Health Effects of Coal-Fired Power Plant Emissions

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Overview

• Background and Key Issues
• What We Know……
• What We Don’t Know……
• EPRI Toxicology Field Studies:
  – TERESA: Toxicological Evaluation of Realistic Emissions of Source Aerosols
  – Tri City CAPS: Tri City Concentrated Ambient Particle Study
• Conclusions
Background and Key Issues

- **PM$_{2.5}$ from power plants:**
  - *Primary* particles: emitted directly from plants; very low due to widespread use of PM controls in the US
  - *Secondary* particles: formed through oxidation of SO$_2$ to sulfate downwind of plants

**KEY ISSUES**

- What is the relative importance of different PM sources and components in adverse health effects?
- How important are power plant emissions in PM$_{2.5}$-related health effects?
What We Know……

• Toxicology:
  – Single component studies: little effect of sulfate in animals or human volunteers except at high concentrations
  – Source-focused studies: use of lab-scale combustors or collected coal fly ash – representativeness?
  – Realistic lab emissions studies (e.g., NERC)
  – Concentrated ambient particle (CAP) studies

• Epidemiology:
  – Associations between sulfate and health effects observed
What We Don’t Know……

• No assessment of the toxicity of actual plant emissions
• No information on the toxicity of actual secondary particles formed through SO$_2$ conversion in the atmosphere
TERESA: Toxicological Evaluation of Realistic Emissions of Source Aerosols

Approach:

• Evaluate toxicity of secondary particles from power plants, at 3 different power plants in the US
• Expose rats to multiple simulated atmospheric conditions
• Examine mobile source emissions using same methods

Project Team:

• EPRI, Harvard School of Public Health

Supported in part by DOE-NETL (Cooperative Agreement DE-FC26-03NT41902)
TERESA Field Setup

Generating Unit

Control Devices (e.g. ESP)

Sampling Port

Primary emissions

Reaction Lab:
- Emissions Aging and Atmospheric Simulation
- Chamber System
- Extensive exposure monitoring

Secondary particles

Toxicology Lab:
- Laboratory Rat Exposures
- Biological responses evaluated
Field Operations at Plant 2
## Exposure Scenarios

<table>
<thead>
<tr>
<th>Code</th>
<th>Scenario</th>
<th>Composition</th>
<th>Simulated Atmospheric Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>Primary</td>
<td>Primary (un-aged) emissions, diluted to ~ 1 ppm SO$_2$</td>
<td>Primary stack emissions</td>
</tr>
<tr>
<td>PO</td>
<td>Primary + oxidized</td>
<td>Primary emissions + •OH</td>
<td>Aged plume, oxidized stack emissions, sulfate aerosol formation</td>
</tr>
<tr>
<td>POS</td>
<td>Primary + oxidized + SOA</td>
<td>Primary emissions + •OH + α-pinene/ozone</td>
<td>Aged plume, unneutralized acidity, secondary organic aerosol (SOA) derived from biogenic emissions</td>
</tr>
<tr>
<td>PONS</td>
<td>Primary + oxidized + neutralized + SOA</td>
<td>Primary emissions + •OH + NH$_3$ + α-pinene/ozone</td>
<td>Aged plume, mixture of neutralized sulfate and SOA</td>
</tr>
<tr>
<td>O</td>
<td>Oxidized</td>
<td>Primary emissions + •OH, no primary PM</td>
<td>Control scenario</td>
</tr>
<tr>
<td>S</td>
<td>SOA</td>
<td>α-pinene/ozone only</td>
<td>Control scenario</td>
</tr>
<tr>
<td>OS</td>
<td>Oxidized + SOA</td>
<td>Primary emissions + •OH + α-pinene/ozone, no primary PM</td>
<td>Control scenario</td>
</tr>
</tbody>
</table>

Plus sham (control animals exposed to air only)
# Exposures Performed

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<tbody>
<tr>
<td><strong>P</strong></td>
<td>May 10-13</td>
<td>June 6-9</td>
<td>August 8-13</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>PO</strong></td>
<td>November 13-15</td>
<td>May 9-12</td>
<td>September 19-22</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>POS</strong></td>
<td>October 4-7</td>
<td>March 21-24 (no SCR)</td>
<td>July 19-22</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>May 3-6 (SCR)</td>
<td>August 14-15</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>PONS</strong></td>
<td>June 22-26</td>
<td>May 31-June 3</td>
<td>July 25-28</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>June 27-30</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>October 11-14</td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>POS</strong></td>
<td>-</td>
<td>July 8, 13</td>
<td>August 16-17</td>
<td>Compromised</td>
</tr>
<tr>
<td></td>
<td></td>
<td>September 8, 9</td>
<td></td>
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<td><strong>OS</strong></td>
<td>-</td>
<td>-</td>
<td>August 28-31</td>
<td>Normal</td>
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<td><strong>O</strong></td>
<td>-</td>
<td>-</td>
<td>September 1-4</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>S</strong></td>
<td>-</td>
<td>-</td>
<td>September 6-9</td>
<td>Normal</td>
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</table>

Total: 78 exposure days
12 rats (6 exposed; 6 filtered air control) per exposure
Summary of Integrated Mass Concentrations

PM mass (μg m⁻³)

Plant 1
Plant 2
Plant 3

1.5
2.5

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Breathing Pattern: Plants 1 and 2

Total: 48 exposures

Plant 1: PO FREQUENCY

Plant 2: PO FREQUENCY

Plant 1: PO TIDAL VOLUME

Plant 2: PO TIDAL VOLUME

P=NS

P=0.06

P=NS

P=0.04
Plant 1: Oxidative Stress in Heart and Lung Tissue

POS
(n=8 in each group)

Boston Particles
(Gurgueira et al., 2002)
(n=4-6 in each group)
Plant 2: Effect of Exposure (POS) on Premature Ventricular Beats

Exposed animal PVBs 86.8% > sham; \( p = 0.05 \)
Summary, Conclusions, and What’s Next

- Plant 1: No effects whatsoever
- Plant 2: Some biological effects with some conditions/scenarios
- Plant 3: Very few effects observed
- Primary PM highest at Plant 3; overall mass highest at Plant 2
- Effects do not appear to be correlated with mass
- See majority of effects in scenarios with secondary organics
  - Effect of SOA alone? No.
  - Interaction of SOA with component of mixture?
  - Additive/synergistic effect?
- Analyses ongoing to understand plant/scenario differences
- Mobile source component in 2008 (funded through the Harvard/EPA PM Center)
Tri City Concentrated Ambient Particle Study (Tri City CAPS)

**Cardiopulmonary Toxicity Induced by Ambient Particulate Matter: Inhalation Toxicology Studies Using a Mobile Particle Concentrator in Regions Dominated by Power Plant and Mobile Source Emissions**

**Approach:**
- Station ambient particle concentrator/mobile lab at 3 locations for 2 seasons
- Expose rats to CAPs for 8 hrs/day for 13 days
- Link responses to PM sources and components

**Project Team:**
- EPRI, Michigan State University, University of Michigan

Supported in part by DOE-NETL (Cooperative Agreement DE-FC26-03NT41902)
Location of Study Sites

Downtown Detroit, MI
Dominated by diesel and gasoline emission-derived PM

Steubenville, OH
Dominated by power plant and local industrial emissions

State Park in NW PA
Rural site; dominated by power plant emissions
Complementary Approach to TERESA

Bottom-up approach (start with controlled sources)

Top-down approach (start with ambient PM, tease out effects of specific sources)
Detroit CAPs Cause Reduced Heart Rate
Summer 2004
Component Analysis

• Looked at the relationship between specific PM components and heart rate
• Reduced heart rate significantly associated with:
  • Integrated CAPs mass
  • “Unidentified mass” (metals + particle-bound water + some portion of organic carbon)
  • Aluminum
  • Cobalt
  • Phosphorus
Conclusions

• Innovative approaches are needed to determine the relative importance of different PM sources and components in adverse health effects
• TERESA: showing some biological effects with power plant emissions under certain conditions/scenarios
• Tri City CAPS: showing CAPs-associated alterations in cardiac function