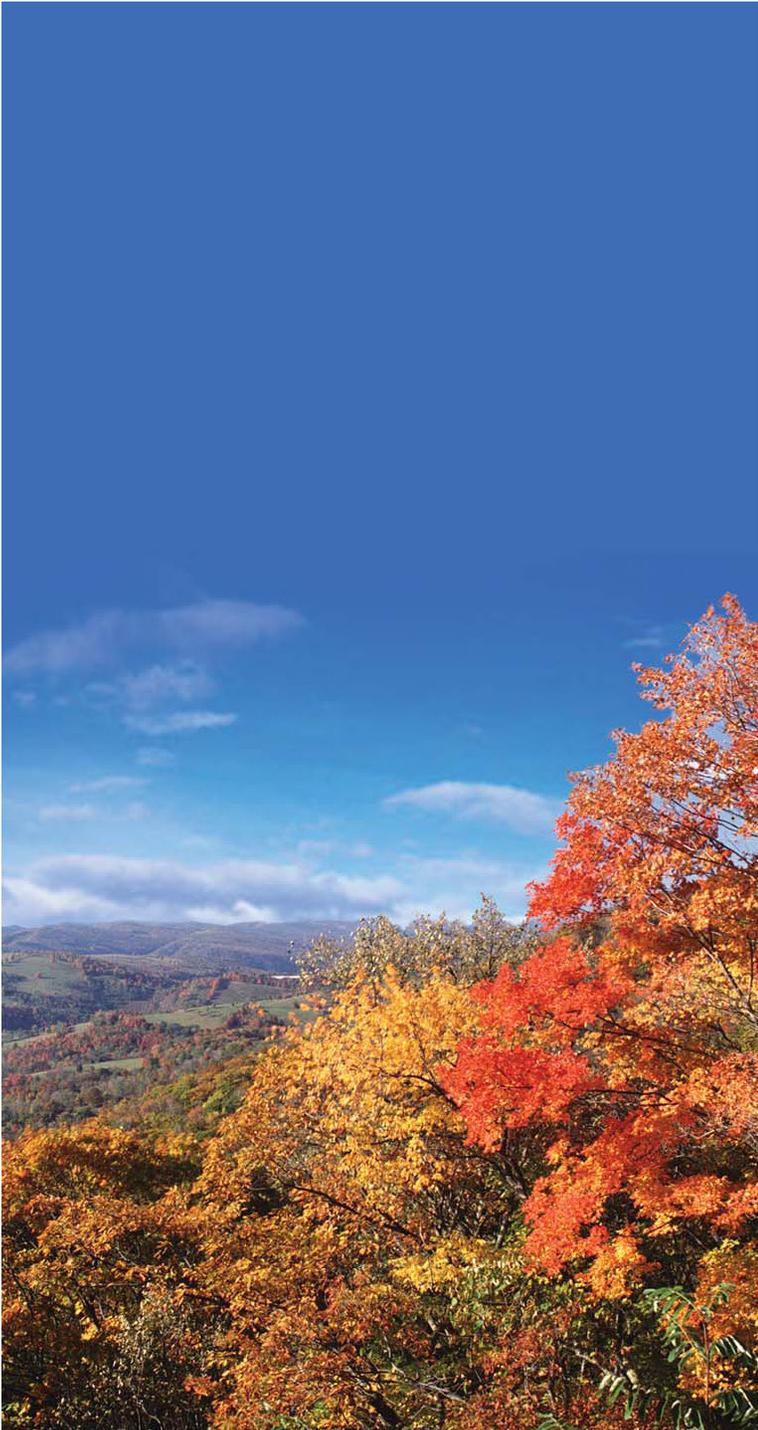




Health Effects of Coal-Fired Power Plant Emissions

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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 SO₂ 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 SO₂ 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

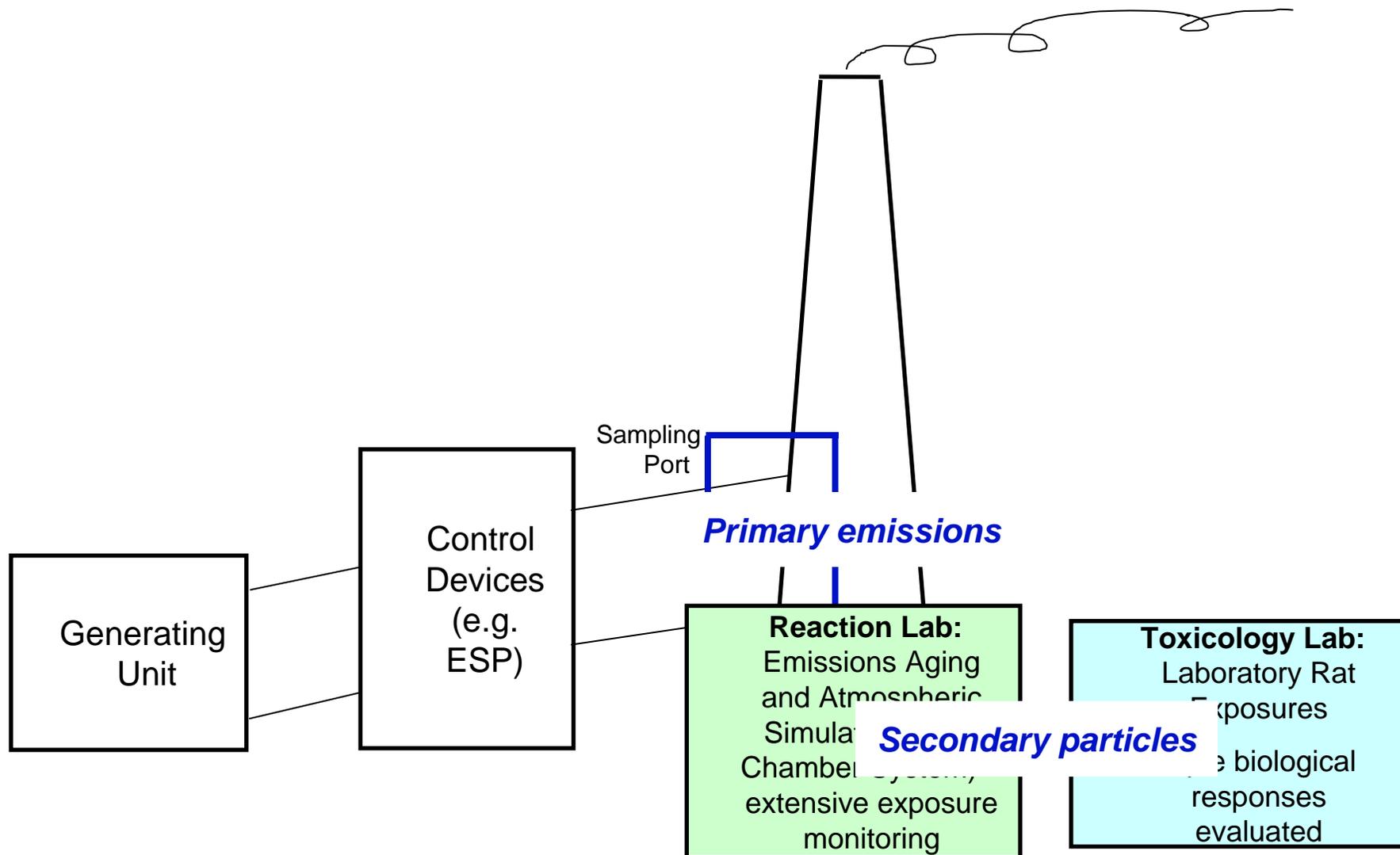


Project Team:

- EPRI, Harvard School of Public Health

**Supported in part by DOE-NETL
(Cooperative Agreement DE-FC26-03NT41902)**

TERESA Field Setup



Field Operations at Plant 2



Exposure Scenarios

Code	Scenario	Composition	Simulated Atmospheric Condition
P	Primary	Primary (un-aged) emissions, diluted to ~ 1 ppm SO ₂	Primary stack emissions
PO	Primary + oxidized	Primary emissions + •OH	Aged plume, oxidized stack emissions, sulfate aerosol formation
POS	Primary + oxidized + SOA	Primary emissions + •OH + α-pinene/ozone	Aged plume, unneutralized acidity, secondary organic aerosol (SOA) derived from biogenic emissions
PONS	Primary + oxidized + neutralized + SOA	Primary emissions + •OH + NH ₃ + α-pinene/ozone	Aged plume, mixture of neutralized sulfate and SOA
O	Oxidized	Primary emissions + •OH, <u>no</u> primary PM	Control scenario
S	SOA	α-pinene/ozone only	Control scenario
OS	Oxidized + SOA	Primary emissions + •OH + α-pinene/ozone, <u>no</u> primary PM	Control scenario

Plus sham (control animals exposed to air only)

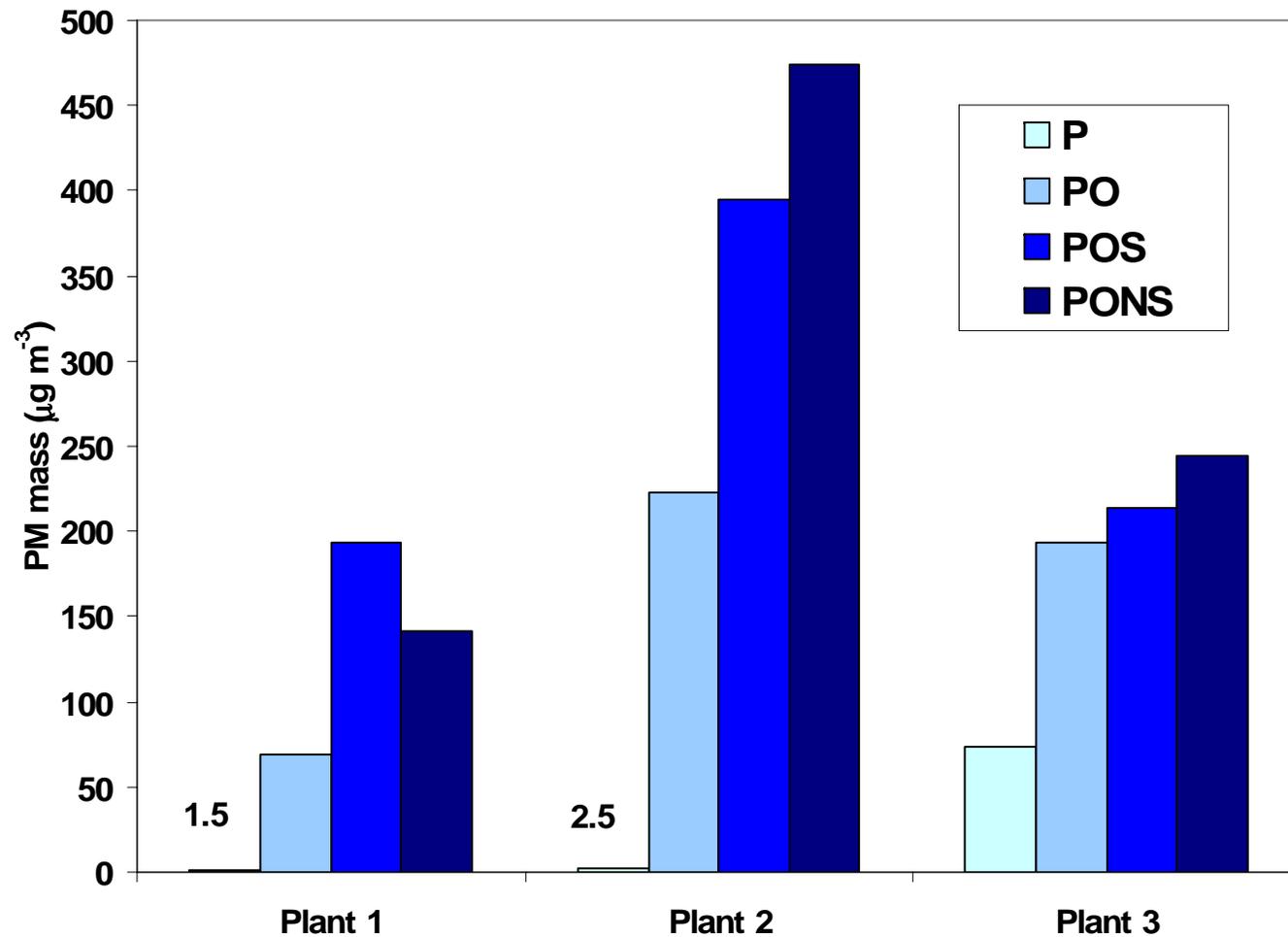
Exposures Performed

Code	Dates (2004) Plant 1	Dates (2005) Plant 2	Dates (2006) Plant 3	Animals Studied
P	May 10-13	June 6-9	August 8-13	Normal
PO	November 13-15	May 9-12	September 19-22	Normal
POS	October 4-7	March 21-24 (no SCR) May 3-6 (SCR)	July 19-22 August 14-15	Normal
PONS	June 22-26 June 27-30 October 11-14	May 31-June 3	July 25-28	Normal
POS	-	July 8, 13 September 8, 9	August 16-17	Compromised
OS	-	-	August 28-31	Normal
O	-	-	September 1-4	Normal
S	-	-	September 6-9	Normal

Total: 78 exposure days

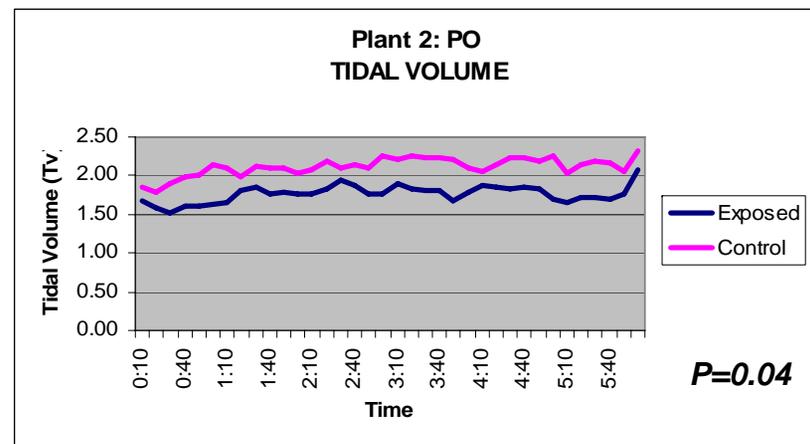
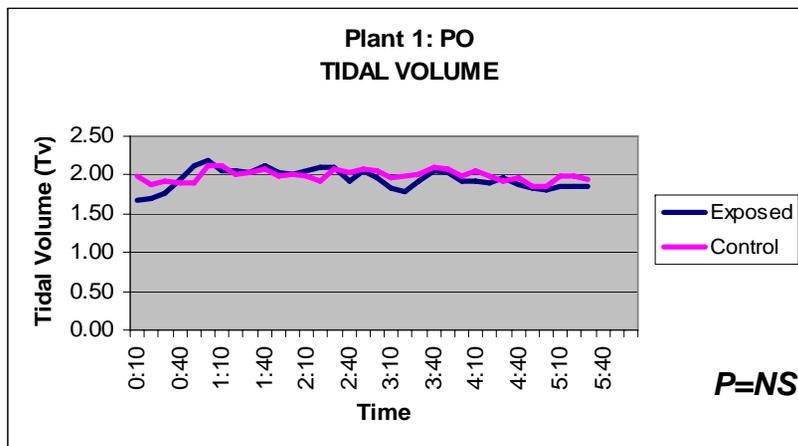
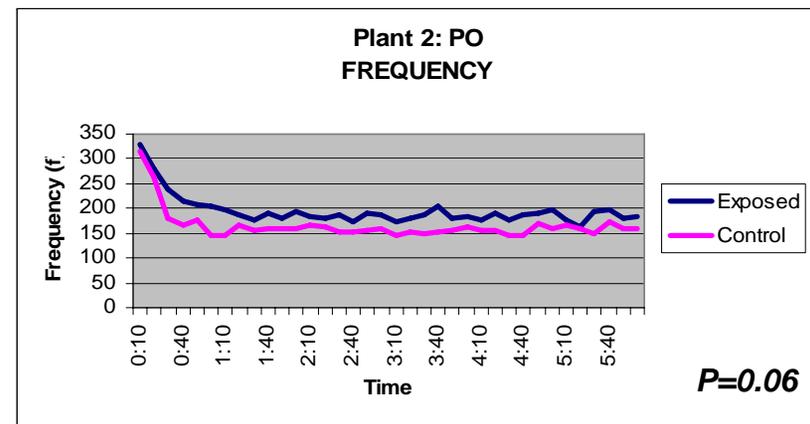
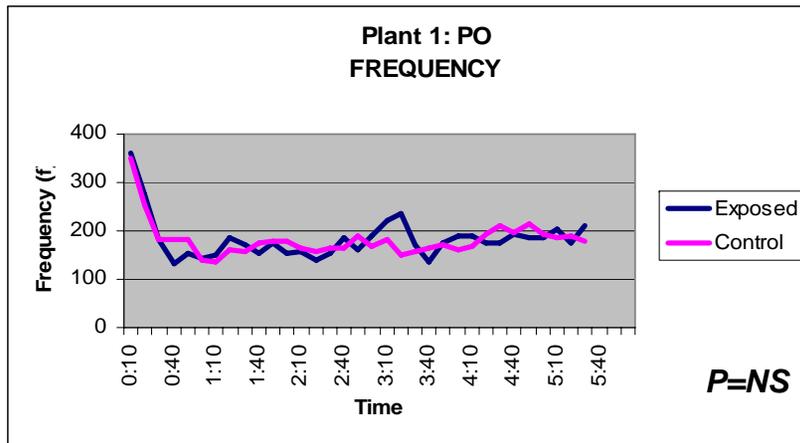
12 rats (6 exposed; 6 filtered air control) per exposure

Summary of Integrated Mass Concentrations



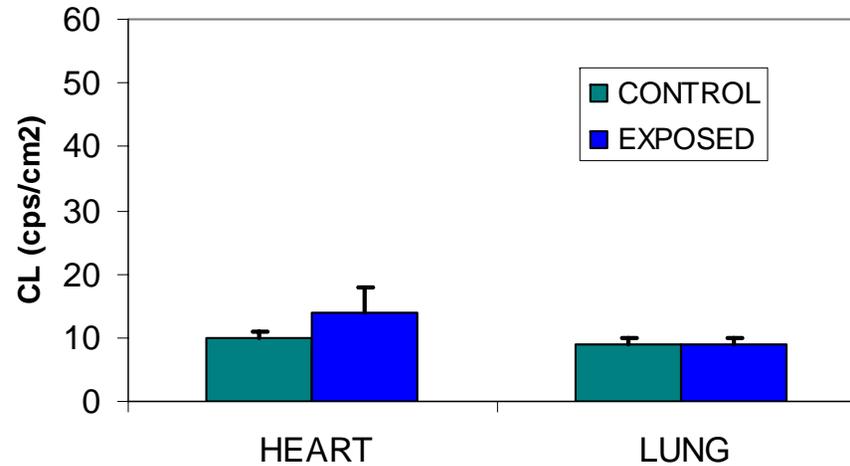
Breathing Pattern: Plants 1 and 2

Total: 48 exposures

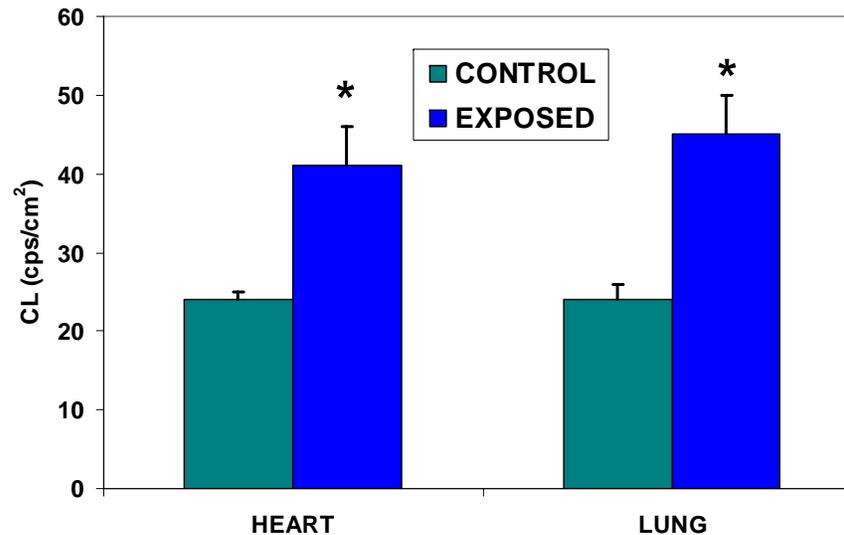


Plant 1: Oxidative Stress in Heart and Lung Tissue

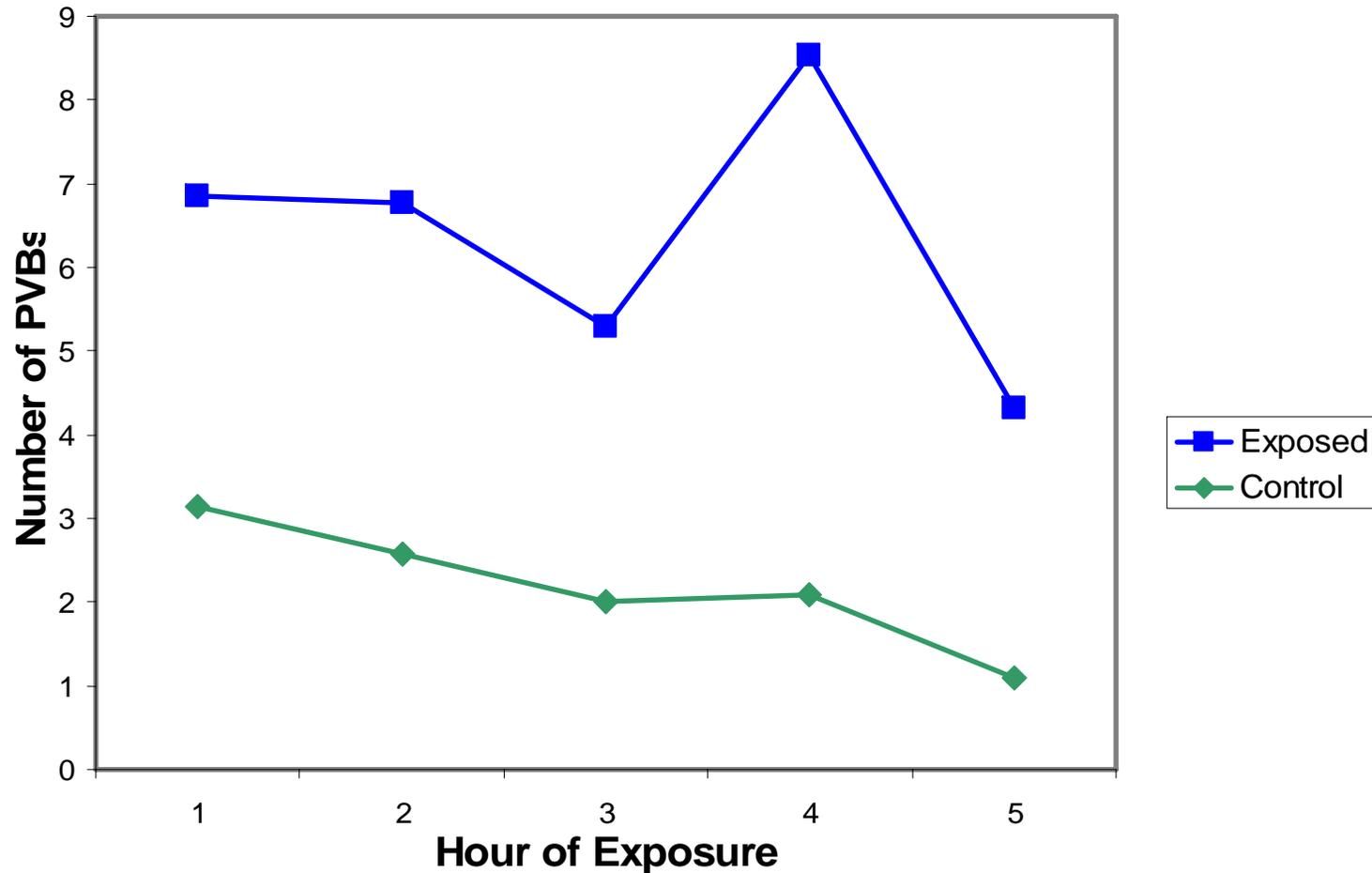
POS
(*n*=8 in each group)



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

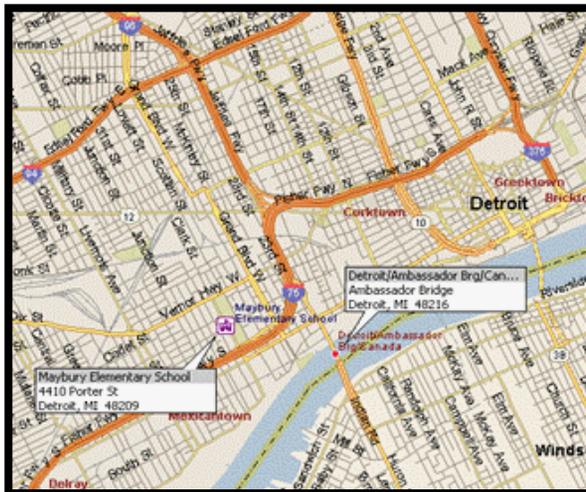
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Location of Study Sites

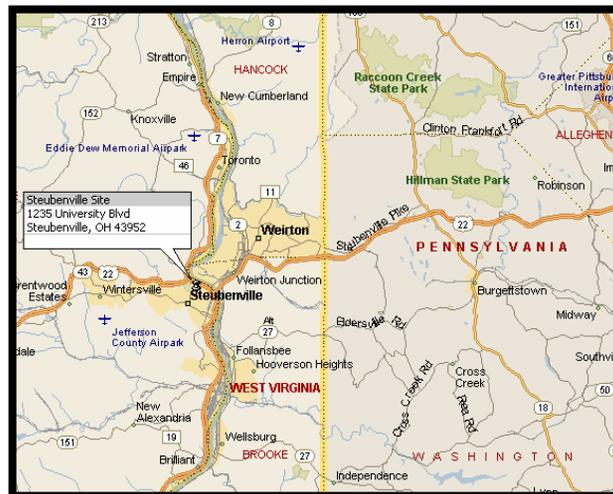
Downtown Detroit, MI

Dominated by diesel and gasoline emission-derived PM



Steubenville, OH

Dominated by power plant and local industrial emissions



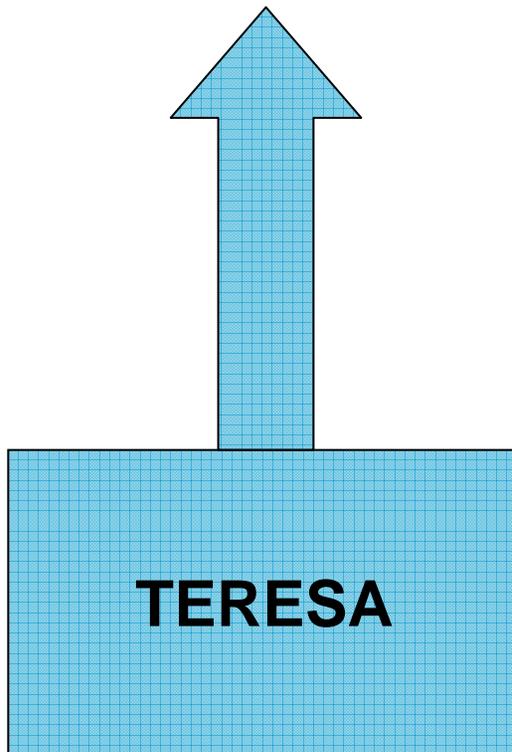
State Park in NW PA

Rural site; dominated by power plant emissions



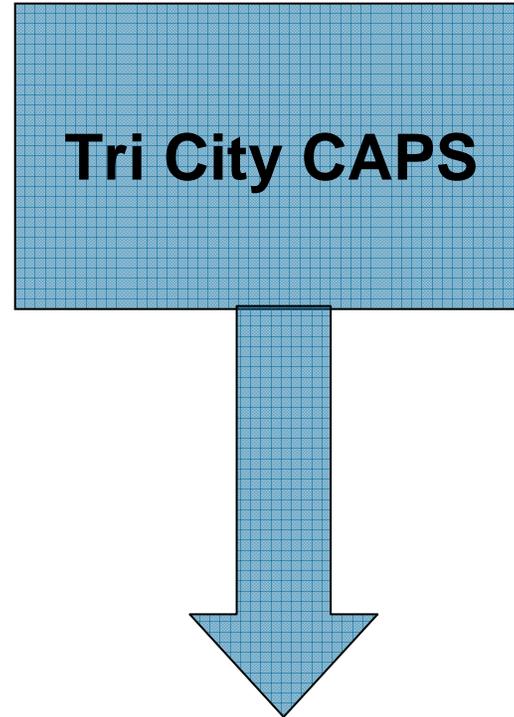
Complementary Approach to TERESA

Bottom-up approach
(start with controlled
sources)

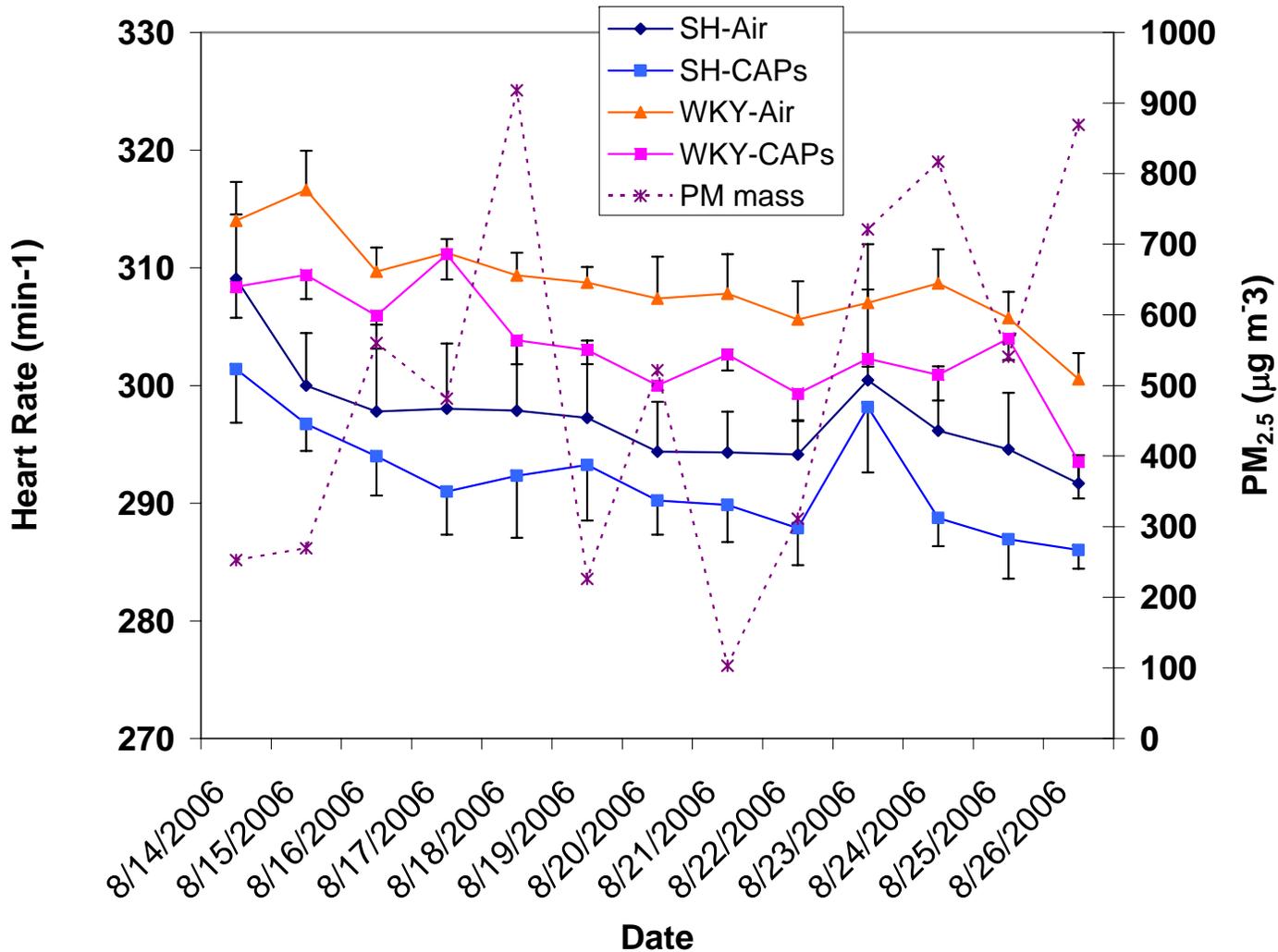


Tri City CAPS

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