

# THE SECOND COLLOQUIUM ON PARTICULATE AIR POLLUTION & HUMAN MORTALITY & MORBIDITY

(May 1-3, 1996, Park City, UT)

## REPORT

to

The California Air Resources Board  
Contract Number 95-323  
(4/1/96 - 3/30/97)

The Air Pollution Health Effects Laboratory,  
Department of Community and Environmental Medicine, and  
Center for Occupational and Environmental Health,  
College of Medicine, University of California,  
Irvine, CA 92697-1825

October, 1996

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## I. INTRODUCTION

# REPORT ON THE SECOND COLLOQUIUM ON PARTICULATE AIR POLLUTION AND HUMAN MORTALITY AND MORBIDITY

Prepared and Edited by : Robert F. Phalen, Richard C. Mannix and Marie C. Tonini

The material in this document was originally prepared for the Second in Colloquium on Particulate Air Pollution and Human Mortality and Morbidity, held May 1-3, 1996, in Park City, Utah. The meeting was sponsored by the Centers for Disease Control and Prevention/NIOSH, and the California Air Resources Board, with co-sponsorship by the U.S. Environmental Protection Agency, and the Centers for Occupational and Environmental Health (Irvine and the University of Utah), the Department of Community and Environmental Medicine--University of California, Irvine, the South Coast Air Quality Management District, the Utah Department of Environmental Quality, Geneva Steel, the Health Effects Institute, the American Automobile Manufacturers Association, and the American Iron and Steel Institute.

The included material covers the colloquium program (104 presentations), all 95 abstracts, and 40 papers prepared for the Colloquium, as well as 10 session summaries and 7 unsolicited commentaries. The material in these Proceedings has not been peer-reviewed. Therefore, the upcoming special issue of Applied Occupational and Environmental Health should be used if one is interested in those papers that were sent through peer-review. For more information concerning this document, please contact:

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## II. PROGRAM

### SECOND COLLOQUIUM ON PARTICULATE AIR POLLUTION AND HUMAN MORTALITY AND MORBIDITY

May 1-3, 1996  
Park City, Utah

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| Session 1:  | Investigational Methods: Strengths & Limitations |
| Session 2:  | Epidemiological Findings                         |
| Session 3:  | Epidemiological Findings (Poster Session)        |
| Session 4:  | Mechanisms of Injury (Poster Session)            |
| Session 5:  | Particle Deposition & Clearance (Poster Session) |
| Session 6:  | Exposure Assessment & Sampling (Poster Session)  |
| Session 7:  | Epidemiological Findings                         |
| Session 8:  | Particle Deposition & Clearance                  |
| Session 9:  | Indoor, Total & Occupational Exposures           |
| Session 10: | Science & Public Policy                          |
| Session 11: | Mechanisms of Injury & Extrapolation             |
| Session 12: | Exposure Assessment & Sampling                   |
| Session 13: | Epidemiological Findings                         |
| Session 14: | Mechanisms of Injury                             |
| Session 15: | Epidemiological Findings & Methods               |
| Session 16: | Exposure Assessment & Sampling                   |
| Session 17: | Exposure Assessment & Sampling                   |
| Session 18: | Indoor, Total & Occupational Exposures           |
| Session 19: | Knowledge Gaps                                   |
| Session 20: | Colloquium Topical Wrap-up                       |

#### Organized by:

Rocky Mountain Center for  
Occupational and Environmental Health  
Department of Family & Preventive Medicine  
University of Utah

Department of Community  
and Environmental Medicine  
and Center for Occupational  
and Environmental Health  
University of California, Irvine

# 2ND COLLOQUIUM ON PARTICULATE AIR POLLUTION & HEALTH PROGRAM

**WEDNESDAY, MAY 1, 1996**

8:00 a.m. INTRODUCTION TO THE COLLOQUIUM: Jeffrey Lee & Robert Phalen

Platform Sessions: 15 minute presentation & 5 minutes for questions

8:15 a.m. **SESSION 1: INVESTIGATIONAL METHODS: STRENGTHS & LIMITATIONS - CELEBRITY THEATER**

Chairs: David Bates & Werner Stöber

- Papers:
- 1.1 Pope, C. Arden III: Epidemiology investigations: Strengths & Limitations.
  - 1.2 Mauderly, J. L.: Toxicology investigations: Strengths & Limitations.
  - 1.3 Schwartz, Joel, Dockery, Neas: Are combustion particles responsible for the associations with daily deaths?
  - 1.4 Cifuentes, Luis A., Lave: Air pollution & mortality: Searching for a threshold in the association.
  - 1.5 Samet, Jonathan, Seger, Kelsall, Xu: Particle epidemiology evaluation project.

Discussion

10:15 a.m. **BREAK**

10:30 a.m. **SESSION 2: EPIDEMIOLOGICAL FINDINGS - CELEBRITY THEATER**

Chairs: Douglas Dockery & Ursula Ackerman-Lieblich

- Papers:
- 2.1 Ito, Kazuhiko, Thurston: Issues in the interpretation of exposure/response curves in time-series PM-health effects studies.
  - 2.2 Moolgavkar, Suresh, Lubeck: Air pollution & hospital admissions in Minneapolis-St. Paul & Birmingham: A Tale of Two Cities.
  - 2.3 Boucher, Kenneth, Lyon: Daily mortality & exposure to PM-10 air pollution, Salt Lake City, Utah 1985-1993. (Presented by Joseph L. Lyon)
  - 2.4 Kalkstein, Laurence, Pope: Synoptic weather modeling & estimates of the exposure-response relationship between daily mortality & particulate air pollution. (Presented by C. Arden Pope, III)
  - 2.5 Dockery, Douglas, Hoek, Schwartz, Neas: Specific air pollutants & the Philadelphia mortality associations.

12:30 p.m. **LUNCH BREAK**

Posters Sessions: 3-5 minutes for presentation

(Posters displayed in Coalition Rooms, posters presented in Silver King Rooms)

1:30 p.m. **SESSION 3: EPIDEMIOLOGICAL FINDINGS (POSTERS & DISCUSSION) - SILVER KING 1**

Chair: George Thurston

- Papers:
- 3.1 Knusten, Synnove, Abbey, Burchette, McDonnell, Lebowitz: Lung function associated with long term ambient particulate pollutants in non-smoking adults - The AHSMDG Study.
  - 3.2 Ricci, Paolo, Ospital: Cancer, cardiovascular & chronic obstructive pulmonary mortality & ambient air pollution: A study of 11 California counties, 1963 to 1992, using monthly data. (Presented by Jean Ospital)
  - 3.3 Hirsch, Alan: Malodor as an air-pollutant: The effects on health.
  - 3.4 Kelsall, Julia, Samet, Zeger, Xu: Air pollution & mortality in Philadelphia, 1974-1988.

- 3.5 Linn, WS, Clark, Anderson, Gong: Short-term particulate exposures & health changes in Los Angeles residents with chronic obstructive pulmonary disease (COPD).
- 3.6 Reichrtova, Eva, Ondrejcekova: Respiratory system response to environmental aerosols. (Not Presented)

1:30 p.m. **SESSION 4: MECHANISMS OF INJURY (POSTERS & DISCUSSION) - SILVER KING 2**

- Chair: Richard Schlesinger
- Papers:
- 4.1 Aust, Ann, Smith: Mobilization of iron from urban particulates leads to the generation of reactive oxygen species & DNA damage *in vitro* & induction of ferritin synthesis in cultured human lung epithelial cells.
  - 4.2 Pinkerton, Kent, Lee, Peake, Buckpitt: Heterogeneity of pulmonary clara cell response to metabolically activated chemicals associated with particulate exposure.
  - 4.3 Ghio, Andrew, Stonehuerner, Pritchard, Guigley, Dreher, Costa: Humic-like substances in air pollution particulates correlate with concentrations of transition metals & oxidant generation.
  - 4.4 Becker, Susanne, Soukup: Regulation of human alveolar macrophage surface receptor expression & phagocytosis by ambient urban air particulates.
  - 4.5 Samet, James, Reed, Ghio, Davlin, Carter, Bromberg, Madden: Effect of residual oil fly ash on prostanoid synthesis by human airway epithelial cells.

1:30 p.m. **SESSION 5: PARTICLE DEPOSITION & CLEARANCE (POSTERS & DISCUSSION) - SILVER KING 3**

- Chair: Gerhard Scheuch
- Papers:
- 5.1 Bennett, William, Zeman: Deposition of fine particles in children spontaneously breathing at rest.
  - 5.2 Asgharian, Bahman, Zhang, Anjilvel: Predictive model of particle deposition in the human upper airway bifurcations.
  - 5.3 Zhang, Lei, Joyner, Asgharian, Morgan: Reconstruction of the central airways of a male F344 rat for airflow & particle transport simulations.
  - 5.4 Menache, Margaret: Inhalability & predicted particle deposition for toxicological experiments.
  - 5.5 Hesterberg, T., Miller, Hart: Deposition & retention of synthetic vitreous fibers in the lung: Investigating the relationship between biopersistence & lung toxicity.
  - 5.6 Sarangapani, Ramesh, Waxler: Particle deposition in the human upper airways.

1:30 p.m. **SESSION 6: EXPOSURE ASSESSMENT & SAMPLING (POSTERS & DISCUSSION) - SILVER KING 4**

- Chair: Melvin Zeldin
- Papers:
- 6.1 Koutrakis, Petros, Wang, Sioutas, Wolfson: Development of a continuous particle mass monitor by measuring pressure drop across filters. (Presented by Constantinos Sioutas)
  - 6.2 Lipfert, Frederick: Trends in airborne particles in the United States.
  - 6.3 Michaels, Robert: Permissible daily airborne particle mass levels encompass brief excursions to the 'London Fog' range which may contribute to daily mortality & morbidity in communities.
  - 6.4 Lillquist, Dean, Roderlechner: Field evaluation of airmetric Mini-vol PM-10 & PM-2.5 samplers.
  - 6.5 Coburn, Timothy, Kelly, Bailey: Reduction in vehicle emissions attributable to alternative transportation fuels & its prospective impact on air quality & public health.
  - 6.6 Hoke, S. H.: A continuous denuder for acid aerosols & gases.
  - 6.7 Hamekoski, Kari, Salonen: Particulate matter in northern climate of Helsinki metropolitan area, Finland. (Presented by Raimo O. Salonen)
  - 6.8 Wright, Dagan: Relationships among EPA sampling methodologies for PM-10 & PM-2.5, & their associations with meteorological variables (temperature, wind-speed, relative humidity).

- 6.9 Meyer, Michael: Federal reference & equivalent particulate matter methods: The importance of standardization.
- 6.10 Cooper, John, Patterson, Towney: Fine, coarse & PM-10 concentrations: Relationships & regulatory implications.
- 6.11 Lihan, Y., Roth, Li: Examination of the legitimacy of using outdoor PM levels as a surrogate for personal exposures in epidemiological studies. (Presented by Daniel H. Roth)

3:15 p.m. **BREAK**

Platform Sessions: 15 minute presentation & 5 minutes for questions

3:30 p.m. **SESSION 7: EPIDEMIOLOGICAL FINDINGS - CELEBRITY THEATER**

Chair: Bert Ostro

- Papers:
- 7.1 Thurston, George, Ito, Gwynn: Associations between PM-10 & mortality in multiple U.S. cities.
  - 7.2 Gwynn, Charon, Burnett, Thurston: A time series analysis of acidic PM & daily mortality & morbidity in the Buffalo, New York region. (Presented by George Thurston)
  - 7.3 Fennelly, Kevin, Bartelson: Cardiopulmonary morbidity associated with particulate air pollution in Denver, Colorado, 1989 - 1992.
  - 7.4 Heyder, J., Brand, Heinrich, Peters, Scheuch, Tuch, Wichmann: Size distribution of ambient particles & its relevance to human health. (Presented by Annette Peters)
  - 7.5 Katsouyanni, Klea, et al: Short-term effects of air pollution on health: A European approach using epidemiologic time-series data. (Presented by Denis Zmirou)

Discussion

3:30 p.m. **SESSION 8: PARTICLE DEPOSITION & CLEARANCE - SILVER KING 1 & 2**

Chair: Annie Jarabek & Burt Snipes

- Papers:
- 8.1 Snipes, M. Burt, James, Jarabek: The 1994 ICRP66 Human Respiratory tract model as a tool for predicting lung burdens from exposures to environmental aerosols.
  - 8.2 Bennett, William, Zeman, Kim, Mascarella: Enhanced deposition of fine particles in COPD patients spontaneously breathing at rest.
  - 8.3 Scheuch, Gerhard: Size dependent particle clearance & retention in the human tracheobronchial region.
  - 8.4 Hofmann, Werner, Bergmann, Koblinger: Modeling deposition & clearance of ultrafine particles in human lungs.
  - 8.5 Guidotti, Tee: The "Cohen hypothesis" revisited: Dust retention, airflow obstruction & disease risk.

Discussion

3:30 p.m. **SESSION 9: INDOOR, TOTAL & OCCUPATIONAL EXPOSURES - SILVER KING 3 & 4**

Chairs: Joan Daisey & Jeffrey Lee

- Papers:
- 9.1 Janssen, Nicole, Hoek, Harssema, Brunekreef: A relationship between personal & ambient fine particle concentrations.
  - 9.2 Brauer, Michael, Marnett, Lang, Bartlett: Continuous assessment of indoor fine particles with a portable nephelometer.
  - 9.3 Lillquist, Dean, Lee, Ramsay, Boucher, Weiss, Lyon: A comparison of indoor/outdoor PM-10 concentrations measured at three hospitals & a centrally located monitor in Utah.
  - 9.4 Hering, Susanne: Indoor/outdoor concentration ratios for fine particle mass & inorganic ions in twelve southern California homes.

Discussion

5:30 p.m. **SUPPER BREAK**

7:30 p.m. **SESSION 10: SCIENCE & PUBLIC POLICY - CELEBRITY THEATER**

Chairs: Joe Mauderly & Daniel S. Greenbaum

- Papers:
- 10.1 McClellan, Roger: The Clean Air Act & criteria pollutants: Are science & legislation out of sync?

- 10.2 Utell, Mark: The Clean Air Act: Clinical plausibility of health effects from criteria pollutants.
- 10.3 Freeman, A. Myrick: The potential role of economics in setting air quality standards.
- 10.4 Zeldin, Melvin D.: Regulatory concerns with meeting particulate matter standards: A local agency perspective.

Panel Discussion Moderator: Joe Mauderly

10:00 p.m. REFRESHMENT SOCIAL

## THURSDAY, MAY 2, 1996

### 8:00 a.m. SESSION 11: MECHANISMS OF INJURY & EXTRAPOLATION - CELEBRITY THEATER

Chair: Mark Utell & Roger McClellan

- Papers:
- 11.1 Friedlander, Sheldon, Yeh: The submicron atmospheric aerosol as a carrier of reactive chemical species: Case of peroxides.
  - 11.2 Kleinman, Michael T., Mautz, Phalen, Bhalla: Toxicity of constituents of PM-10 inhaled by aged rats.
  - 11.3 Dreher, Kevin, Jaskot, Richards, Lehmann, Hoffman, Costa: Pulmonary toxicity of size-fractionated urban ambient air particulate matter (PM).
  - 11.4 Godleski, John, Sioutas, Kader, Koutrakis: Death from inhalation of concentrated ambient air particles in animal models of pulmonary disease.
  - 11.5 Scöber, Werner, Miller, McClellan: Requirements for a credible extrapolation model derived from health effects in rats exposed to particulate air pollution: A way to minimize the risks of human risk assessment?

Discussion

### 8:00 a.m. SESSION 17: EXPOSURE ASSESSMENT & SAMPLING - SILVER KING 1-3 (Note Session time change)

Chair: Michael Kleinman

- Papers:
- 17.1 Lipfert, Frederick, Wyzga: Simulation studies on the effects of exposure error on environmental epidemiology.
  - 17.2 Reiss, Richard, Roberts, Lurmann, Wright: An evaluation of PM-10 measurements made by tapered element oscillating microbalances in southern California.
  - 17.3 Gordon, Terry, Fang, Gerber, Chen: The use of a centrifugal concentrator in ambient PM-10 toxicology studies.
  - 17.4 Ding, Yiming, Cui, Lee, Eatough: Fine particulate n-nitroso & nitrite organic compounds in the atmosphere.
  - 17.5 Noble, Christopher, Prather: Aerosol time-of-flight mass spectrometry: A new method for performing real-time characterization of aerosol particles.

Discussion

10:00 a.m. BREAK

### 10:15 a.m. SESSION 12: EXPOSURE ASSESSMENT & SAMPLING - CELEBRITY THEATER

Chairs: William Wilson & Glen Cass

- Papers:
- 12.1 Allen, George, Abt, Koutrakis: Assessment of the temporal relationship between daily summertime ultra-fine particulate count concentration with PM-2.5 and black carbon soot in Washington D.C.

- 12.2 Wilson, William: Exposure measurements relevant to epidemiology: Total exposure to ambient fine particles.
- 12.3 Lundgren, Dale, Rich, Hlaing: PM-1, PM-2.5 & PM-10 aerosol chemistry vs. meteorology for Phoenix, Arizona.
- 12.4 Salmeen, Irving, Ball, Japar: Automobile particulate emissions: Challenges in assessing impacts on particulate air pollution & public health.
- 12.5 Miguel, Ann, Cass, Weiss, Glovsky: Latex allergens in tire dust & in air-borne particles.

12:15 p.m. **LUNCH BREAK**

1:15 p.m. **SESSION 13: EPIDEMIOLOGICAL FINDINGS - CELEBRITY THEATER**

Chair: Aaron Cohen & Dane Westerdahl

- Papers:
- 13.1 Li, Yuanzhang, Roth: The analysis of the association between air pollutants & hospital admission in Birmingham, Alabama, 1986 -1990. (Presented by Daniel H. Roth)
  - 13.2 Peters, John, Avol, Navidi, London, Thomas: Respiratory morbidity in twelve southern California communities with differing levels & types of air pollution.
  - 13.3 Hoek, G., Roemer, Brunekreef: The PEACE study (1): Acute effects of air pollution on peak flow.
  - 13.4 Abbey, David, Nishino, McDonnell, Lebowitz: Development of chronic productive cough or asthma as associated with long term ambient particulate pollutants in non-smoking adults - the AHSMOG study.
  - 13.5 Pekkanen, Juha, Timonen, Hosiokangas, Ruuskanen, Salonen: Effects of PM-10, black smoke, & resuspend dust on pef among asthmatic children. (Presented by Raimo O. Salonen)

Discussion

1:15 p.m. **SESSION 14: MECHANISMS OF INJURY - SILVER KING 1-3**

Chair: Burt Snipes

- Papers:
- 14.1 Schlesinger, Richard: Acidity: Potential contributor to increased mortality/morbidity associated with particulate air pollution?
  - 14.2 Frampton, Mark, Morrow, Zelikoff, Schlesinger, Utell: Inhalation of sulfuric acid alters alveolar macrophage function in humans. (Presented by Mark Utell)
  - 14.3 Devlin, Robert, Carter, Ghio, Samet, Reed: In vitro exposure of human airway epithelial cells to an urban air particulate pollutant induces IL-6, IL-8, & TNF $\alpha$  mRNA & protein expression.
  - 14.4 Ghio, Andrew, Carter, Samet, Reed, Quay, Dailey, Richards, Devlin: Respiratory epithelial cell production of lactoferrin after in vitro exposure to an air pollution particle is metal dependent.

Discussion

3:15 p.m. **BREAK**

3:30 p.m. **SESSION 15: EPIDEMIOLOGICAL FINDINGS & METHODS - SILVER KING 1 & 2**

Chair: George Thurston

- Papers:
- 15.1 Ostro, Bart, Lipsett, Das: Particulate matter & asthma: A quantitative assessment of the current evidence.
  - 15.2 Roemer, Willem, Hoek, Brunekreef: The PEACE study (2): Acute effects of air pollution on respiratory symptoms.
  - 15.3 Wyzga, Ronald, Lipfert: The Philadelphia story (continued): Themes & variations.
  - 15.4 Valberg, Peter, Watson: A confounding role for indoor air pollutants in the associations of ambient particulate matter (PM) with daily morbidity & mortality.
  - 15.5 Rodan, Bruce, Karch: Methodological limitations in particulate air pollution epidemiology based on pulmonary function testing.

Discussion

**3:30 p.m. SESSION 16: EXPOSURE ASSESSMENT & SAMPLING - SILVER KING 3 & 4**

**Chairs:** Beverly Cohen & Constantinos Sioutas

- Papers:**
- 16.1 Dasgupta, Purnendu: Automated particle collection & analysis: Measurement of aerosol acidity.
  - 16.2 Sioutas, Constantinos, Koutrakis, Ferguson, Kim, Burton: Harvard/EPA ambient fine & ultrafine particle concentrators for conducting inhalation exposure studies.
  - 16.3 Koenig, Jane, Haller, Larson, Norris, Westberg, Claiborn: Size & chemical characterization of airborne particulate matter in Spokane.
  - 16.4 Zhang, Junfeng, Lioy, Suh, Koutrakis: Diurnal variations in & correlations among particulate matter, sulfate, sulfur dioxide, & ozone at a site in metropolitan Philadelphia.
  - 16.5 Cohen, Beverly, Li: Electric charge on ambient ultrafine particles.

**3:30 p.m. SESSION 18: INDOOR, TOTAL & OCCUPATIONAL EXPOSURES - CELEBRITY THEATER**

**Chair:** Gregory Wagner

- Papers:**
- 18.1 Bahadori, Tina, Koutrakis: Personal, indoor & outdoor exposures to particulate matter of ten COPD patients living in private residences. (Based on results from the Nashville Particulate Personal Monitoring: A Pilot Study.)
  - 18.2 Avol, Edward, Navidi, Colome: Indoor & outdoor residential sampling of PM-10 & PM-2.5 aerosols in southern California.
  - 18.3 Stieb, David, Brook, Broder, Judek, Burnett: Personal exposure of adults with cardiorespiratory disease to particulate acid & sulfate in Saint John, New Brunswick, Canada.
  - 18.4 Pauluhn, Jurgen: Hazard identification & risk assessment of pyrethroids in the indoor environment.
  - 18.5 Ramsay, James, Lilquist: The effects of building ventilation types & human activity patterns on indoor PM-10.

Discussion

**5:30 p.m. SUPPER BREAK**

**7:30 p.m. DISCUSSION WITH SESSION CHAIRS PANEL - CELEBRITY THEATER**  
(Includes discussion of future meetings)

**Chair:** David Bates

**8:30 p.m. END OF DAY 2**

## **FRIDAY, MAY 3, 1996**

**8:00 a.m. SESSION 19: KNOWLEDGE GAPS (WORKSHOP CONSENSUS REPORT) - CELEBRITY THEATER**

**Chair:** Morton Lippmann & David Bates

- Papers:**
- 19.1 Driscoll, Kevin: Inherent toxicity of PM components - Roles of particle size & composition.
  - 19.2 Cassee, Flemming: Host responsiveness in health and disease.
  - 19.3 Bates, David: Critical determinants of responses in human populations.
  - 19.4 Wilson, William: Ambient air.
  - 19.5 Soderholm, Sidney: Indoor air - occupational.
  - 19.6 Bachmann, John: Establishing revised PM NAAQS.
  - 19.7 Phalen, Robert: Establishing revised size-selective OELs.
  - 19.8 Pope, C. Arden III: Epidemiologic opportunities and limitations.

Discussion

10:30 a.m. **BREAK**

10:45 a.m. **SESSION 20: COLLOQUIUM TOPICAL WRAP-UP - CELEBRITY THEATER**

Chair: Robert Phalen & Jeffrey Lee

Comments: Dockery, Douglas: Epidemiology summary  
Schlesinger, Richard: Mechanisms of injury summary  
Snipes, Burt: Particle deposition & clearance summary  
Wilson, William: Exposure assessment & sampling summary  
Daisey, Joan: Indoor, total & occupational relationships summary  
Wagner, Gregory: Occupational research gaps summary

Discussion

12:45 p.m. **END OF COLLOQUIUM**

**Please remember to hand in your Colloquium evaluation and research needs forms before departing.**

### III. ABSTRACTS

#### 1.1

**PRESENTER AND AUTHOR:** C. Arden Pope, III

**TITLE:** Epidemiology Investigations of the Health Effects of Particulate Air Pollution: Strengths and Limitations

**ABSTRACT**

There have been many epidemiological studies that have investigated the health effects of particulate air pollution. This paper provides a simple framework to categorize the basic study designs of most of the currently available studies of the health effects of particulate air pollution and briefly discusses the common methods of statistical analysis. Within this framework it outlines the basic strengths and limitations associated with the currently available epidemiological evidence. Both the strengths and limitations of the epidemiological studies stem largely from the use of people who are living in uncontrolled environments, and who are exposed to complex mixtures of particulate air pollution. Inherent to these studies are at least four basic limitations including: 1) limited information about biological mechanisms, 2) relatively meager information regarding linkages between ambient and personal exposures, 3) difficulty of disentangling independent effects or potential interactions between highly correlated risk factors, and 4) inability to fully explore the relative health impacts of various constituents of particulate pollution. Associations of cardiopulmonary health outcomes with particulate air pollution that have been observed in the epidemiological studies provide only one important part of the full picture. A more complete understanding of the health effects of particulate air pollution will require important contributions from toxicology, exposure assessment, and other disciplines. Nevertheless, the pattern of cardiopulmonary health effects associated with particulate air pollution that has been observed by the epidemiological studies is currently the strongest evidence of the potential health effects of this pollution.

PRESENTER: Joe L. Mauderly

TITLE: Toxicology Investigations: Strengths and Limitations

AUTHOR: Mauderly, J L

ABSTRACT

The strengths and limitations of laboratory-based toxicology investigations of the health impacts of particulate air pollution are those common to all laboratory experiments. The greatest strength is the ability to dissect a complex problem into its component parts and conduct a sequence of hypothesis-driven experiments which, together, yield an understanding of the processes by which phenomena observed in population studies occur. The accompanying weaknesses are 1) the difficulty of developing the correct hypothesis, and 2) the difficulty of constructing an experiment to address the hypothesis definitively. The second greatest strength is the ability to conduct the experiment under carefully-planned, well-controlled circumstances. The corresponding weaknesses are 1) the need to understand which variables to control, and 2) the danger of eliminating outcomes which require the interactions of variables which were eliminated. The third greatest strength is the ability to select the experimental model and level of organization to study (eg, molecule, cell, tissue, organ, subject). The inherent weakness is the ubiquitous uncertainty about applicability of results from the model system to the population or exposure situation of concern. The three cardinal responsibilities of the experimentalist to the community are: 1) rationality of experimental design; 2) reliability of results; and 3) validity with which the results may be extrapolated to the population and exposure of concern. Unfortunately, there is often a tendency to dwell on the second responsibility and give insufficient attention to the first and third. When exposures are occurring, rather than potential, toxicology and epidemiology are both empowered when conducted as a partnership.

**TITLE:** Are combustion particles responsible for the associations with daily deaths?

**AUTHORS:** Schwartz J, Dockery D, Neas M

**ABSTRACT**

While a score of studies have reported associations between airborne particles and daily deaths, they have not identified the characteristics of particles responsible for these effects. We have investigated this question using fine and coarse particle mass concentrations measured every other day for eight years in each of six metropolitan areas (Boston, MA; St. Louis, MO; Knoxville, TN; Steubenville, OH; and Madison, WI). Hydrogen ion concentrations were measured daily for one year in each location. Poisson regressions controlling for smooth functions of time, temperature, humidity, and indicators of day of week, rain and snow were estimated in each location, and the estimated pollution effects combined across cities. An interquartile range (IQR) change in  $PM_{2.5}$  ( $13.7 \mu g/m^3$ ) was associated with a 2% increase in daily deaths (95% CI 1.5% to 2.7%). An IQR increase in sulfate particle mass ( $5.8 \mu g/m^3$ ) was associated with a 1.2% increase (95% CI 0.7%-1.8%). In contrast, coarse mass ( $10.6 \mu g/m^3$ ) defined as  $PM_{10}-PM_{2.5}$  and hydrogen ion ( $18.9 \text{ nmole}/m^3$ ) showed weak associations 0.5% (95% CI -0.1% to 1.1%) and 0.2% (95% CI -0.6% to 0.9%) respectively. We interpret these finding as indicating that daily mortality is specifically associated with fine combustion particles, that these associations are not restricted to sulfate particles, and that the associations are not attributable to the total acidity of the fine particles. These analyses were supported by Environmental Protection Agency Cooperative Agreement R and a contract from the Environmental Criteria Assessment Office.

PRESENTER: Luis A. Cifuentes

TITLE: Air pollution and mortality: searching for a threshold in the association.

AUTHORS: Cifuentes, L A , Lave, L B

ABSTRACT

A growing number of time-series analysis have shown an association between ambient concentrations of particulate matter and increased human mortality. Some of these studies have been used by EPA as the basis for the proposed NAAQS standard for particulate matter. However, few of the studies have considered in depth the possible existence of a threshold in the effects. In this work we analyze data for three cities that have different characteristics of weather and pollution : Philadelphia, PA; Birmingham, AL; and Santiago, Chile. We control for weather and seasonal effects for each individual cities, and also use a single model across all cities. Particulate matter was measured as TSP in Philadelphia,  $PM_{10}$  in Birmingham, and both  $PM_{10}$  and  $PM_{2.5}$  in Santiago. Other pollutants included in the analysis are  $SO_2$  and  $O_3$  for Philadelphia, and  $SO_2$ ,  $O_3$  and CO for Santiago. We explored the existence and level of a threshold using three different approaches: (a) including a discrete variable representing the quintiles of particulate matter concentrations, (b) dividing the data in different subsets according to their particulate matter concentrations, and (c) modeling particulate matter concentrations as a continuous piecewise linear function. The results indicate an important influence of the observations with higher levels of particulate matter, and support the hypothesis of the existence of a threshold level in the association.

PRESENTER: Jonathan Samet

TITLE: Particle Epidemiology Evaluation Project

AUTHORS: Samet J, Zeger S, Kelsall J, Xu J

ABSTRACT

The Particle Epidemiology Evaluation Project was implemented in 1994 to address critical issues related to the observational studies on particulate air pollution and mortality. It has two phases: Phase IA which was directed at replication and validation of selected studies and Phase IB included analyses of data for Philadelphia. Phase IA was directed at replicating and validating selected published reports on particulate air pollution and mortality. The project included verifying one of the previously analyzed data bases (Philadelphia, 1973-1980), replicating analyses for key locales, and assessing the sensitivity of findings to analytic assumptions and model specification choices. The data set and analyses were replicated. We also found that the qualitative effects of TSP and SO<sub>2</sub> were not sensitive to model specification; we did find interdependence of their effects. In Phase IB, we have shown that findings are relatively insensitive to the approach for control of effects of weather on mortality. The general findings with regard to pollution were unchanged, comparing empiric approaches with synoptic categorization of weather. In Phase IB, we have also analyzed a data set for Philadelphia, 1974-1988, which includes TSP, SO<sub>2</sub>, NO<sub>2</sub>, CO, and O<sub>3</sub>. We found moderate correlations among the primary combustion pollutants. Using an analytic strategy that more finely controlled for time trends than earlier approaches, we found effects of either TSP or SO<sub>2</sub> considered alone or in combination with O<sub>3</sub> on mortality. Ozone had consistent effects on mortality, even following control for other pollutants. A less plausible association with lagged CO was also apparent in the data independent of TSP/SO<sub>2</sub> and ozone. We conclude that air pollution was associated with increased mortality during the years 1973-1988; significant effects were present for either TSP or SO<sub>2</sub>, considered as representing the complex of primary combustion pollutants, and for O<sub>3</sub>.

PRESENTER: Kazuhiko Ito

TITLE: Issues in the Interpretation of Exposure/Response Curves in Time-Series PM-Health Effects Studies

AUTHORS: Ito, K, Thurston, G

ABSTRACT

A number of time-series studies have reported significant associations between particulate matter (PM) and morbidity/mortality in various locales in the U.S. and in other countries. These studies have typically reported short-term excess increases in deaths or hospital admissions as the percent increase of total deaths or admissions per certain unit PM concentrations (e.g., relative risk per 100  $\mu\text{g}/\text{m}^3$  of PM<sub>10</sub>). Some studies also reported estimated exposure/response curves for ranges of PM levels. However, there are unresolved issues that may be critical in interpreting these relative risks and exposure/response curves for risk assessment. These include: 1) exposure misclassifications within metropolitan areas (i.e., spatial variability) of PM, co-pollutants, and weather variables; 2) effect modifications and confounding by co-pollutants and weather variables; 3) the extent of premature deaths (i.e., "harvesting"); 4) potential underestimation of risk estimates for at-risk sub-populations; and, 5) statistical power to detect health effects. In this presentation, these issues are outlined and illustrated using example mortality/PM<sub>10</sub> data from Chicago, IL and Steubenville, OH. Results include the differential spatial variability for PM, co-pollutants, and temperature; underestimation of relative risks for sub-populations, and the effect of small datasets (both in terms of the number of days and daily mortality counts available) on the statistical power to detect a given PM slope size. Alternative possible interpretations of these risk estimates and exposure/response curves are given, and their implications to risk assessment discussed.

**PRESENTER:** Suresh H. Moolgavkar

**TITLE:** Air pollution and hospital admissions in Minneapolis-St. Paul and Birmingham: A tale of two cities

**AUTHORS:** Moolgavkar, S, Luebeck, G

**ABSTRACT**

Recent studies have found associations between air pollution and hospital admissions for respiratory diseases, particularly COPD and pneumonia. In this paper we examined the relationship between various components of air pollution and daily hospital admissions for COPD and pneumonia among the elderly in Minneapolis-St. Paul and Birmingham during the period 1986-1991. As in previous papers, we used parametric and semi-parametric Poisson regression to analyze the data. We found no consistent associations between specific components of air pollution and hospital admissions for respiratory diseases. Furthermore, we found that the results of the analyses were sensitive to methods used for control of weather and temporal trends. We conclude that it is not possible to isolate the contributions made by individual components of air pollution to daily hospital admissions for respiratory disease in these two cities.

PRESENTER: Joseph L. Lyon

TITLE: Daily mortality and exposure to PM<sub>10</sub> air pollution, Salt Lake City, Utah 1985-1993.

AUTHORS: Boucher, K, Lyon, J

ABSTRACT

Earlier studies found an association between exposure to high levels of PM<sub>10</sub> air pollution and all causes mortality (minus accidental deaths) among residents of Utah County, Utah between 1985-1993. We examined the same association for Salt Lake County, Utah, the adjacent urban county, and compared the results to the Utah County results. There is substantial air mixing between the two counties and the number of days when PM<sub>10</sub> levels were 75  $\mu\text{g}/\text{m}^3$  or greater were similar, 9.6% and 11.8% respectively. The chemical composition of particulate air pollutants was similar for the major criteria pollutants with exception of a 4% higher level of NO and 4% lower levels of organic carbon in Utah County. For all causes mortality (estimated from a Poisson regression model for PM<sub>10</sub> levels of 50  $\mu\text{g}/\text{m}^3$ ) the rate ratio (RR) for Salt Lake County was 1.02 and for Utah County was 1.06. Using different lags (days after exposure) ranging from 1 to 20 days, mortality in Utah County peaked at day two (RR = 1.05) and then declined to 0.97 at day eight. Mortality in Salt Lake County increased gradually peaking at day 14 (RR = 1.04) and was then stable. RR for cause specific mortality were also estimated for the two counties using a five day moving average. For Salt Lake County the RR for deaths from respiratory, cardiovascular, and other causes were 1.03, 1.04, and 1.00. For Utah County they were 1.11, 1.07, and 1.03. RR for place of death was also examined. For Salt Lake County the RR was 1.01 for hospital deaths, 1.04 for nursing home deaths, and 1.01 for deaths at home. For Utah County the RR were 1.07, 1.06, and 1.05. Despite similar indices of air pollution, the two adjacent counties demonstrate a different mortality pattern for exposure to PM<sub>10</sub> during the same period.

**PRESENTER:** C. Arden Pope, III

**TITLE:** Synoptic Weather Modeling and Estimates of the Exposure-Response Relationship Between Daily Mortality and Particulate Air Pollution

**AUTHORS:** Kalkstein LS, Pope, CA III.

**ABSTRACT:**

This study estimated the association between particulate air pollution and daily mortality in Utah Valley using the synoptic climatological approach to control for potential weather effects. This approach was compared with alternative weather modeling approaches. Although seasonality explained a significant amount of variability in mortality, other weather variables explained only a very small amount of additional variability in mortality. The synoptic climatological approach performed as well or slightly better than alternative approaches to controlling for weather. However, the estimated effect of particulate pollution on mortality was mostly unchanged or slightly larger when synoptic categories were used to control for weather. Furthermore, the shape of the estimated dose-response relationship was similar when alternative approaches to controlling for weather were used. The associations between particulate pollution and daily mortality were not significantly different from a linear exposure-response relationship that extends throughout the full observed range of pollution.

**PRESENTER:** Douglas W. Dockery

**TITLE:** Specific Air Pollutants and the Philadelphia mortality associations.

**AUTHORS:** DW Dockery, G Hoek, J Schwartz, LM Neas. *Harvard School of Public Health, Boston, MA. Univ. of Wageningen, The Netherlands.*

**ABSTRACT:**

Numerous studies have reported an association between daily mortality and air pollution in Philadelphia, although the specific pollutant responsible for these associations remains undefined. Fine particle mass ( $PM_{2.5}$ ) pollution was measured daily in Philadelphia starting in 1992, along with concentration of each of the criteria pollutants. Daily nontrauma deaths in the city of Philadelphia for 1992 and 1993, adjusted parametrically for trend, season, temperature and dew point, were associated with  $PM_{2.5}$  concentrations on the previous day (2.0% increase in deaths associated with each  $10 \mu g/m^3$  increase in  $PM_{2.5}$ , 95% CI 0.4% to 3.5%,  $p = .01$ ). These results were confirmed using nonparametric regression. A weaker association was found for  $PM_{10}$  (1.2% per  $10 \mu g/m^3$ ,  $p = .06$ ), but no association was found with coarse particles ( $p = .85$ ) nor with total aerosol acidity ( $p = .57$ ). Of the gaseous pollutants, there was no association with  $SO_2$  ( $p = .78$ ), and a negative association with CO. There was a positive association with  $O_3$  (0.9% per  $10 \mu g/m^3$ ) which appeared to be independent of and additive to the  $PM_{2.5}$  associations. These findings point to a specific effect of fine particles with a possible additional effect of ozone.

This abstract is supported by NIEHS Grant ES06239 and EPA Cooperative Agreements CR11650 and CR822050.

**PRESENTER:** Synnøve Knutsen

**TITLE:** Lung function associated with long term ambient particulate pollutants in nonsmoking adults - The AHSMOG Study

**AUTHORS:** Knutsen SF, Abbey DE, Burchette RJ, McDonnell WF, Lebowitz MD.

**ABSTRACT**

Since 1977 nonsmoking California Seventh-day Adventists (N = 6,340) have been followed to study development of respiratory disease associated with long-term ambient concentrations of air pollutants (Chest 1984;86:830-838). Statistically significant associations between development of airway obstructive disease (AOD) symptoms and both total and fine particulates have been found (Inhalat Toxicol 1995;7:19-34); however, no lung function data has been available. In 1993 spirometry was performed on a sample (1,391) of subjects. Gender specific prediction equations for lung function measures specific to this study population were formed from regressions on age, height, and armspan for healthy nonsmoking individuals. Ambient concentrations of pollutants were estimated using monthly interpolations (1973-1993) from fixed site monitoring stations to zip code centroids of home and work locations of study participants.

Gender specific multiple linear regressions were used to study associations between lung function measures and particulates  $< 10\mu\text{m}$  in diameter ( $\text{PM}_{10}$ ) adjusting for covariates. Covariates included history of environmental tobacco smoke exposure, education, parental AOD or hayfever, occupational exposures, indoor sources, and time spent outdoors. Suspended sulfates ( $\text{SO}_4$ ), ozone ( $\text{O}_3$ ), and sulfur dioxide ( $\text{SO}_2$ ) were also used as covariates in multipollutant models. For females, no associations were observed between % predicted  $\text{FEV}_1$  ( $\text{PPFEV}_1$ ) and  $\text{PM}_{10}$ . When adding the other pollutants to the model, only  $\text{SO}_2$  showed a negative association of borderline significance ( $p < .10$ ). For males,  $\text{PM}_{10}$  was significantly ( $p < .05$ ) associated with decreased  $\text{PPFEV}_1$  in males whose parents had AOD or hayfever. In the multiple linear regression, an increase in  $\text{PM}_{10}$  of 42 days/year in excess of  $100\mu\text{g}/\text{m}^3$  was associated with a decrease of -5.48% [95% confidence interval (CI): -8.86, -2.10] in  $\text{PPFEV}_1$ . When  $\text{SO}_4$  was added to the  $\text{PM}_{10}$  model, an increase of  $7.5\mu\text{g}/\text{m}^3$  was associated with a decrease in  $\text{PPFEV}_1$  of 5.86% (95% CI: -12.77, 1.04) ( $p = .096$ ), and when  $\text{SO}_2$  was added it was associated with a 4.97 (95 CI: -10.44, 0.50) ( $p = .07$ ) decrease in  $\text{PPFEV}_1$ ; the effects of  $\text{PM}_{10}$  remained virtually unchanged in both models. Adjusting for  $\text{O}_3$  in the two-pollutant models increased previously described associations but  $\text{O}_3$  was not independently associated with  $\text{PPFEV}_1$  in these multipollutant models.

**PRESENTER:** Jean Ospital

**TITLE:** Cancer, cardiovascular and chronic obstructive pulmonary mortality and ambient air pollution: a study of 11 California counties, 1963 to 1992, using monthly data

**AUTHORS:** Ricci, P, F, Ospital, J

**ABSTRACT**

This paper discusses the continuing development of a data base of monthly air pollution, climatic, and mortality time series from 1963 to 1992, for California, and analyses based on these data bases. The counties included are, in the South: Los Angeles, Riverside, San Bernardino, Orange, San Diego; and, in the North, Contra Costa, Marin, Santa Clara, San Francisco, San Mateo, and Alameda. The air pollutants, a significant increase in the number included from the data bases discussed in a previous paper, are: Total Suspended Particulate Matter,  $PM_{10}$ , Pb,  $O_3$  and Total Oxidants,  $SO_4$ ,  $SO_{4-10}$ ,  $NO_{3-10}$ , and CO, Soiling Index (SI, as Coefficient of Haze (COH)), and light scattering (LS). Climatic data include temperature and relative humidity. The mortality rates, developed from unstandardized daily mortality counts by county of residence at time of death, are adjusted by gender, race, and age and are standardized by the 1980 California population. These rates are divided into four age groups: all ages, less than 25 years old, 25 to 44 years old, and older than 44 years. The mortalities are those resulting from (selected) all causes, all cancers, lung and respiratory cancers, cardiovascular diseases, and chronic obstructive pulmonary diseases. Smoking trends, and trends for other risk factors, are also included, as are outdoor and indoor activity patterns leading to exposure. The monthly time series results indicate that counties with temporally lower levels of air pollution have higher mortality rates than those counties with higher levels of air pollution. Moreover, although the trends for disease-specific mortality rates are increasing, the corresponding trends for the etiologically relevant air pollutants are decreasing. However, there also are some positive relationships, as in the case of between cardiovascular diseases and exposure to lead. Nevertheless, placed in the context of cost-risk-benefit balancing and apportionment of risk to the emitting sources of the pollutants, the overall results suggest that there are unanswered questions that require additional scrutiny

PRESENTER: Alan R. Hirsch, MD

TITLE: Malodor as an Air-Pollutant: The Effects on Health

AUTHOR: Hirsch, Alan R

ABSTRACT

Malodor may present with a direct neurotoxic or indirect (stress) effect impacting upon the organisms health through:

(1) Annoyance. While many people are not bothered by malodor, those with impaired olfactory ability tend to perceive the hedonics of a malodor in even a more unpleasant fashion.

(2) Physical Health Effects. Studies suggest that exposure to malodors may induce adverse health effects including: depression, nausea, vomiting, headaches, coughing, insomnia, impaired appetite, the exacerbation of asthma, permanent olfactory loss, cardiovascular effects and immune function compromise.

At a mobile park adjacent to a Navy landfill in Port Orchard, Washington, we found that chronic exposure to intermittent malodor was associated with cortical and subcortical dysfunction as manifest by encephalopathy, limbic encephalopathy and cephalgia.

(3) Psychiatric Effects. Psychiatric effects of malodors have been reported to include: aggression, confusion and depression. This is demonstrated by adverse psychiatric events coinciding with malodorous air pollution including: increased number of motor vehicle accidents, family disturbances, psychiatric hospital admissions and 911 emergency calls.

Persons who have recently undergone a major stressful event or suffering chronic diseases may even be more vulnerable to the psychological effects of malodorous air pollution.

In a study for the Illinois EPA and the Attorney General's Office, State of Illinois, of a mulching site southeast of Chicago we found that on days that malodor wafted to the school across the street, there was an increase in behavioral problems in the school children.

(4) Community Health/Social Effects. Exposure to unpleasant odors has been reported to increase helplessness, reduce motivation, capacity to cope with other stressors, frustration tolerance, task performance, learning ability, and promote familial disharmony. All of these potential effects of malodor would induce a disadvantageous social condition for growth of the individual and stability of the family unit.

PRESENTER: Julia Kelsall

TITLE: Air pollution and mortality in Philadelphia, 1974-1988.

AUTHORS: Kelsall, J, Samet, J, Zeger, S, Xu, J

ABSTRACT

Many analyses have now been conducted to assess the association between air pollution and mortality; a number of these studies have used data from Philadelphia. In this new systematic analysis of Philadelphia data, 1974-1988, a Poisson regression model was built, taking account of the effects of time, weather and pollutants sequentially. The data set comprised total daily mortality counts further subdivided by cause (cardiovascular, respiratory, other) and age ( $<65$ ,  $65-74$ ,  $\geq 75$ ), and daily measurements of temperature, dew point temperature, total suspended particulates (TSP), sulfur dioxide ( $\text{SO}_2$ ), nitrogen dioxide ( $\text{NO}_2$ ), carbon monoxide (CO) and ozone ( $\text{O}_3$ ). Generalized additive models were used to allow the incorporation of smooth relationships of mortality with time, and fitted models were compared using Akaike's Information Criterion (AIC), a measure of prediction error.

After controlling for time and weather, examination of the relationships among the pollutants revealed that TSP,  $\text{SO}_2$ ,  $\text{NO}_2$  and CO had moderately high day-to-day positive correlations (0.4 to 0.7) whereas  $\text{O}_3$  was positively correlated with the other pollutants in the summer (0.0 to 0.4) and negatively correlated in the winter (-0.4 to -0.5). When included simultaneously in the model, we found that the means of current and previous days' levels of TSP,  $\text{SO}_2$ ,  $\text{NO}_2$  and  $\text{O}_3$  had statistically significant effects on total mortality. The effects of TSP and  $\text{SO}_2$  were diminished when in a model together. Surprisingly, there was an association of the mean CO level of 3 and 4 days previously with mortality. We also found a negative effect of  $\text{NO}_2$  which was only apparent when included in a model along with TSP or  $\text{SO}_2$ . We conclude that in order to more successfully identify the individual effects of the pollutants, we must investigate a wide range of sites for which the relative levels and correlations of pollutants in the mixture substantially differ.

**PRESENTER:** William S. Linn

**TITLE:** Short-Term Particulate Exposures and Health Changes in Los Angeles Residents with Chronic Obstructive Pulmonary Disease (COPD)

**AUTHORS:** Linn, WS, Clark, KW, Anderson, KR, Gong, H

**ABSTRACT**

To help understand the epidemiologic relationship between particulates and mortality/morbidity, we studied daily exposures, time-activity patterns, and health status in Los Angeles area residents with severe COPD. Volunteers (N = 45) recorded activity and symptoms in diaries, and measured lung function and blood pressure 3 times/day, for 4-day periods (Thursday through Monday mornings) in summer or fall. Concurrent personal, inside-home, and outside-home  $PM_{10}$  measurements were taken during one 24-hr interval;  $PM_{2.5}$  was measured during the preceding or following 24 hr. Background  $PM_{10}$  was determined from the nearest of 6 local monitoring stations. Passive personal monitors estimated cumulative exposures to pollutant gases. Temperature and humidity were monitored inside and outside homes, and at nearest stations. Holter electrocardiograms were obtained for 24 hr in each subject. Preliminary air monitoring results (24-hr averages in  $\mu g/m^3$ ) showed a mean (range) of 55 (23-174) for nearest-station  $PM_{10}$ , 53 (5-149) for outside-home  $PM_{10}$ , 37 (14-86) for inside-home  $PM_{10}$ , and 42 (17-105) for personal  $PM_{10}$ .  $PM_{2.5}$  measurements averaged near 2/3 of corresponding  $PM_{10}$ . Nearest-station  $PM_{10}$  correlated highly with outside-home concentrations ( $r = 0.77$  for  $PM_{10}$ ,  $0.71$  for  $PM_{2.5}$ ), less highly with indoor concentrations ( $r = 0.30$  for  $PM_{10}$ ,  $0.64$  for  $PM_{2.5}$ ) or personal concentrations ( $r = 0.38$  for  $PM_{10}$ ,  $0.29$  for  $PM_{2.5}$ ). Indoor and personal concentrations correlated ( $r = 0.59$  for  $PM_{10}$ ,  $0.74$  for  $PM_{2.5}$ ), as expected since subjects spent >90% of their time indoors. Physical activity occupied  $\approx 14\%$  of subjects' time, but was of low intensity, judging from electrocardiograms. Self-rated overall clinical status did not vary significantly day to day, and did not covary significantly with same-day  $PM_{10}$  at the nearest station. Some lung function indices varied significantly with time; most did not covary with  $PM_{10}$ , although there was a suggestion of reduced peak flow with increasing  $PM_{10}$ .

PRESENTER: Eva Reichrtova

TITLE: Respiratory system response to environmental aerosols

AUTHORS: Reichrtova, E, Ondrejckova, I

ABSTRACT

Because it is difficult to simulate complex ambient atmospheres in the laboratory, we carried out non-traditional environmental studies using domestic rabbits (housed in special cages constructed for the whole-body respiratory exposure) in an effort to screen for potential health hazards due to environmental aerosols. Comparisons between animal environmental and chamber exposures (to aerosols created from the real waste particles) were done. Respiratory system response to a 6-months inhalation of environmental aerosols was investigated in three regions polluted by emissions derived from a nickel smelter, a magnesite-processing plant, and from a mercury-recycling plant, respectively. All parameters investigated in the exposed animals were compared to non-exposed animals. The count of PAM (pulmonary alveolar macrophages (PAM)), their enzymes activities and PAM Fc receptors (FcR) activity were investigated. The count of PAM and their lysosomal enzyme activities were significantly increased in dependence on the duration of exposures in all three regions. Therefore the count of PAM has been suggested as a suitable biomarker for both the particulate air pollution and the respiratory system reaction to environmental aerosols. In contrast to the increased PAM phagocytic activity, a significant depression was found in the antibody-mediated rosette formation by PAM in the rabbits exposed to environmental aerosols derived from the nickel smelter and from the mercury-producing plant. Using the method of scanning electron microscopy (SEM), non-uniform changes in the mucosal relief of the trachea were found in rabbits exposed to metal aerosols in all regions investigated. In addition, morphological changes seen via SEM on the tracheal mucosa were consistent with x-ray microanalysis of metals deposited on tracheal relief. The inhalatory exposure of rabbits was assessed by analyses of metals which characterized environmental aerosols, using atomic absorption spectrometry (AAS). Metal contents were analyzed in tissue samples taken out from lungs and other body tissues of the exposed and non-exposed animals. As inorganic iron compounds were present in all aerosols investigated, a magnetometry was used to screen for magnetic particles burden of the lungs. The findings approved the AAS iron analyses. In groups of children 9-10 years of age, exposed to environmental aerosols, changes in local respiratory immunity as well as in the whole immune response were found. In the area of the nickel smelter, a significant increase in the respiratory diseases approved epidemiologically and by the elevation of serum antiproteases activities, was found. In the children residing in the locality of the mercury-recycling plant, an increase in respiratory diseases incidence, a significant depression of salivary lysozyme and SIgA concentrations accompanied with changes in serum proteins were found.

PRESENTER: Ann Aust

TITLE: Mobilization of Iron from Urban Particulates Leads to the Generation of Reactive Oxygen Species and DNA Damage *In Vitro* and Induction of Ferritin Synthesis in Cultured Human Lung Epithelial Cells

AUTHORS: Aust, A, Smith, K.

ABSTRACT

The molecular mechanism by which airborne particulates cause an increased incidence of respiratory symptoms, decreased pulmonary function, and increased mortality is not known. In crocidolite asbestos, iron has been shown to be responsible for biochemical reactivity, such as generation of oxygen radicals, lipid peroxidation, and DNA damage *in vitro*, which were greatly enhanced when iron was mobilized by chelators, e.g. citrate. Some of the acute biological effects of asbestos in cultured cells, such as cytotoxicity, lipid peroxidation, DNA damage, and increased production of nitric oxide, also appeared to depend upon the iron associated with the fibers. Since airborne particulates other than asbestos also contain iron, some of the acute effects after inhalation may be the result of iron-catalyzed generation of reactive oxygen species (ROS). To determine whether iron associated with airborne particulates can be mobilized, particulates (Standard Reference Material #1649 from NIST), containing 3% iron by weight, were incubated with 1 mM citrate, EDTA or ascorbate in 50 mM NaCl, pH 7.5. In one hour, 26, 49 or 5 nmoles of iron/mg of particulate was mobilized with the various chelators, respectively. Under the same conditions, crocidolite asbestos released 4, 30, or 0.5 nmoles of iron/mg of fiber, respectively. The urban particulate was also compared with crocidolite in its ability to generate reactive oxygen species to cause strand breaks in  $\phi$ X174 RFI DNA. In 30 min, the particulate at 0.1 mg/ml in ascorbate (1 mM) and EDTA (1 mM) generated ROS to a level almost comparable to crocidolite at 10 times the concentration. To determine whether iron was mobilized from urban particulates in cultured human lung epithelial cells (A549), cells were exposed to the particulates (100  $\mu$ g/cm<sup>2</sup>) for 24 h and the level of ferritin, the intracellular iron storage protein, was determined. The ferritin levels in the treated cells were 7 times higher than in the untreated controls. This strongly suggests that iron was mobilized within the cultured cells. If this iron is reactive to generate ROS as the iron in the DNA strand break assays, this may result in cell damage leading to acute respiratory symptoms and increased mortality. (Supported by Grants ES05782 and ES05814 from the National Institute of Environmental Health Sciences)

PRESENTER: Kent E. Pinkerton

TITLE: Heterogeneity of pulmonary clara cell response to metabolically activated chemicals associated with particulate exposure.

AUTHORS: Pinkerton, K.E., Lee, C.H., Peake, J.L., Buckpitt, A.R.

ABSTRACT

Nonciliated bronchiolar epithelial (Clara) cells have a number of important functions in the lungs including metabolism of inhaled xenobiotic materials via the cytochrome P450 monooxygenase system. Clara cells are among a small number of cells in the lungs which undergo postnatal differentiation. Exposure to environmental tobacco smoke (ETS) during perinatal development significantly alters both the activity and expression of cytochrome P450 1A1 in the lungs of rats. Those chemicals responsible for P450 activation have been found to reside in the particulate phase of ETS. The purpose of this study was to determine Clara cell responses to exposure to aged and diluted sidestream cigarette smoke (ADSS) from birth to 120 days of age in the lungs of rats. Of particular interest was the impact of cell location and particulate concentration on the response of the Clara cell to ETS. Research cigarettes (1R4F, University of Kentucky) were automatically smoked in a machine using a 35 ml puff volume of 2 seconds duration, once per minute. The sidestream smoke coming from the smoldering end of each cigarette was collected and aged for two minutes prior to further dilution with filtered air. Animals were exposed to 100, 300, 500, or 1,000 mg/m<sup>3</sup> of total suspended particulates (TSP) for 6 hours/day, 5 days/week. The lungs of animals were examined at 21, 70, and 120 days of age. Immunohistochemical staining of paraffin-embedded tissue sections was done using antibodies for Clara cell secretory protein (CCSP) and cytochrome P450 1A1. CCSP is a specific marker for Clara cells and was found to be present in the lungs at all ages observed. In contrast, P4501A1 antibody staining of cells was not noted in the lungs of filtered air control rats at any age or in rats 21 days of age exposed to any concentration of ADSS. By 70 days of age, animals exposed to the highest concentrations of ADSS (i.e., 300, 500, and 1000 mg/m<sup>3</sup> TSP) demonstrated P4501A1 positive staining of selected Clara cells in a concentration-dependent fashion. P4501A1 staining was most prominent in the upper airways in contrast to the terminal bronchioles of the lungs. By 120 days of age, P4501A1 cell staining frequency was the highest observed at any age. Staining of Clara cells in the upper airways with P4501A1 was noted for a limited number of cells following exposure to 100 mg/m<sup>3</sup> TSP. P4501A1 positive cells were also noted in the terminal bronchioles of animals exposed to 500 and 1000 mg/m<sup>3</sup>. A striking finding was the induction of P4501A1 in virtually every Clara cell found on or near airway bifurcation ridges in contrast to those Clara cells lining the other portions of the bronchial tree. These findings suggest that at least part of the heterogeneity noted in the response of Clara cells to inhaled particulates is due to cell location in the lungs, particulate concentration, and duration of exposure.

**PRESENTER:** Andrew J. Ghio

**TITLE:** Humic-like substances in air pollution particulates correlate with concentrations of transition metals and oxidant generation.

**AUTHORS:** Ghio, AJ, Stonehuemer, J, Pritchard, RJ, Quigley, DR, Dreher, KL, Costa, DL.

**ABSTRACT**

We tested the hypotheses that: 1) an incomplete oxidation of carbon based fossil fuels during their combustion produces humic-like substances (HLS) which can be present in air pollution particulates and confer a capacity to complex metals; 2) air pollution particulates collected on PM<sub>10</sub> filters can be associated with concentrations of first row transition metals; 3) particulates can catalyze the production of free radicals by cycling these transition metals through two stable valence states; and 4) concentrations of transition metals and oxidant generation by air pollution particulates increase with the content of HLS associated with these particles. HLS were isolated by alkali extraction. The content of these substances in combustion products of coal, diesel, oil, and wood were  $3.1 \pm 0.8\%$ ,  $4.7 \pm 1.0\%$ ,  $1.0 \pm 0.1\%$ , and  $8.2 \pm 0.6\%$  respectively. Similarly, filters with sequestered air pollution particulates contained HLS ranging from 0.0 to 7.1%. Elemental analysis of these materials isolated from both products of fuel combustion and sequestered particulate disclosed values of C, H, N, and O consistent with an HLS. There were correlations between HLS content and ionizable concentrations of metals, quantified using inductively coupled plasma emission spectroscopy, associated with particulates sequestered on filters. Similarly, HLS content correlated with the absorbance of oxidized products of deoxyribose demonstrating an affiliation between these substances and free radical generation by sequestered particulate. We conclude that HLS, a potential organic metal chelator, can be isolated from air pollution particulates. Concentrations of acid-soluble transition metals and *in vitro* oxidant generation correlated with the content of these substances collected on filters. This abstract of a proposed presentation does not necessarily reflect EPA policy.

**PRESENTER:** Susanne Becker

**TITLE:** Regulation of human alveolar macrophage surface receptor expression and phagocytosis by ambient urban air particulates.

**AUTHORS:** Susanne Becker and Joleen Soukup.

# **ABSTRACT**

Episodes of high particulate air pollution levels correlate with increased hospitalization due to respiratory disease, including pneumonia. We hypothesized that inhaled particulate matter of an aerodynamic diameter  $< 10$  micron (PM10) affect the expression of surface receptors on alveolar macrophages (AM) involved in inflammation and antimicrobial defenses, thereby exacerbating symptoms of respiratory disease. To test this hypothesis, AM were exposed *in vitro* to particulates (100 ug/ml/5x10<sup>5</sup> cells) collected from 3 different urban centers (UAP), to oil fly ash (OFA) a likely component of PM10, and to volcanic ash (VA). Cells were also exposed to LPS (10 ng/ml) as all UAP contained trace amounts of this bioactive material. Expression of beta-2 integrin chains CD18, CD11a, CD11b, CD11c, beta-1 integrin CD29, CD54 (ICAM-1), and of CD14 (endotoxin receptor) was assessed after 18 hours. Particle-exposed cells were also given *Candida albicans* and the phagocytic and chemiluminescence response toward this organism determined. Results of receptor expression experiments are summarized :

Particle	% of control receptor expression						
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	CD18	CD11a	CD11b	CD11c	CD29	CD54	CD14
UAP	<u>73*</u>	80	<u>46*</u>	<u>86*</u>	<u>78*</u>	<u>145*</u>	<u>56*</u>
OFA	113	<u>78*</u>	113	124	<u>52*</u>	125	103
VA	117	94	110	118	91	95	<u>205*</u>
LPS	127*	90	120	126	89	<u>202*</u>	<u>234*</u>

Mean (n = 4-5 experiments) \* significantly different from untreated control cells (p < .05).

These data show that exposure to OFA downregulates CD11a and CD29, while VA have no effect on these receptors but upregulates CD14. On the other hand, UAPs significantly downregulate expression of CD18, CD11b, and CD11c, all of which have been shown to be involved in phagocytic events of opsonized microorganisms and in cell-cell interactions. CD29 and CD14 are also downregulated by UAP while CD54 is upregulated. Furthermore, phagocytosis of *C.albicans* is inhibited in UAP-exposed AM as is the respiratory burst possibly because of lower beta-2 integrin levels. The above data indicate that receptor expression is differently modulated by the different particles and by LPS. Modulation of specific receptor is likely to be caused by individual components of the heterogeneous UAP. Identifying the responsible bioactive components will improve the understanding of adverse health effects of PM10.

This is an abstract of a proposed presentation and does not reflect EPA policy.

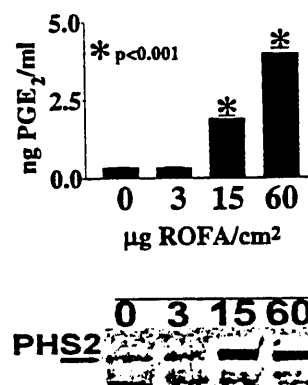
**PRESENTER:** James M. Samet

**TITLE:** EFFECT OF RESIDUAL OIL FLY ASH ON PROSTANOID SYNTHESIS BY HUMAN AIRWAY EPITHELIAL CELLS.

**AUTHORS:** Samet, J, Reed, W, Ghio, A, Devlin, R, Carter, J, Bromberg, P, Madden, M.

**ABSTRACT**

Residual oil fly ash (ROFA) is a toxic respirable particulate air pollutant derived from the combustion of fuel oil. To determine the effect of ROFA exposure on airway epithelium, we studied eicosanoid metabolism in the human airway epithelial cell (AEC) line BEAS 2B cultured in serum-free media on plastic and exposed *in vitro* to 0-60 mg/cm<sup>2</sup> ROFA. Treatment with ROFA for 24 hours induced a marked, dose-dependent increase in immunoreactive prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) release BEAS cells (shown at right). LDH release was not significantly altered. BEAS treated with ROFA for 24 hours and then pulsed for 30 min with [<sup>3</sup>H]arachidonate exhibited a striking increase in [<sup>3</sup>H]-prostanoid synthesis, indicating increased prostaglandin H synthase (PHS) activity. Western blots showed a dose-dependent increase of PHS2 protein in BEAS treated with ROFA (shown at right). RT-PCR detected earlier ROFA-induced increases in the level of PHS2 (but not PHS1) mRNA in BEAS. Similar effects were observed using cultured primary human AEC. These data show that exposure of AEC to non-cytotoxic levels of ROFA increases PGE<sub>2</sub> synthesis through enhanced expression of PHS2. These changes may modulate the toxic effects of ROFA inhalation. Supported by EPA CR 817643. This is an abstract of a proposed presentation and does not necessarily reflect EPA policy.



PRESENTER: William D. Bennett

TITLE: Deposition of fine particles in children spontaneously breathing at rest

AUTHORS: WD Bennett and KL Zeman

ABSTRACT

Recent epidemiological studies suggest that children may be more susceptible to effects of inhaled particulate matter. To determine if children receive an increased lung dose of particles compared to adults we have been measuring fractional deposition (DF) of fine particles in children, age 8-18 (n=22), and adults, age 19-35 (n=12). Each subject inhaled 2um monodisperse, carnauba wax particles while following a breathing pattern previously determined by respiratory inductance plethysmography for that subject (i.e. that subject's spontaneous pattern at rest). Breath-by-breath DF (ratio of particles not exhaled/total particles inhaled) was determined by photometry at the mouth. Among the children there was no variation in DF with subject age or height. DF for the youngest children (age 8-14) (n=11) vs. the older children (age 14-18) (n=11) was  $.23 \pm .08$ (sd) and  $.20 \pm .03$  respectively (NS), also not different from the adults; DF =  $.22 \pm .09$ . On the other hand, the rate of deposition normalized to lung surface area,  $nD_{rate}$ , tends to be greater (40%) in the younger vs. older children and adults for resting breathing of these particles ( $p=0.07$ ). The variable  $nD_{rate}$  is a function of the DF, the subject's minute ventilation, and his/her lung size. The increase in  $nD_{rate}$  in the younger children is due to their higher minute ventilation in relation to their lung size. These results should prove useful in determining age-relative risks that may be associated with the inhalation of pollutant particles in ambient air. Supported by USEPA Cooperative Agreement CR817643. This is an abstract of a proposed presentation and does not necessarily reflect EPA policy.

PRESENTER: Bahman Asgharian

TITLE: Predictive model of particle deposition in the human upper airway bifurcations

AUTHORS: Asgharian, B, Zhang, L, Anjilvel, S

ABSTRACT

Particle deposition in single and double bifurcating airways due to inertial impaction was studied numerically for inspiratory flows. A single bifurcating airway with a varying bifurcation angle and parent-to-daughter tube diameter ratio was constructed. The flow field in this geometry was solved numerically using a finite-element method (FIDAP, Fluid Dynamics International, Evanston, IL). A computer program was developed to determine particle trajectory and deposition efficiency in this flow field. Inlet flow velocity profile, flow Reynolds number, and bifurcation angle were found to have substantial effects on particle deposition. A double bifurcation model was also constructed to investigate the effect of dichotomous branching pattern on particle deposition. The geometry was generated for a  $30^\circ$  bifurcation angle and a parent-to-daughter airway diameter ratio of 1.4 and 1.25 in the first and second airway bifurcations, respectively. The flow field in this geometry was solved using FIDAP for various flow Reynolds numbers. The calculated flow field data were used to simulate particle trajectory in the airways. By simulating a large number of particles, the deposition efficiencies at the first and second bifurcations were obtained. The velocity profile at the inlet of the parent tube was found to affect deposition efficiency at the first bifurcation. Deposition efficiency at the second bifurcation showed a weak dependence on inlet flow profile. It fell between the deposition efficiency curves for parabolic and uniform inflows in a single bifurcating airway having the same geometry as the second generation of the double bifurcating airway.

PRESENTER: Lei Zhang

TITLE: Reconstruction of the central airways of a male F344 rat for airflow and particle transport simulations

AUTHORS: Zhang, L, Joyner, D, Asgharian, B, Morgan, K

ABSTRACT

To accurately predict particle deposition in the rat tracheobronchial tree and to extend the work of the Chemical Industry Institute of Toxicology on airflow simulations in the rat nasal airways and upper respiratory tract, a three-dimensional computer model of the central airways of a male F344 rat was reconstructed from the serial step sections of the rat lung cast for airflow and particle transport simulations. The rat (15 weeks old, 290 g) was euthanized using CO<sub>2</sub>, and a cast was made by injecting a silicone rubber solution into the lung through the trachea. The cast was carefully trimmed to leave only the central airways (main and lobar bronchi). The trimmed cast was serial-sectioned at 150  $\mu\text{m}$  per slice. The sections were photographed, and the images were digitized to generate two-dimensional coordinates of the perimeters of the airway cross sections. By aligning the sections, three-dimensional coordinates of the airways were obtained. A three-dimensional mesh of the geometry was created that resembled the original geometry closely. The flow field in the meshed geometry was obtained using a commercially available finite element software, FIDAP (Fluid Dynamics International, Evanston, IL). Inspiratory flow rates within the physiological range for the rat were selected. Flow distribution to each lobar bronchia was approximately in proportion to the corresponding lobar volume. A computer program was developed that used the flow field data to calculate particle transport and deposition in this geometry. Simulations were performed for different particle sizes with aerodynamic diameters from 2.05 to 11.6  $\mu\text{m}$ . At an inspiratory flow rate of 440 ml/min, the total deposition fraction in the airways increased from 3.0 to 32.3% when the particle diameter increased from 2.05 to 11.6  $\mu\text{m}$ . Deposition was significant at the bifurcation sites due to impaction, and deposition patterns in the airways were more complicated than those in idealized dichotomous bifurcation models.

PRESENTER: Margaret Ménache

TITLE: Inhalability and predicted particle deposition for toxicological experiments

AUTHORS: Ménache, MG

ABSTRACT

A slight but statistically significant increase in mortality associated with elevated levels of particulate matter has been observed in a number of epidemiological studies. Toxicological experiments have not shown mechanistic or other relationships that would provide biological plausibility for the observed human responses.

In using toxicological data to understand the human responses, dosimetric models provide estimates of the dose delivered to a target site in the respiratory tract. The estimated dose will depend on inhalability (that fraction of the ambient air that is predicted to enter the upper respiratory tract) as well as other factors such as the appropriate dose metric (e.g., fractional deposition, fractional deposition per unit surface area, number of particles per macrophage).

Inhalability has been studied experimentally (using mannequins) and theoretically for humans in the work environment for many years. A single experiment has also been reported in the literature using human subjects breathing quietly in still air. No studies have been performed explicitly for laboratory animals.

Using the human exposure in still air and animal data from a particle deposition study, we have presented two inhalability equations. These equations indicate that, for humans breathing quietly in still air, inhalability is  $> 95\%$  for particles with aerodynamic diameters ( $d_{ae}$ )  $< 11 \mu\text{m}$ . In laboratory animals, inhalability is predicted to be  $> 95\%$  for particles with  $d_{ae} < 0.7 \mu\text{m}$  but only  $45\%$  for particles with  $d_{ae} < 10 \mu\text{m}$ . Inhalability of a  $3 \mu\text{m}$  particle is estimated to be about  $77\%$ .

This analysis of inhalability suggests that it is an important effect for exposures involving particles with  $d_{ae} > 1 \mu\text{m}$  in animals studies, and, therefore, for animal-to-human extrapolation. Consideration of fractional deposition adjusted for inhalability indicates that the effects are most pronounced in the upper respiratory tract.

This abstract does not reflect US EPA policy.

PRESENTER: Thomas W. Hesterberg

TITLE: Deposition and Retention of Synthetic Vitreous Fibers in the Lung: Investigating the Relationship between Biopersistence and Lung Toxicity

AUTHORS: Hesterberg, TW, Miiller, WC, and Hart, GA

ABSTRACT

Chronic inhalation studies of synthetic vitreous fibers (SVFs) showed that some fiber types produced lung disease (refractory ceramic fibers) while others did not (fiber glass). Steady state lung fiber levels were comparable for the different fiber types and rates of disappearance of fibers from the lung did not correlate with lung toxicity. Thus, rates of fiber break-down and clearance are probably not the only determinants of lung toxicity. In a short-term inhalation study, animals were exposed to high levels of SVFs or crocidolite asbestos for five days and then held without further exposure for a year, during which time some animals were periodically killed to determine the disappearance rate of fibers from the lung. After 365 days, >95% of long (>20 $\mu$ m) SVFs had disappeared from the lung compared to only 17% of long crocidolite fibers. Longer SVFs disappeared more rapidly than short SVFs, suggesting that long fibers were dissolving or breaking. Mean diameters and lengths of the SVFs decreased with time, while the mean diameter of crocidolite remained unchanged and its mean length showed an apparent increase, probably related to macrophage mediated clearance of short fibers. Leaching of oxides occurred in the fibrous glasses and slag wool and correlated with morphological changes in the fibers over time. No chemical or morphological changes were observed with crocidolite fibers. These changes in SVF number, chemistry and morphology demonstrate the relatively low biological persistence of some SVFs. The physical and chemical transformation of synthetic vitreous fibers may be critical determinants of their potential toxicity to the lung and pleura. Fibers that are rapidly dissolved and broken are thought to be less toxic due to their short residence time in the lung. In addition, it is well known that short fibers are less toxic at the cellular level than long fibers. Finally, fibers that are changed chemically, i.e. leached fibers, may be inherently less toxic than unleached fibers.

PRESENTER: Ramesh Sarangapani

TITLE: Particle deposition in the human upper airways

AUTHORS: Sarangapani, R, and Wexler, AS

ABSTRACT

Recent studies have reported association between particulate air pollution and daily mortality. Children, elderly, and asthmatics represent a sensitive population developing acute lung function deficits after exposure to near ambient levels of pollutants in urban areas. However, the precise agents - size, composition or both - is as yet unclear based on the available data. The upper airways (comprising the nasal cavity, pharynx, and the larynx) filter and condition the inspired air, and are an important respiratory defense system. In this report we present the flow characteristics in the upper airways and explore the filtering capacity of this region for particles in the size range of  $0.01\text{-}10.0\ \mu\text{m}$ . To mathematically model the flow in the nasal cavity an anatomically accurate finite element mesh of the human upper airways was constructed from a CAT scan of a healthy adult male. The 3D Navier-Stokes equation was then solved numerically using a computational fluid dynamics package FIDAP (FDI, Evanston, IL). Flow simulation was done assuming turbulent flow conditions in the upper airways, under different breathing conditions. Finally the equations of motion for the particles were solved to evaluate the particle trajectories and determine the particle deposition patterns.

PRESENTER: Constantinos Sioutas

TITLE: Development of a continuous particle mass monitor by measuring pressure drop across filters.

AUTHORS: Koutrakis, P, Wang, PY. Sioutas, C, Wolfson, JM.

ABSTRACT

We have developed a continuous mass measurement method based on monitoring the pressure drop across a porous membrane (Nuclepore) filter over a period of time. Aerosol samples are first drawn through a diffusion dryer that reduces the sample relative humidity to 40% or less, and subsequently through a series of porous membrane filters. The increase in the pressure drop with mass loading has been studied for different sampling flow rates, pore size, and particle density, for particles in the size range 0.1-2.0  $\mu\text{m}$ . Filters of pore size from 1 to 8  $\mu\text{m}$  were tested for sampling flows varying from 0.7 to 16 LPM. The test aerosols used in the experiments included PSL latex, ammonium sulfate, sodium chloride, and indoor air particles. Aerosol mass concentrations ranged from 8.0 to 132.0  $\mu\text{g}/\text{m}^3$ . Our results showed that by appropriately choosing the face velocity and pore size of the filter, interception becomes the dominant mechanism for particle deposition. This makes the pressure drop independent of particle size and only dependent on the mass concentration of the sampled aerosol. The pressure drop per unit time and particle concentration averages to 0.0088 ( $\pm$  0.0005) inches  $\text{H}_2\text{O}/\text{hr}/(\mu\text{g}/\text{m}^3)$  for particles in the range 0.1-2.0  $\mu\text{m}$ . Our continuous mass monitoring method can detect as little as 5  $\mu\text{g}/\text{m}^3$  in about 1 hour.

We have also developed a theory that explains the mechanisms for increasing the pressure drop across Nuclepore filters with mass loading. Our sampler was also validated in field tests by comparing its performance to collocated continuous (Tapered Element Oscillating Microbalance, TEOM) and time-integrated (Harvard Impactor, HI) samplers. Excellent agreement was found between our continuous sampler and HI for 3-hour sampling intervals, whereas HI and the continuous mass monitor were systematically measuring higher  $\text{PM}_{2.5}$  concentrations than those determined with the TEOM, presumably due to volatilization losses of the latter method.

PRESENTER: Frederick W. Lipfert

TITLE: Trends in airborne particles in the United States

AUTHOR: Frederick W. Lipfert

**ABSTRACT**

This paper examines trends in population exposures to airborne particles in the United States, for the period 1940-1990. Changes in emissions from specified source categories and trends in ambient monitoring data are considered, both as national averages and for specific locations. To address the question of trends by particle size, particulate matter (PM) sources are segregated into two groups: those that emit primarily combustion-derived particles and those that emit the larger particles typically produced by certain industrial processes. Substantial downward trends are seen in both groups. The primary source of ambient trend data is the database for total suspended particle (TSP) measurements; these trends are supplemented by comparing 1970 data from the Cascade Impactor Network with 1980 data from the Inhalable Particulate Network to provide trends by particle size during the period immediately after implementation of the Clean Air Act. Trends in coefficient of haze (a measure of small carbonaceous particles) are also compared. The paper concludes that there have been substantial improvements in population exposures to all particle sizes during this period, primarily in the locations having the worst air quality. It thus follows that epidemiological studies designed to study chronic health effects must recognize differential rates of air quality improvement over time.

PRESENTER: Robert A. Michaels

TITLE: Permissible Daily Airborne Particle Mass Levels Encompass Brief Excursions To the 'London Fog' Range Which May Contribute To Daily Mortality and Morbidity In Communities

AUTHOR: Michaels, R. A.

ABSTRACT

Recent studies associate 24-hour  $PM_{10}$  levels within EPA's  $150\text{-}\mu\text{g}/\text{M}^3$  standard with mortality and morbidity. These findings remain unexplained. This study documents the contribution of brief particle mass excursions to 24-hour average levels, and evaluates the possible public health significance of such excursions. Two technologies were identified for short-term measurement of particle mass. Ten 24-hour periods distributed among three locations were examined with  $PM_{10} < 150\text{ }\mu\text{g}/\text{M}^3$ . At all three locations, and in six of 10 days, excursions exceeded  $150\text{ }\mu\text{g}/\text{M}^3$ . Fifteen-minute excursions approached  $2,000\text{ }\mu\text{g}/\text{M}^3$ , surpassing the 1952 London fog despite regulatory control of particles at the 24-hour time frame. Toxicology literature confirmed the harmfulness of brief exposure to particles in the range of observed excursions. Fine particle excursions are followed by prolonged internal exposure of lungs which cannot efficiently clear them. Prolonged internal exposure may correlate with prolonged, potentially lethal, cardiopulmonary stress, especially for the frail elderly and infirm. An examination of policy documents revealed consistency of observed short-term health effects of particles with EPA criteria for air pollutant standard setting. However, EPA has focused upon protracted toxicological causes, whereas this study suggests that shorter-term mechanisms may cause or contribute to causing the unexplained effects. The study thus supports the recommendation that EPA further consider imposing a one-hour particle standard, despite Agency uncertainty that *"the majority of effects"* observed after daily exposure would occur after briefer exposures. A one-hour mass limit of  $300\text{ }\mu\text{g}/\text{M}^3$  might enhance protection against acknowledged short-term effects, avoiding a burdensome change in the  $150\text{ }\mu\text{g}/\text{M}^3$  24-hour mass limit.

**PRESENTER:** Dean Lillquist

**TITLE:** Field Evaluation of Airmetrics Minivol PM<sub>10</sub> and PM<sub>2.5</sub> Samplers

**AUTHORS:** Lillquist, D.R. and Rederlechner, N.

**ABSTRACT**

AIRmetrics has recently developed a portable particulate air sampler. Samplers are: small, lightweight, battery powered, very quiet, and relatively inexpensive. Depending on the preseparator head used, each sampler has the ability to be set up for TSP, 10 $\mu$ m or 2.5 $\mu$ m cut size sampling. These features make the sampler an attractive research tool, ideal for both ambient saturation studies utilizing numerous sampling locations in an air shed as well as studies requiring indoor/outdoor measurements. During the winter of 1995-1996, AIRmetrics samplers, equipped with both PM<sub>10</sub> and PM<sub>2.5</sub> sampling heads were compared to a Sierra-Andersen Series 240 Dichotomous sampler. Three AIRmetrics PM<sub>10</sub>, three AIRmetrics PM<sub>2.5</sub> and a Sierra-Andersen Dichotomous sampler were run side-by-side. Fourteen sampling events were captured over a window of eight weeks. This study design allowed for both intra-sampler variability of the AIRmetrics samplers and inter-sampler comparison to an EPA reference method. Results revealed the accuracy and precision of these samplers and help to establish their use in specialized applications measuring particulate matter of various cut sizes.

The results revealed:

1) The AIRmetrics PM<sub>10</sub> sampler gave consistent but higher ( $\sim +11\%$ ) results compared to the SA PM<sub>10</sub> and; 2) The AIRmetrics PM<sub>2.5</sub> sampler demonstrated more variability and there was difficulty in establishing a consistent correlation between it and the Sierra-Andersen PM<sub>2.5</sub> sampler.

PRESENTER: Timothy C. Coburn

TITLE: Reduction in vehicle emissions attributable to alternative transportation fuels and its prospective impact on air quality and public health

AUTHORS: Coburn, T. C., Kelly, K. J., and Bailey, B. K.

ABSTRACT:

The Clean Air Act of 1990 requires the composition of the U. S. Federal vehicle fleet to shift towards clean fuel vehicles beginning in 1998. That year, 30% of all vehicles in the fleet must be CFVs, increasing in successive years to 100% by 2001. The Energy Policy Act of 1992 further constrains the Federal fleet to include alternative fuel vehicles (AFVs), up to 25% of the total beginning in 1996, with the percentage rising to 75% in the year 2000. CFVs and AFVs are vehicles which are designed to operate on fuels other than conventional gasoline or diesel, and which contribute lower levels of pollutants to the atmosphere than their conventional-fuel counterparts. The U. S. Environmental Protection Agency establishes and maintains the Federal emissions standards which CFVs and AFVs are required to meet. In addition to actual placement of vehicles, the Energy Policy Act, along with the Alternative Motor Fuels Act (AMFA) of 1988, directs the U. S. Department of Energy to collect and report data pertaining to the overall performance of AFVs. The objective of this effort is to monitor alternative fuel technology and to promote its development to insure that the ultimate objectives of the legislation are met. A significant component of the Federal data collection effort on AFVs involves emissions testing. A large program of tests was recently completed, some of results of which are summarized in this presentation. Comparative information is presented on the levels of regulated exhaust emissions, toxic constituents, particulate matter, and ozone forming potential for various vehicle types, makes, and models operating on several alternative fuels around the country. Comparisons are also made to the corresponding levels of pollutants emitted by standard vehicles operating on conventional gasoline and diesel. The information obtained to date indicates that original equipment vehicles designed to operate on alternative fuels emit lower levels of pollutants than their gasoline or diesel counterparts; and that, for some combinations of fuel and vehicle type, the reduction is substantial. Based on this evidence, communities having a large concentration of alternative fuel vehicles may exhibit reduced incidences of disease and health disorders that are related to exhaust emissions. Other benefits, such as reduced costs of medical care, reduced insurance premiums, and a generally-improved business climate, are likely to be realized.

PRESENTER: Steven H. Hoke

TITLE: A Continuous Denuder for Acid Aerosols and Gases

AUTHORS: Hoke, S.H.

ABSTRACT

A continuous flow denuder has been developed to extract water soluble acid aerosols and gases from the atmosphere prior to analysis. To avoid interaction between the aerosol or gas to be measured and the surface walls of the denuder, it has been designed so that there is minimal surface interaction prior to extraction. This denuder was designed specifically to support the real-time halogen acid aerosol/gas analyzer developed by the Army or any analyzer which requires extraction of an aerosol or gaseous analyte into aqueous solution prior to analysis. The denuder body is constructed of plexiglas G acrylic material. Trapping solution is pumped to the denuder entrance at a rate of 2.5 milliliters per minute. Air is drawn into the denuder at 2.5 liters per minute by means of a vacuum source. The air and liquid meet at the denuder entrance and are immediately drawn into the extraction chamber where turbulent flow conditions exist. Any aerosols or gases in the air sample that are soluble in the trapping solution are extracted from the air into the liquid phase in the extraction chamber. The advantages of this device are that it provides for a small, non-breakable, continuous flow denuder which can be operated in any orientation. The extraction efficiency of this device compares favorably with results obtained using a midjet impinger for sampling various halogen acid aerosols and gases. An adapter has been designed to slip over the denuder body for the purpose of measuring air flow through the device without disrupting the normal operation of the denuder. This denuder when used in conjunction with the real-time acid aerosol/gas analyzer can provide real-time concentration profiles from exposures of an episodic nature.

PRESENTER: Raimo O. Salonen

TITLE: Particulate Matter in Northern Climate of Helsinki Metropolitan Area, Finland

AUTHORS: Hämeikoski, K, Salonen, R O

ABSTRACT

TSP has been measured with high volume samplers in Helsinki Metropolitan Area since 1978, and PM<sub>10</sub> has been measured with Wedging PM<sub>10</sub> high volume samplers since 1987. Continuous PM<sub>10</sub> measurements with Beta-gauge and oscillating microbalance monitors have been conducted since 1991 at different sites.

Stack and tailpipe emissions of PM are low in the area due to district heating, effective control measures and lack of major industry. Indirect emissions from the surfaces of paved roads are a large PM source due to street sanding and use of studded tires in winter. PM<sub>10</sub> and especially TSP concentrations show clear seasonal behaviour, in which the highest concentrations occur in spring and somewhat lower peaks in fall. The spring peaks coincidence with melting of snow and drying of streets. The fall peaks are probably due to the start of street sanding and the beginning of the use of studded tires.

During the recent years there has been a downward trend in TSP concentration, which is probably due to improved street maintenance especially in spring. Still, the highest annual TSP average concentration in 1994 was 83  $\mu\text{g}/\text{m}^3$  in urban traffic environment, while the corresponding PM<sub>10</sub> concentration was 28  $\mu\text{g}/\text{m}^3$ . The highest 24-hour PM<sub>10</sub> concentration reached 150  $\mu\text{g}/\text{m}^3$  in 1994. When the 24-hour concentrations were compared to the PM<sub>10</sub> levels used in the newest WHO health effect assessment, the three-day-average PM<sub>10</sub> concentration of 50  $\mu\text{g}/\text{m}^3$  was exceeded on six separate occasions and 100  $\mu\text{g}/\text{m}^3$  was exceeded once.

According to preliminary results, resuspension seems to have a major impact on PM<sub>10</sub> concentrations especially in spring, and therefore the PM<sub>10</sub> concentrations in the area are only partly combustion-related. A comprehensive research programme has been started this spring to study the chemical composition and size distribution of PM<sub>10</sub>, and to estimate the contribution of different sources to PM<sub>10</sub>. These results will be used also in future epidemiological studies. The main question is, whether these, relatively high 24-hour PM<sub>10</sub> concentrations are as harmful as the recent epidemiological studies would suggest.

PRESENTER: Dagan Wright

TITLE: Relationships among EPA sampling methodologies for PM<sub>10</sub> and PM<sub>2.5</sub>; and their associations with meteorological variables (temperature, wind-speed, relative humidity)

AUTHOR: Wright, Dagan

ABSTRACT

Even with the strict EPA guidelines for reference and equivalent sampling methodology, variation between manual versus automated sampling methods exist. The purposes of the study are to look at variations of measurements between sampling methodologies and their associations with meteorological variables (temperature, relative humidity, and wind speed), to observe the ratio of PM<sub>2.5</sub> to PM<sub>10</sub> and their associations with meteorological/methodology variables, and to observe which automated sampling method comes in closer agreement to the manual sampling method used by the state. The following EPA approved reference manual samplers are located with a meteorological station at one site: Sierra-Anderson Model 1200 PM<sub>10</sub> High-Volume Air Sampler System (HIVOL) and Sierra-Anderson SA241M Dichotomous Samplers. The following EPA reference/equivalent automated samplers, each automated sampler working at different time periods, were also located on the same site as the manual samplers: TEOM Series 1400 PM<sub>10</sub> instrument and Anderson Instruments Model FH621-N PM<sub>10</sub> Beta Attenuation Monitor (BETA). The following results were observed: the variation of the relationship of TEOM to HIVOL was associated with a model (R Square = 0.269,  $p < 0.0001$ ) that includes temperature, relative humidity, and wind speed; the state adjusted measurements of the TEOM was on the average 1.46 times greater than the HIVOL reading; the ratio of PM<sub>2.5</sub> to PM<sub>10</sub> is weakly associated with relative humidity (R Square = 0.073,  $p < 0.0001$ ); comparisons of the automated sampling methods showed the BETA had a the highest correlation to the HIVOL (R Square = 0.944,  $p < 0.0001$ ), and measurement of the ratio of PM<sub>2.5</sub> to PM<sub>10</sub> had a standard deviation of 0.186. Linear relationships exist between sampling-method variations, the ratio of PM<sub>2.5</sub> to PM<sub>10</sub>, and meteorological variables. These linear models can be elaborated in future research. The BETA (automated sampler) mirrored the HIVOL (state manual sampler) to a greater degree.

PRESENTER: Michael B. Meyer

TITLE: Federal reference and equivalent particulate matter methods:  
the importance of standardization

AUTHOR: Michael B. Meyer

ABSTRACT

Recent published epidemiological health studies have increased the concern over human health-related issues associated with airborne particulate matter. In addition, the current reviews of particulate-based air quality regulations in the United States and Europe have triggered an increased focus on the collection, measurement and identification of ambient particulate matter. Advances in PM mass monitoring instrumentation have made possible enhanced ambient monitoring in terms of temporal resolution and chemical speciation. With the introduction of new methods, however, comes the inevitable desire to compare and contrast performance and field data with historical measurements. While this strategy is important to provide a link to the historical data base, ensuing discussions frequently focus upon individual data bases instead of the fundamental scientific basis for measurement technologies. A brief review of current U.S. EPA reference and equivalent PM<sub>10</sub> methods will be presented. Emphasis is placed on each of the method's sampling and measurement procedures, with particular attention paid to method variations and probable causes. With particulate matter, unlike criteria gaseous pollutants, a potential exists for what is defined as particulate matter to change its mass as a result of unstable residence on the filter due to light volatile substances (e.g. water, aromatic hydrocarbons, secondary aerosols, organic carbon) associated with the particulate matter and filter media. Some have speculated that particulate matter retention will become an acute issue with the possible introduction of a new fine particulate matter (PM<sub>2.5</sub>) standard in 1997. Particulate matter measurements are often used to identify health risk and to protect public health through control measures determined and enforced by regulatory agencies. Thus, it is critical to carefully define the 'filter history' or physical conditions under which particulate matter is both sampled and measured. Field data from recent research programs suggest that the current federal PM<sub>10</sub> reference method does not indicate particulate matter pollution in a consistent fashion. The arbitrary choice of sampling and equilibration parameters leads to a method that is inherently ill-defined. The importance of standardizing key parameters such as sampling temperature, filter face velocity, equilibration temperature and relative humidity is shown. In the absence of such standardization, the measurement of particulate matter mass concentrations can, at times, vary by as much as a factor of two or more.

PRESENTER: John A. Cooper

TITLE: Fine, coarse, and PM10 concentrations: relationships and regulatory implications

AUTHORS: Cooper, J.A., Patterson, B.C, Tawney, C.W.

ABSTRACT

The adequacy of the current PM10 standard is being reviewed by the U.S. Environmental Protection Agency and a possible new PM2.5 standard is being considered. Two essential aspects of this review are the relationships between PM10, fine (PM2.5) and coarse particles, and the PM2.5 concentration that is equivalent to the current PM10 standards. A non-rural PM10, PM2.5 and coarse particle database consisting of 12,059 records has been compiled, evaluated and PM10 equivalency defined. The results of our analysis indicates that on a national basis, PM2.5 concentrations are not correlated with PM10 concentrations and that the PM2.5 concentration equivalent to the current PM10 24-hour standard concentration is about 95  $\mu\text{g}/\text{m}^3$  or greater. In addition, the median PM2.5 concentration as percent of PM10 for the subset of PM10 samples greater than 100  $\mu\text{g}/\text{m}^3$  with the low coarse fraction is 62.6%. These results have significant implications with regards to implementing possible new standards and defining non-attainment airsheds. Defining the PM2.5 concentration equivalent to the current PM10 standard concentration, for example, is particularly important because it defines the status quo or benchmark for comparing concentrations in a possible new PM2.5 measurement scale. The database used for this analysis and the PM10, PM2.5, and coarse particle relationships will be described and the implications discussed.

PRESENTER: Yuanzhang Li

TITLE: Examination of the legitimacy of using outdoor PM levels as a surrogate for personal exposures in epidemiological studies

AUTHORS: Lihan Y, Roth HD, Li Y

ABSTRACT

A major premise in PM epidemiological studies is that outdoor levels are closely linked with personal exposures over time. This is questionable, however, because individuals spend a great deal of time indoors at home, at work, or in other microenvironments. To examine the soundness of the association between outdoor levels and personal exposures, we have studied PTEAM data from Azusa and Riverside, California. In addition we have reviewed exposure studies by Janssen, et al. (1995) on Wageningen data and by Liou, et al. (1990) on New Jersey data. In Azusa, the association between outdoor levels and personal exposures was negative but statistically insignificant. In Riverside, the association was marginally significant but was at such a low level that the exposure data could have easily distorted epidemiological results. Liou, et al. (1991) found no strong statistical association between outdoor levels and personal exposures in 8 out of 14 study subjects. Finally, Janssen, et al. (1995) did not report enough data to make any judgements about the association between outdoor levels and personal exposures. Given all these findings, it is questionable whether outdoor measurements can be used as a surrogate for personal exposures in epidemiological studies.

PRESENTER: George D. Thurston

TITLE: Associations Between PM-10 and Mortality in Multiple U.S. Cities

AUTHORS: Thurston, G.; Gwynn, R.C.; Ito, K.

ABSTRACT

Recent analyses have indicated an association between elevated concentrations of air pollutants, including particulate matter less than 10  $\mu\text{m}$  in aerodynamic diameter (PM10), and increased human mortality. Some reviews have suggested that PM10 mortality effects are similar from place to place, despite variations in PM10 and population composition. However, few past PM10 studies have fully considered the potentially confounding influences of other pollutants, and differing analytical methods among the papers make direct quantitative comparisons and broad conclusions difficult.

In this work, we have developed and analyzed a comprehensive and consistent database of daily air pollution, weather, and mortality data for the period 1981-1990 in multiple major cities spread throughout the 48 contiguous U.S. states. These cities, each having differing weather, pollution, and population characteristics, include: New York City, Atlanta, Houston, St. Louis, Chicago, Detroit, Minneapolis, San Francisco, and Los Angeles. The pollutants considered in each city include PM10, carbon monoxide (CO), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>) and ozone (O<sub>3</sub>). The data were analyzed in a consistent manner, giving directly comparable time-series regression results for these pollutants. Positive PM10-mortality associations were seen in all of these cities, but the size and significance of the RR estimates associated with PM10 varied across cities. Various methods of seasonality adjustment were investigated, but found to cause little variation in PM10 results. The inclusion of other pollutants caused the PM10 RR's to decline somewhat, though PM10 was less affected than other pollutants.

PRESENTER: George D. Thurston

TITLE: A time series analysis of acidic PM and daily mortality and morbidity in the Buffalo, NY region.

AUTHORS: Gwynn, R.C., Burnett, R.T., Thurston G.D.

ABSTRACT

A key unresolved question in the particulate matter (PM) - mortality and PM - morbidity associations reported by recent time-series studies is: What characteristic(s) of PM is (are) responsible for these health effect associations? A component which may provide one biologically plausible mechanism for PM causality is aerosol acidity ( $H^+$ ). For example, acidic aerosols were elevated during the London, England "killer" fog episodes of the 1950's and 60's. While a number of studies have shown that  $H^+$  is associated with increased morbidity in the U.S. and Canada, no studies have considered a long enough series of daily  $H^+$  measurements to meaningfully test whether  $H^+$  is or is not associated with excess acute mortality at present-day ambient  $H^+$  levels in North America.

In this work, we considered a two-and-a-half year record of daily  $H^+$  measurements (May 1988-Oct. 1990) collected in the Buffalo, NY region in a Poisson time-series analysis of total and respiratory daily mortality and hospital admissions. Other air pollutants considered in the analysis included: PM<sub>10</sub>, ozone, carbon monoxide, sulfur dioxide, and nitrogen dioxide. Various modeling techniques were used to control for confounding of effect estimates due to seasonality, weather and day-of-week effects. Of the pollutants considered,  $H^+$  was most consistently significantly ( $p < 0.05$ ) associated with adverse health effects across mortality and hospital admissions categories. Relative risks (RR's) associated with increases in pollutants equal to the maximum minus the mean concentration were estimated (e.g., 345 nmoles/ $m^3$  for  $H^+$ , 65  $\mu g/m^3$  for PM<sub>10</sub>). Respiratory hospital admissions RR's associated with  $H^+$  from the various models ranged from 1.30-1.50, while those for respiratory mortality ranged from 1.49-1.60. Total mortality RR's associated with  $H^+$  ranged from 1.15-1.18, while those associated with PM<sub>10</sub> ranged from 1.07-1.10. Simultaneous regression with gaseous air pollutants had minimal effects on the  $H^+$  coefficients. These analyses indicate that  $H^+$  may be a significant contributor to the acute adverse morbidity and mortality health effects associated in the past with PM mass.

PRESENTER: Kevin Fennelly

TITLE: Cardiopulmonary morbidity associated with particulate air pollution in Denver, Colorado, 1989-92

AUTHORS: Fennelly, K, Bucher Bartelson, B

ABSTRACT

Cardiac and pulmonary mortality has been associated with concentrations of particulate matter (PM) in urban air pollution; fewer data are available regarding the association of PM with hospitalizations for cardiopulmonary diseases. Recently, hospital admissions for congestive heart failure have been associated with ambient carbon monoxide (CO) concentrations. We tested the hypothesis that both CO and PM concentrations are positively associated with cardiopulmonary hospital admissions in Denver, Colorado. We obtained daily data for hospital admissions, criteria air pollutants, weather variables, pollen and spore counts, and an index of flu-like illnesses for the period from May 1, 1989 through December 15, 1992. We analyzed the data using a generalization of Poisson regression which allows for serial correlation. The model included variables for day of the week, seasonal correction, and time trend as well as the above covariates. Both  $PM_{10}$  (PM less than 10 microns) and CO were associated with cardiopulmonary hospitalizations in a single pollutant model. There were no significant associations with a control outcome, non-respiratory cancer admissions. The relative risk (RR) associated an increase of 100  $mcg/m^3$  of  $PM_{10}$  was 1.13 (1.04, 1.22; 95% confidence interval). The RR associated with an increase of 5 parts per million of CO was 1.11 (1.02, 1.20). When both pollutants are included in the model, only  $PM_{10}$  remained significant ( $p=0.013$ ). We found similar results when we excluded the only 4 days when the daily  $PM_{10}$  concentration exceeded 100  $mcg/m^3$ , which is below the current U.S. National Ambient Air Quality Standard. We conclude that particulate air pollution in Denver is positively associated with hospital admissions for cardiopulmonary diseases at concentrations below the current U.S. federal standard.

**PRESENTER:** Gerhart Scheuch

**TITLE:** Size distribution of ambient particles and its relevance to human health

**AUTHORS:** Heyder, J, Brand, P, Heinrich, J, Peters, A, Scheuch, G, Tuch, T, Wichmann, E

**ABSTRACT**

Recently it has been recognized that ambient particles might play a greater role in pollution-induced respiratory responses than previously thought. Fine or even ultrafine particles might be more "toxic" than coarse particles and the "physical" toxicity of these particles might even exceed their "chemical" toxicity. Consequently, more insight into health related aspects of particulate air pollution will be obtained by correlating respiratory responses with mass and number concentration of ambient particles.

On 145 days during the winter season 1991/92, mean size distributions of ambient particles in the range 0.01 - 2.5  $\mu\text{m}$  were determined with a differential mobility analyser and an optical particle counter in Erfurt, a town in East Germany. During this period 79% of the particles were smaller than 0.1  $\mu\text{m}$  in diameter. The corresponding mean mass concentrations were calculated assuming an average particle density of 1.5 g cm<sup>-3</sup>. 82% of the mass concentration were associated with particles in the size range 0.1 - 0.5  $\mu\text{m}$ . Since the variation of particle number concentration was not highly correlated with the variation of particle mass concentration ( $r=0.51$ ), these values were correlated with daily mean expiratory peak flow rates of 27 non-smoking, asthmatic residents of Erfurt. Elevated particle number concentration was closer associated with a decrease in expiratory peak flow rate than elevated particle mass concentration. Thus, ultrafine particles may indeed play a role in air pollution-induced alterations of respiratory lung function.

**PRESENTER:** Denis Zmirou

**TITLE:** Short-term effects of air pollution on health: a European approach using epidemiologic time-series data

**AUTHORS:** Katsouyanni, K, Zmirou, D, Spix, C, Sunyer, J, Schouten, JP, Ponka, A, Anderson, HR, Le Moullec, Y, Wojtyniak, B, Vigotti, MA, Bacharova, L

**ABSTRACT**

The APHEA (Air pollution on Health: European Approach) project is an attempt to provide quantitative estimates of the short-term health effect of air pollution, using an extensive data base from 10 different European countries, which represent various social, environmental, meteorological and air pollution situations. Within the framework of the project, the methodology of analyzing epidemiological time series data, as well as that of performing meta-analysis, are further developed and standardized.

Data have been collected from 15 European cities with a total population exceeding 25 million. The exposure data consist of daily measurements of black smoke, sulphur dioxide, suspended particles, nitrogen dioxide and ozone (each available in several, though not all, cities) from already existing monitoring networks. There is substantial variability in air pollution mixtures and air pollutant levels in participating cities. The mean (24 h) levels of SO<sub>2</sub> range 27-327  $\mu\text{g}/\text{m}^3$  in the winter season, and those of black smoke range 15-292  $\mu\text{g}/\text{m}^3$ , the mean (1 h) levels of ozone in the summer season range 32-166  $\mu\text{g}/\text{m}^3$ . The outcome data are daily counts of total and cause-specific deaths (respiratory and cardiovascular conditions). Data on potential confounders (mainly meteorological and chronological variables) are also collected. Poisson regression allowing for autocorrelation and overdispersion is used in the analysis, after control of seasonality, other periodic patterns and other confounding effects. City specific results are summarized after evaluation of their heterogeneity.

The results concerning total mortality and particulate matter pollution are consistent with those from the U.S. although the relative risks are at the lower range of the U.S. estimates. A 50  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> and black smoke (BS) is associated with an increase of 2.1% and 2.9% [2.1-3.7 = 95% CI] in total mortality in Western European cities. A larger effect of BS is observed for respiratory mortality: RR = 1.04 [1.00-1.08] and for cardiovascular mortality RR = 1.04 [1.01-1.06]. Despite greater average concentrations of particles, central European cities do not show significant associations, raising interesting questions as to the nature of the pollution make-up that is related to health effects. Similarly, some seasonal variability of the relative risks suggest different contributions of the sources of particles. Sulphur dioxide shows consistently greater associations with mortality than particulates.

PRESENTER: MB Snipes

TITLE: The 1994 ICRP66 human respiratory tract model as a tool for predicting lung burdens from exposures to environmental aerosols.

AUTHORS: Snipes, MB, James, AC, Jarabek, AM

ABSTRACT

The new ICRP human respiratory tract model (ICRP66, 1994) was used to predict deposition rates for the extrathoracic, tracheobronchial, and alveolar-interstitial (AI) regions of the respiratory tract for specific environmental aerosols, and to model particle retention in the AI region for chronic exposures. Trimodal (fine, intermodal, and coarse) environmental aerosols reported for Philadelphia, PA, and Phoenix, AZ, were selected for comparison. Because both acute and chronic effects of ambient aerosols are of concern, deposited and retained dose metrics were estimated and presented as mass or number of particles per unit of regional surface area and lung mass, respectively. Modeling was done for eight demographic groups, including a group having symptoms consistent with cardiopulmonary disease, using predicted daily ventilation patterns appropriate for each group. To estimate particle burdens in the AI region from chronic exposures, dissolution-absorption half-times for the fine, intermodal, and coarse modes of the example aerosols were assumed to be 10, 100, and 1000 days, respectively. Default ICRP66 values for particle physical clearance parameters were used. Ventilation patterns greatly influenced predicted particle deposition rates, especially in the younger cohorts. Modeling results indicated substantial differences between the two environmental aerosol deposition and accumulation patterns for the intermodal and coarse modes, but very similar patterns for the fine modes. These similarities in predicted deposition patterns for the fine aerosol modes suggest that any differences in biological responses to these environmental aerosols may be associated with differences in their chemical constituents. Implications for comparisons of distributions of mass and particle numbers for environmental aerosols collected by samplers with different size cut-points will be discussed.

PRESENTER: William D. Bennett

TITLE: Enhanced deposition of fine particles in COPD patients spontaneously breathing at rest

AUTHORS: WD Bennett , KL Zeman, CS Kim, and J Mascarella

ABSTRACT

Particulate air pollution has been linked to acute increases in mortality among individuals with preexisting cardiorespiratory disease. These individuals may receive an increased dose of particles to their lungs compared to healthy subjects. We measured fractional deposition (DF) of inhaled, fine particles in subjects with moderate-severe COPD (n=13) and an age-matched group of subjects (n=11) with normal pulmonary function, mean age = 62 vs. 67 yrs. and FEV1(%pred) = 33 vs. 90 respectively. Each subject inhaled 2 $\mu$ m monodisperse, carnauba wax particles while following a breathing pattern previously determined by respiratory inductance plethysmography for that subject (i.e. that subject's spontaneous pattern at rest). DF (ratio of particles not exhaled /total particles inhaled) was determined by photometry at the mouth. COPD patients had greater DF than normals, 0.40 $\pm$ 0.16(sd) vs. 0.26 $\pm$ 0.06, p<.02 . The COPD patients also had an increased resting minute ventilation for DF measurements compared to normals, mean  $\dot{V}_E$  = 11.2 l/min vs. 7.4 l/min. As a result, deposition rate,  $D_{rate}$ , proportional to particles depositing/time, was nearly 2.5 times greater in COPD subjects relative to normal,  $D_{rate}$  = 4.6  $\pm$  2.5 and 1.9  $\pm$  0.8 respectively, p<.005. Among the COPD subjects,  $D_{rate}$  increased with increasing specific airway resistance, sRaw (r = .70, p < .01). These data indicate that at rest COPD patients receive a substantially increased dose of inhaled, fine particles compared to normals that increases with severity of their airways disease. Supported by USEPA Cooperative Agreement CR817643. This is an abstract of a proposed presentation and does not necessarily reflect EPA policy.

PRESENTER: Gerhard Scheuch

TITLE: Size dependent particle clearance and retention in the human tracheobronchial region

AUTHOR: Scheuch, G

ABSTRACT

In a series of experiments nonsoluble, radiolabelled aerosol particles were administered to volunteers as a 'bolus' at a preselected point in a breathing cycle. During a short period of breathholding the particles were deposited at the chosen lung depth, VL, which is determined by the volume that is inhaled following the aerosol bolus. Inhaling the bolus near the end of the breath,  $VL < 100\text{ml}$ , ('shallow bolus') the particles deposit in the tracheobronchial and extrathoracic airways. Measuring the retention of the particles in the lung with a sensitive collimated lung counter, two distinct phases of particle clearance were found. Particles that cleared in a rapid phase left the lungs with half-times of about 2.5 hours, while particles in the slow phase were cleared with half times of about 5 - 30 days.

The fraction of fast-clearing particles strongly depends on particle size. While particles with geometric diameters,  $d$ , larger than about  $6\text{ }\mu\text{m}$  were cleared almost completely within 24 hours, about 50% of particles with  $d = 2.5\text{ }\mu\text{m}$  were still found in the lungs after one day. The results suggest that there is a slow phase of clearance from the airways. This slowly cleared fraction of the tracheobronchial deposit decreases with increasing particle size.

Preliminary results with ultrafine particles ( $^{111}\text{InCl}$ ),  $d = 30\text{ nm}$ , inhaled with the shallow bolus inhalation technique, yielded a fraction of about 80% of slowly cleared particles.

The longer residence time of small and ultrafine particles in the tracheobronchial region is of direct practical relevance to human health, because it increases the dose of inhaled pollutants applied to the airway epithelium.

PRESENTER: Werner Hoffman

TITLE: Modeling deposition and clearance of ultrafine particles in human lungs

AUTHORS: Hofmann W, Bergmann R, Koblinger L

ABSTRACT

Epidemiological studies have recently indicated that inhalation of ambient ultrafine particles may cause statistically observable health effects despite the relatively small mass deposited in the lungs. These results suggest that health effects may be related to the number of particles or to their total surface area rather than to the mass deposited. It has also been speculated that people with lung diseases may be more affected by particulate air pollution than healthy adults.

The theoretical simulations of deposition and clearance of inhaled particles in the human lungs are based on a stochastic transport, deposition and clearance model, which accounts for the randomness of the human airway structure. For the investigation of deposition and clearance in diseased lungs, appropriate morphometric scaling factors were applied, considering also related differences in breathing patterns.

To study potential effects of ultrafine particles, a given mass of inhaled particulate matter is divided among an increasing number of particles with correspondingly decreasing diameters, ranging from 10 ( $\mu$ )m down to 1 nm. The distribution of the various particle sizes with respect to number, surface area and mass has been computed for individual bronchial and alveolar airway generations. Three major observations can be made: (i) the dependence of physical deposition mechanisms on particle size produces significantly different particle deposition patterns throughout the lung; (ii) this size effect in the tracheobronchial region is further enhanced by the dependence of the fast and slow cleared fraction on particle size; and, (iii) for a given particle size, the distribution of total particle surface area and number of deposited particles is shifted toward more distal airways, relative to the mass distribution.

In conclusion, if health effects are related to the number of particles deposited in a given airway or to the total surface area available for chemical reactions, then ultrafine particles are more effective pollutants than large particles having the same mass.

**PRESENTER:** Tee L. Guidotti

**TITLE:** The "Cohen Hypothesis" Revisited: Dust Retention, Airflow Obstruction and Disease Risk

**AUTHOR** Tee L. Guidotti

**ABSTRACT**

In 1978 Cohen advanced a mechanistic theory, based on epidemiologic observations, for which evidence gathered since has been supportive. Cohen's theory suggests that a toxic exposure (most commonly but not exclusively cigarette smoking) may in genetically susceptible individuals produce a fundamental lesion expressed as (but not restricted to) airways obstruction; this lesion may then result in accumulation of potential carcinogenic activity in airways and in the circulation as a consequence of impaired host defenses and reduced clearance. Although cancer risk was explained in the hypothesis, other health outcomes are also postulated. Tests of hypotheses based on this theory have confirmed an association between airways obstruction and indices of cancer risk (mortality, incidence, frequency of bronchial epithelial metaplasia in high risk groups). Cohen's theory predicts that individuals with airways obstruction should have higher detectable and biologically significant levels of biomarkers for exposure or effect, controlling for present smoking and intake of agents inhibiting carcinogenesis and restricting age and sex, than nonobstructed individuals. Cigarette smoking is the ideal exposure by which to test the hypothesis because it is more prevalent, readily quantifiable, and accessible in numbers than solely occupational exposures, and because it provides a generalizable model based on an extreme situation of carcinogenic burden. We proposed a test of this hypothesis in 1984, using cigarette smokers with and without airways obstruction. A limited pilot study performed in 1983 confirmed the feasibility of the protocol and provided data on group means and variances.

**PRESENTER:** Nicole Janssen

**TITLE:** Relationship between personal and ambient fine particle concentrations

**AUTHORS:** Janssen NAH, Hoek G, Harssema H, Brunekreef B

**ABSTRACT**

A study was conducted on the relationship between repeated measurements of personal and ambient PM10. Averaging time was 24 hours and measurements were spaced one week apart. Participants were 37 non-smoking adults with no occupational exposure (age 50-70) and 45 children (age 10-12). For each subject 4-8 measurements were obtained. This design allowed for correlating personal and ambient PM10 *within* individuals. Individual regression analysis was used. The median intercept, slope and Pearson R were:

adults:  $PM10_{PERSONAL} = 32 + 0.53 \times PM10_{AMBIENT}$ ;  $R=0.50$

children:  $PM10_{PERSONAL} = 74 + 0.55 \times PM10_{AMBIENT}$ ;  $R=0.62$

Excluding days with exposure to ETS improved the correlation, resulting in a median Pearson R of 0.71 for adults and 0.73 for children.

A similar, but smaller, study was conducted on respirable particles ( $D_{50}: \pm 3 \mu m$ ), including 13 children. An even better correlation was found, with a median Pearson R of 0.86 using all measurements, and 0.91 after excluding days with exposure to ETS.

The mean personal PM10 concentration was  $61 \mu g/m^3$  for adults ( $n=262$ ; range 11-173  $\mu g/m^3$ ) and  $104 \mu g/m^3$  for children ( $n=301$ ; range 35-230  $\mu g/m^3$ ). The mean of the corresponding PM10 concentrations in ambient air was about  $40 \mu g/m^3$ . For the adults, "living near a busy road", "time spent in traffic" and "exposure to ETS" explained 69% of the variance in the difference between personal and ambient PM10, with a non significant intercept of  $4 \mu g/m^3$ . For the children, the difference could largely be explained by high PM10 concentrations in the classroom, "exposure to ETS" and "physical activity". For respirable particles the differences between personal and ambient concentrations were smaller; on average  $11 \mu g/m^3$  for all children and  $5 \mu g/m^3$  after excluding days with exposure to ETS.

The results show a reasonably high correlation between personal and ambient PM10 within individuals, with large but attributable differences between personal and ambient concentrations. A small study on respirable particles suggests better correlations and smaller differences between personal and ambient concentrations than for PM10.

**PRESENTER:** Michael Brauer

**TITLE:** Continuous assessment of indoor fine particles with a portable nephelometer

**AUTHORS:** Brauer, M, 't Mannetje, A, Lang, B, Bartlett, K

**ABSTRACT:**

Locations where relationships between ambient particle concentrations and health outcomes have been observed share combustion processes as a common particulate source. Since individuals spend the majority of their time indoors, fine particles generated in indoor combustion processes (cooking, smoking, woodburning,) are also important for health effects assessment.

Used with filter sampling, continuous monitoring of fine particles improves exposure assessment by characterizing the impact of time-varying indoor sources. We evaluated a continuous monitor, a portable nephelometer, for the assessment of indoor particulate levels. Simultaneous sampling with PM<sub>10</sub> and PM<sub>2.5</sub> impactors was undertaken to determine the relationship between particle light scattering coefficient ( $\sigma_{sp}$ ) and particle mass concentration in field and environmental chamber settings. Measurements were conducted in 20 restaurants and bars with different smoking restrictions and in the kitchens of 5 homes in Vancouver, in 6 homes in rural British Columbia with modern woodburning stoves, and in 22 homes in rural Mexico where biomass was burned for cooking. Outdoor measurements were made to assess indoor:outdoor ratios. Chamber studies evaluated nephelometer measurements of Environmental Tobacco Smoke and particles produced from toasting bread and frying foods.

$\sigma_{sp}$  and particle mass were highly correlated ( $r^2$  values of 0.73 - 0.98) over a wide range of concentrations (7 - 1600  $\mu\text{g}/\text{m}^3$ ). Since light scattering is most efficient for 0.1 -1.0  $\mu\text{m}$  particles, the correlation was greater for PM<sub>2.5</sub> than PM<sub>10</sub>. Different  $\sigma_{sp}$  vs. particle mass slopes were observed for the different sampling environments, reflecting the influence of particle composition on light scattering. However, in similar indoor environments, the relationship between particle light scattering and mass concentration was consistent enough to use independent nephelometer measurements as estimates of mass concentrations. The fast-response, continuous measurement, and datalogging capabilities of a portable nephelometer, make it a useful tool for the assessment of indoor particle exposures.

PRESENTER: Dean Lillquist

TITLE: A Comparison of Indoor/Outdoor  $PM_{10}$  Concentrations Measured at Three Hospitals and a Centrally Located Monitor in Utah

AUTHORS: Lillquist DR, Lee JS, Ramsay JR, Boucher K, Weiss Z and Lyon L

ABSTRACT

This research measured daily 24-hour  $PM_{10}$  concentrations at various locations in Salt Lake City, Utah from December 1994 to May 1995. Between four and six indoor locations were sampled at each of three hospitals. Indoor data were compared to outdoor roof data of each respective hospital and to the City's central monitoring location. The study goals were to: 1) evaluate variation of  $PM_{10}$  concentrations at four different outdoor sampling locations across Salt Lake valley; 2) determine if a centrally located monitor can predict  $PM_{10}$  concentrations across the valley; 3) evaluate variation in  $PM_{10}$  concentrations inside hospitals to see if a single indoor sampler can estimate exposure for an entire hospital; 4) compare indoor/outdoor roof concentrations at three hospitals to determine if an outdoor roof sampler can be used to predict indoor concentrations; and 5) determine if a centrally located monitor can predict concentrations indoors in the three hospitals. Results from outdoor samplers indicate that variation occurred between the four sites. When regressed against the central sampler, the slopes of each hospital were linear but statistically different. Because the three hospitals had similar y-intercepts, at low outdoor  $PM_{10}$  concentrations the central monitor predicted hospital roof concentrations. As outdoor  $PM_{10}$  concentrations increased, the central monitor had higher concentrations and overestimated  $PM_{10}$  relative to the three hospital sites. Results from indoor samplers indicate large variation in  $PM_{10}$  concentrations both within and between hospitals. Data indicate that one indoor location does not adequately represent the variation in indoor  $PM_{10}$  concentrations and that outdoor  $PM_{10}$  account for a varying percentage of indoor  $PM_{10}$  concentrations. Finally, it appears that  $PM_{10}$  concentrations at different indoor hospital locations cannot be predicted by one central outdoor  $PM_{10}$  sampling location.

PRESENTER: Susanne Hering

TITLE: Indoor - outdoor concentration ratios for fine particle mass and inorganic ions in twelve southern California homes

AUTHORS: Susanne V. Hering, Aerosol Dynamics Inc., Berkeley, CA and Ed Avol, University of Southern California School of Medicine, Los Angeles, CA.

ABSTRACT

Airborne concentrations of fine particle mass, sulfate, nitrate, and ammonium ion, and the concentrations of gaseous nitric acid were measured as part of a pilot study to test a low-flow rate, two-week sampler for indoor measurements. This sampler had been developed to provide low-cost outdoor monitoring for chemical characterization of inorganic fine aerosols for epidemiology studies, and was adapted to indoor monitoring for this study. The sampler consists of a single-jet impactor that provides a sharp precut at  $2.3\text{ }\mu\text{m}$ , followed by a 8mm-long carbonate-coated glass honeycomb denuder to capture the gaseous nitric acid, a Teflon filter to collect particles, and a carbonate-impregnated quartz fiber filter to collect volatilized particulate nitrate. Samplers were deployed inside and immediately outside of twelve homes in southern California, and operated continuously for two weeks. Replicate samples for fine particles were collected at one-half of the homes. Approximately one-half of the homes had air conditioning, most had pets. Only one home had a smoker. All measurements were made in the late summer and fall. Fine particle sulfate was generally comparable indoors and outdoors, while indoor/outdoor ratios of nitrate and fine mass were more variable. For sulfate, indoor/outdoor concentration ratios varied from 0.6 to 0.8 for air conditioned houses, and from 0.9 to 1.0 for homes without air conditioning. Ratios for fine particle mass and nitrate were more variable, but generally the indoor/outdoor ratio for fine particle mass was greater than for sulfate in the same home. For the one home with a smoker, the indoor fine particle mass concentration was five times higher than outdoors, while indoor and outdoor sulfate were the same. Nitric acid concentrations were consistently lower indoors for all homes. The sulfate data indicate efficient penetration of outdoor fine particles into these southern California homes, while the mass data indicate that the additional contribution from indoor sources is important.

**PRESENTER:** Roger O. McClellan

**TITLE:** The Clean Air Act and Criteria Pollutants: Are Science and Legislation Out of Sync?

**AUTHORS:** McClellan, Roger O.

**ABSTRACT**

Post-World War II rapid expansion of the U. S. economy and the marked increase in use of motor vehicles resulted in increased levels of air pollution, especially in urban areas. Soon a number of researchers began investigating the association between various constituents of air pollution and human health effects, both in the field and the laboratory. Out of these investigations came recognition that many air pollution issues had national dimensions. This gave impetus to the passage of the Clean Air Act (CAA) of 1970. A key provision of the act concerned the establishment of National Ambient Air Quality Standards (NAAQSs) for individual pollutants, such as ozone and carbon monoxide, and the mixture called particulate matter (PM). The CAA stimulated further research on individual pollutants. Over the years, the background criteria documents used for setting the NAAQSs have been periodically revised. The NAAQSs for ozone and PM are now being considered for revision. In both cases, some data points to health effects at quite low exposure concentrations of individual pollutants. Other investigations and interpretations emphasize the difficulty of ascribing effects to single pollutants and suggest that the observed effects are really due to the interactive effects of the total air pollutant mixture. This raises serious questions as to future research directions and legislative (and related regulatory) actions. Has our emphasis on single pollutants resulted in our failing to direct adequate attention to understanding the effects of the total mixture of air pollutants? Beyond a shift in research priorities, are we also prepared to explore alternative regulatory strategies that consider mixtures of air pollutants rather than single pollutant regulations? This presentation will pose some of these questions and suggest avenues for further research and possible regulatory changes.

**PRESENTER:** Mark J. Utell

**TITLE:** The Clean Air Act: Clinical Plausibility of Health Effects  
from Criteria Pollutants

**AUTHOR:** Utell, M

**ABSTRACT**

The language of the Clean Air Act mandates policy that links science and medical judgment with regulation. To protect individuals from "dirty" air, policy criteria include: a) identifying susceptible groups; b) establishing no-effect levels (highly intertwined with susceptible groups); c) building an adequate margin of safety; and d) defining adverse health outcomes. Over the past twenty years, these criteria have been highly effective largely because they have generated the necessary scientific underpinnings for regulation. Ozone, sulfur dioxide, carbon monoxide and lead standards possess biologic and clinical plausibility. In contrast, the recent observation that low levels of particulate matter are associated with increased respiratory morbidity and mortality is perplexing. Biologic plausibility is suspect as neither clinical experience nor review of the literature identify a supportable pathophysiologic mechanism to explain the relationship between inhaled particles and mortality. The complex nature of particulate matter calls into question the current strategy of regulating pollutants on an individual basis. It is time to start investigating "air pollution" and not individual pollutants. Thus, despite the effectiveness of the Clean Air Act in protecting health, it is appropriate to re-examine the approaches to air pollution regulation especially in the absence of a sound scientific and clinical basis.

PRESENTER: A. Myrick Freeman III, PhD

TITLE: The potential role of economics in setting air quality standards

AUTHOR: Freeman, A. M.

Abstract:

Economic analysis can help to answer two important policy questions with regard to controlling air pollution. The first is the retrospective question of whether we have gotten our money's worth out of the reductions in pollution achieved under the Clean Air Act since 1970. In other words, have the benefits been greater than the costs? Earlier reviews undertaken in the late 1970s and early 1980s suggested that the answer was "yes" for particulate matter and sulfates but "no" for ozone. EPA's ongoing retrospective study of the CAA may provide a more definitive answer to this question.

The second question concerns the relationship between the benefits and costs associated with possible changes in air quality standards being considered now. This question is becoming increasingly important as evidence accumulates that there is no clear threshold for health effects for particulate matter and ozone. Recent advances in our knowledge of the relationship between health effects and air pollution at population levels and in our understanding of the values people place on changes in health risk are making it possible for economists to contribute to the discussion of where to set ambient air quality standards. These advances are reflected in three recent major studies of the environmental costs of generating electricity (the Department of Energy/Resources for the Future Fuel Cycle Study, the European Commission Externe Fuel Cycle Study, and the New York State Environmental Externalities Cost Study) and two prospective studies of the benefits associated with reducing levels of particulate matter (by the American Lung Association) and sulfates (by EPA). All of these studies use similar health effects data and economic methods.

The ALA study estimates the monetized health benefits of reducing PM10 concentrations in the US from present levels (or the current federal standard, if it is lower) to the California standards to about \$10.8 billion per year. The EPA study estimates the monetized health benefits of the reductions in sulfate concentrations expected under Title IV to be about \$10.6 billion per year in 1997. While it must be acknowledged that there are substantial uncertainties in such estimates, I believe that information of this sort can shed light on the difficult choices and tradeoffs involved in setting ambient air quality standards.

PRESENTER: Melvin D. Zeldin

TITLE: Regulatory concerns with meeting particulate matter standards: A local agency perspective

AUTHOR: Melvin D. Zeldin

ABSTRACT:

The Clean Air Act Amendments (CAA), enacted in 1990, set in motion specific requirements for attaining the National Ambient Air Quality PM10 Standards. Currently, the U.S. Environmental Protection Agency (EPA) is reviewing the adequacy of the PM10 standards, and is considering a new fine particulate standard. Unlike other criteria pollutants, particulate matter is a "grab bag" of pollutants, ranging from directly emitted smoke, dust, soot, and metallic particles to secondarily formed sulfate, nitrate, and organic particles. Virtually all polluting sources contribute in some fashion to ambient particulate matter. Each area of the country may have unique combinations of particulates, and hence planning for attainment requires a fundamental knowledge of the particulate components – even though the standards are based on mass only. While EPA has established reference and equivalent methods for mass determinations, the investigative requirements for particulate matter can be particularly costly and labor intensive. Even with special studies of this nature, issues can be raised regarding representativeness, temporal resolution, and accountability for artifacts. This, coupled with considerable uncertainty in many area source emissions inventories for particulate matter, make local planning efforts extremely difficult and complex.

To meet these challenges, the South Coast Air Quality Management District (AQMD) has undertaken a year-long monitoring and emissions inventory improvement study. The monitoring program, conducted during 1995, utilized special monitors to account for positive and negative artifacts, and to gather representative spatial and temporal data for both PM10 and PM2.5. This data allows for comparisons between PM10 and PM2.5 ambient levels as measured in a complex urban atmosphere where over 10 million people reside. The results indicate that most secondary particles are dominant in the fine particulate fraction, but that crustal components are mostly in the coarse mode (i.e., greater than 2.5 microns). Also comparisons of the current PM10 standards to various possible thresholds for a new PM2.5 standard show more severe exceedances for PM2.5 should lower PM2.5 thresholds be used.

PRESENTER: Sheldon K Friedlander

TITLE: The submicron atmospheric aerosol as a carrier of reactive chemical species: case of peroxides

AUTHORS: Friedlander, S K, Yeh, E K, Department of Chemical Engineering, UCLA

ABSTRACT

The submicron atmospheric aerosol carries short-lived, reactive chemical species, including hydrogen peroxide and organic peroxides, at concentrations as high as one millimolar in the associated water. This conclusion is based on equilibrium calculations and (limited) experimental data. Hydrogen peroxide and hydroxymethylhydroperoxide ( $\text{HOCH}_2\text{OOH}$ ) are especially likely to be found in the aerosol (aqueous) phase because of their very high Henry's law constants. Aerosol phase concentrations of  $\text{H}_2\text{O}_2$  fall within a range in which significant biochemical effects have been observed when cells are bathed with  $\text{H}_2\text{O}_2$  solutions. This may help explain the results of epidemiological studies that have shown that adverse health effects are associated with fine aerosols and/or sulfates. The submicron sulfate containing aerosol is itself frequently a product of chemical reactions involving  $\text{H}_2\text{O}_2$ , hence a surrogate for the peroxide and associated reactive species. That is, the epidemiological results may signal a response to atmospheric peroxides rather than to sulfates. This hypothesis supports reduction of the total submicron aerosol mass as a way to reduce health effects because the total submicron mass is closely linked to the aqueous component that carries the reactive species. To test this hypothesis, studies are needed of the effects of exposures of cellular layers and/or animals to submicron  $\text{H}_2\text{O}_2$  containing aerosols that also contain salts such as ammonium sulfate in the pH range 2 to 6.

**PRESENTER:** Michael T. Kleinman

**TITLE:** Toxicity of Constituents of PM10 Inhaled by Aged Rats

**AUTHORS:** Kleinman, MT, Mautz, WJ, Phalen, RF and Bhalla, DK

**ABSTRACT**

The EPA has estimated that exposures to particles smaller than 10  $\mu\text{m}$  in mass median aerodynamic diameter (MMAD) (PM10) may contribute to approximately 60,000 deaths per year, based upon epidemiological associations. The biological mechanisms for effects of components of PM10 on human health are not firmly established. There is evidence that acidic and combustion-generated components, which are found in the fine particle fraction ( $\leq 2.5 \mu\text{m}$  diameter) are among those which are the most toxic.

This study examined the toxicology of PM10 components using laboratory-generated aerosols containing acidic ammonium bisulfate (ABS), and resuspended carbon black (C), a surrogate for combustion-generated carbonaceous aerosols. Since the human population most affected by PM10 appears to be elderly, this study used geriatric rats (about 24 months old) as a model. *In vivo* subchronic 4 week nose-only exposures (4 hr/d, 3 d/wk) of rats ( $n=10$  per endpoint per atmosphere) were performed with atmospheres containing either C or ABS at concentrations of 60 and 70  $\mu\text{g}/\text{m}^3$ , respectively, or to a mixture of C + ABS. Ozone ( $\text{O}_3$ ), might be an important co-pollutant with PM10, therefore mixtures of C +  $\text{O}_3$  and C + ABS +  $\text{O}_3$  were also tested. Control groups were exposed to purified air or to  $\text{O}_3$  alone. The particle size was 0.5  $\mu\text{m}$  mass median aerodynamic diameter, for both C and ABS aerosols. The following hypotheses were examined: (1) The mixture of carbon and acidic sulfate particles will have greater deleterious effects than will the carbon particles alone; and (2) The effects of the acidic sulfate and carbon particle mixture combined with 0.15 ppm  $\text{O}_3$  will be more deleterious than the additive effects of the particle mixture and the  $\text{O}_3$  portions of the mixture.

Biological endpoints which are related to the etiology or presentation of lung diseases were examined. These included permeability, inflammatory responses, macrophage functions, collagen synthesis, mucus production, and lung morphometry. Lung permeability was not significantly changed following exposures to any of the pollutant atmospheres, nor was there significant evidence of infiltration of alveolar spaces by inflammatory cells. Exposures to particle atmospheres, in the absence of  $\text{O}_3$ , did not significantly affect macrophage functions, however macrophages from rats exposed to either C +  $\text{O}_3$ , or C + ABS +  $\text{O}_3$  showed functional changes that were significantly altered in comparison to those from rats exposed to  $\text{O}_3$  alone. The  $\text{O}_3$ -containing mixtures induced significant changes in collagen concentrations in lung tissue and resulted in significant increases in lung cell turnover rates; both changes are suggestive of irritant effects on the lung. Changes in both of these parameters were greatest following exposures to the mixture which contained ABS.

This study demonstrated significant biological effects in geriatric rats due to exposure to atmospheres containing components of the fine-particle fraction of PM10, *i.e.* carbon and ammonium bisulfate particles. Ozone (0.15 ppm) exacerbated the effects of this mixture. The results of the study supported both hypotheses. (Supported by the California Air Resources Board)

PRESENTER: Kevin Dreher

TITLE: Pulmonary toxicity of size-fractionated urban ambient air particulate matter (PM)

AUTHORS: Dreher K, Jaskot, R, Richards, J, Lehmann, L, Hoffman, A, Costa, D

ABSTRACT

Lung injury induced by size-fractionated urban ambient air PM was examined in order to identify particle characteristics responsible for its toxicity. A massive air sampler fractionated and simultaneously collected Washington, D C. ambient air PM into <1.7, 1.7-3.7 and 3.7-20  $\mu$ m MMAD size ranges. The <1.7  $\mu$ m fraction had the highest sulfate content, highest percentage of soluble transition metals (TM) and was found to be the most acidic of the three ambient air PM size fractions. Rats were exposed to each air PM fraction by intratracheal instillation (2.5 mg/rat). Bronchoalveolar lavage (BAL) was performed on rats 24 hours post-exposure and lavage samples analyzed for protein, lactate dehydrogenase albumin and cellular content. The <1.71  $\mu$ m fraction induced a greater degree of lung injury by producing larger increases in BAL protein, albumin, red blood cell and eosinophil levels when compared to the 1.7-3.7 and 3.7-20  $\mu$ m air PM fractions. All fractions induced similar amounts of pulmonary neutrophil and macrophage influx. In contrast to the other size air PM fractions, a leachate prepared from the <1.7  $\mu$ m fraction was able to induce similar increases in BAL protein, albumin, red blood cell and eosinophil levels. Also in contrast to the other size air PM fractions, washing of the <1.7  $\mu$ m particles with H<sub>2</sub>O significantly attenuated these responses. These data indicate that the Washington, D.C. <1.71  $\mu$ m ambient air PM fraction resembles certain emission source particles due to its acidic nature, high sulfate and soluble TM content as well as its ability to induce a greater degree of acute lung injury, (This abstract does not reflect EPA policy)

**PRESENTER:** John J. Godleski

**TITLE:** Death from inhalation of concentrated ambient air particles in animal models of pulmonary disease.

**AUTHORS:** Godleski J, Sioutas C, Katler M, Koutrakis P

**ABSTRACT**

Epidemiologic studies have found increased mortality associated with particulate air pollution. To test the biologic plausibility of this association, normal rats, rats with monocrotyline-induced pulmonary inflammation (50 mg/kg SC), and rats with SO<sub>2</sub>-induced chronic bronchitis (250 ppm SO<sub>2</sub>, 6 wks) were exposed to concentrated air particles (CAPs) or filtered air for 3 consecutive days, 6 hrs/day. The concentrating system of Sioutas et al (Env Hlth Perspect 1995; 103:171) was used.

Parameter\\Group	Control	Monocrotyline	Chronic Bronchitis
CAPs ( $\mu\text{g}/\text{m}^3 \pm \text{SD}$ )	245 $\pm$ 70	228 $\pm$ 89	288 $\pm$ 64
% Mortality	0%	19%*	37%**
Pathologic Findings on Death or Sacrifice	<ul style="list-style-type: none"> <li>- No inflammation</li> <li>- Minimal Bronchoconstriction</li> </ul>	<ul style="list-style-type: none"> <li>- Acute inflammation in alveoli &amp; interstitium</li> <li>- Some Bronchoconstriction</li> </ul>	<ul style="list-style-type: none"> <li>- Airway inflammation, increased mucus</li> <li>- Marked bronchoconstriction</li> <li>- Interstitial edema</li> <li>- Pulmonary vascular congestion</li> </ul>

\* Chi square  $p=0.05$ . \*\*  $p < 0.01$  - disease group with CAPs compared to control no lung disease-with CAPs or control air with disease.

Deaths occurred during exposure and overnight. Inflammation was found in groups with disease, but animals exposed to CAPs exhibited increases in inflammatory parameters and bronchoconstriction. Bronchoconstriction, visible buckling of the epithelium, was enumerated in all groups comparing the number of constricted airways in animals dying spontaneously with those who survived exposures but were killed afterwards. Animals with chronic bronchitis had the most evidence of airway constriction. In comparing death versus killed, bronchoconstriction was significantly increased in the disease groups (monocrotyline  $p=0.007$ ; chronic bronchitis  $p=0.02$ ). Thus, ambient particle inhalation can cause death in rats with disease, and inflammation as well as airway constriction appear to be important in the response.

PRESENTER: Werner Stober

TITLE: Requirements for a credible extrapolation model derived from health effects in rats exposed to particulate air pollution - a way to minimize the risks of human risk assessment?

AUTHORS: Stober W, Miller FJ, McClellan RO

# ABSTRACT

For some years, several regulatory agencies have attempted to develop rather simple mathematical and biostatistical models to yield quantitative assessments of human risk derived from data of experimental rat exposures to typical particulate air pollutants. In our view, these models have failed to adequately consider some important biological aspects. These assessments are based on comparisons between the low levels of exposure of humans and the intentionally high levels used in rat exposure studies. However, four crucial questions have not been addressed in past efforts: a) What constitutes the effective dose of particulate matter following long biopersistence in the lung? b) Is the effective dose mechanistically the same in humans and rats? c) Are the dose-response curves linear, and if not, are the nonlinearities the same for humans and rats? d) Are the models suitable for extrapolating from high to low exposure and from rats to humans?

As mechanistic similarity will remain an open question for some time, so will the lack of suitable data for human retention continue to impede accurate rat-to-man extrapolations. For the other problems, however, improvements are possible and necessary. Invariably, published procedures treat particles as chemically active agents whether they are soluble or not. Focusing on insoluble particles, this paper shows that particle mass accumulated in the lung is not *per se* the relevant effective dose. Such a dose must be related to a target tissue or cell population and is represented by an integral of the total particulate surface and its residence time in the target area. This is a theoretically more reliable approach to effective dose than using the retained mass. Besides, there is plausible experimental evidence available to support this position. Based on this effective relative dose, it can be shown from rat lifetime exposures that even crude dose-response relationships for lung tumor incidence show a strong non-linearity. No-threshold probit analyses of rat data indicate that ubiquitous exposure concentrations for rats at diesel soot levels between 40 and 85  $\mu\text{g}/\text{m}^3$  are "virtually safe" from lung cancer incidence. This must be a very conservative assessment, because in reality, this suspicious, nonchemical, nongenotoxic, rat-specific carcinogenicity can be seen only under heavy lung overload and involves most likely a no-effect threshold. Another major drawback of current risk assessment procedures involves the use of inappropriate lung retention models. Most frequently, postexposure models have been applied to chronic exposures. Almost all models are "models of data" that, according to a definition of DiStefano and Landaw (1984), are only acceptable for interpolations (e.g., the radiological applications of the ICRP model). However, physiologically based "models of systems" discriminating between various compartments in the pulmonary region give plausible disposition patterns of the retained particles and thus more credible extrapolations.

PRESENTER: George Allen

TITLE: Assessment of the temporal relationship between daily summertime ultra-fine particulate count concentration with PM<sub>2.5</sub> and Black Carbon Soot in Washington DC

AUTHORS: Allen, G. Abt, E, Koutrakis, P

ABSTRACT

Recent epidemiological evidence has shown a significant relationship between ambient daily particulate mass concentrations and human health effects as measured by cardio-pulmonary morbidity and mortality. Most of the research aimed at determining causal agents focuses on the combustion-related component of PM<sub>10</sub>. Some studies have suggested that ultra-fine aerosols (typically less than 0.15  $\mu\text{m}$  diameter) may be an important factor in the observed health effects of particle mass, either by their count concentration or by other mechanisms. The temporal relationship between PM<sub>2.5</sub>, ultra-fine particles (number concentration) and black carbon soot (BC) was characterized during the summer of 1994 in Washington, DC. Particles smaller than 0.7  $\mu\text{m}$  aerodynamic diameter were measured with a TSI model 3934L scanning mobility particle sizer, and a TSI model 3310A laser-Doppler velocimeter was used for particles between 0.7 and 10  $\mu\text{m}$ . A TEOM continuous mass analyzer was used to measure PM<sub>2.5</sub> in conjunction with integrated 24 hour low-volume PM samplers. BC was measured by optical attenuation on a filter using an Aethalometer. The temporal relationship between daily means of particle count concentration, mass concentration, and BC mass concentration is presented. Preliminary analysis shows no correlation between daily (mean 24 hour) particle number concentration and PM<sub>2.5</sub> or BC, and an  $R^2$  of 0.25 between PM<sub>2.5</sub> and BC. These results are consistent with the lack of temporal variability (on a daily basis), local sources, and short ( $\ll 1$  day) lifetimes of ultra-fine particles compared to the sources (regional and long-distance transport) and lifetimes (many days) of the larger particles (0.4 to 1  $\mu\text{m}$ ) that dominate the fine PM concentration. If the lack of an association between count and mass concentration holds over other seasons and locations, it would be more difficult to accept theories of mechanisms for PM health effects which rely on a temporal association between ultra-fine particles and PM<sub>2.5</sub> or PM<sub>10</sub>.

**PRESENTER:** William E. Wilson

**TITLE:** Exposure measurements relevant to epidemiology: total exposure to ambient fine particles

**AUTHOR:** William E. Wilson

**ABSTRACT:**

Recent epidemiological studies have found associations between day-to-day variations in ambient PM and day-to-day variations in health outcomes. There may well be a link between a person's exposure to PM and health outcomes. However, for epidemiological results based on ambient measurements to be valid, there must be a link between a person's exposure to PM and ambient PM concentrations. Personal exposure to PM is usually greater than ambient PM concentrations and has a low and non-significant correlation with ambient PM concentrations. Personal exposure studies generally seek to model personal exposure in terms of time-weighted exposure to PM in various environments, outdoors, indoors, in transit, at work, etc. However, personal exposure may also be thought of in terms of exposure to particles from various sources, i. e. to ambient-generated particles while outdoors (ambient-outdoor), to ambient-generated particles which have infiltrated indoors (ambient-indoor), to particles due to a person's presence or activities (personal cloud), and to particles generated indoors independent of a person's presence or activities (indoor-generated). When this is done it becomes clear that concentrations measured at an ambient monitoring site are likely linked to personal exposure to ambient fine-mode particles (outdoors plus indoors) but, in general, not to personal exposure to ambient coarse-mode particles, to personal cloud particles or to indoor-generated particles. Thus, it is likely that time series epidemiological studies, using daily ambient PM concentrations, provide information mainly on the association between health outcomes and exposure to fine mode ambient particles. Results from personal exposure research may be used to improve the linkage between ambient concentrations and total exposure to ambient particles. Penetration rates of ambient particles indoors and the indoor lifetimes of particles as a function of size may be used to estimate the concentration of ambient-indoor particles under equilibrium conditions as a function of air exchange rates. This information may be used with personal or community activity patterns to estimate the personal exposure to ambient particles as a function of ambient concentrations. Such techniques might improve the associations found between ambient PM concentrations and health outcomes.

PRESENTER: Dale A. Lundgren

TITLE: PM<sub>1</sub>, PM<sub>2.5</sub> and PM<sub>10</sub> Aerosol: Chemistry vs. meteorology for Phoenix, Arizona

AUTHORS: Lundgren Dale A., Rich , Thomas, A., and Hlaing, Daniel N.

ABSTRACT

Intermodal aerosol mass and chemistry are of concern in a setting a particulate matter (PM) standard at 1.0  $\mu\text{m}$  (PM<sub>1</sub>) or 2.5  $\mu\text{m}$  (PM<sub>2.5</sub>) and in correlating human health effects with an aerosol measurement (PM<sub>1</sub>, PM<sub>2.5</sub> and PM<sub>10</sub>).

Phoenix aerosol was sampled using the new PM<sub>10</sub>/PM<sub>2.5</sub>/PM<sub>1.0</sub> trichotomus sampler developed by Dr. Virgil Marple at the University of minnesota. Separate samples of 1.0 to 2.5  $\mu\text{m}$ , 2.5 to 10  $\mu\text{m}$  and the PM<sub>1</sub> (<1.0  $\mu\text{m}$ ), PM<sub>2.5</sub> (<2.5  $\mu\text{m}$ ) and PM<sub>10</sub> (<10  $\mu\text{m}$ ) particulate matter were obtained (on Teflon filters), weighed and chemically analyzed. Chemical composition of the 1.0 to 2.5  $\mu\text{m}$  size fraction was compared with that of the PM<sub>1</sub> (<1.0  $\mu\text{m}$ ) fraction and that of the 2.5 to 10  $\mu\text{m}$  fraction in order to estimate the contribution of Coarse Mode (>2.5  $\mu\text{m}$ ) and Find Mode (<1.0  $\mu\text{m}$ ) aerosol to this intermediate (1.0 to 2.5  $\mu\text{m}$ ) size range. Summer days were separated into low (approximately 20  $\mu\text{g}/\text{m}^3$ ), medium (approximately 40  $\text{mg}/\text{m}^3$ ) and higher (approximately 80  $\mu\text{g}/\text{m}^3$ ) PM<sub>10</sub> aerosol concentration groups. Aerosol Find and Coarse Mode mass fraction and chemical composition was correlated with meteorology and other variables, which help describe and predict the intermodal measurements. Data sets will be presented, correlated and discussed.

**PRESENTER:** Irving Salmeen

**TITLE:** Automobile particulate emissions: Challenges in assessing impacts on particulate air pollution and public health.

**AUTHORS:** Irving Salmeen, James Ball, Steven Japar

**ABSTRACT**

The association between particulate air pollution and adverse health effects is important to the motor vehicle industry because vehicles contribute directly and indirectly to PM<sub>10</sub>. Understanding the impacts of motor vehicle exhaust on particulate air pollution requires knowledge of the emission rates and properties of particles directly emitted in the exhaust, so-called primary emissions, and knowledge of atmospheric reactions which produce so-called secondary particles from gaseous exhaust and evaporative emissions. These are somewhat understood for early to mid-1980s vintage vehicles, and the contribution of vehicle exhaust to PM<sub>10</sub> in Los Angeles air of the 1980s has been estimated. Retrospective understanding is important, but the vehicle manufacturing industry obviously needs to be concerned with the future. Thus with a view toward understanding likely future impacts, we review here what is known about primary particulate emissions from late-model and current production US and European passenger cars. We will discuss mainly experiments conducted by Ford Research Laboratories because we are not aware of similar studies published by others. Except for some new preliminary data on particle number distributions, most of the experiments reviewed herein have been published.

Until now, there has been little incentive to study primary particulate emissions from new gasoline cars because the PM<sub>10</sub> emission rates have been less than 10 mg/mi since the mid 1980s, a level much lower than the emissions standard of 80 mg/mi. In contrast, particulate emission rates from new diesel-powered passenger cars, while much lower than those of their ten year old cousins, are a continuing concern because they are typically ~100 mg/mi. There is a problem in knowing what to study because there is as yet no hypothesis as to the properties of particles important in eliciting adverse health effects. Thus the studies to characterize emissions are at the observation and classification stage, borrow heavily from the techniques developed during the early 1980s, and are fraught with doubts that the right questions are being asked. Nonetheless, the state of the art has yielded the following data: Particulate mass emission rates under standard laboratory test conditions from several Ford vehicles spanning the 1989 to 1995 model years were ~5 mg/mi, which is at the edge of detectability by conventional gravimetric methods. Specific faults deliberately induced in the electronic ignition system and catalyst system in a 1990 Taurus did not appreciably affect the particulate emission rates or particle composition, a result which may simply mean that laboratory tinkering does not mimic field failure. Particle emission rates from two well-maintained Escorts and two Explorers were about constant at ~5 mg/mi over 100,000 miles, suggesting that it is at least possible in principle for a car to have low emissions over its lifetime. Thermogravimetric analysis of particles from a 1990 Taurus suggested that the particles were generally similar in composition to those from 1980s vintage gasoline and diesel powered passenger cars, although the data do not permit detailed comparisons, especially of the organic composition. The diesel-powered current production European passenger cars showed emission rates about 100mg/mi. The diesel - engineering community expects these rates to be near 60 mg/mi by the 1997 model year.

We have begun to study the particle number emission rates and the corresponding number size distributions. Preliminary data for a vehicle which meets the California ULEV standards showed that the number distributions peak at ~50 nm except when the engine is first cranked (cold start) where the distribution peaks at 150 nm. The emissions are easily detectable during cold start, but with a hot catalyst, the emissions are nearly imperceptible above the background particulate levels, except during hard accelerations.

PRESENTER: Ann Miguel

TITLE: Latex allergens in tire dust and in airborne particles

AUTHORS: Miguel, A, Cass, G, Weiss, J, Glovsky, M

ABSTRACT

The prevalence and severity of latex allergy has increased dramatically in the last 15 years due to exposure to natural rubber products. Although historically this health risk has been elevated in hospital personnel and patients, a recent survey has indicated a significant potential risk for the general population. Procuring a wide-spread source for latex exposure we have considered tire debris. We have searched for the presence of latex allergens in passenger car and truck tire tread, in debris deposited from the atmosphere near a freeway and in airborne particulate matter samples representative of the entire year 1993 at two sites in the Los Angeles basin. After extraction of the samples with PBS, a modified-ELISA inhibition assay was used to measure relative allergen potency and Western blot analyses were used to identify latex allergens. The inhibition studies with the human IgE latex assay revealed inhibition by the tire tread source samples and ambient freeway dust as well as control latex sap and latex glove extracts. Levels of extractable latex allergen per unit of protein extracted were about 2 orders of magnitude lower for tire tread as compared to latex gloves. Western blot analyses using binding of human IgE from latex-sensitive patients showed a band at 34-36 kDa in all tire and ambient samples. Long Beach and Los Angeles air samples showed 4 additional bands between 50 -135 kDa. Alternative Western blot analyses using rabbit IgG raised against latex proteins showed a broad band at 30-50 kDa in all samples with additional bands in the urban air samples similar to the IgE results. A latex cross-reactive material was identified in mountain cedar. In conclusion, the latex allergens or latex cross-reactive material present in sedimented and airborne particulate material, derived from tire debris, generated by heavy urban vehicle traffic could be important factors in producing latex allergy and asthma symptoms associated with air pollution particles.

PRESENTER: Yuanzhang Li

TITLE: The analysis of the association between air pollutants and hospital admission in Birmingham Alabama: 1986-1990

AUTHORS: Li Y, Roth HD

ABSTRACT

We examined the association between hospital admissions of persons 65 and older and selected air pollutants in Birmingham for the years 1986 to 1990. Because of the availability of data, different pollutants were considered in different years. TSP was taken into account in 1986 and 1987;  $PM_{10}$  from 1987 through 1990;  $SO_2$  in 1986, 1987, and 1990; and  $O_3$  and CO in all the years of study. Other aerometry variables considered included temperature, humidity and barometric pressure. In addition to total admissions, other causes of admission considered in the analysis included Poisson auto-regression and regression of the deviation from a 19-days' weighted moving average. Analyses were also performed for different lag structures and combinations of pollutants.

The associations between  $PM_{10}$  and hospital admissions differed by cause, lag, statistical model, etc. In general, even in cases where  $PM_{10}$  was the only pollutant considered in the analysis, it was not a significant predictor of previous day's  $PM_{10}$  on pneumonia using a Poisson regression model controlling at least four days' weather variables linearly, and the current day's  $PM_{10}$  on cancer using Poisson regression model controlling dummy temperature. In all the other dozens of analyses performed,  $PM_{10}$  was not a significant predictor of hospital admissions.

Also the predictive power of  $PM_{10}$  varied from year to year. For example, for hospital admissions due to pneumonia, the regression coefficient of the previous day's  $PM_{10}$  is positive but not significant (0.00151 with standard error 0.00228) in 1987; negative but not significant (-0.00119 with standard error 0.00217) in 1988; positive but not significant (0.00027 with standard error 0.00207) in 1989; and negatively significant (-0.00431 with standard error 0.00212) in 1990. the overall coefficient for the period of 1987 to 1990 is negative but not significant (-0.00033 with standard error 0.001). The inconsistent effect from year to year was also true for other causes of admissions, other lags of the pollutant, etc. Hence, drawing a conclusion from a special data set by using a special model is misleading. In addition, our results are not consistent with those from an earlier analysis of the Birmingham morbidity data.

PRESENTER: John M. Peters

TITLE: Respiratory morbidity in twelve southern California communities with differing levels and types of air pollution

AUTHORS: Peters, JM, Avol, EL, Navidi, WC, London, SJ, Thomas, DC

ABSTRACT

To study the possible chronic respiratory effects of air pollutants in Southern California, 3676 school children from 12 communities were evaluated. We focused on four outdoor pollutants -ozone, PM10, acids, and NO2. Twelve communities, identified on the basis of historical monitoring information were selected to represent extremes of exposure and possible pollutant profiles. Monitoring stations were set up in the 12 communities to measure the 4 pollutants. In each community, about 150 fourth graders, 75 seventh graders, and 75 tenth graders were enrolled. Questionnaires were used to collect data on residential histories, historic and current health status, patterns of physical activity and housing characteristics. Lifetime estimates of exposure were made by combining existing monitoring data with residential histories. The pulmonary function of each child was measured during the season assumed to have the lowest pollution (spring) and in the morning before pollution levels reached their highest.

The school absence records were also monitored for each child and parents of absentees were called to ascertain reasons for absence for a sub-sample. The absences were categorized into health-related and non-health-related. If they were health related we sub-categorized into respiratory illnesses.

Prevalence rates of respiratory disease did not vary among communities according to air pollution patterns or levels. The risk of asthma was higher in subjects who were males; American Indians, African Americans, the medically insured; in those with pets or pests; in those living in homes with water damage, mildew or smokers; in families of higher income and when a parent had asthma. Life-time history or recent air pollution levels were not associated with asthma prevalence. Bronchitis prevalence was associated with lifetime exposure to PM10 (RR 1.05, 95%CI 1.02-1.09,  $p = .004$ ).

There were no clear relationships between PM10 exposure and pulmonary function level. Increased lifetime exposure to PM10 was significantly associated with an increase in the number of absences due to respiratory illness.

PRESENTER: Gerard Hoek

TITLE: The PEACE Study (1): Acute effects of air pollution on Peak Flow

Authors: Hoek, G, Roemer, W, Brunekreef, B

ABSTRACT

The PEACE study (Pollution Effects on Asthmatic Children in Europe) is a multi-center study of 14 institutes in 10 European countries, partly funded by the EU ENVIRONMENT programme. One of the objectives was to collect new information on the short-term health effects of air pollution on the respiratory health of susceptible children and to make a comparison of these health effects between urban and non-urban locations. Panels of children of 6-12 year old with chronic respiratory symptoms were selected in an urban and a non-urban location on basis of a questionnaire. During the winter of 1993-1994 the children filled out a diary about respiratory symptoms and medication use and performed twice daily PEF measurements. At fixed background locations 24 hour air pollution concentrations were monitored in the two locations. The monitored components were PM10, Black Smoke, SO<sub>2</sub>, NO<sub>2</sub> and NO. Mean 24 hour concentration of PM10 and SO<sub>2</sub> in the measurement period ranged from 13 to 100 µg/m<sup>3</sup> and 3 to 114 µg/m<sup>3</sup> respectively in the locations.

The analysis of the relationship between air pollution and PEF was performed on group level for each panel separately. A regression analysis with adjustment for first order autocorrelation and the number of reporting children as weight was performed with the daily mean population deviation as dependent variable and (lagged) 24 hour air pollution concentrations as independent variable. Time trend, minimum temperature and day of the week were included as confounders. The results in most of the separate panels gave no clear indication of a negative short term effect of air pollution on PEF. When calculating a combined effect estimate over the separate panels, however, a small negative effect of PM10 appears. At this stage, the separate effect estimates have not been investigated with respect to factors which might explain a difference in response between or within the panels.

PRESENTER: David Abbey

TITLE: Development of chronic productive cough or asthma as associated with long term ambient particulate pollutants in non-smoking adults - The AHSMOG Study.

AUTHORS: Abbey DE, Nishino N, McDonnell WF, Lebowitz MD.

ABSTRACT

Standardized respiratory symptoms questionnaires were completed by 3,091 nonsmoking California Seventh-day Adventists in 1977, 1987 and 1992. Ambient concentrations of air pollutants were estimated from 1973-1992 for included total suspended particulates (TSP), 1973-1987; particulates  $< 10\mu\text{m}$  in diameter (PM10), indirectly estimated from TSP, 1973-1987, and directly estimated from monitored PM10, 1987-1992; suspended sulfates ( $\text{SO}_4$ ) 1977-1992; sulfur dioxide ( $\text{SO}_2$ ) 1973-1992; 8-hour average of ozone ( $\text{O}_3$ ) 1973-1992. Adjustments to ambient concentrations have been made for time spent indoors. Chronic productive cough was defined as cough with sputum production on most days, for at least three months/year, for two-years or more. To be classified as having asthma, individuals must have been told by their physician that they had asthma as well as having a history of wheezing. Using indirect estimates of PM10 prior to 1987 and direct estimates after 1987, multiple logistic regression models adjusting for covariates have been used to study development of chronic productive cough 1977-1992 (191 new cases) and development of asthma 1977-1992 (106 new cases). For chronic productive cough, gender specific analyses indicated similar effects of PM10 and covariates for both genders. Hence, a pooled analyses was conducted using gender as a covariate. The relative risk (RR) for developing chronic productive cough was 1.57 [95% confidence interval (CI): 1.08, 2.27] for an increase of 42 days/year in excess of  $100 \mu\text{g}/\text{m}^3$  of PM10, while no association was found with  $\text{O}_3$ ,  $\text{SO}_2$ , or  $\text{SO}_4$ .

Gender specific analyses for development of asthma showed small nonsignificant positive associations with PM10 in both genders. Neither  $\text{SO}_4$  nor  $\text{SO}_2$  were significant when added to these models. However, when  $\text{O}_3$  was added to the models, the RR for females for PM10 increased to 3.23 (95% CI: 1.43, 6.48) while the RR for  $\text{O}_3$  was  $< 1$ . For males, RR for PM10 remained nonsignificant and RR for  $\text{O}_3$  was 3.85 (95% CI: 0.62, 18.70) for a 5pphm increase in the daily 8-hour  $\text{O}_3$  average. Gender differences may be the result of gender differences in exposure.

**PRESENTER:** Raimo O. Salonen

**TITLE:** Effects of PM10, black smoke, and resuspended dust on pef among asthmatic children

**AUTHORS:** Pekkanen, J, Timonen, K, Hosiokangas, J, Ruuskanen, J, Salonen, R.O

### **ABSTRACT**

In northern areas, dust from melting snow and street sanding causes spring dust episodes with high PM10 levels. Whether these spring dust episodes are associated with similar health effects to PM10 from combustion processes was analyzed by using data from a daily follow-up of peak expiratory flows (PEF) among 39 asthmatic children aged 7–12 years in the center of Kuopio, Eastern Finland, in 1994.

The follow-up extended from winter, when ambient particles originate mainly from combustion processes, to spring, when particles are mainly resuspended dust. There was one high spring dust episode (maximum PM10  $158 \mu\text{g}/\text{m}^3$ ). As this peak dominated all regression models, only results excluding this peak are presented.

The daily averages (max.) of PM10 and Black Smoke were  $19.7 (61.6) \mu\text{g}/\text{m}^3$  and  $12.6 (56.9) \mu\text{g}/\text{m}^3$ . The elemental composition of PM10 samples on every third day was analyzed with ICP-MS and the sources of PM10 solved by receptor modeling. In these models, aluminium was a good marker for particles from resuspended dust. Based on the aluminium content of the filters, the PM10 concentration originating from resuspended dust was estimated for 36 days (mean  $7.2$ , max  $44.9 \mu\text{g}/\text{m}^3$ ).

The association of 24-hour particulate concentrations with average population deviations in morning and evening PEF was analyzed with first-order autoregressive models, adjusting for time trends, temperature, humidity, weekend, and pollen. During the study period (82 days), PM10 and Black Smoke tended to be associated with decline in morning PEF, but not with evening PEF. In contrast, resuspended dust PM10 concentration (36 days) tended have a more immediate effect on evening PEF.

In conclusion, the present results from northern climate suggest that low concentrations of ambient particles from combustion processes may cause delayed lung function impairment among asthmatic children, whereas resuspended dust particles might have a more immediate effect on lung function. This hypothesis needs to be tested in future studies.

PRESENTER: Richard B. Schlesinger

TITLE: ACIDITY: POTENTIAL CONTRIBUTOR TO INCREASED MORTALITY/MORBIDITY ASSOCIATED WITH PARTICULATE AIR POLLUTION?

AUTHOR: Richard B. Schlesinger

ABSTRACT

Epidemiological studies have strongly suggested that ambient particle-associated acidity, generally in the form of acid sulfates, may be causally related to total mortality during historical air pollution episodes, and to increases in acute bronchitis and exacerbation of asthma observed more recently. However, experimental studies have only been partially successful in providing some mechanistic basis for such associations. Controlled exposures of humans have shown alterations in lung clearance function and pulmonary mechanics in some studies, while controlled exposures of animals have shown alterations in airway responsiveness, mucociliary transport, pulmonary macrophage function and host defense against microbes, regulation of internal pH, production of cytokines, and changes in airway epithelial histology. A number of the biological alterations in animals appear to be analogous to those noted in humans with asthma or bronchitis. However, most controlled studies show significant biological effects only at exposure concentrations well above those found in ambient air, resulting in concern that these studies are not adequately mimicking exposure scenarios encountered in the epidemiological studies. There are a number of possible reasons for the apparent "discrepancy" between the epidemiological and controlled studies of acid aerosols. These involve considerations of particle size, the physical state of the acidic particle (pure acid droplet or an acidic surface layer on a solid particle), the influence of co-pollutants and the use of normal vs. compromised host models. This paper discusses these factors in relation to the biological plausibility of exposure to acidic particles as causative agents in the increased mortality/morbidity suggested as due to ambient exposures to PM<sub>10</sub>, and relates effects in toxicological studies to those found in epidemiological and controlled clinical studies.

**PRESENTER:** Mark J. Utell

**TITLE:** Inhalation of sulfuric acid alters alveolar macrophage function in humans

**AUTHORS:** Frampton, M, Morrow, P, Zelikoff, J, Schlesinger, R, Utell, M

**ABSTRACT**

Particulate matter in the atmosphere has been associated with increased respiratory morbidity and mortality, and sulfuric acid aerosols comprise the predominant particulate species in many industrialized areas. Animal studies have demonstrated effects of  $\text{H}_2\text{SO}_4$  aerosol exposure on alveolar macrophage function, but extrapolation of these findings to humans has remained problematic. As part of a collaborative study to compare the results of  $\text{H}_2\text{SO}_4$  exposure in humans and animals under similar conditions, we exposed 12 healthy nonsmoking volunteers to aerosols of either  $\text{H}_2\text{SO}_4$  or NaCl,  $1 \text{ mg/m}^3$  for 3 hours, with intermittent exercise. Bronchoalveolar lavage (BAL) was performed 1 hour after exposure; total and differential cell counts were determined in both the first lavage aliquot (bronchial lavage) and the last three pooled lavage aliquots (alveolar lavage). Alveolar macrophages (AM) were examined for viability, surface adherence, release of superoxide anion ( $\text{O}_2^-$ ) and hydrogen peroxide ( $\text{H}_2\text{O}_2$ ), phagocytosis of serum-opsonized latex particles, and expression of surface receptors important in host defense. Cell recovery in both bronchial and alveolar lavage samples were similar following NaCl and  $\text{H}_2\text{SO}_4$  exposure, indicating the absence of an airway inflammatory response to the acidic aerosol. Percent of AM adherent to plastic decreased following  $\text{H}_2\text{SO}_4$  exposure vs NaCl ( $90.0 \pm 1.8$  vs  $94.2 \pm 1.0$  %,  $p < 0.05$ ). Release of  $\text{O}_2^-$  by AM stimulated with opsonized zymosan decreased following  $\text{H}_2\text{SO}_4$  exposure vs NaCl ( $2.74 \pm 0.27$  vs  $3.25 \pm 0.29$  nmol,  $p < 0.05$ ). Cell viability, release of  $\text{H}_2\text{O}_2$ , and phagocytosis were unaffected by the exposure. Concentration of total protein and albumin in BAL fluid was unchanged, indicating no effect on epithelial permeability. Effects of  $\text{H}_2\text{SO}_4$  exposure on AM release of  $\text{O}_2^-$  in these studies are similar to findings in animals, and suggest that exposure to  $\text{H}_2\text{SO}_4$  aerosols may impair respiratory host defense against infection.

PRESENTER: Robert B. Devlin

TITLE: In vitro exposure of human airway epithelial cells to an urban air particulate pollutant induces IL-6, IL-8, and TNF $\alpha$  mRNA and protein expression.

AUTHORS: Devlin, R.B., Carter, J., Ghio, A.J., Samet, J., and Reed, W.

ABSTRACT:

Episodes of high particulate air pollution correlate with increased mortality and morbidity. We hypothesize that particulate air pollutants induce a strong inflammatory response which can contribute to the effects observed in epidemiological studies. Residual oil fly ash (ROFA) is a particulate pollutant found in many urban settings. It has been reported to induce inflammation and lung damage when instilled into rats. Surprisingly, in vitro studies with human alveolar macrophages (AM) show that ROFA does not induce inflammatory mediators, even though it is actively phagocytosed by the AM. In this study human airway epithelial cells were exposed to concentrations of ROFA that did not cause significant cell damage, as measured by LDH release. Within 2 hours after addition of ROFA, significant amounts of IL-6, IL-8, and TNF $\alpha$  could be detected in cell supernatants. This is the result of increased mRNA expression for these 3 inflammatory mediators, as measured by quantitative RT-PCR. The inflammatory response of epithelial cells to ROFA is transition metal dependent since addition of deferoxamine almost totally ablates the increases in cytokine mRNAs and proteins. These experiments demonstrate that ROFA can induce the synthesis of inflammatory cytokines in human airway epithelial cells, which is dependent on the metal content of ROFA. The magnitude of response to ROFA is nearly an order of magnitude larger than that observed in epithelial cells exposed to ozone, a potent oxidant. This is an abstract of a proposed presentation and does not necessarily reflect EPA policy.

**PRESENTER:** Andrew J. Ghio

**TITLE:** Respiratory epithelial cell production of lactoferrin after *in vitro* exposure to an air pollution particle is metal dependent.

**AUTHORS:** Ghio, AJ, Carter, JD, Samet, JM, Reed, W, Quay, J, Dailey, LA, Richards, JH, Devlin, RB

**ABSTRACT**

Iron is essential to normal cell function. Living systems control the oxidative stress associated with this metal by storing it in ferritin. To transport the iron, the cell uses either transferrin or lactoferrin. We tested the hypothesis that lactoferrin and ferritin are produced by respiratory epithelial cells after *in vitro* exposure to an air pollution particle and that this production is metal dependent. The particle used was an oil fly ash collected downstream from the cyclone of a power plant burning a low sulfur residual oil. Normal human bronchial epithelial cells (Clonetics) were grown on 12 well plates (Costar) and exposed for two and twenty-four hours to oil fly ash. Cells were lysed, ultracentrifuged, and the isolated RNA reverse-transcribed. Resultant cDNA was PCR amplified for 24, 36, and 25 cycles for GAPDH, lactoferrin, and ferritin, respectively, using gene specific primers. Supernatant was obtained for assays of lactoferrin and ferritin protein concentrations. There was a significant, dose-dependent increase in lactoferrin mRNA at 24 hours. Ferritin has an iron-responsive element; control of this protein is posttranscriptional and there was no dose-dependent increase in its mRNA. There were significant increases in the concentrations of both proteins in the supernatants. Inclusion of 100  $\mu$ M deferoxamine with 100  $\mu$ g/ml oil fly ash prevented increases in both lactoferrin mRNA and elevations of lactoferrin and ferritin proteins. We conclude that respiratory epithelial cells produce lactoferrin in response to exposure to an air pollution particle and that this production is metal dependent. This abstract of a proposed presentation does not necessarily reflect EPA policy.

PRESENTER: Bart Ostro

TITLE: Particulate Matter and Asthma: A Quantitative Assessment of the Current Evidence

AUTHORS: Ostro, B, Lipsett, M, Das, R

ABSTRACT

Numerous reports document significant world-wide increases in asthma morbidity and mortality from the late 1970s to the early 1990s. Various social and environmental factors, including exposure to indoor and outdoor pollutants and allergens, have been postulated as partial explanations of increasing asthma trends. Although air pollution concentrations have not generally increased over this period, other factors, including increases in poverty and decreases in regular medical care, may render individuals more susceptible to effects from exposures. There is a substantial literature linking exposure to several ambient air pollutants with respiratory effects in asthmatics. These effects have been most clearly demonstrated in controlled exposure studies with sulfur dioxide; effects are less consistently associated with exposure to ozone, nitrogen dioxide and acidic sulfates. Since the chemical composition and size distribution of PM<sub>10</sub> vary markedly with time and location, the impact of these heterogeneous mixtures on asthmatics is difficult to ascertain from chamber studies. In addition, asthmatic participants in chamber studies do not represent the entire spectrum of age, severity and behavioral responses associated with this condition. Epidemiologic studies, however, have repeatedly demonstrated associations of particulate matter with exacerbation of asthma including ecological time-series analyses of emergency room visits and hospital admissions, as well as panel studies examining associations with peak flow, medication use, and asthma symptoms. This paper first reviews several clinical studies on particulate matter and asthmatics. Then, the biological plausibility of a causal relationship is explored in a discussion of the pathophysiologic pathways through which inhaled particles may affect the respiratory status of asthmatics. Next, we review the advantages and disadvantages of studying asthmatics in epidemiologic studies of air pollution. Finally, we summarize the quantitative results from epidemiologic studies linking particulate matter to several adverse asthma outcomes. For example, existing research indicates that mean levels of particulate matter may be associated with increases of 2 to 5 percent in hospital admissions for asthma, from 5 to 10 percent in emergency room visits, and up to 60 percent in asthmatic symptoms.

PRESENTER: Willem Roemer

TITLE: The PEACE Study (2): Acute effects of air pollution on respiratory symptoms

Authors: Roemer, W, Hoek G, Brunekreef, B

ABSTRACT

The PEACE study (Pollution Effects on Asthmatic Children in Europe) is a multi-center study of 14 institutes in 10 European countries, partly funded by the EU ENVIRONMENT programme. For a description of the study design, see The PEACE Study (1) abstract.

Mean 24 hour concentration of PM<sub>10</sub> and SO<sub>2</sub> in the measurement period ranged from 13 to 100 µg/m<sup>3</sup> and 3 to 114 µg/m<sup>3</sup> respectively in the locations.

Daily prevalence and incidence of respiratory symptoms and medication use were calculated for each panel separately. The association between daily variation in incidence and prevalence has been analysed using logistic regression methods taking into account first order autocorrelation of the residuals. Dependent variables were incidence or prevalence, and (lagged) 24 hour air pollution concentrations as independent variables. Time trend, minimum temperature and day of the week were included as confounders. In most of the panels the results showed no effect of air pollution on daily incidence or prevalence of symptoms. When calculating a combined effect estimate over the separate panels no effect on incidence seems to appear. The data on prevalence are still being analyzed. The separate effect estimates of the panels have not yet been investigated with respect to factors which might explain differences in response between or within the panels.

PRESENTER: Ronald E. Wyzga

TITLE: The Philadelphia Story (Continued): Themes and Variations

AUTHORS: Wyzga, Ronald E. and Lipfert, Frederick W.

ABSTRACT

This paper has two objectives: first, to attempt to reconcile the various daily mortality analyses that have been presented for Philadelphia (the count stands at eight at this writing), and second, to present some new analyses involving additional pollutants (CO, NO<sub>x</sub>, and SO<sub>4</sub><sup>2-</sup>). Some of these measurements were taken in the suburbs around Philadelphia rather than in the city per se, so that attenuated relationships might have been expected; these results are considered in the context of estimated exposure errors. The paper emphasizes the magnitudes of the mortality effects attributed to air pollution, rather than their statistical significance. This is done in several different contexts: each pollutant considered individually along with weather factors, and the joint vs. individual contributions of various combinations of pollutants, taken two, three, and four pollutants at a time. These differences are then considered in the contexts of correlations among pollutants and of their estimated exposure errors.

**PRESENTER:** Peter A. Valberg

**TITLE:** A confounding role for indoor air pollutants in the associations of ambient particulate matter (PM) with daily morbidity and mortality

**AUTHORS:** Valberg, PA, Watson, AY

**ABSTRACT**

It has not been possible to causally link outdoor PM with changes in daily morbidity/mortality because of our inability to identify a toxic agent that is consistent across all the epidemiologic studies, and that has been validated by laboratory toxicology. An alternative explanation is possible: Varying levels of ambient PM may be correlated to conditions that directly modify morbidity/mortality rates or promote the production of, and increase people's exposure to, lung toxins. That is, the same changes in weather and human behavior that give rise to "day-to-day fluctuations in ambient particulate matter" can also give rise to changes in physiologic factors, such as stress, and in exposures, such as to indoor air. PM levels may thus only be a marker for other, causal factors. Weather fluctuations leading to cold stress or heat stress result in an increasing fraction of time spent indoors. Moreover, inclement weather promotes the use of climate-control systems, which can generate increased indoor levels of potentially toxic airborne particles. At the same time, weather stress tends to increase outdoor PM levels because of the increased use of vehicles and an increased demand for electric power for heating or air conditioning. Therefore, personal exposure to indoor toxins, may be linked to outdoor PM concentrations. The major significance of indoor air contaminants is that, unlike outdoor PM, toxicological and epidemiological studies of indoor air support the biological plausibility of morbidity and mortality risk for indoor allergens, bioaerosols, and perhaps environmental tobacco smoke (ETS). In evaluating causality in the epidemiologic studies, one must consider the factors that drive outdoor PM fluctuations in the first place to determine if the observed morbidity/mortality changes can be caused by pathways other than the hypothetical frank toxicity from inhalation of ambient PM.

PRESENTER: Bruce Rodan

TITLE: Methodological limitations in particulate air pollution epidemiology based on pulmonary function testing.

AUTHORS: Rodan, B, Karch, N

ABSTRACT

Pulmonary function tests (PFT) constitute an important measure in research efforts to determine if, and at what level, particulate matter (PM) air pollution, or one of its chemical constituents or subfractions, can cause adverse effects on the human lung. Epidemiological investigations employing PFTs have generally demonstrated small decrements in pulmonary function in the range of less than 1 to 2 percent at high ambient PM exposures, in addition to studies reporting no decrements. The numerous sources of PFT variability and their magnitude mandate caution in determining the meaning of these small changes in PFT indices. This presentation updates the authors' previously published review (Inhal. Toxicol. 1995;7:1269-1291) of methods employed in contemporary epidemiological studies of PM air pollution to account for alternative causes of PFT changes. Emphasis is placed on the potential for meteorological variables, indoor air pollution exposures, aeroallergens, respiratory infections and psychogenic factors to affect PFT results. Any differential distribution of sources of variability over time or between exposed and control populations could bias or confound study results. A progressive development of methodological rigor in considering potential confounding factors has improved the assessment of PFT effects attributable to PM pollution. No studies were completely satisfactory, however, reflecting the fundamental discrepancy between the extent of inherent and extraneous variability in PFT indices compared to the small size of the pulmonary effects being measured. A surprising number of studies also lacked a control group independent of exposed subjects acting as their own controls. As a result, the PM literature relying on PFTs can only be considered suggestive of a small adverse effect, requiring confirmation from mechanistic studies or from controlled chamber exposures.

**PRESENTER:** Purnendu K. Dasgupta

**TITLE:** Automated Particle Collection And Analysis: Measurement of Aerosol Acidity

**AUTHORS:** Dasgupta, P. K.

**ABSTRACT**

There is increasing evidence that aerosol acidity exerts a strongly synergistic role in the adverse health effects posed by oxidants such as ozone. Gas phase acidity, such as due to  $\text{HNO}_3$  or  $\text{SO}_2$  is largely removed by the upper respiratory tract that behaves much like a wet diffusion denuder. Aerosols penetrate to the deep lung and as the aerosol is deposited, the drop in local pH considerably increases the reactivity of  $\text{O}_3$  due to the increased oxidation potential. Thus far, measurement of aerosol acidity in anything approaching short time duration measurements have been very difficult. Recently, we have developed a number of approaches to continuous collection of atmospheric aerosol and near real-time analysis of the soluble constituents of the aerosol by chromatographic means. Aerosol acidity can be measured in an automated fashion, from the difference between the total cation and the anion equivalents. Detection limits in single digits  $\text{ng/m}^3$  of strong acid  $\text{H}^+$  can be attained. The LOD is affected by the amount of background aerosol (e.g.  $(\text{NH}_4)_2\text{SO}_4$  and  $\text{NH}_4\text{NO}_3$ ) present,  $\text{NH}_4\text{HSO}_4$  is measured as a mono-equivalent strong acid. The instrument has been successfully used to study acidity of aerosols in indoor air. Kerosene-fueled space heaters do cause the formation of acid aerosols which have further been identified as primarily due to  $\text{H}_2\text{SO}_4$ . Simpler direct chromatographic means of measuring  $\text{H}^+$  are under study and will also be discussed.

PRESENTER: Constantinos Sioutas

TITLE: Harvard/EPA ambient fine and ultrafine particle concentrators for conducting inhalation exposure studies

AUTHORS: Sioutas, C, Koutrakis, P, Ferguson, ST, Kim, CS, Burton, RM

ABSTRACT

This paper presents a summary of technologies developed jointly by Harvard and EPA that concentrate ambient fine ( $0.1 < d_p < 2.5 \mu\text{m}$ ) and ultrafine ( $d_p < 0.1 \mu\text{m}$ ) particles for conducting human and animal inhalation exposure studies. Concentration enrichment is achieved by drawing fine or ultrafine particles through a series of virtual impactors. To concentrate fine particles, each impactor contains the majority of ambient fine mass (i.e.,  $0.1 < d_p < 2.5 \mu\text{m}$ ) in a bleed flow (minor flow), typically 20% of the total flow entering the stage. A dilution system is added to condition the temperature and relative humidity of the concentrated aerosol prior to supplying it to the exposure chamber. The intake sampling flow is as high as 5500 liters/min and the system delivers an aerosol enriched in concentration by a factor of 25-30 at flows as high as 200 liters/min. The fine particle concentrators were characterized using outdoor and indoor air samples as test aerosols. Fine mass and sulfate concentrations at the outlet of the concentrating system were compared to the ambient fine mass and sulfate levels, determined using Harvard-Marple impactors. The experimental tests have demonstrated that the overall concentration enrichment achieved by our system is by a factor of 9.5 ( $\pm 1.5$ ) and 26.3 ( $\pm 3.5$ ), for a two- and three-stage system, respectively. Furthermore, the concentrated aerosol is delivered into the exposure chamber at a pressure of 0.94 atmospheres, thus making it possible to conduct human exposures at essentially atmospheric pressure.

To concentrate ultrafine particles using virtual impaction, we first grow the particles by passing them over a pool of warm water ( $50^\circ\text{C}$ ) to achieve saturate the aerosol with warm vapor. Subsequently, the aerosol is drawn through a cooling tube ( $10^\circ\text{C}$ ) that allows the ultrafine particles to grow to micron-size. The grown particles are then drawn through a virtual impactor to be separated from the surrounding air. The effect of parameters such as sampling flow rate, ultrafine particle chemical composition, and aerosol concentration on the collection efficiency and losses of our system will be presented.

PRESENTER: J Q Koenig

TITLE: Size and Chemical Characterization of Airborne Particulate Matter in Spokane

AUTHORS: J Q Koenig\*, L. Haller,, T. Larson\*, G., Norris\*, H. Westberg, and C. Claiborn, Laboratory for Atmospheric Research, Department of Civil and Environmental Engineering, Washington State University, Pullman, WA 99164-2910.

ABSTRACT

Recent health effects studies suggest that the present air quality standard for aerosols, which is based upon respirable particles, does not adequately protect public health; thus the standard is currently under review. In July, 1994, a comprehensive study was initiated in Spokane to study the relationship between various size fractions and chemical components of atmospheric aerosol and health effects. This study is one of the most comprehensive particulate matter studies conducted to date, and is one of a limited number of studies that have been conducted in the arid west, where presumably a significant portion of the aerosol will be derived from geological materials.

Continuous fine and coarse particulate matter measurements are made at two locations (one is in an industrial area, and the other is in a residential area). At the residential site, particulate matter smaller than 1.0 mm, and ultrafine particles are also continuously monitored, and daily 24-hr. samples of fine and coarse particulate matter are collected and analyzed for a variety of chemical species including elemental components, ionic species, soluble iron, elemental and organic carbon, and acidity.

Preliminary results indicate that windblown dust enhances both the fine and coarse fractions of particulate matter in Spokane. Seasonal trends in chemical composition and size characterization will be examined. The relationships between the 24-hour average values and peak hourly values, as well as differences between weekday and weekend levels, will also be discussed.

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Funded by the Washington State Department of Ecology, the US EPA, and the American Lung Association of Washington

**PRESENTER:** Junfeng (Jim) Zhang

**TITLE:** Diurnal variations in and correlations among particulate matter, sulfate, sulfur Dioxide, and ozone at a site in Metropolitan Philadelphia

**AUTHORS:** Zhang, J, Lioy, P, Suh, H, Koutrakis, P

**ABSTRACT**

Epidemiological studies have demonstrated associations between daily mortality and ambient particulate concentrations. More accurate conclusions, however, could be anticipated if the exposure data, instead of concentration data, were available and used in the analysis. Most measurements of particulate matter have been based on samples collected over periods of 24 hours. Since people usually do not spend an entire day outdoors, it would be inappropriate to assess human exposures to ambient air pollutants by using the 24-hour-averaged concentrations. Therefore, it is necessary to understand diurnal distributions of the pollutants concerned. As part of the Metropolitan Aerosol Acidity Characterization Study, hourly concentrations of  $PM_{10}$ ,  $PM_{2.5}$ , and sulfate have been measured at a site in Metropolitan Philadelphia during the summer of 1992. The measurement technique employed for  $PM_{10}$  and  $PM_{2.5}$  was the Rupprecht and Patashnick model 1400 Tapered Element Oscillation Microbalance (TEOM®), and that employed for particulate sulfate was the Harvard Continuous Sulfate/Thermal Speciation (CSTS) system. Maximum concentrations of  $PM_{2.5}$  and sulfate were observed to occur during afternoon hours when children are more likely to play outside and thus most directly exposed. Coarse particles ( $PM_{10} - PM_{2.5}$ ) seemed to have a peak concentration at around 6-7 am, which may reflect a local source in the early morning, most likely the local traffic.  $PM_{10}$  turned to have high concentrations in the early morning due to the peak coarse particle concentrations. In the afternoon,  $PM_{10}$  concentrations gradually increased due to the increase in  $PM_{2.5}$  concentrations.

We have also measured  $O_3$  and  $SO_2$  simultaneously at the site. Through the Pearson correlation analysis, we have found that sulfate was positively correlated with  $O_3$  ( $r=0.65$ ),  $PM_{2.5}$  ( $r=0.78$ ), and  $PM_{10}$  ( $r=0.37$ ), respectively. Since a large fraction of  $PM_{10}$  was  $PM_{2.5}$ ,  $PM_{10}$  and  $PM_{2.5}$  were positively correlated ( $r=0.55$ ). Sulfate and  $SO_2$  were strongly correlated if pairing  $SO_2$  concentration of Hour  $t$  with sulfate concentration of Hour  $(t+7)$  for the analysis (sulfate peak concentration occurred 7 hours later from when  $SO_2$  peak concentration occurred). Strong correlations among these species suggest that concentrations of one species could be estimated from concentrations of others for this site by regression analyses.

PRESENTER: Beverly S. Cohen

TITLE: Electric Charge on Ambient Ultrafine Particles

AUTHORS: Cohen, B.S., and Li, W.

ABSTRACT

The particle number concentration in ambient air is dominated by nanometer sized particles and most airborne particles carry a few charges. Our deposition studies in hollow airway casts demonstrated that electrical forces can result in significantly enhanced deposition of charged ultrafine particles in human airways. Thus, the charge distribution on an aerosol can effect the dose of an inhaled toxicant by affecting the quantity and pattern of particle deposition in the human respiratory system.

There are currently few data on which to base estimates of the charge fraction of ordinary ambient ultrafine aerosols. The magnitude of the effect on the dose of inhaled submicrometer particles can be estimated by assuming a theoretical particle charge distribution, for example the Boltzmann distribution for equilibrium conditions. However, when charge distributions are measured, equilibrium conditions are not normally met; a larger fraction of charged particles and an excess of charge of one sign is common.

We monitored the aerosol charge distribution in our laboratory (Lanza Labs, NYU, Tuxedo, NY) to test whether the indoor aerosol charge distribution differs substantially from the Boltzmann distribution. A Differential Mobility Analyzer and Ultrafine Condensation Particle Counter (TSI 3010 and TSI3025 respectively, TSI, St. Paul, MN) were used in series to measure the number concentration of charged particles in a specific size range. The measured number concentrations were compared with and without a  $^{85}\text{Kr}$  charge neutralizer in the line. The difference between the two concentrations indicated the difference between the natural aerosol charge distribution and a Boltzmann distribution.

We found that the fractions of charged particles were about 50% -100% higher than predicted in the size range 10 nm - 500 nm. The fraction of naturally charged particles with electrical mobility equal to a singly charged 125 nm particle for example was about 60% higher than that of a Boltzmann distribution.

PRESENTERS: Frederick W. Lipfert

TITLE: Simulation studies on the effects of exposure error on environmental epidemiology

AUTHORS: Lipfert FW, Wyzga RE

ABSTRACT

This paper examines the distributions of personal, indoor, and outdoor ambient exposures to air pollution to develop some exploratory simulation strategies for time-series studies of acute events. These distributions are described in terms of three parameters: the mean values, the standard deviations (in normalized coordinates), and the correlations with coincident outdoor measurements. The need to distinguish spatial (i.e., interpersonal) from temporal variability is recognized. Probabilistic simulations are then performed to explore how the results of epidemiology studies might change if they had been based on personal exposures instead of outdoor ambient measurements. The criteria of interest include the regression slopes, their standard errors, and indicators of linearity. The importance of city size is considered, in which the trend in spacial homogeneity of outdoor air quality in smaller cities must be contrasted with the likelihood of increased heterogeneity of personal exposure when fewer events are considered each day. This aspect of the problem may also be extended to consideration of individual diagnostic or age groups.

PRESENTER: Richard Reiss

TITLE: An evaluation of PM<sub>10</sub> measurements made by tapered element oscillating microbalances in Southern California

AUTHORS: Reiss, R, Roberts, P, Lurmann, F, Wright, D

ABSTRACT

The Tapered Element Oscillating Microbalance (TEOM) is a convenient and cost-effective method for collecting ambient PM<sub>10</sub> data on a daily basis. As part of the Southern California Children's Health Study, hourly PM<sub>10</sub> measurements are being made with TEOMs at 13 sites in central and southern California to characterize daily PM<sub>10</sub> in the communities. To avoid problems with water collecting and later evaporating off the filter of the TEOM, the instruments are operated at 50°C. There was a concern that operation at this elevated temperature may result in the volatilization of some material such as ammonium nitrate and organic species, which would result in a bias towards low PM<sub>10</sub> measurements. There was also concern that the bias would depend on the chemical composition of the PM which varies between communities and seasons. In order to investigate this possibility, a comparison of PM<sub>10</sub> data collected using TEOMs and High Volume Samplers (HiVol) was performed. Collocated 24-hr average HiVol PM<sub>10</sub> measurements were available every sixth day at three sampling sites. HiVol PM<sub>10</sub> measurements were also available at seven sites within 10 km of the TEOM monitoring sites for use in the comparison.

Good agreement between collocated TEOM and HiVol PM<sub>10</sub> measurements was found at the Atascadero site in central California, which is one of the least polluted sites. However, during the winter the TEOM data were slightly lower than the HiVol data at Atascadero which is probably due to the volatilization of organic material in wood smoke PM. The comparison for sites in the Los Angeles area showed poorer agreement, with the TEOM measurements being consistently lower than the HiVol measurements. The largest bias was observed at Rubidoux in Riverside County which has recorded some of the highest PM<sub>10</sub> concentrations in the Los Angeles Basin. This bias was highly correlated ( $r^2 = 0.89$ ) with the nitrate concentration from the HiVol filters, which suggests that ammonium nitrate volatilization explains the low TEOM measurement at this site. At other sites such as Long Beach, lower correlations were found between the TEOM - HiVol difference and the HiVol nitrate, suggesting that other components were volatilized. As a result of the bias in the TEOM measurements at the study sites, regression equations were developed for each study site to normalize the hourly TEOM measurements to the HiVol. This adjustment procedure is necessary to ensure that consistent data will be used throughout the study sites for exposure assessment.

PRESENTER: Terry Gordon

TITLE: The use of a centrifugal concentrator in ambient PM10 toxicology studies.

AUTHORS: T Gordon, C P Fang, H Gerber, and L C Chen. NYU Medical Center, Tuxedo, NY and Gerber Scientific, Reston, VA.

ABSTRACT

Epidemiologic studies have provided strong evidence that episodic exposure to ambient particulate matter is associated with increases in morbidity and mortality. These adverse effects have been demonstrated at concentrations far below the NAAQS and, thus, the biological plausibility of these effects has been questioned. For the purpose of exposing test animals to relevant and reproducible exposure concentrations of PM10, we have developed a centrifugal concentrator which can concentrate ambient particles up to 20-fold. A high volume blower is used to deliver ambient air to the inlet manifold and the entrained particles travel along a concentric annulus formed by a stationary solid outer cylinder and a porous inner cylinder rotating at high speed (5K to 15K rpm). Suction applied at one end of the porous shaft causes the dispersion medium (air) to pass through the porous cylinder and into the shaft. Since the rotational velocity of the particles is comparable to that of the rotating cylinder near its surface, the particles move radially outward due to the centrifugal force, as well as down the concentric annulus and inward due to the suction of air into the rotating porous cylinder. The particles reach their highest concentration near the outlet manifold, where they enter the exposure chamber under positive pressure ( $\sim 0.1$ " H<sub>2</sub>O). Except for nominal coarse particle loss due to impaction and diffusional loss of ultrafine particles, the increase in particle concentration is simply the ratio of the flow rates for the inlet air and the exhaust air drawn through the porous cylinder. We have used the Gerber concentrator to deliver concentrated ambient PM10 from NYC to a nose-only exposure chamber and examined the concentrating effect across ambient particle sizes.

**PRESENTER:** Yiming Ding

**TITLE:** Fine particulate n-nitroso and nitrite organic compounds in the atmosphere

**AUTHORS:** Ding, Y., Cui, W., Lee, M.L., Eatough, D.J.

**ABSTRACT**

Photochemistry between gas phase organic compounds,  $\text{NO}_x$  and ozone is expected to result in the formation of nitrogen oxide containing compounds of toxicological importance. Classes of compounds which may form include nitro-, N-nitroso- and nitrite- substituted organic compounds. Many of these compounds will be labile, semi-volatile organic compounds in equilibrium between the gas and particulate phases in the atmosphere. The phase distribution of these potentially toxic semi-volatile organic compounds may be determined using diffusion denuder sampling technology. The total concentration of N-nitroso compounds in a collected samples is determined using N-nitroso specific denitrosation reactions followed by detection of the NO with a TEA. The denitrosation chemistry can also be used to determine nitrite compounds. Differentiation between total N-nitroso- and nitrite- containing compounds is done using sulfamic acid as a nitrite specific reagent. Specific nitro and N-nitroso organic compounds are detected using supercritical fluid chromatography coupled to a thermal energy analyzer using nitro- and nitroso-specific chemiluminescence detection. These analytical techniques for the sampling and determination of total N-nitroso and nitrite material have been used for the quantification of these compounds in both fine particles and the gas phase in samples collected in Provo, Utah. The results indicate that the majority of the N-nitroso and nitrite organic compounds present in fine particulate matter in the urban area studied are semi-volatile organic compounds which are lost from particles during sampling. Furthermore, the concentrations of these fine particulate compounds are comparable to the concentrations of gas phase N-nitroso and nitrite organic species. Detailed analyses of nitrogen, nitro-, nitrite and nitroso-organic semi-volatile compounds can be expected to improve our understanding of the etiology of observed health effects associated with exposure to ambient fine particles.

PRESENTER: Christopher A. Noble

TITLE: Aerosol Time-of-Flight Mass Spectrometry: A New Method for Performing Real-Time Characterization of Aerosol Particles

AUTHORS: Noble, C. A., Prather K. A.

ABSTRACT:

We have recently developed a technique, aerosol time-of-flight mass spectrometry (ATOFMS), which can be used for the direct determination of the size and chemical composition of individual aerosol particles in real-time. Such measurements are made *in-situ* by combining a unique dual-laser aerodynamic particle sizing system to size and track each particle in the instrument and time-of-flight mass spectrometry for single particle composition analysis. In experiments to date, ATOFMS has been used for the analysis of model inorganic and organic aerosol systems, ambient aerosol particles, and environmental tobacco smoke particles drawn directly into the instrument. In this presentation, the emphasis will be on ambient aerosol sampling, including compositionally-resolved particle size distributions of ambient aerosol particles as a function of time, showing definitive size/composition correlations. Information at this level of detail showing chemical speciation in single particles as a function of size may allow for more directed health effects studies to determine specific effects of aerosol constituents for particles of a given size.

PRESENTER: Tina Bahadori

TITLE: Personal, Indoor, and Outdoor Exposures to Particulate Matter of Ten COPD Patients Living in Private Residences (Based on Results from the *Nashville Particulate Personal Monitoring: a Pilot Study*)

AUTHORS: Bahadori, T, Koutrakis, P

ABSTRACT

This proposed paper discusses findings from the Nashville Personal Monitoring Study, and examines the relationship between personal exposures and both indoor and outdoor concentrations of particulate matter. Furthermore, it serves as an opportunity to reflect on issues related to the accurate measurement and characterization of the personal exposures of sensitive individuals.

During the summer of 1995, a study was conducted to characterize the personal  $PM_{2.5}$  and  $PM_{10}$  exposures of individuals with chronic obstructive pulmonary disease (COPD), living in Nashville, Tennessee. Ten COPD patients residing in private residences were monitored for six days each. For these individuals, personal exposures, as well as ambient and indoor concentration of  $PM_{2.5}$  and  $PM_{10}$  were measured. In addition, data was collected to determine the air exchange rates within the residences, as well as the individual's time/activity patterns during the monitoring period.

The study participants (3 women, 7 men) ranged in age from  $<40$  to  $>80$ . All were current non-smokers living with a spouse or other caretaker in their own air-conditioned homes. Three were currently on oxygen therapy. Nine were disabled or retired, but relatively active. Preliminary analyses of the data indicate that mean personal exposures were  $33 \mu g/m^3$  for  $PM_{10}$  and  $22 \mu g/m^3$  for  $PM_{2.5}$ , virtually identical to the mean outdoor concentrations of 32 and  $23 \mu g/m^3$ , respectively. Indoor mean concentrations were lower, at 22 and  $16 \mu g/m^3$ , respectively. There was, however, no correlation between personal and outdoor concentrations of the two size fractions. The correlation between personal and indoor concentrations of  $PM_{2.5}$  ( $R^2 = 0.48$ ;  $n = 30$ ;  $p < 0.0001$ ) were good, and much higher than that of  $PM_{10}$  ( $R^2 = 0.08$ ;  $n = 29$ ;  $p < 0.0001$ ).

While this study once again highlighted the importance of direct measurement as a means for assessing personal exposure, it also made evident the difficulties associated with the collection of accurate and statistically interpretable data from the subpopulations at risk. Senior citizens or those mobility-restricted due to respiratory illness, while at risk, may be too fragile to endure the stresses associated with direct and detailed measurement of personal exposures. Thus, special care and attention is required in the design and execution of such studies to render them meaningful and valuable.

PRESENTER: Ed Avol

TITLE: Indoor and outdoor residential sampling of PM<sub>10</sub> and PM<sub>2.5</sub> aerosols in southern California

AUTHORS: Avol E, Navidi W, Colome S

ABSTRACT

In conjunction with a multi-year study to track the lung health development of Southern California school children and document their exposure to ambient ozone, respirable particles, nitrogen dioxide, and acids, filter samples were collected in and around a subset of subjects' homes. Sampling was performed in four greater Los Angeles communities historically prone to episodes of elevated ambient levels of ozone and respirable particles. Samples were collected on two occasions for 24hrs both inside and outside subject's homes during June through November 1994. Sampling was conducted using 37mm Teflo filters in impactors designed to remove particles larger than 10 or 2.5  $\mu\text{m}$  at a flow rate of 4 LPM. A total of 61 indoor and outdoor (I/O) PM<sub>2.5</sub> samples were collected, while 87 I/O PM<sub>10</sub> measurements were made. A sub-set of home samples (n=23) was collected with co-located I/O PM<sub>10</sub> and PM<sub>2.5</sub> samplers. Inter-quartile indoor PM<sub>10</sub> levels ranged from 24 to 47  $\mu\text{g}/\text{m}^3$ , while inter-quartile outdoor PM<sub>10</sub> values were observed to be 18 to 45  $\mu\text{g}/\text{m}^3$ . Three homes were observed with 24hr indoor PM<sub>10</sub> values exceeding 100  $\mu\text{g}/\text{m}^3$ . Inter-quartile indoor and outdoor PM<sub>2.5</sub> levels ranged from 10 to 22 and 7 to 19  $\mu\text{g}/\text{m}^3$  respectively. Three 24hr indoor PM<sub>2.5</sub> samples were measured at over 75  $\mu\text{g}/\text{m}^3$ . Co-located indoor PM<sub>10</sub> and PM<sub>2.5</sub> samples were poorly correlated ( $R^2 = 0.40$ ) in non-smoking homes, and driven by single extreme samples in smoking homes. Outdoor co-located PM<sub>10</sub> and PM<sub>2.5</sub> samples were highly correlated ( $R^2 = 0.86$ ). Median I/O PM<sub>10</sub> and PM<sub>2.5</sub> ratios of 1.05 and 1.10, respectively, suggested that the majority of homes sampled did not have significant indoor sources of either PM<sub>10</sub> or PM<sub>2.5</sub>. However, samples collected in a small subset of homes, especially homes inhabited by smokers, were elevated.

**PRESENTER:** David Stieb

**TITLE:** Personal exposure of adults with cardiorespiratory disease to particulate acid and sulfate in Saint John, New Brunswick, Canada

**AUTHORS:** Stieb, D, Brook, J, Broder, I, Judek, S, Burnett R

**ABSTRACT:**

**BACKGROUND:** Saint John is the site of an ongoing study of the relationship between cardiorespiratory emergency department (ED) visits and airborne particles, including particulate acid and sulfate.

**PURPOSE:** To assess the extent to which fixed site monitors reflect average personal exposure to fine particle sulfate ( $\text{SO}_4^{2-}$ ) and acidity ( $\text{H}^+$ ) among adults visiting the ED with cardiorespiratory disease.

**METHODS:** Study participants had made an ED visit for cardiorespiratory disease in the previous 12 months and resided within approximately 5 km of the fixed monitor which records the highest  $\text{H}^+$  and  $\text{SO}_4^{2-}$  concentrations in Saint John. Twenty-one volunteers wore personal annular denuders, during the period 7:30 a.m. to 6 p.m. (mean duration 7.6 hours) for up to 4 separate days between July 6 and August 11, 1995. Subjects completed a time-activity diary for each sampling period.

**RESULTS:** Subjects ranging from 49 to 85 years of age completed a total of 62 sampling periods. The mean proportion of time spent indoors, outdoors and in vehicles was respectively 81.8, 7.6 and 10.5 percent. Mean personal  $\text{SO}_4^{2-}$  (29.8 nmol/ $\text{m}^3$ ) and  $\text{H}^+$  concentrations (17.1 nmol/ $\text{m}^3$ ) were lower than measurements at fixed site monitors (mean  $\text{SO}_4^{2-}$  49.1 nmol/ $\text{m}^3$ ; mean  $\text{H}^+$  37.6 nmol/ $\text{m}^3$ ). The correlation ( $R^2$ ) between mean personal and fixed site  $\text{SO}_4^{2-}$  was 0.90 ( $p < 0.0001$ ), and high daily concentrations measured at the fixed site were reflected in high mean personal concentrations. For  $\text{H}^+$  there was little correlation between mean personal and fixed site measurements ( $R^2 = 0.06$ ;  $p = 0.29$ ), although some high personal measurements corresponded to high fixed site concentrations.

**CONCLUSIONS:** In this population of adults visiting an ED with cardiorespiratory disease, fixed site  $\text{SO}_4^{2-}$  monitors appear to accurately reflect daily variability in average personal  $\text{SO}_4^{2-}$  exposures. Personal exposure to  $\text{H}^+$  does not appear to be well-represented by fixed site monitors.

PRESENTER: Jürgen Pauluhn

TITLE: Hazard identification and risk assessment of pyrethroids in the indoor environment

AUTHORS: Pauluhn, J.

ABSTRACT

Pyrethroids often have greatly varying activities, depending on the route of administration (oral, dermal, inhalational). Particularly the  $\alpha$ -cyano-pyrethroids have additional specific features, for example the sensory irritation potential within the respiratory tract, that can be adequately quantified by inhalation testing only. Sensory irritation is considered to be one of the most important endpoints for risk assessment of  $\alpha$ -cyano-pyrethroids in the indoor environment. Measurements were taken during acute and repeated exposure of rats to evaluate whether this endpoint is threshold concentration-dependent or cumulative dose-dependent. To reproduce exposure scenarios resulting from *worst-case* pest control measures on carpets, the dislodgment of pyrethroid laden dust particles was studied on a small-scale test model. The findings obtained support the conclusion that  $\alpha$ -cyano-pyrethroids act only as sensory irritants. Concomitant respiratory tract inflammation and ensuing changes in susceptibility - which is a common finding in chemical sensory irritants - was not found, consequently, the activity of  $\alpha$ -cyano-pyrethroids in "equitoxic doses" may differ considerably after inhalational and oral administration. The carpet studies showed that there was no specific enrichment of pyrethroids in the total dust fraction, even after sustained, extreme mechanical loading (continuous brushing for approximately 18 hrs). The lack of a correlation between absolute (mg pyrethroid/m<sup>3</sup> air) and relative (mg pyrethroid/kg dust) airborne dust concentrations as well as the low dislodgment of pyrethroids following pest control measures from pretreated carpets showed that sedimented house dust is not a suitable matrix for risk assessment of pyrethroids in such an environment and therefore pyrethroid determinations in house dust (vacuum cleaner bag analyses) are considered a poor indicator for the assessment of potential, inhalational exposure of the general population to pyrethroids.

PRESENTER: James R. Ramsay

TITLE: The Effects of Building Ventilation Types and Human Activity Patterns on Indoor  $PM_{10}$ .

AUTHORS: James R. Ramsay and Dean R. Lillquist

ABSTRACT

Indoor and ambient airborne  $PM_{10}$  concentrations in two Salt Lake hospitals were examined January through May 1995. Seven samplers were located at each hospital. One ambient sampler was located on the roof at each hospital. Data analysis showed that the two hospitals had significantly ( $p > 0.05$ ) different ambient  $PM_{10}$  concentrations ( $26\mu g/m^3/24hr$  versus  $19\mu g/m^3/24hr$ ). Six indoor samplers were located in areas with different levels of human activity and different air filtration systems. Human activity levels were scored low, moderate, or high. Three types of air filtration systems were identified: 1) Low efficiency (LE), 2) Bag filters (BF), 3) HEPA. At each hospital either the effectiveness of the LE and the BF systems, or the effectiveness of the BF and HEPA filtration systems were compared. In both comparisons the type of air filtration system significantly ( $p > 0.05$ ) affected the concentration of indoor  $PM_{10}$ . The average  $PM_{10}$  concentrations in the LE/BF comparison were  $18\mu g/m^3/24hr$  and  $13\mu g/m^3/24hr$  respectively. The average  $PM_{10}$  concentrations in the BF/HEPA filtration comparison were  $18\mu g/m^3/24hr$  and  $11\mu g/m^3/24hr$  respectively. Large variation in the  $PM_{10}$  concentrations among sampler sites within a common air filtration system were observed. In the BF and HEPA filtration environments the  $PM_{10}$  levels were positively correlated with increasing human activity. The increase in  $PM_{10}$  attributable to moderate human activity in a HEPA environment was  $1.15\mu g/m^3/24hr$  ( $p > 0.1$ ). In a BF environment the increase in  $PM_{10}$  at a high activity site, compared to a moderate site, was  $5\mu g/m^3/24hr$  ( $p > 0.05$ ). An estimate of the proportion of indoor  $PM_{10}$  attributable to ambient sources was made. The average  $PM_{10}$  concentration of a HEPA site was subtracted from the average  $PM_{10}$  concentrations of BF sites with the same human activity level as the HEPA site. The difference in the two averages,  $6\mu g/m^3/24hr$  (35%), is assumed to be due to infiltration of ambient  $PM_{10}$  through the BF.