. Our primary interest was the within-subject and within-session effect of $PM_{2.5}$. STATA software was used.

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Exhaled nitric oxide in subjects with respiratory disease is associated with levels of $PM_{2.5}$ and black carbon in Seattle

—Karen Jansen

The association between particulate matter (PM) and health is primarily based on large epidemiological studies. Panel studies allow individual measurements of exposure and health in selected susceptible subjects. One such multi-city panel study, designed to evaluate geographical differences in PM, was recently conducted in New York City, Irvine, CA, and Seattle, WA.

Indoor, outdoor, personal, and central site $PM_{2.5}$ were monitored with Harvard impactors or Marple PEMS. Light absorbing carbon (LAC), a measure shown in Seattle to represent black carbon from motor vehicles and woodstoves, was measured using an integrated plate reader.

In Seattle, the NW PM Center used exhaled nitric oxide (eNO) to document airway inflammation in sixteen subjects aged 60 to 86 years. Subjects with asthma (N = 7) or COPD (N = 9) participated during the winter of 2002-3. An offline exhaled breath sample was collected daily from each subject for twelve days during potentially high PM periods (24 hour maximum 44 ug/m^3).

Within-subject associations between eNO and air pollution metrics were analyzed using a linear mixed effects model with random intercept, controlling for age, relative humidity, and temperature.

For subjects with asthma, a 10 ug/m^3 increase in outdoor $PM_{2.5}$ was associated with a 4.2 ppb increase in eNO (95% CI: 1.3, 7.1) and a 1 ug/m^3 increase in outdoor, indoor, and personal LAC was associated with a 2.3 ppb increase in eNO (95% CI: 1.08, 3.57), a 4.0 ppb increase in eNO (95% CI: 2.02, 5.91), and a 1.2 ppb increase between PM or LAC and changes in eNO in subjects with COPD. Older subjects with asthma may be more susceptible to PM-induced airway inflammation than their counterparts with COPD. Light absorbance is useful for looking at associations between constituents of PM and health outcomes.

Using light as a tool

We can measure the amount of black or graphitic carbon—often referred to as elemental carbon—in the air by taking advantage of its light-absorbing capabilities.

It is generally agreed that black carbon is the major contributor to light absorption by airborne particles. This light absorption can easily be measured by a nondestructive optical technique known as the integrating plate method (IPM).

Particles are first collected on a 35mm filter, which is inserted between a light source and a detector. If we know 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 can deduce the concentration of black carbon in the air during sampling.

The contribution of black carbon to light extinction varies by time and place, based on the distribution of the combustion sources that produce black carbon. Because wood-burning fireplaces and diesel vehicles are major sources of black carbon, areas with large numbers of these two sources would be expected to have more black carbon in the atmospheric aerosol than other locations. In areas where wood burning is significant, more particulate black carbon would be expected in winter than in summer.

As part of our panel studies, we have measured the concentration of black carbon in airborne particles using these methods. We find that measures of light absorbing carbon (LAC) are associated with increases in exhaled nitric oxide in adult subjects with asthma who participated in an exposure assessment/health effects study in Seattle in 2002–03 (Fields, 2003; Jansen et al, 2004).

Similar changes were not seen in the adult subjects who had chronic obstructive pulmonary disease (COPD) but not asthma. Use of inhaled corticosteroid medication by the subjects with asthma did not appear to modify the relationship between LAC and eNO as opposed to findings in children with asthma where inhaled corticosteroid use mitigated the eNO response.

For further reading

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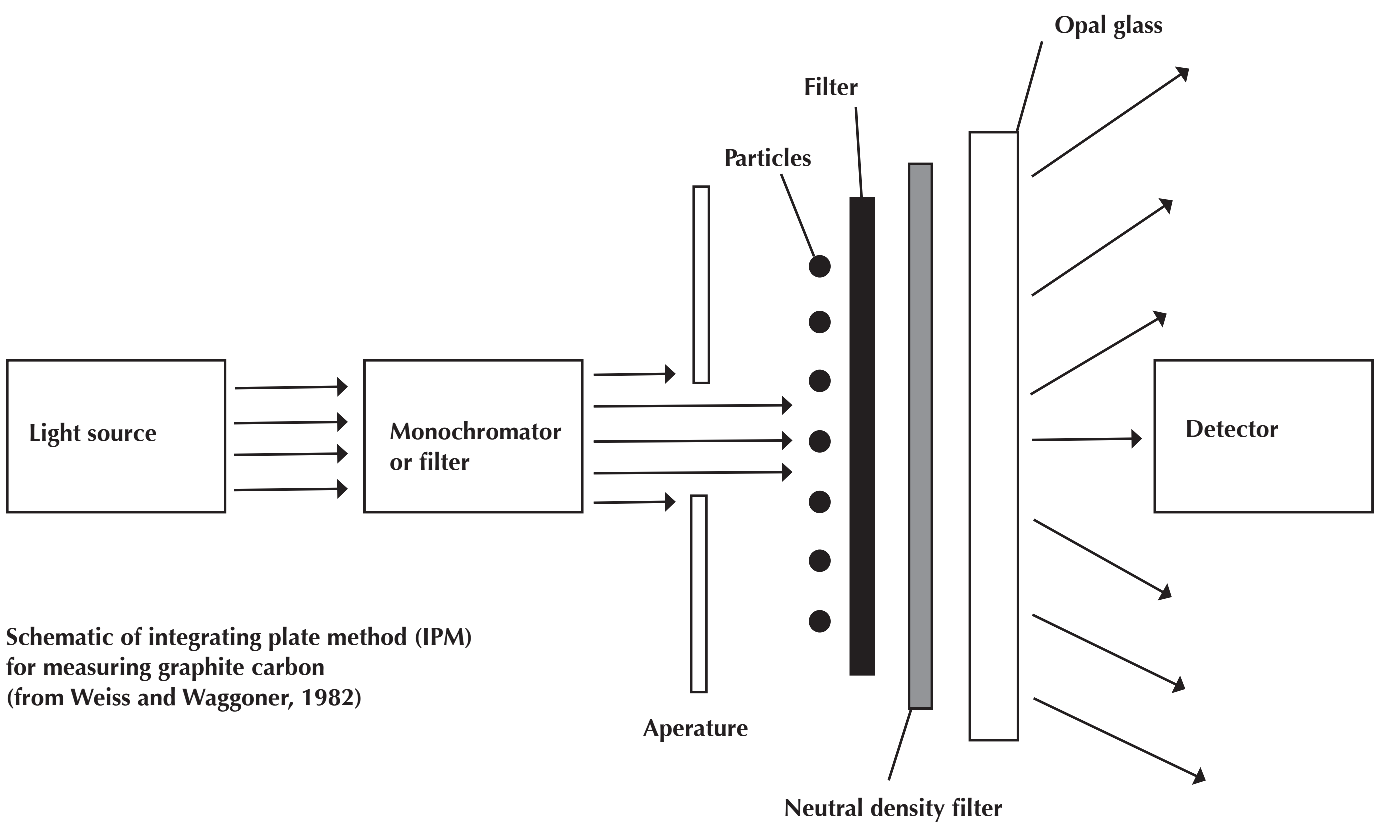

Schematic of integrating plate method (IPM) for measuring graphite carbon (from Weiss and Waggoner, 1982)

2003–04 news, events & publications

Events & Visitors

2003

February

Seminar: Dick Hoskins, PhD, geographer and director of the GIS (Geographic Information Systems) and Spatial Epidemiology group, Office of Epidemiology, Washington State Department of Health, *Geographical information and spatial epidemiology*

April

Seminar: Teal Hallstrand, MD, MPH, assistant professor, Division of Pulmonary & Critical Care, UW, *Functional genomic analysis of exercise-induced bronchoconstriction*

May

Seminar: Lillian Calderon-Garciduenas, PhD, Department of Environmental Sciences & Engineering, University of North Carolina-Chapel Hill, and National Institute of Pediatrics, Mexico City, *Air pollution: Noses, lungs, hearts, and brains*

Seminar: Bert Brunekreef, PhD, professor of Environmental Epidemiology, Institute for Risk Assessment Sciences, The Netherland's Utrecht University, *Dutch cohort studies on air pollution and health*

Seminar: Chris Simpson, PhD, assistant professor, Department of Environmental & Occupational Health Sciences (DEOHS), UW, *Organic molecular markers in air pollution research*

Seminar: Hao Liu, Biostatistics PhC, *Source apportionment and health effect model*

Workshop: Isabelle Romieu, PhD, Pan American Health Organization, México; David Bates, MD; and Jonathan Samet, MD. Informal review of current findings of the NW PM Center

PM/Cardiovascular Mortality Meeting, convened by Joel Kaufman, MD, MPH, associate professor DEOHS, UW, with David Bates, MD; Bert Brunekreef, PhD; Annette Peters PhD, Ralph Delfino, MD, MPH; Joel Schwartz, PhD; Doug Dockery, PhD; Jane Koenig, PhD; Tim Larson, PhD; Lianne Sheppard, PhD; Thomas Lumley, PhD; and Jeff Sullivan, MD

July

Seminar: Tim Takaro, MD, MPH, acting assistant professor, Medicine and DEOHS, UW, *Uses of proximity to roadways in air pollution research*

September

Public Meeting, Fall 2002 agricultural burning health effects study in Pullman, presentation of study implementation by Candis Claiborn, PhD, Department of Civil & Environmental Engineering, WSU, with Joel Kaufman and David Kalman, professor and chair, DEOHS, UW

October

Seminar: Stephan van Eeden, MD, PhD, FRCPC, assistant professor, Department of Internal Medicine, University of British Columbia, *Blood vessels and particulate matter air pollution*

Seminar: Christopher Gill, PhD, professor of Chemistry, Malaspina University-College, Nanaimo, British Columbia, and codirector, Applied Environmental Research Laboratory, *Membrane introduction mass spectrometry*

2004

January

Seminar: David Kittelson, PhD, professor, Department of Mechanical Engineering, and director, Center for Diesel Research, University of Minnesota, *Understanding nanoparticles from combustion*

February

Seminar: Sverre Vedal, MD, MPH, *Particulate matter health effects: What now?*

Seminar: Kim Oanh, PhD, Asian Institute of Technology, Thailand, *Preliminary findings of the Asian regional air quality monitoring network (AIRPET) on PM level, composition, and source apportionment*

March

Seminar: Sarah L. Rees, PhC, Carnegie Mellon University Department of Engineering & Public Policy, *Two case studies in air quality: Performance of the federal reference method for fine particulate matter and air quality impacts of light duty diesel vehicles*

Presentations

2003

March

PM 2003/AAAR 4th PM Colloquium, Pittsburgh PA, Tim Larson, *Source apportionment of personal exposure to $PM_{2.5}$ using CMB and PMF,* and Joellen Lewtas, PhD, USEPA, *Development of urinary metabolite biomarkers to assess population exposure to $PM_{2.5}$ from various combustion sources*

April

Atmospheric Sciences Colloquium: Jane Koenig, *Assessing health effects of air pollutants*

AAAR PM Meeting, Pittsburgh, PA, Robert Elleman, Atmospheric Sciences graduate student, *CMAQ PM modeling for the Pacific Northwest 2001 (PNW2001) Field Campaign*

May

American Thoracic Society Annual Conference, Karen Jansen, MS, research scientist, DEOHS, UW, *The use of breath condensate as a non-invasive measure of airway inflammation in field studies*

September

International Society for Polycyclic Aromatic Compounds, Amsterdam, The Netherlands, Joellen Lewtas, *Evaluation of urinary PAH metabolites as biomarkers of exposure to $PM_{2.5}$ from combustion sources*

October

CMAQ Users' Meeting, ResearchTriangle Park, NC, Robert Elleman, *Comparison of ACM and MRF Boundary Layer Parameterizations in MM5*

November

Malaspina Univ. College, Nanaimo, BC, Chris Simpson, *Chemical speciation and source apportionment of particulate air pollution*

December

University of Georgia, Athens GA, Chris Simpson, *Molecular tracers for woodsmoke, and a urinary biomarker for woodsmoke exposure*

6th National Environmental Public Health conference, Atlanta, GA, Chris Simpson, *Molecular tracers for woodsmoke, and a urinary biomarker for woodsmoke exposure*

2004

January

Conference: Diesel Exhaust & Human Health: Current Scientific & Policy Issues, Joel Kaufman, cosponsored by American Lung Association of Washington and the UW Pediatric Environmental Health Specialty Unit

UBC-UW joint Conference on Occupational and Environmental Health, Semiahmoo, Chris Simpson, *Molecular tracers for woodsmoke, and a urinary biomarker for woodsmoke exposure*

2004 American Meteorological Society Annual Meeting, Seattle, Robert Elleman, *Analysis of CMAQ PM modeling during the Pacific Northwest 2001 (PNW2001) Field Campaign*

Honors

Sally Liu, ScD, DEOHS, UW, 2003 International Society of Exposure Analysis Joan Daisey Award for Outstanding Contributions to the Science of Human Exposure Analysis by a Young Scientist

Air Toxics Team, EPA Region X recognition of their "outstanding work in the field of air toxics," members include Chang-fu Wu, Timothy Larson, Alison Cullen, and Sally Liu

Holly Janes, Biostatistics graduate student, won the student paper competition from the Environmental Statistics Chapter at the Joint Statistical Meetings, the largest gathering of statisticians held in North America. The paper, *Overlap bias in the case-crossover design, with application to air pollution exposures,* will be published soon in *Statistics in Medicine*

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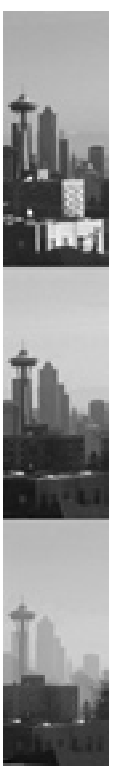
Smoke, Dust & Haze



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