Fine PM Health Effects Studies – Policy Significance

- Epidemiologic studies provide key science basis for setting primary (health-based) PM NAAQS

- PM component health studies have potential to provide insight for potential future regulation of toxic PM components and sources

- Intervention studies inform assessments of potential health outcome & health cost benefits of reductions in PM (accountability)
Fine PM Health Effects

- Total, C-P, Lung Cancer Mortality
- Lung Function
- Cardio-Pulmonary Hospital Admissions
- Cardio-Pulmonary ER Visits
- CV parameter effects
- Birth Outcomes
Six Cities Follow-up Study
(Laden et al., AJRCCM, 2006)

- Portage, WI; Topeka, KS; Louisville, KY; Kingston, TN; Watertown, MA; Steubenville, OH.


- Ability to examine effect of fine PM reductions over study period
Six Cities Cohort Follow-up

Mortality Risk Ratio vs. PM$_{2.5}$ ($\mu$g/m$^3$)

Orange = Period 1
Yellow = Period 2

(Laden et al, AJRCCM, 2006)
TABLE 3. ADJUSTED PROPORTIONAL HAZARD MORTALITY RATE RATIOS AND 95% CONFIDENCE INTERVALS FOR A 10-μg/m³ INCREASE IN AVERAGE AMBIENT PM$_{2.5}$ OVER THE ENTIRE FOLLOW-UP (1974–1998) AND THE RATE RATIOS FOR AVERAGE PM$_{2.5}$ IN PERIOD 1 AND THE DECREASE IN LEVELS BETWEEN THE TWO PERIODS

<table>
<thead>
<tr>
<th>Cases</th>
<th>Entire Follow-Up Average PM$_{2.5}$</th>
<th>Period 1 Average PM$_{2.5}$</th>
<th>Decrease in Average PM$_{2.5}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total mortality</td>
<td>2,732</td>
<td>1.16 (1.07–1.26)</td>
<td>1.18 (1.09–1.27)</td>
</tr>
<tr>
<td>Cardiovascular*</td>
<td>1,196</td>
<td>1.28 (1.13–1.44)</td>
<td>1.28 (1.14–1.43)</td>
</tr>
<tr>
<td>Respiratory*</td>
<td>195</td>
<td>1.08 (0.79–1.49)</td>
<td>1.21 (0.89–1.66)</td>
</tr>
<tr>
<td>Lung cancer*</td>
<td>226</td>
<td>1.27 (0.96–1.69)</td>
<td>1.20 (0.91–1.58)</td>
</tr>
<tr>
<td>Other</td>
<td>1,115</td>
<td>1.02 (0.90–1.17)</td>
<td>1.05 (0.93–1.19)</td>
</tr>
</tbody>
</table>

For definition of abbreviations, see Table 2.
Rate ratios have been adjusted for age in 1-yr categories, sex, current smoker, current pack-years of smoking, former smoker, former pack-years of smoking, less than high school education, and linear and quadratic terms for body mass index.


† Average PM$_{2.5}$ calculated as the average of Six Cities monitoring data for available years 1980–1988 and PM$_{2.5}$ estimated from Aerometric Information Retrieval System and extinction data for years where Six Cities data were not available.

‡ Average PM$_{2.5}$ in Period 1 calculated as the average from 1980–1985, the years where there are monitoring data for all cities, decrease in average PM$_{2.5}$ (average Period 2 (1990–1998) – average Period 1).

(Laden et al., AJRCCM, 2006)
Long-term Fine PM & CV Effects in Women
(Miller et al., NEJM, 2007)

- ~66,000 women
(1994 – 98)
- 36 cities
- 2000 PM2.5 data
- Avg. 6 year follow-up

<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.
WHI Study vs. Other Long-term Cohort Studies

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

- 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

(CARB, 2006)
Cohort Studies – CV Mortality

RR for CV mortality associated with a 10-μg/m³ in long-term PM₂.₅

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. 2002)
Ischemic HD (Pope et al. 2004)
Dysrhythmias, Heart failure, Cardiac arrest (Pope et al. 2004)
Cardiopulmonary (Jerrett et al. 2005)
Ischemic HD (Jerrett et al. 2005)

ACS LA cohort
Non Fatal Cardiovascular (Miller et al. 2004)
Fatal Cardiovascular (Miller et al. 2004)

Women’s Health Initiative

(Pope & Dockery, JAWMA, 2006)
Fine PM & Hospital Admissions
(Dominici et. al., JAMA, 2006)

- Medicare data – 11.5 million people 65+
- 204 counties
- 1999 - 2002
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 µg/m³ PM2.5 reduction

### Table 2. Annual Reduction in Admissions Attributable to a 10-µg/m³ 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 µm in aerodynamic diameter.

*Per 10-µg/m³ reduction in PM$_{2.5}$.
Are There Health Effects At PM2.5 Levels Below the 2006 Daily PM2.5 NAAQS?

<table>
<thead>
<tr>
<th>Reason for Hospital Admission</th>
<th>Lag Day No.*</th>
<th>National Average Relative Rate, PE (95% PI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>All Days</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>0</td>
<td>0.81 (0.30 to 1.32)</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>0</td>
<td>0.86 (−0.06 to 1.79)</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>2</td>
<td>0.44 (0.02 to 0.86)</td>
</tr>
<tr>
<td>Heart rhythm</td>
<td>0</td>
<td>0.57 (−0.01 to 1.15)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>0</td>
<td>1.28 (0.78 to 1.78)</td>
</tr>
<tr>
<td>COPD</td>
<td>0</td>
<td>0.91 (0.18 to 1.64)</td>
</tr>
<tr>
<td>Respiratory tract infection</td>
<td>2</td>
<td>0.92 (0.41 to 1.43)</td>
</tr>
</tbody>
</table>

Abbreviations: COPD, chronic obstructive pulmonary disease; PE, point estimate; PI, posterior interval; PM<sub>2.5</sub>, particulate matter of less than or equal to 2.5 µm in aerodynamic diameter.

*Results are reported for the lag at which the greatest effect of PM<sub>2.5</sub> was estimated.
†Percentage change in hospital admission rates per 10-µg/m<sup>3</sup> increase in PM<sub>2.5</sub> concentration for all Medicare enrollees (aged >65 years).

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

- Regional differences in PM2.5 mass equivalent effects on cardiovascular (N.E.) and respiratory-related hospital admissions (S.E. & West)
- 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)
Effect of PM2.5 on COPD and Asthma in Seattle
(Trenga et al., Chest, 2006)

Panel study:
• 57 older adults (24 w/COPD, 33 w/o); non-smokers living w/ non-smokers
• 17 asthmatic children (11 w/medication, 6 w/o)
• Personal, indoor, outdoor and central site PM2.5 and PM coarse measurements (1999 – 2002): 5-10 day 24-hr. monitoring periods
  – Median PM$_{2.5}$ values
    • 3 central sites – 10.3 µg/m$^3$
    • Outdoor – 8.6 µg/m$^3$
    • Indoor – 7.6 µg/m$^3$
    • Personal – 8.5 µg/m$^3$
• Lung function measured 13 times each year 1 & 2
Effect of PM2.5 on COPD and Asthma in Seattle
(Trenga et al., Chest, 2006)

Older Adult Lung Function (COPD & No COPD) controlling for CO and NO2

• Association between decrease in lung function (FEV1) and central site PM2.5
  – All adults (-35.5 mL, 0 lag; -40.4 mL, 1-day lag)
  – COPD (-70.8 mL, 1-day lag)

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
Fine PM and Preterm Birth
(Huynh et al., Paediatr Perinat Epidemiol, 2006)

- Preterm birth: 24 – 36 weeks gestation
- Preterm birth linked to infant mortality and morbidity, possible chronic adult disease
- ~11,000 preterm CA births, 1999 – 2000
- Each preterm birth matched to 3 normal births (39 - 44 week controls)
- Maternal fine PM exposure – closest PM monitor ≤ 5 miles; CO county data
- 15% increase in risk of preterm birth per 10 µg/m³ PM2.5 (total gestation; adjusted)
• ~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
• ~5% increased risk for low birth weight associated with gestational, 2nd & 3rd trimester PM2.5 exposure (IQR)
• Controlling for CO reduces but does not eliminate PM2.5 association
• Effect of PM2.5 on LBW greater for Black mothers compared to White
“…the results from large, multi-city studies suggest that there is no strong evidence of a clear threshold for PM mortality effects. Some single city studies provide some suggestive hints for possible thresholds, but not in a statistically clear manner. More data need to be examined with alternative approaches…but, in the meantime, the use of linear PM effects models appears to be appropriate.”

(EPA 2004 PM CD)
Figure 8-30. Concentration-response curves for PM$_{10}$ mortality relationships in 20 largest U.S. cities (1987-1994), for total (Total) mortality, cardiovascular and respiratory (CVDRESP) mortality, and other-causes (Other) mortality. The concentration-response curves for the mean lag, current day, and previous day PM$_{10}$ are denoted by solid lines, squared points, and triangle points, respectively.

Source: Dominici et al. (2003a).

Figure 8-31. Posterior probabilities of thresholds for each cause-specific mortality and for mean PM$_{10}$, 20 largest U.S. cities, 1987-1994. Total = total nonaccidental mortality; CVDRESP = cardiovascular mortality and respiratory mortality; Other = mortality from other causes.

Source: Dominici et al. (2003a).
Selected Concentration-Response Relationships from Various Studies of Long-term Exposure

(a) U.S. cross-sectional mortality

(b) Harvard Six-cities (Laden et al. 2006)

(c) ACS cohort (Pope et al. 2002)

(d) Children's Lung Growth (Gauderman et al. 2004)

(adaptations from original publications rescaled for comparison purposes) (Pope & Dockery, JAWMA, 2006)
Figure 3. Combined random-effect estimated of the dose–response relationship between MI emergency hospital admissions and PM$_{10}$, computed by fitting a piecewise linear spline, with slope changes at 20 µg/m$^3$ and 50 µg/m$^3$. 
Q. Fine Particle Components, Organic Carbon Compounds and Human Health – How Much Do We Know?

A. A Bit, But Really Not Very Much
Percent of PM2.5 composition by component for yearly, winter, and summer averages, by region (Bell et al. EHP 2007)
µg/m³

R. Peng,
Unpublished data
<table>
<thead>
<tr>
<th>Measure or constituent</th>
<th>Concentration range (µg/m³)</th>
<th>Major sources</th>
<th>Epidemiology studies</th>
<th>Toxicology studies</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass</td>
<td>4–25</td>
<td>—</td>
<td>X</td>
<td>X</td>
<td>Increased interest in ultrafine particles recently</td>
</tr>
<tr>
<td>Sulfate</td>
<td>1.5–6</td>
<td>Fuel combustion</td>
<td>x</td>
<td>X</td>
<td>Sulfate is identified mainly with ammonium salts</td>
</tr>
<tr>
<td>Nitrate</td>
<td>0.5–5</td>
<td>Fuel combustion</td>
<td>—</td>
<td>x</td>
<td>Nitrate is identified mainly with ammonium salts</td>
</tr>
<tr>
<td>Acidity</td>
<td>0.01–0.2</td>
<td>Secondary sulfate or nitrate</td>
<td>x</td>
<td>x</td>
<td>Acidity in the atmosphere varies from sulfuric acid to strong acids; the latter are highly transient in the air.</td>
</tr>
<tr>
<td>Metal salts or oxides</td>
<td>&lt;0.1–2</td>
<td>Soil dust, road dust, and industrial processes</td>
<td>x</td>
<td>x</td>
<td>Soil dust components are believed to be mainly oxides; industrial metals vary as oxides or salts</td>
</tr>
<tr>
<td>Black or elemental carbon (EC)</td>
<td>1–2</td>
<td>Fuel combustion</td>
<td>—</td>
<td>x</td>
<td>Black carbon in the atmosphere is not truly elemental in nature but is likely to be a mix of elemental material and oily char from combustion</td>
</tr>
<tr>
<td>Organic matter (OM)</td>
<td>1–6</td>
<td>Fuel combustion; industrial and biological material</td>
<td>—</td>
<td>x</td>
<td>The operationally defined differentiation of EC and OC is ambiguous, but is the key measure of both components in monitoring</td>
</tr>
<tr>
<td>Other unidentified</td>
<td>0.5–4</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>From closure of chemical mass balance</td>
</tr>
</tbody>
</table>

Note: The listed composition includes a typical range of annual average ambient concentration components that are estimated to provide a reconstructed particle mass balance.

*Large number of studies (X); small number of studies (x).

*Mass reported is based on the FRM filter method, which does not account accurately for semi-volatile components, including nitrate and SVOC. These may be a factor in exposure, but have not been investigated.

*Sulfate derives from primary emissions of acidic material associated with metals such as Fe, Ni, and V from fossil fuel combustion such as residual oil, and secondarily from oxidation of sulfater dioxide emissions in the atmosphere. The former is believed to be a minor (<5%) fraction of total PM-associated sulfate.

*Black and organic carbon monitored in United States and elsewhere only after 1999. The measurement method is operationally defined in terms of a differential response to heating of filter samples.

*By convention, OM is given as 1.4 x organic carbon concentration. The factor of 1.4 actually is believed to be variable depending on the age and mix of sources of OC. Includes biological debris, e.g., fine particle bacteria, virus, detritus, waxy material, etc., also includes a large number of chemical species from primary and secondary sources, of which only perhaps 20–25% by mass have been identified.

*Organics investigated to date focused mainly on diesel exhaust particles, PAH, and biological indicators such as endotoxin.

*Believed to include adjustment for organic matter, bound water, chlorides, and other salts, as well as uncertainties in metal compound composition.

*A few epidemiological studies have inferred a relationship to carbon through traffic proximity studies, for example, and identification with transportation using a PM marker for gasoline.
Atlanta Air Pollution and ER Visits

- EPRI-funded ARIES Study
- 31 Atlanta area hospitals
- Detailed pollutants: mid-1998 to mid-2000

(Metzger et al., 2004)

Risk of CV ER visit per 2µg/m³ PM2.5 OC:
- ~3% (all CVD)
- ~5% (CHF)

(Peel et al., 2005)

Risk of pneumonia ER visit per 2µg/m³ PM2.5 OC = ~3%
Air Pollution and NYC Asthma ED Visits (2006)

- NYSDoH – funding: ATSDR, NYSERDA
- Lower Manhattan, S. Bronx
- Pollutants: sulfur dioxide, sulfates, ozone, nitrogen oxides, aldehydes, nitrous acid, nitric acid, hydrochloric acid, ammonium, metals, elemental carbon, organic carbon, hydrogen ion, pollen, mold spores, PM2.5, PM10, chromium, nickel, manganese, iron, particle number
## Table 4a. Relative Risks and 95% Confidence Intervals for Asthma ED Visits as Function of 5-Day Mean Air Pollution and Bioaerosols from Single-Pollutant Models

Note: Exposure increments used to compute RRs were the two-community average concentrations (Table 3). Bold text indicates statistical significance at the 0.05 level.

<table>
<thead>
<tr>
<th>Air Contaminant</th>
<th>Bronx</th>
<th>Manhattan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max 8-hour O$_3$</td>
<td>1.06 (1.01, 1.10)</td>
<td>1.06 (0.94, 1.19)</td>
</tr>
<tr>
<td>Max 8-hour O$_3$ (warm season)</td>
<td>1.08 (1.03, 1.12)</td>
<td>1.04 (0.91, 1.19)</td>
</tr>
<tr>
<td>NO$_2$</td>
<td>1.10 (1.01, 1.18)</td>
<td>0.95 (0.72, 1.25)</td>
</tr>
<tr>
<td>SO$_2$</td>
<td>1.11 (1.06, 1.17)</td>
<td>0.99 (0.88, 1.12)</td>
</tr>
<tr>
<td>FRM PM$_{2.5}$</td>
<td>1.05 (1.01, 1.10)</td>
<td>1.04 (0.94, 1.15)</td>
</tr>
<tr>
<td>Max PM$_{2.5}$</td>
<td>1.09 (1.03, 1.15)</td>
<td>1.04 (0.91, 1.18)</td>
</tr>
<tr>
<td>Coarse PM</td>
<td>1.02 (1.00, 1.04)</td>
<td>1.02 (0.98, 1.07)</td>
</tr>
<tr>
<td>Sulfate</td>
<td>1.03 (1.00, 1.06)</td>
<td>1.05 (0.98, 1.13)</td>
</tr>
<tr>
<td>pH</td>
<td>0.99 (0.98, 1.00)</td>
<td>0.99 (0.95, 1.02)</td>
</tr>
<tr>
<td>Elemental (Soot) Carbon</td>
<td>1.04 (0.99, 1.09)</td>
<td>1.06 (0.94, 1.19)</td>
</tr>
<tr>
<td>Organic Carbon</td>
<td>1.05 (0.93, 1.17)</td>
<td>1.20 (0.96, 1.49)</td>
</tr>
<tr>
<td>Total Metals</td>
<td>1.02 (0.99, 1.05)</td>
<td>1.02 (0.91, 1.15)</td>
</tr>
<tr>
<td>Total Aldehydes</td>
<td>1.02 (1.00, 1.04)</td>
<td>1.03 (0.96, 1.10)</td>
</tr>
<tr>
<td>Total Pollen</td>
<td>1.00 (1.00, 1.00)*</td>
<td>1.01 (1.00, 1.02)</td>
</tr>
<tr>
<td>Total Mold</td>
<td>1.01 (0.99, 1.03)</td>
<td>1.01 (0.97, 1.06)</td>
</tr>
</tbody>
</table>

*When RR and CI bounds appear equal, it is due to rounding.
### Table 5. Relative Risks from Regressions Based on Daily Maximum Hourly (SO₂ and NO₂) or Daily Maximum 3-Hour (Elemental and Organic Carbon) Exposures

Note: Bold text indicates statistical significance at the 0.05 level.

<table>
<thead>
<tr>
<th>Contaminant</th>
<th>Increment used to calculate RR</th>
<th>Bronx</th>
<th>Manhattan</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO₂ (ppm)</td>
<td>0.0492</td>
<td>1.12 (1.04, 1.20)</td>
<td>0.97 (0.75, 1.25)</td>
</tr>
<tr>
<td>SO₂ (ppm)</td>
<td>0.0227</td>
<td>1.07 (1.03, 1.12)</td>
<td>0.96 (0.86, 1.07)</td>
</tr>
<tr>
<td>Elemental (Soot) Carbon (µg/m³)</td>
<td>1.9787</td>
<td>1.05 (1.01, 1.09)</td>
<td>1.05 (0.95, 1.16)</td>
</tr>
<tr>
<td>Organic Carbon</td>
<td>3.7014</td>
<td>1.05 (0.95, 1.16)</td>
<td>1.10 (0.92, 1.32)</td>
</tr>
</tbody>
</table>
Fine PM Components & Mortality
(Ostro et al., EHP, 2006)

- PM mass and components: 2000 to 2003
- Cause of Death: all-causes, heart and lung disease, and all-cause for greater than 65 years old.
- Fresno, Kern, Riverside, Sacramento, San Diego, Santa Clara
  - 8.7 Million people, 25% of California’s population
- Health effects measured up to 3 days after pollution episode

Fine PM Components & Mortality

(Ostro et al., EHP, 2006)
Possible Mechanism for PM Health Effects

Ambient Particles in Lung

- Sensory Nerves
- Epithelial Cells
- Other Lung Tissues

- Airway Effects
- Dysfunction Endothelial
- Acute Response
- Blood Coagulability

CARDIAC & RESPIRATORY EFFECT

Inflammatory Cell Activation
### PM, Components and CV Effect Indicators

(Rückerl et al., AJRCCM, 2006)

- Exposure measure = 1IQR
- CRP*: indicator of inflammatory response linked to coronary heart disease
  - AP = ~3% risk
  - UFP = ~2% risk
- ICAM-1**: endothelial dysfunction indicator; predicts acute coronary events
  - EC = ~4% risk
  - OC = 3% risk
  - PM10 = 3% risk
  - PM2.5 = ~2% risk

(Sarnat et al., Occup. Environ. Med., 2006)

- Cardiac arrhythmia in elderly
- PM2.5, sulfate, EC, CO, O3, SO2, NO2
- 70% increase in risk of arrhythmia for sulfate only (5 day IQR)

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* C-reactive protein; **Intercellular adhesion molecule 1
So What Does This All Mean?

- Recent PM2.5 studies confirm earlier evidence of mortality and morbidity (especially CV-related)
- Longer-term chronic studies find larger effects than short-term acute studies
- Finer exposure resolution (i.e. within city ΔPM2.5) produces larger effects 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
- Increasing emphasis on sensitive populations
  - Existing CV and respiratory disease
  - Diabetics
  - Fetus
  - Women?
- Emerging evidence for various PM components and mechanisms of adverse effects
- Limited evidence so far for role of OCs in fine PM health effects
"Thank God! A panel of experts!"