

Addressing Uncertainties in PM Epidemiological Studies

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1997: Top 9 Reasons Not to Regulate PM2.5

- Time Series Associations confounded
- Exposure uncorrelated with ambient
- All Harvesting
- Thresholds
- No Mechanism/Biological Plausibility
- Only due to Some Particles, will Regulate Wrong Ones
- Don't know who is Susceptible
- Only 2 Cohort Studies/Faked
- Don't know if lower PM2.5 means fewer deaths

Confounding in Time Series

- Case-Crossover/Matching
- Exposure Studies
- Hierarchical Modeling Approach
- NMMAPS/APHEA

Case-Crossover Analysis

- Match each death with control day nearby in time
 - Schwartz and Bateson (1999,2001) showed how to choose so can control for Season
 - Lumley (2000) showed how to choose so avoid Selection Bias
 - Can Match on Same Concentration of Other Pollutant or Temperature

14 Cities with Daily PM10

- Controls Matched on Temperature
 - 0.39% (0.19—0.58) Increase per 10 $\mu\text{g}/\text{m}^3$ PM10 (Schwartz, OEM 2004)
- Controls Matched on Other Pollutants:

• CO	0.53%	[0.04, 1.02]
• O ₃	0.45%	[0.12, 0.78]
• NO ₂	0.78%	[0.42, 1.15]
• SO ₂	0.81%	[0.47, 1.15]
- Schwartz, EHP 2004
- Two day mean gives larger effects
- Not confounded

Statistical Approaches to Exposure Error

- Two arguments about exposure
 - Ambient poor surrogate for personal exposure
 - Better measured pollutant will “steal” effect from worse measured pollutant
- Zeger et al (2000)
 - Stealing very unlikely
 - Bias is downward

Hierarchical Approach

- Suppose we fit Single Pollutant Models in many Cities
- Control for Confounding by Second Pollutant Across City in Meta-analysis
- Advantage-Reduces Effect of Measurement Error (Schwartz and Coull, Biostatistics 2003)

Application: Fine Vs Coarse Particles

Air Pollution and Daily Deaths in Six US Cities (1996)
Fine Particles not Coarse (large) ones

Particle Measure	Standard Estimate	Corrected Estimate
PM2.5	.0149 (.00197)	.0342 (.00287)
Coarse Mass	-.00206 (.00491)	-.0235 (.00616)

Application: Reanalyze NMMAPS

- 90 Cities

- Slightly larger effect of PM10 (less measurement error)

- Zeka and Schwartz, EHP in press

- <http://ehp.niehs.nih.gov/docs/2004/7286/abstract.html>

Exposure and Confounding

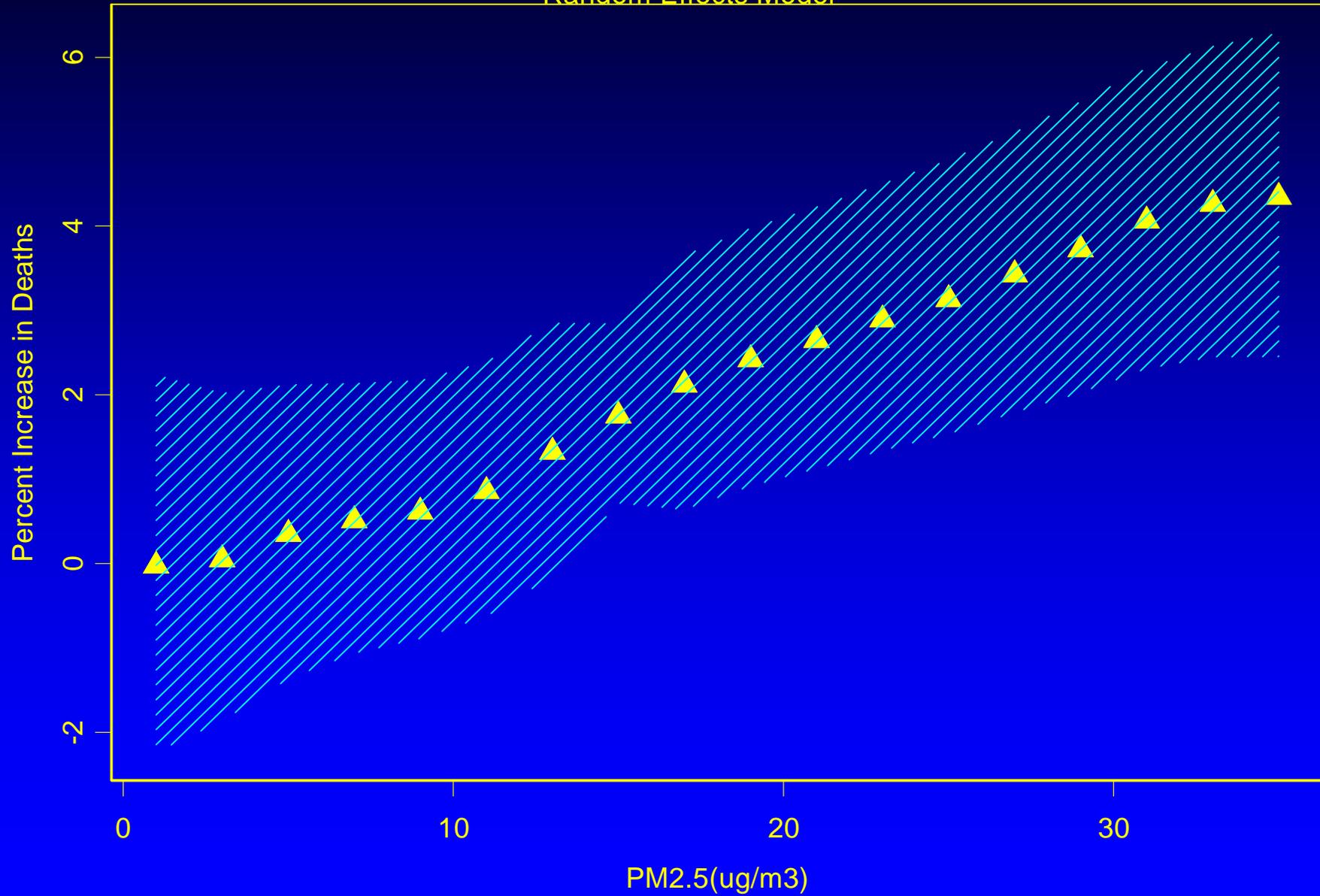
- In Baltimore and Boston
 - Ambient Ozone, NO₂, SO₂ are better predictors of Exposure to PM_{2.5} than of Exposure to themselves
 - NO₂ and CO better predict traffic particles
 - Ozone better predicts Sulfates
- Suggests in Eastern US two pollutant models are just source apportionment for PM effects, and need personal monitoring to study gases

Threshold?

- Combine data across multi-city studies

Dose Response between PM2.5 and Daily Deaths

Random Effects Model

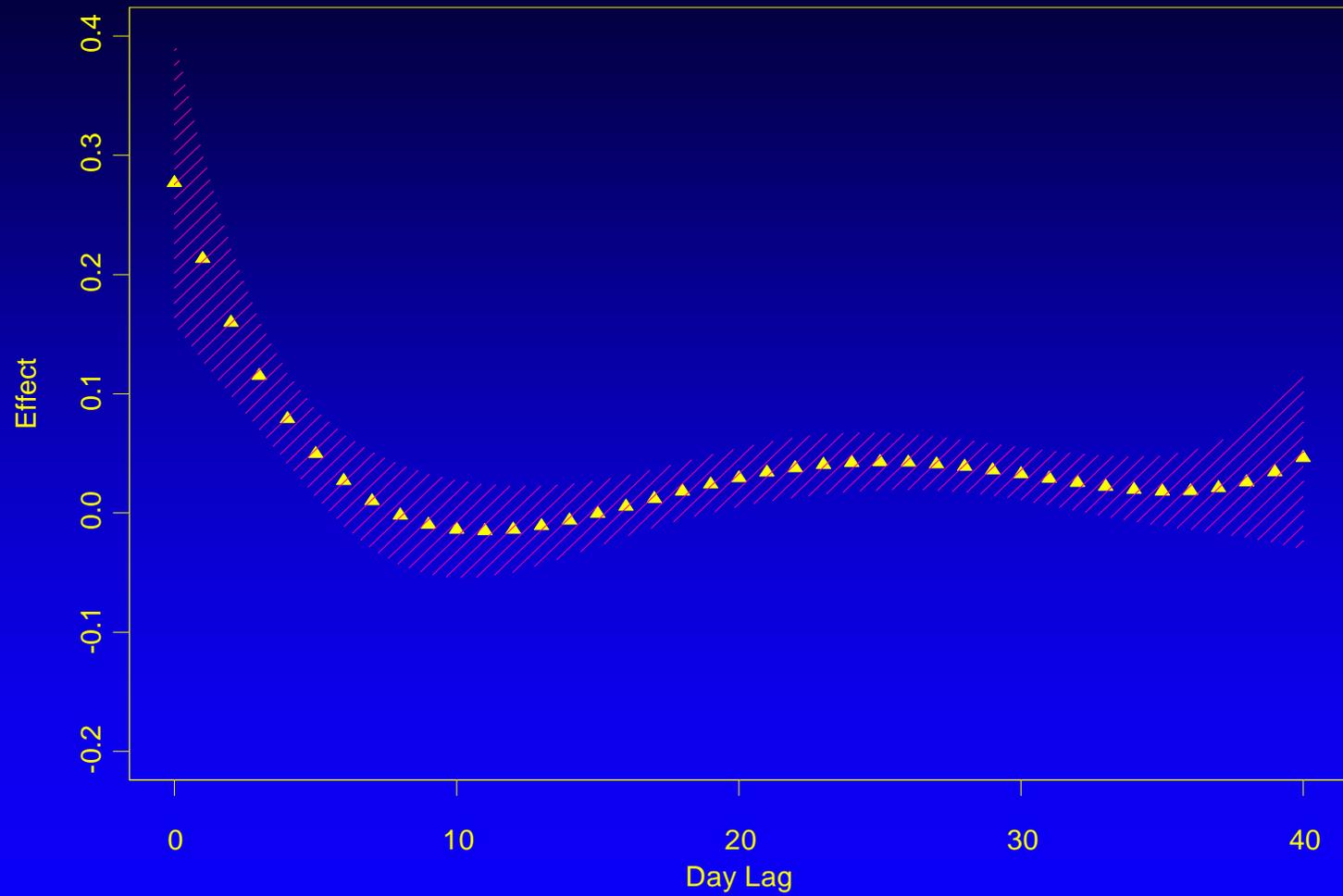


Harvesting?

Combine over 10 European Cities

Look at effects out to 40 days

4 Degree Distributed Lag in 10 Cities - Random Effect Model

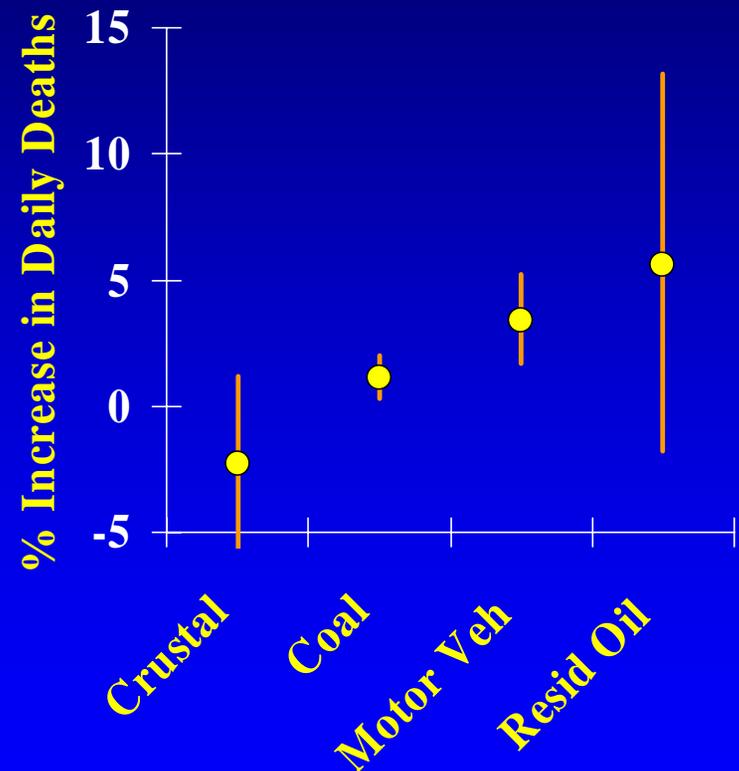


Only Some Sources produce Toxic Particles?

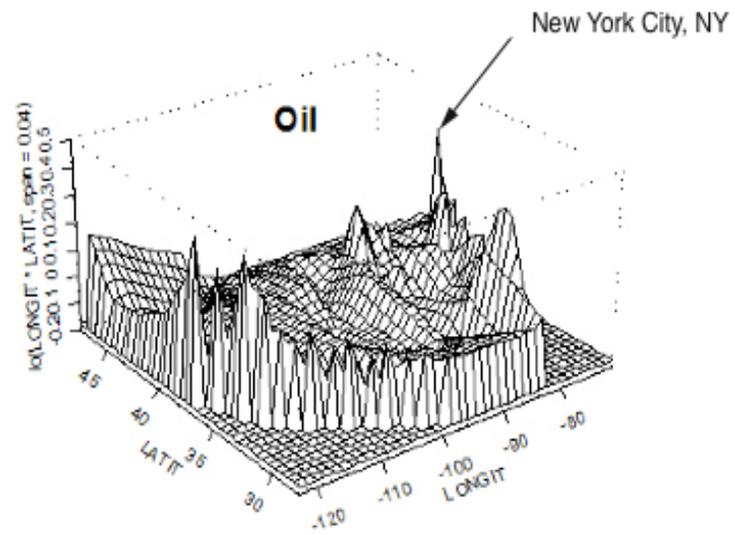
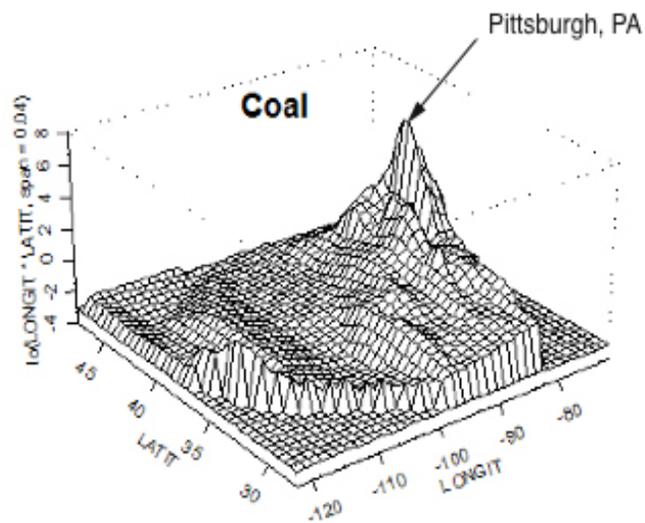
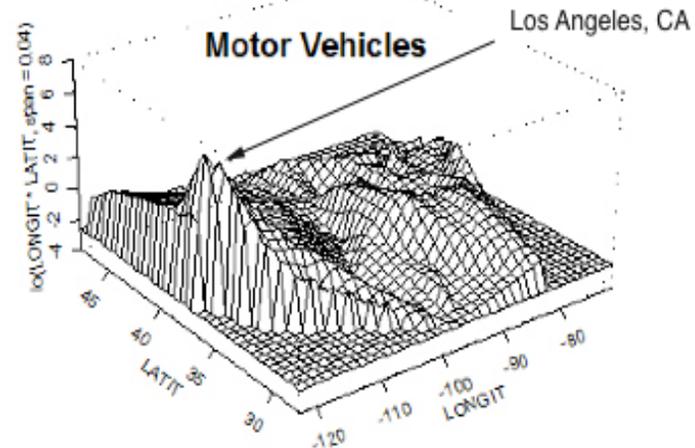
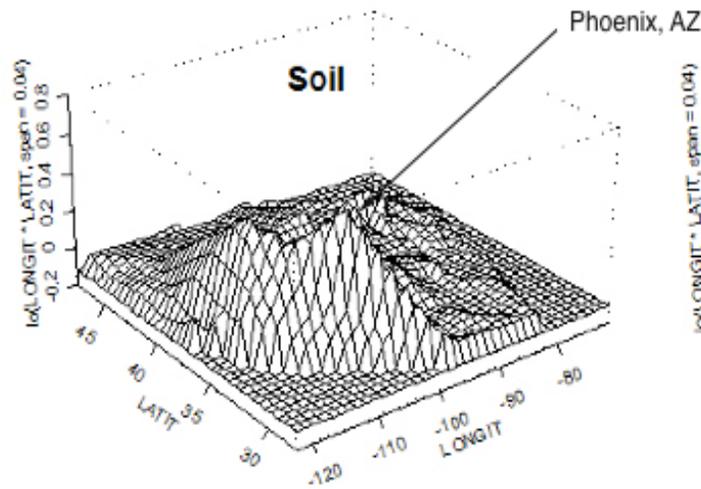
Laden et al, EHP 2000

Traffic, Coal and Residual Oil particles all toxic

Seattle Studies → Wood Smoke Toxic



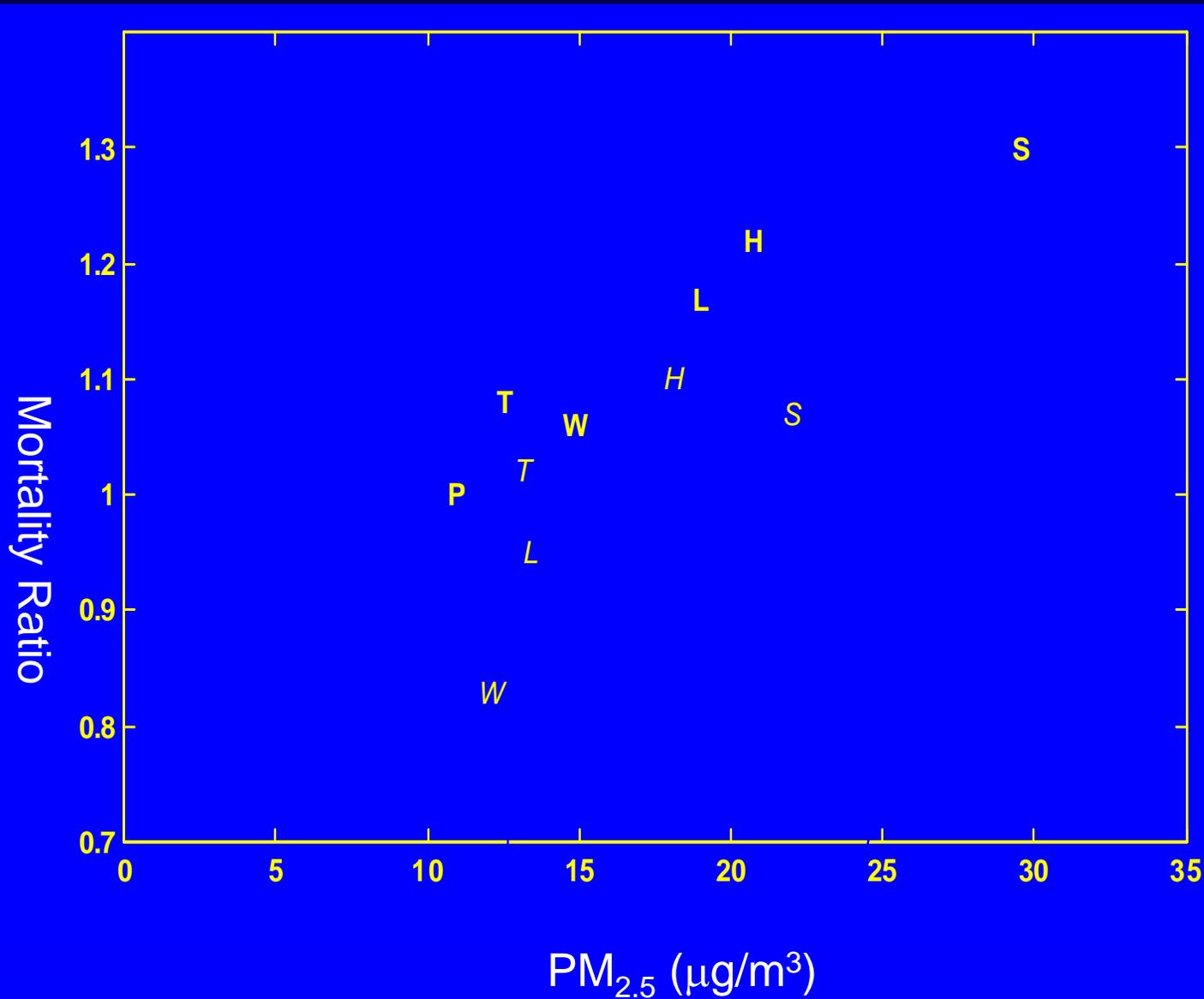
Spatial U.S. Variability of PM_{2.5} Factors



Cohort Studies

- Reanalyzed and found to be Robust (HEI)
- New Cohort in Netherlands finds effects of traffic particles on mortality
- Children's Health Study finds Air Pollutants (including particles) impair Lung Growth in Children

Mortality and Air Pollution in 6 US Cities in 2 Followup Periods



So Where Are We?

Time Series Confounding? ✓	Exposure Uncorrelated with Ambient? ✓ ?	Harvesting? ✓
Thresholds? ✓	Biologically Plausible? ✓ ?	Which Particles? ✓ ?
Susceptibility? ✓ ?	Cohort Studies? ✓ ?	Only Correlation? ✓

Future Epidemiologic Studies

- Susceptibility
 - New groups (Pregnancy Outcomes, Diabetics, etc)
- Mechanisms of Toxicity
 - Use drugs, etc to test pathways
- Separate out Different Sources/Characteristics of Particles
- Chronic Effects (sources, pathways, etc)