Clearing the Air on Particulate Matter: PM 2.5 (Part 2)

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Health Effects of PM2.5

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Source-to-Response Framework (PM Example)

Sources of Airborne Particulate Matter or Gaseous Precursor Emissions

Indicator in Ambient (Outdoor) Air (e.g. Mass Concentration)

Personal Exposure

Dose to Target Tissues

Human Health Response

Mechanisms determining emissions, chemical transformation (including formation of secondary particles from gaseous precursors), and transport in air

Human time-activity patterns, indoor (or microenvironmental) sources and sinks of particulate matter

Deposition, clearance, retention and disposition of particulate matter presented to an individual

Mechanisms of damage and repair
Air Pollution: Dimensions of Concern

Comfortable
Asymptomatic

Risk

Diseased

Exposed

Uncomfortable
Symptomatic

Healthy

Risk

Not Exposed
ATS Adverse Health Effects of Air Pollution Definition

- Any effect on mortality – consideration of the extent of life-shortening
- Permanent loss of lung function
- Reversible loss of lung function with symptoms
- Symptoms associated with negative impact on QoL or change in clinical status
- Detectable negative effect on clinical status
- Measurable negative impact on quality of life
- Shift in risk factor distribution (risk profile of exposed population) even in absence of frank illness

Adapted from American Thoracic Society, 2000
“...initiation of, or contribution to, the inducement, or aggravation of asthma, emphysema, chronic bronchitis, congenital abnormalities of the lung, impairment of the body's defense mechanism, coronary heart disease and/or hypertensions, impaired fetal development, harm to red blood cells and anemias, and accelerated aging.”

House of Representatives – Committee Report 95-294
Clean Air Act Amendments of 1977
Frailty Models

- Chronic effect: air pollution affects the size of the frail pool
- Acute effect: air pollution increases risk for hospitalization and mortality
Health Effects Pyramid

- Death
- Hospitalizations
- Emergency Room Visits
- Asthma Attacks
- Impaired Breathing
- Reduced Lung Function

NUMBER OF PEOPLE AFFECTED
Smaller particles penetrate:

- Nose
- Pharynx
- Larynx
- Trachea
- Bronchi/Bronchioles

Gas Exchange Airways
Lymphatic System
### Three Disciplinary Approaches for Obtaining Health Information

<table>
<thead>
<tr>
<th>Discipline</th>
<th>Population</th>
<th>Strengths</th>
<th>Weaknesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidemiology</td>
<td>Communities</td>
<td>Natural exposure</td>
<td>Difficulty of quantifying exposure</td>
</tr>
<tr>
<td></td>
<td>Diseased Groups</td>
<td>No extrapolations</td>
<td>Many covariates and confounders</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Susceptible groups</td>
<td>Minimal dose-response data</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Long-term, low-level effects</td>
<td>Association versus causation</td>
</tr>
<tr>
<td>Clinical Studies</td>
<td>Experimental Healthy or Diseased Subjects</td>
<td>Controlled exposure</td>
<td>Artificial exposure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Few covariates</td>
<td>Acute effects only</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vulnerable persons</td>
<td>Hazards</td>
</tr>
</tbody>
</table>

*Air Pollution Health Study Methodologies* (modified from Vallero, *Fundamentals of Air Pollution*, 4th ed.)
Particles And Health: What Do Studies Find?

- Mortality: Total; Cardio-Pulmonary; Lung Cancer & Infant (long-term exposure only)

- Cardio-Pulmonary Hospital Admissions & ED Visits
  - Ischemic heart disease
  - Congestive heart failure

- CV Parameter Effects

- Lung Function Decrements In Children
Particles And Health: What Do Studies Find? (2)

• Asthma Development

• Respiratory Symptoms & Medication Use
  – Asthma symptoms
  – Cough
  – Shortness of breath
  – Chest tightness

• Adverse Birth Outcomes
  – Low birth weight
  – Pre-term birth
  – Intrauterine growth restriction
### Table 2-1. Summary of Causal Determinations for PM$_{2.5}$

<table>
<thead>
<tr>
<th>Exposure Duration</th>
<th>Outcome</th>
<th>Causality Determination</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Long-term</strong></td>
<td>Mortality</td>
<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular Effects</td>
<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Respiratory Effects</td>
<td>Likely to be Causal</td>
</tr>
<tr>
<td></td>
<td>Reproductive and Developmental Effects</td>
<td>Suggestive</td>
</tr>
<tr>
<td></td>
<td>Cancer, Mutagenicity, Genotoxicity Effects</td>
<td>Suggestive</td>
</tr>
<tr>
<td><strong>Short-term</strong></td>
<td>Mortality</td>
<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular Effects</td>
<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Respiratory Effects</td>
<td>Likely to be Causal</td>
</tr>
<tr>
<td></td>
<td>Central Nervous System Effects</td>
<td>Inadequate</td>
</tr>
</tbody>
</table>

PM2.5 Health Effects: Causal Assessment
Harvard “Six Cities” Study

P Portage, WI  L St. Louis, MO
T Topeka, KS  H Harriman, TN
W Watertown, MA  S Steubenville, OH

Dockery et al, NEJM, 1993
Six Cities Cohort Follow-up

Orange = Period 1
Yellow = Period 2

PM$_{2.5}$ (µg/m$^3$)

Mortality Risk Ratio

St. Louis
Watertown
Topeka
Kingston
Steubenville
Portage

(Laden et al, AJRCCM, 2006)
PM & Mortality Risk: ACS II Study

Table 2. Adjusted Mortality Relative Risk (RR) Associated With a 10-μg/m³ Change in Fine Particles Measuring Less Than 2.5 μm in Diameter

<table>
<thead>
<tr>
<th>Cause of Mortality</th>
<th>1979-1983 (95% CI)</th>
<th>1999-2000 (95% CI)</th>
<th>Average (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause</td>
<td>1.04 (1.01-1.08)</td>
<td>1.06 (1.02-1.10)</td>
<td>1.06 (1.02-1.11)</td>
</tr>
<tr>
<td>Cardiopulmonary</td>
<td>1.06 (1.02-1.10)</td>
<td>1.08 (1.02-1.14)</td>
<td>1.09 (1.03-1.16)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>1.08 (1.01-1.16)</td>
<td>1.13 (1.04-1.22)</td>
<td>1.14 (1.04-1.23)</td>
</tr>
<tr>
<td>All other cause</td>
<td>1.01 (0.97-1.05)</td>
<td>1.01 (0.97-1.06)</td>
<td>1.01 (0.95-1.06)</td>
</tr>
</tbody>
</table>

*Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet. CI indicates confidence interval.
Long-term PM2.5 Cardiovascular Health Studies

Harvard Six-cites cohort
- Cardiopulmonary (Dockery et al. 1993)
- Cardiovascular (Laden et al. 2006)

ACS multi-city cohort
- Cardiopulmonary (Pope et al. 1995)
- Cardiovascular (Pope et al. 2004)
- Ischemic HD (Pope et al. 2004)
- Dysrhythmias, Heart failure, Cardiac arrest (Pope et al. 2004)

ACS LA cohort
- Cardiopulmonary (Jerrett et al. 2005)
- Ischemic HD (Jerrett et al. 2005)

Women’s Health Initiative
- Non Fatal Cardiovascular (Miller et al. 2004)
- Fatal Cardiovascular (Miller et al. 2004)

Pope & Dockery, JAWMA, 2006
PM2.5 Reductions & Increased Life Expectancy

- Avg. > life expectancy = 2.7 years
- Multiple factors associated with life expectancy increase
- Air quality changes account for as much as 15% of > life expectancy
- < 10 $\mu$g/m$^3$ PM2.5 = > 0.6 yr life expectancy

Pope et al. 2009

Figure 4. Changes in Life Expectancy for the 1980s–1990s, Plotted against Reductions in PM$_{2.5}$ Concentrations for 1980–2000.
Dots and circles labeled with numbers represent changes in population-weighted mean life expectancies at the county level and metropolitan-area level, respectively. The solid and broken lines represent regression lines with the use of county-level and metropolitan-area-level observations, respectively. The metropolitan areas are coded by.
Fine PM & Hospital Admissions

- Medicare data – 11.5 million people 65+
- 204 counties
- 1999 - 2002

(Dominici et. al., JAMA, 2006)
Percent Change in Hospital Admission Rates Per 10 μg/m³ PM2.5 Increase By Subregions

(Dominici et al. JAMA, 2006)
Percent Change in Hospitalization Rates per 10 μg/m³ Increase in PM2.5 for the US Eastern and Western Regions

(Dominici et al., JAMA, 2006)
~10,000 avoided C-P hospital admissions annually per 10 \( \mu g/m^3 \) PM2.5 reduction

### Table 2. Annual Reduction in Admissions Attributable to a 10-\( \mu g/m^3 \) Reduction in the Daily PM\(_{2.5} \) Level for the 204 Counties in 2002

<table>
<thead>
<tr>
<th>Cause-Specific Hospital Admissions</th>
<th>Annual No. of Admissions</th>
<th>Annual Reduction in Admissions (95% PI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular disease</td>
<td>226,641</td>
<td>1,836 (680 to 2,992)</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>70,061</td>
<td>602 (−42 to 1,254)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>346,082</td>
<td>1,523 (69 to 2,976)</td>
</tr>
<tr>
<td>Heart rhythm</td>
<td>169,627</td>
<td>967 (−17 to 1,951)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>246,598</td>
<td>3,156 (1,923 to 4,389)</td>
</tr>
<tr>
<td>COPD</td>
<td>108,812</td>
<td>990 (196 to 1,785)</td>
</tr>
<tr>
<td>Respiratory tract infection</td>
<td>226,620</td>
<td>2,085 (929 to 3,241)</td>
</tr>
</tbody>
</table>

Abbreviations: COPD, chronic obstructive pulmonary disease; PI, posterior interval; PM\(_{2.5} \), particulate matter of less than or equal to 2.5 \( \mu m \) in aerodynamic diameter.

*Per 10-\( \mu g/m^3 \) reduction in PM\(_{2.5} \).

( Dominici et al., JAMA, 2006 )
Medicare Study Findings

• Regional differences in PM2.5 mass equivalent effects on cardiovascular and respiratory-related hospital admissions

• Implication of results for differences in fine PM sources/composition?

• Substantial national health benefits estimated for fine PM reductions (even at levels below current daily PM2.5 NAAQS)
Fine PM and Low Birth Weight

- ~360,000 Connecticut and Massachusetts births 1999 – 2002

- Criteria air pollution data: 1998 – 2002

- Daily AQ data - PM10 & PM2.5 data filled with weekly avgs.

- Exposures estimated over gestational period and by trimester

Bell et al., EHP, 2007
Figure 1. Change in birth weight per IQR increase in gestational exposure, using the linear model with all births (solid line, $n = 358,504$) and first births only (dashed line, $n = 129,282$). The point reflects the central estimate; the vertical line represents the 95% confidence interval.
Fine PM and Low Birth Weight

Findings

- Low birth weight associated with gestational, 2\textsuperscript{nd} & 3\textsuperscript{rd} trimester PM2.5 exposure
- Controlling for CO reduces but does not eliminate PM 2.5 association
- Effect of PM2.5 on LBW greater for Black mothers compared to White
PM Inhalation

Blood
- Altered rheology
- Increased coagulability
- Translocated particles
- Peripheral thrombosis
- Reduced oxygen saturation

Heart
- Altered cardiac autonomic function
- Increased dysrhythmic susceptibility
- Altered cardiac repolarization
- Increased myocardial ischemia

Lungs
- Inflammation
- Oxidative stress
- Accelerated progression and exacerbation of COPD
- Increased respiratory symptoms
- Effected pulmonary reflexes
- Reduced lung function

Vasculature
- Atherosclerosis, accelerated progression of and destabilization of plaques
- Endothelial dysfunction
- Vasoconstriction and Hypertension

Systemic Inflammation Oxidative Stress
- Increased CRP
- Proinflammatory mediators
- Leukocyte & platelet activation

Brain
- Increased cerebrovascular ischemia

Multiple mechanistic pathways have complex interactions and interdependencies

Pope & Dockery, JAWMA, 2006
PM2.5 and Cardiovascular/Respiratory Mortality

RR associated with a 10 μg/m³ increase in PM2.5 for 1979 to 1983, 1999 to 2000, and 1979-2000 average, respectively

## PM2.5 Mortality and Smoking

Adjusted RRs and 95% CIs for a 10 µg/m³ Increase in PM$_{2.5}$ (Average) and for Former and Current Smoker (vs Never Smoker) for Various Cause-of-Death Categories

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>PM$_{2.5}$ (95% CI)</th>
<th>Former Smoker (95% CI)</th>
<th>Current Smoker (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cardiovascular diseases plus diabetes</td>
<td>1.12 (1.08–1.15)</td>
<td>1.26 (1.23–1.28)</td>
<td>1.94 (1.90–1.99)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>1.18 (1.14–1.23)</td>
<td>1.33 (1.29–1.37)</td>
<td>2.03 (1.96–2.10)</td>
</tr>
<tr>
<td>Dysrhythmias, heart failure, cardiac arrest</td>
<td>1.13 (1.05–1.21)</td>
<td>1.18 (1.12–1.24)</td>
<td>1.72 (1.62–1.83)</td>
</tr>
<tr>
<td>Hypertensive disease</td>
<td>1.07 (0.90–1.26)</td>
<td>1.21 (1.07–1.37)</td>
<td>2.13 (1.86–2.44)</td>
</tr>
<tr>
<td>Other atherosclerosis and aortic aneurysms</td>
<td>1.04 (0.89–1.21)</td>
<td>1.63 (1.45–1.84)</td>
<td>4.21 (3.71–4.78)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>1.02 (0.95–1.10)</td>
<td>1.12 (1.06–1.18)</td>
<td>1.78 (1.67–1.89)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>0.99 (0.86–1.14)</td>
<td>1.05 (0.94–1.16)</td>
<td>1.35 (1.20–1.53)</td>
</tr>
<tr>
<td>All other cardiovascular diseases</td>
<td>0.84 (0.71–0.99)</td>
<td>1.22 (1.09–1.38)</td>
<td>1.78 (1.56–2.04)</td>
</tr>
</tbody>
</table>

2004 Pope Study Implications for PM2.5-related Mortality Mechanisms

- Absolute risk of PM larger for smokers v. non-smokers

- Long-term PM exposure contributes to progression of CV disease v. exacerbation of COPD

- Primary mechanisms for long-term PM2.5 exposure-related mortality:
  - Pulmonary/systemic inflammation
  - Accelerated atherosclerosis
  - Altered cardiac autonomic functions
Summary: Fine Particles and Health

- Recent PM2.5 studies confirm earlier evidence of mortality and morbidity (especially CV-related)

- Longer-term chronic exposure studies find larger effects than short-term acute exposure studies

- Finer exposure resolution (i.e. within city ΔPM2.5) produces larger effect estimates than national (between city) estimates

- Interindividual variability in response to PM2.5 exposure makes detection of dose-response threshold (if it exists) unlikely at population level
Summary: Fine Particles and Health (2)

• Increasing emphasis on sensitive populations
  – Elderly
  – Children
  – Existing CV and respiratory disease
  – Low SES populations
  – Fetus & Neonates

*************************************
  – Diabetics
  – Obese
  – Genetic factors (antioxidant response, enzymes, procoagulants)
  – Smokers

• Emerging evidence for various PM components (e.g., elemental carbon, metals) and mechanisms for adverse effects
Particles And Health: What Do We Need To Know?

- How much lifespan is lost from long-term exposures?
- Do long-term exposures adversely affect adult lung function?
- What are the mechanisms for the effects of PM on health?
- Is there a threshold for health effects?
- What is the role of co-pollutants?
- What characteristics of particles determine toxicity?
  - Size
  - Number
  - Chemistry (secondary inorganic PM; metal content)
  - Solubility
  - Surface area and reactivity
  - Other characteristics
Questions?
Fine Particulate Matter and Vulnerable Populations

Andrew Stolbach, MD
Assistant Professor
Department of Emergency Medicine
Johns Hopkins University, School of Medicine
PM 2.5 in Vulnerable Populations

http://www.carepress.com/safe_t_toons1

"This means something but I can't remember what!"

http://petcaretips.net/picture-canary-coal-mine.html
PM 2.5 in Vulnerable Populations

- Children
- Elderly
- Cardiovascular disease
- Lung disease
- Obesity
- Low socioeconomic status
PM 2.5 in Vulnerable Populations

- Children
  - High activity levels
  - High minute ventilation
  - High lung surface area

- Elderly

- CV disease

- Lung disease

- Obesity

- SES

PM 2.5 in Vulnerable Populations

Children

Asthmatic Children (Meds and No-meds) controlling for CO and NO₂

- Association between decrease in lung function (FEV₁) and PM2.5
  - All children (-45.9 mL, 0 lag)
  - No-med children (-75.9 mL, 0 lag)
  - General similar results for PEF, MMEF
PM 2.5 in Vulnerable Populations

- Early and lifetime exposures to PM 2.5 associated with reduced PEF and FEF at 25% and 50% of vital capacity

Oftedal B et al. Epidemiology 2008
PM 2.5 in Vulnerable Populations

Children

Elderly

CV disease

Lung disease

Obesity

SES

Figure 3. Indoor PM concentrations, asthma symptoms, and rescue medication use: multivariate models (coarse module adjusted for age, sex, race, parent education level, season, indoor fine PM, ambient fine PM, ambient coarse PM; fine module adjusted for age, sex, race, parent education level, season, indoor coarse PM, ambient coarse PM, ambient fine PM).

<table>
<thead>
<tr>
<th>Vulnerable Populations</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Children</td>
<td></td>
</tr>
<tr>
<td>Elderly</td>
<td></td>
</tr>
<tr>
<td>CV disease</td>
<td></td>
</tr>
<tr>
<td>Lung disease</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td></td>
</tr>
</tbody>
</table>

- Reduced pulmonary clearance of PM
- Higher prevalence of other comorbidities
PM 2.5 in Vulnerable Populations

Figure 2. Percent increase in risk and 95% CIs of HF admissions and readmissions associated with 10 μg/m³ of PM$_{2.5}$ lagged moving average of 14 days stratified by gender, age, and length of hospital stay. Closed and open circles are estimates from models including and excluding weather variables, respectively.
PM 2.5 in Vulnerable Populations

Risk for Death from Cardiovascular Disease per 10 μg/m³ Increase in PM2.5

- 76% for Older Women
- 13% for Older Women
- 19% for Whole Population
- 28% for Whole Population

Sources:
- Miller et al. 2007, Women’s Health Initiative Study
- Krewski et al. 2000, American Cancer Society’s Study
- Krewski et al. 2000, Six Cities Study
- Laden et al. 2006, Harvard Six Cities Study
Elderly exposed to fine particulate matter in an experimental setting had decreased heart rate variability immediately and at 24 hours.

Decreased heart rate variability may predict dysrhythmias.

The effect was absent in young subjects.
PM 2.5 in Vulnerable Populations

- PM 2.5 causes inflammation, which exacerbates coronary artery disease
- Inflammation increases platelet activation and decreases activity of tissue plasminogen activator
- In animal models there is rapid progression of atherosclerosis and faster plaque rupture
- Studies show PM 2.5 is related to carotid intima thickness in people

Children

Elderly

CV disease

Lung disease

Obesity

SES

Kunzli et al. Environmental Health Perspectives 2005
PM 2.5 in Vulnerable Populations

- In Beijing, increases in PM 2.5 concentration were associated with increase emergency department visits for cardiovascular disease (OR 1.005, 95% CI: 1.001-1.009)

• In Utah, short-term exposure to PM 2.5 was associated with increased hospitalization in those with CHF

PM 2.5 in Vulnerable Populations

- PM 2.5 was associated with decreased heart rate variability in individuals with the metabolic syndrome, but not those without

Children

Elderly

CV disease

Lung disease

Obesity

SES

http://www.ummm.edu/careguides/00284.htm

Park SK. Environ Health Perspect 2010
PM 2.5 in Vulnerable Populations

### Table 3. Estimated Hazard Ratios for the Time to the First Cardiovascular Event or Death Associated with an Exposure Increase of 10 μg per Cubic Meter in the Level of Fine Particulate Matter (PM$_{2.5}$).

<table>
<thead>
<tr>
<th>Outcome</th>
<th>No. of Events</th>
<th>Hazard Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Overall</td>
<td>Between Cities</td>
</tr>
<tr>
<td>First cardiovascular event</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any cardiovascular event†</td>
<td>1816</td>
<td>1.24 (1.09–1.41)</td>
</tr>
<tr>
<td>Coronary heart disease‡</td>
<td>1268</td>
<td>1.21 (1.04–1.42)</td>
</tr>
<tr>
<td>Cerebrovascular disease§</td>
<td>600</td>
<td>1.35 (1.08–1.68)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>584</td>
<td>1.06 (0.85–1.34)</td>
</tr>
<tr>
<td>Coronary revascularization</td>
<td>949</td>
<td>1.20 (1.00–1.43)</td>
</tr>
<tr>
<td>Stroke</td>
<td>554</td>
<td>1.28 (1.02–1.61)</td>
</tr>
<tr>
<td>Death from cardiovascular cause</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any death from cardiovascular cause</td>
<td>261</td>
<td>1.76 (1.25–2.47)</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Definite diagnosis</td>
<td>80</td>
<td>2.21 (1.17–4.16)</td>
</tr>
<tr>
<td>Possible diagnosis</td>
<td>59</td>
<td>1.26 (0.62–2.56)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>122</td>
<td>1.83 (1.11–3.00)</td>
</tr>
</tbody>
</table>

* All analyses evaluated the time until the first event in the category. All estimates were adjusted for age, race or ethnic group, educational level, household income, smoking status, systolic blood pressure, body-mass index, and presence or absence of diabetes, hypertension, or hypercholesterolemia.
† Events include myocardial infarction, coronary revascularization, stroke, death from coronary heart disease (both definite and possible diagnosis), and cerebrovascular disease. The sum of events in each category may be greater than the total number of events, since some subjects had both coronary and cerebrovascular events.
‡ Events include myocardial infarction, coronary revascularization, and death from coronary heart disease.
§ Events include stroke and death from cerebrovascular disease.

Miller et al., NEJM, 2007
Older adults with COPD exposed to PM 2.5 had bigger decreases in their pulmonary function tests than those without COPD.
### PM 2.5 in Vulnerable Populations

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children</td>
<td>• 57 older adults (24 w/COPD, 33 w/o); non-smokers living w/ non-smokers</td>
</tr>
<tr>
<td>Elderly</td>
<td>• Personal, indoor, outdoor and central site PM2.5 and PM coarse measurements (1999 – 2002): 5-10 day 24-hr. monitoring periods</td>
</tr>
<tr>
<td>CV disease</td>
<td>- Median PM$_{2.5}$ values</td>
</tr>
<tr>
<td></td>
<td>• 3 central sites – 10.3 µg/m$^3$</td>
</tr>
<tr>
<td></td>
<td>• Outdoor – 8.6 µg/m$^3$</td>
</tr>
<tr>
<td></td>
<td>• Indoor – 7.6 µg/m$^3$</td>
</tr>
<tr>
<td></td>
<td>• Personal – 8.5 µg/m$^3$</td>
</tr>
<tr>
<td>Lung disease</td>
<td>• Lung function measured 13 times each year 1 &amp; 2</td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>SES</td>
<td></td>
</tr>
</tbody>
</table>
**PM 2.5 in Vulnerable Populations**

- **Children**
  - Elderly
    - Association between decrease in lung function (FEV$_1$) and central site PM2.5
  - CV disease
    - All adults (-35.5 mL, 0 lag; -40.4 mL, 1-day lag)
    - COPD (-70.8 mL, 1-day lag)

- Lung disease

- Obesity

- SES

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*Trenga et al., Chest, 2006*
PM 2.5 in Vulnerable Populations

- Obesity, which is associated with chronic inflammation and increased minute ventilation, may be synergistic with PM 2.5 exposure
- More data are needed
Low socioeconomic status is associated with decreased access to health care, fresh food.
PM 2.5 in Vulnerable Populations

- High school graduation rate was associated with increased cardiovascular mortality from PM 2.5.
PM 2.5 in Vulnerable Populations

- Decreased median household income associated with increased mortality from PM 2.5

Children
Elderly
CV disease
Lung disease
Obesity
SES
What We Can Recommend to Patients Regarding Exposure to Outdoor PM 2.5

- Pay attention to air quality
- Avoid strenuous exercise in during times of poor air quality
- Avoid exercise near highways during peak traffic
What We Can Recommend to Patients Regarding Exposure to Outdoor PM 2.5

<table>
<thead>
<tr>
<th>AQI Value</th>
<th>Actions To Protect Your Health From Particle Pollution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good (0–50)</td>
<td>None</td>
</tr>
<tr>
<td>Moderate (51–100)</td>
<td>Unusually sensitive people should consider reducing prolonged or heavy exertion.</td>
</tr>
</tbody>
</table>
| Unhealthy for Sensitive Groups (101–150) | The following groups should reduce prolonged or heavy outdoor exertion:  
  - People with heart or lung disease  
  - Children and older adults  
  Everyone else should limit prolonged or heavy exertion. |
| Unhealthy (151–200)           | The following groups should avoid all physical outdoors:  
  - People with heart or lung disease  
  - Children and older adults  
  Everyone else should avoid prolonged or heavy exertion. |
| Very Unhealthy (201–300)      | The following groups should remain indoors and keep activity levels low:  
  - People with heart or lung disease  
  - Children and older adults  
  Everyone else should avoid all physical activity outdoors. |
What We Can Recommend to Patients Regarding Exposure to Indoor PM 2.5

Watch the 3 S-Words:

- Smoking
- Sweeping
- Stove
• In 1990, officials in Dublin banned the sale of bituminous coal

  – Compared daily air pollution, weather, and deaths for 6 year period before and after the ban

  – Avg. reductions: BS = ~36μg/m³ (71%)

  – \( \text{SO}_2 = \) ~11μg/m³ (34%)

What We Can Do About Exposure to Outdoor PM 2.5

- There was no change in the number of accidental deaths

**Respiratory Deaths**
- 15%

**Cardiac Deaths**
- 10%

Scientists estimated that adopting stricter PM 2.5 standards in Europe would result in decreased mortality in adults over 30 years old.

- Proposed Standard: 25 μg/m³ → 0.4%
- 15 μg/m³ → 1.6%
- 10 μg/m³ → 3.0%

Questions?
Addressing Other Risk Factors in PM2.5 Health Studies (Pope et. al. 2002)