Public Health Implications of Ammonia Emissions

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Outline of talk

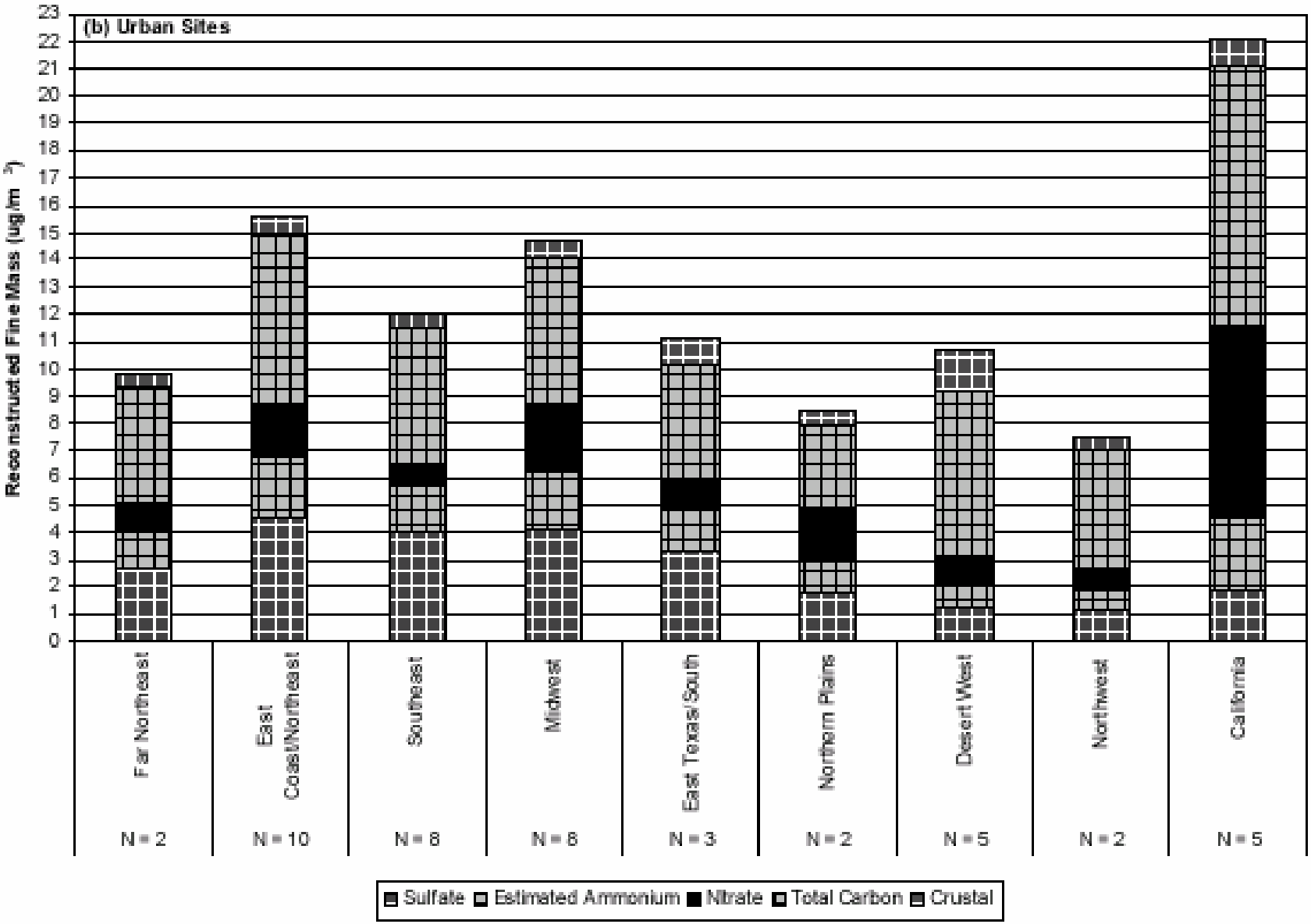
- Ammonia health effects (direct)
- PM health effects (indirect)
  - General evidence
  - Potential for differential toxicity
- Influence of ammonia concentrations/controls on PM-related health impacts
- Conclusions

Ammonia toxicity (IRIS)

- No oral RfD, carcinogenicity assessment
- Inhalation RfC = 0.1 mg/m^3
  - Based on NOAEL of 9.2 ppm in study of respiratory effects in soda ash facility
  - Respiratory lesions seen in rats, with LOAEL of 1.9 mg/m^3
- Other health risks: burns, coughing, throat irritation at high concentrations (> 50 ppm)
  - Minimal effects expected at ambient concs

Role of ammonia in PM

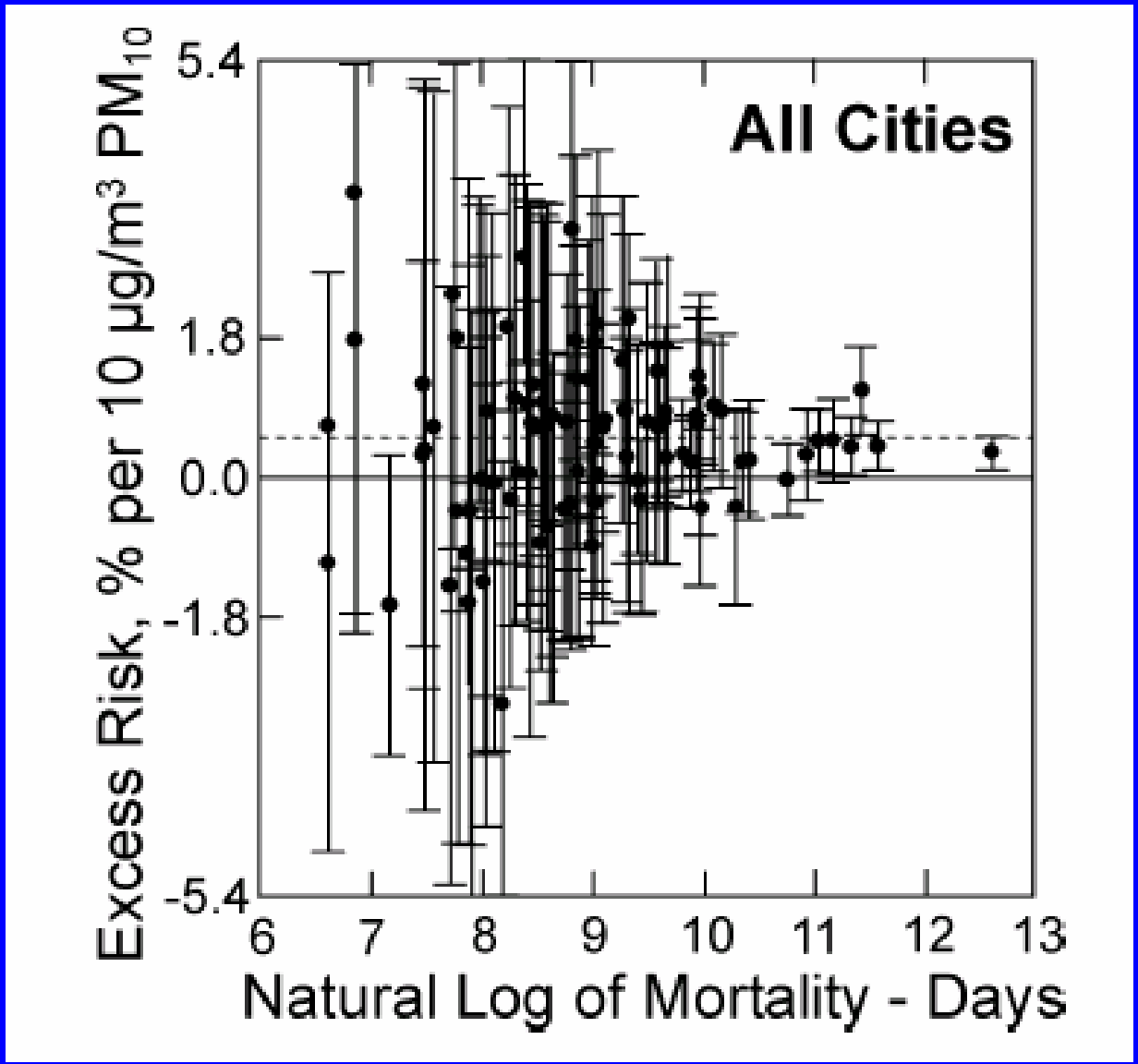
- Involved in formation of secondary particulate matter
  - Ammonium nitrate
    - Gas-phase nitric acid + gas-phase ammonia
  - Ammonium sulfate
    - Gas-phase sulfuric acid + gas-phase ammonia
- Will influence gas/particle balance, acidity and composition


(b) Urban Sites
Reconstructed Fine Mass (ug/m 3)
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
Far Northeast
N = 2
East Coast/Northeast
N = 10
Southeast
N = 8
Midwest
N = 8
East Texas/South
N = 3
Northern Plains
N = 2
Desert West
N = 5
Northwest
N = 2
California
N = 5
Sulfate
Estimated Ammonium
Nitrate
Total Carbon
Crustal

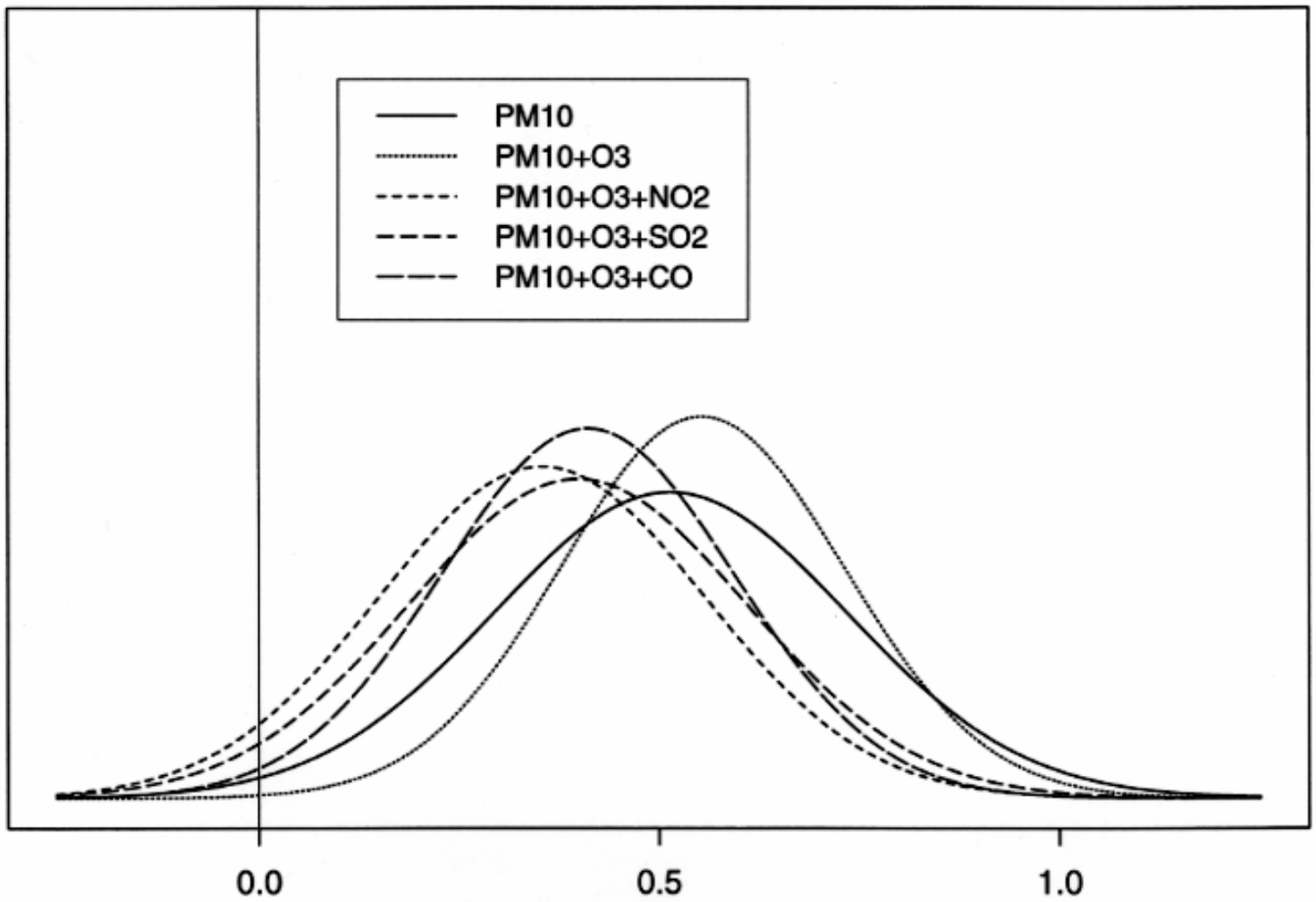
PM health effects - General

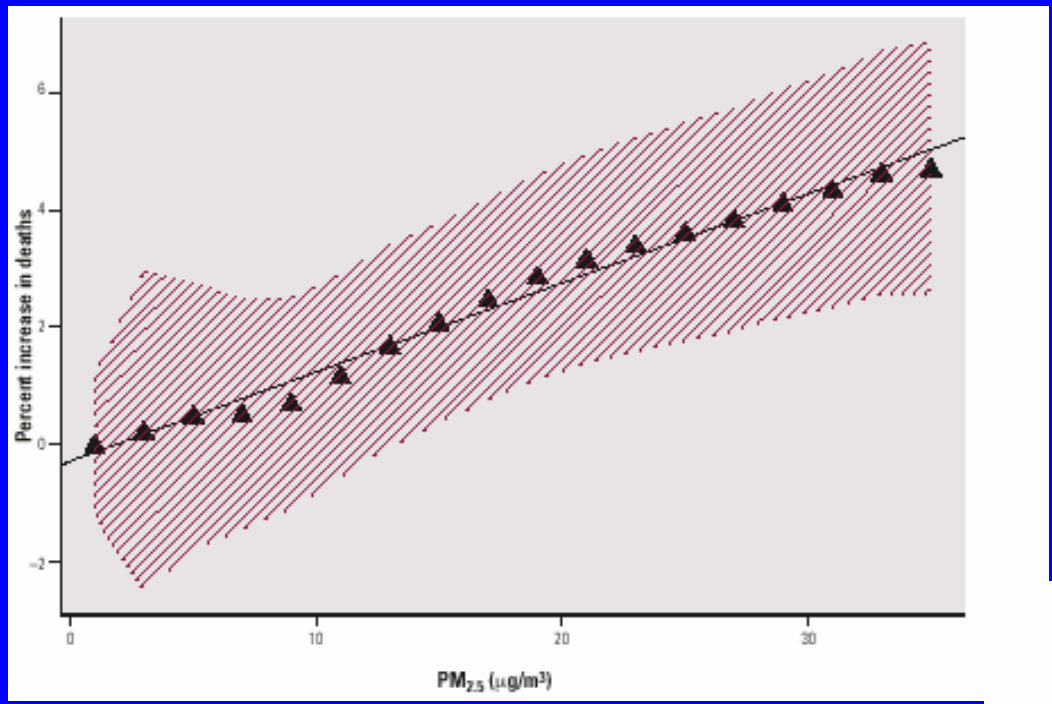
- Past studies have found acute and chronic mortality from PM to be the most significant health effects (from a valuation perspective)
  - Acute: Due to short-term exposure
  - Chronic: Due to long-term exposure
- Generally linked with $PM_{2.5}$
  - Ammonium sulfate and ammonium nitrate entirely in fine fraction
- C-R functions derived from epidemiology

Numerous acute studies

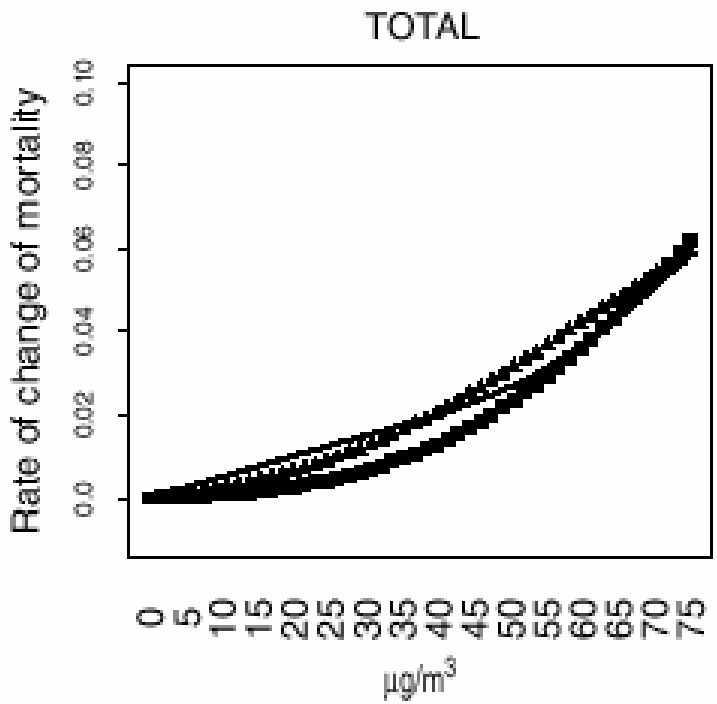


Effects independent of other pollutants

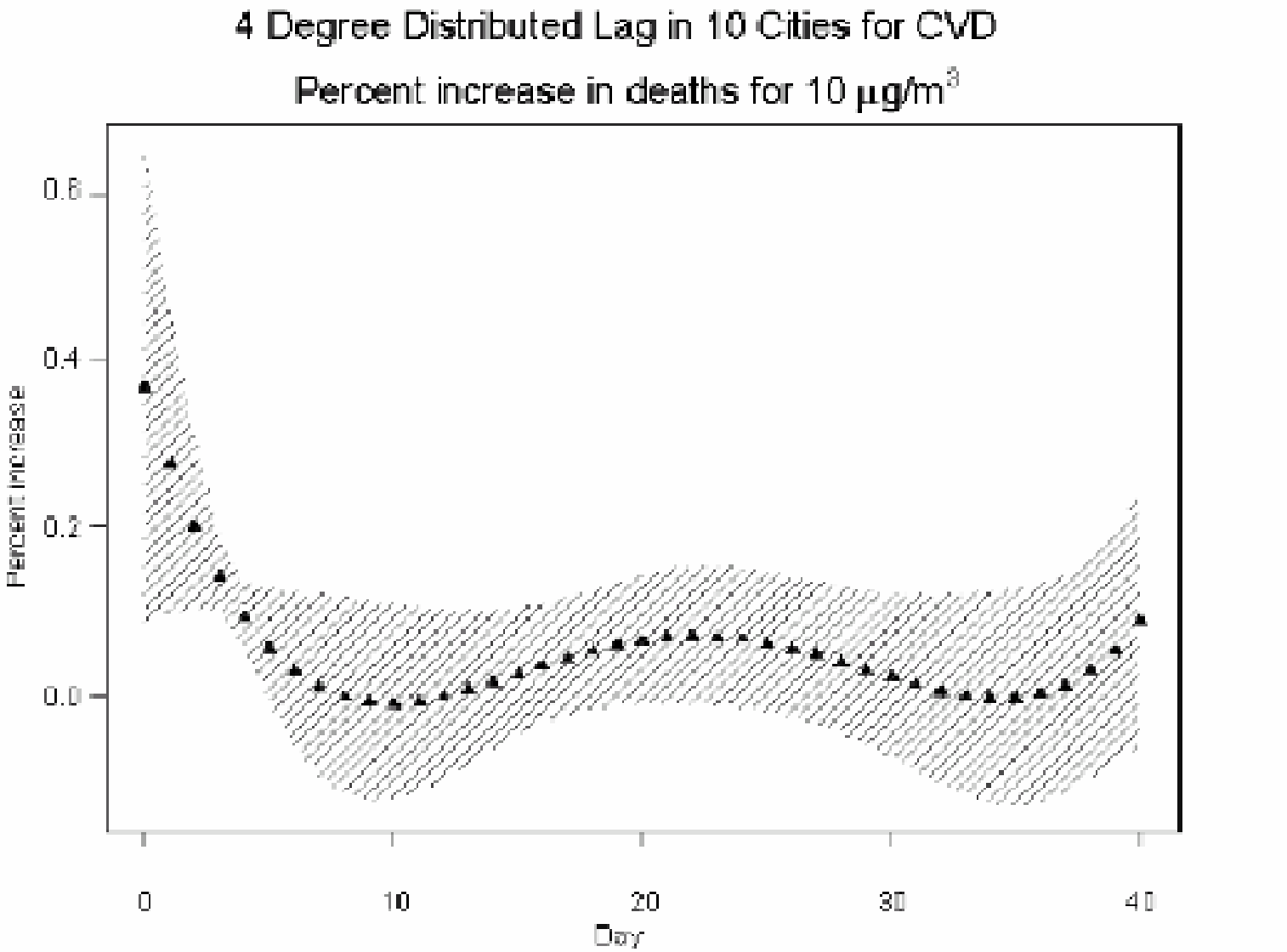




Little evidence of threshold



No evidence of harvesting



Evidence for short-term morbidity

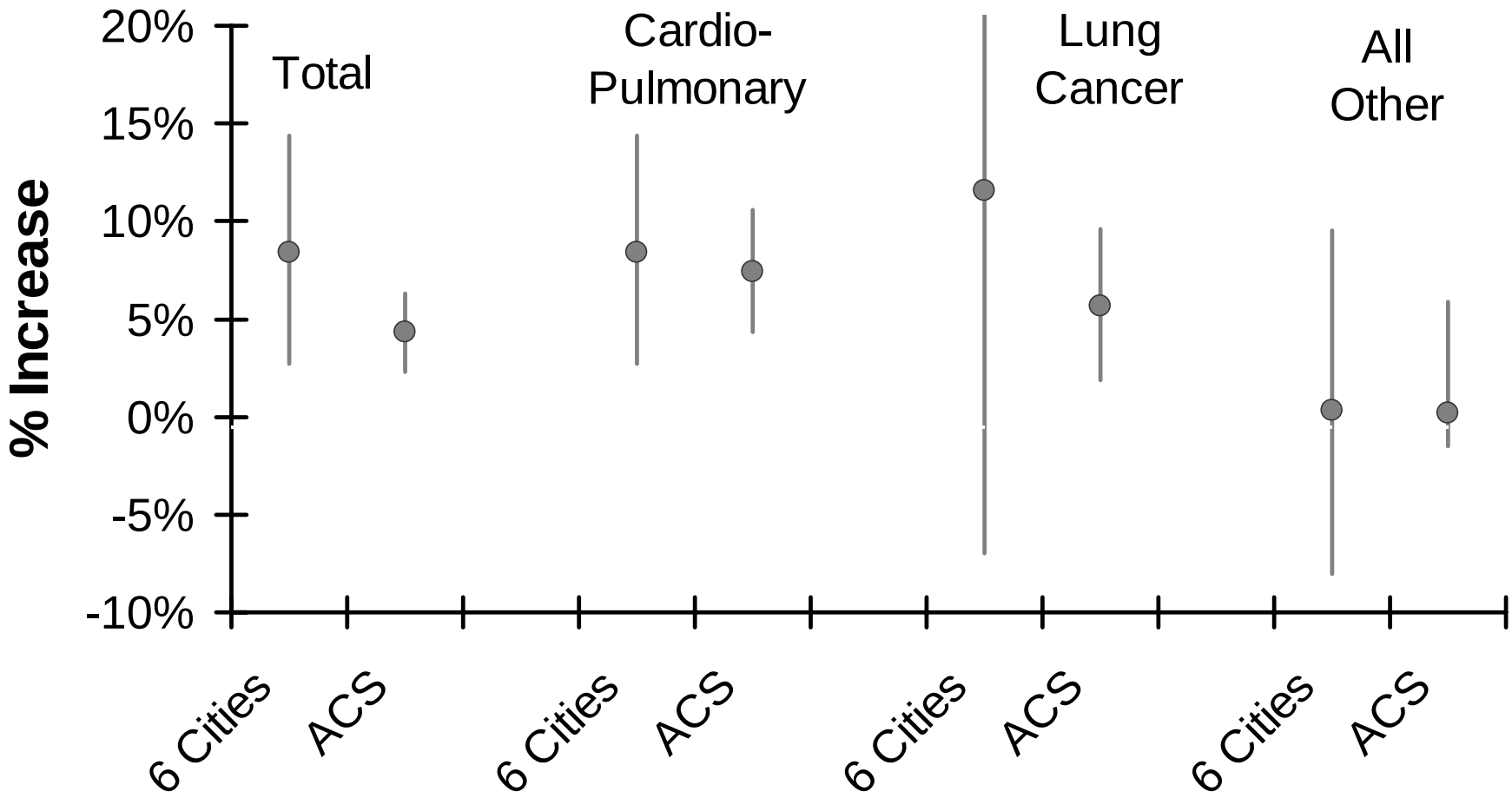
- Numerous health endpoints linked with PM in time-series studies
  - Cardiovascular and respiratory hospitalizations
  - Emergency room visits
  - Asthma attacks
  - Restricted activity days
  - Upper/lower respiratory symptoms

Cohort mortality studies

- Follow a group of people over time and analyze deaths and air pollution levels after controlling for potential confounders such as smoking, education, obesity, and occupation
  - Different confounding concerns than time-series studies
- Many fewer cohort studies have been conducted due to time and expense involved
- Two U.S. studies are mainly referenced:
  - Six Cities & American Cancer Society

Increase in Lifetime Mortality Risk

Effect of 6.5 μ g/m^3 $PM_{2.5}$



Issue of differential toxicity

- $PM_{2.5}$ regulated on a mass basis, but increasing interest in whether toxicity differs by constituent
- When thinking of ammonia, primary concern is for ammonium sulfate (AS) and ammonium nitrate (AN)
  - Related question: does acidity matter?

Time-series evidence for AS

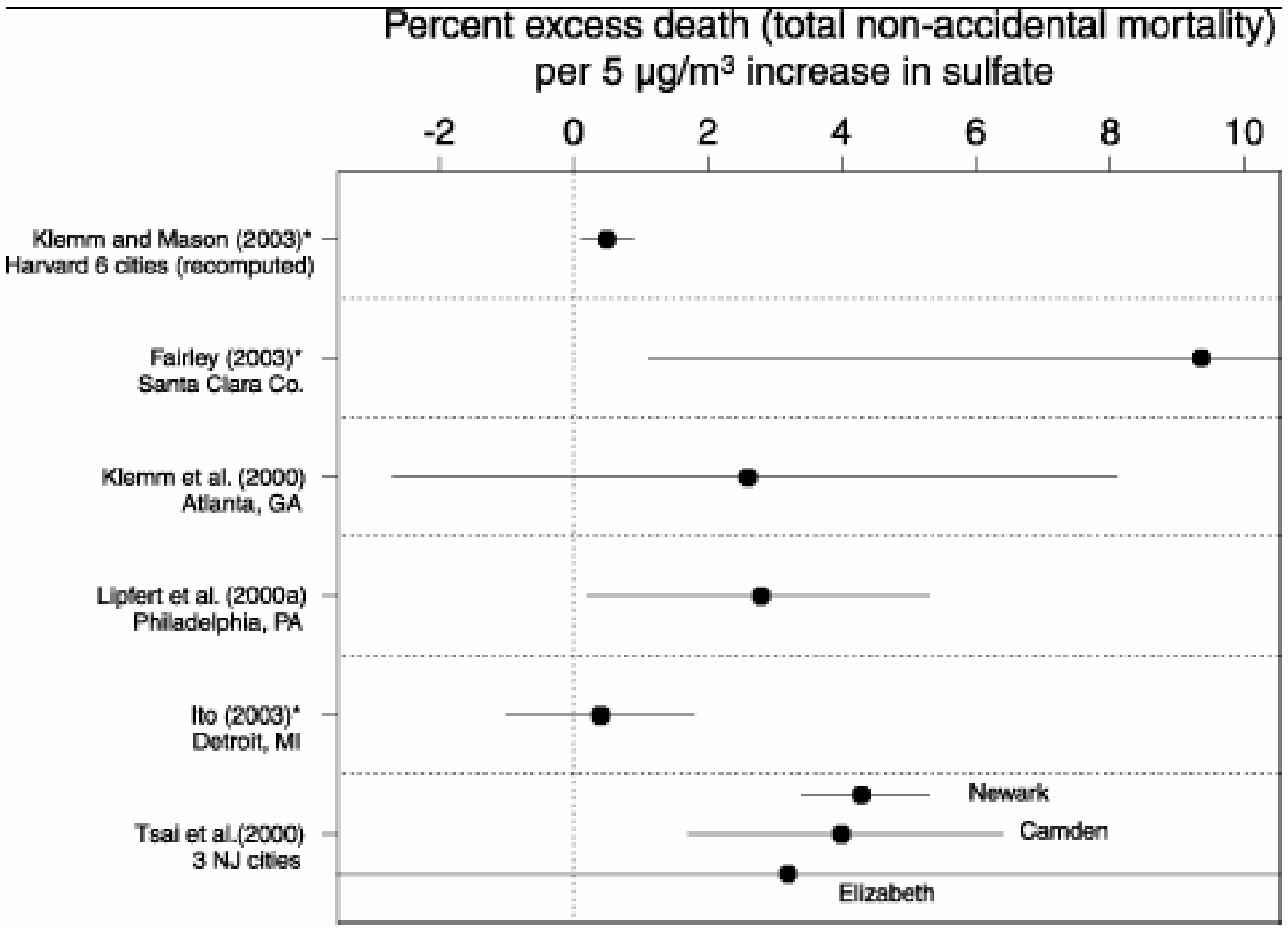


Figure 8-8. Excess risks estimated per 5 µg/m^3 increase in sulfate, based on the studies in which both $PM_{2.5}$ and $PM_{10-2.5}$ data were available.

Cohort evidence for AS

- Positively associated with mortality in Six Cities, ACS
  - HEI Reanalysis: $PM_{2.5}$, sulfate, and SO_2 all associated with mortality
- Insignificant in AHSMOG, but with central RR estimate for males between ACS and Six Cities values

Effects of ammonium nitrate

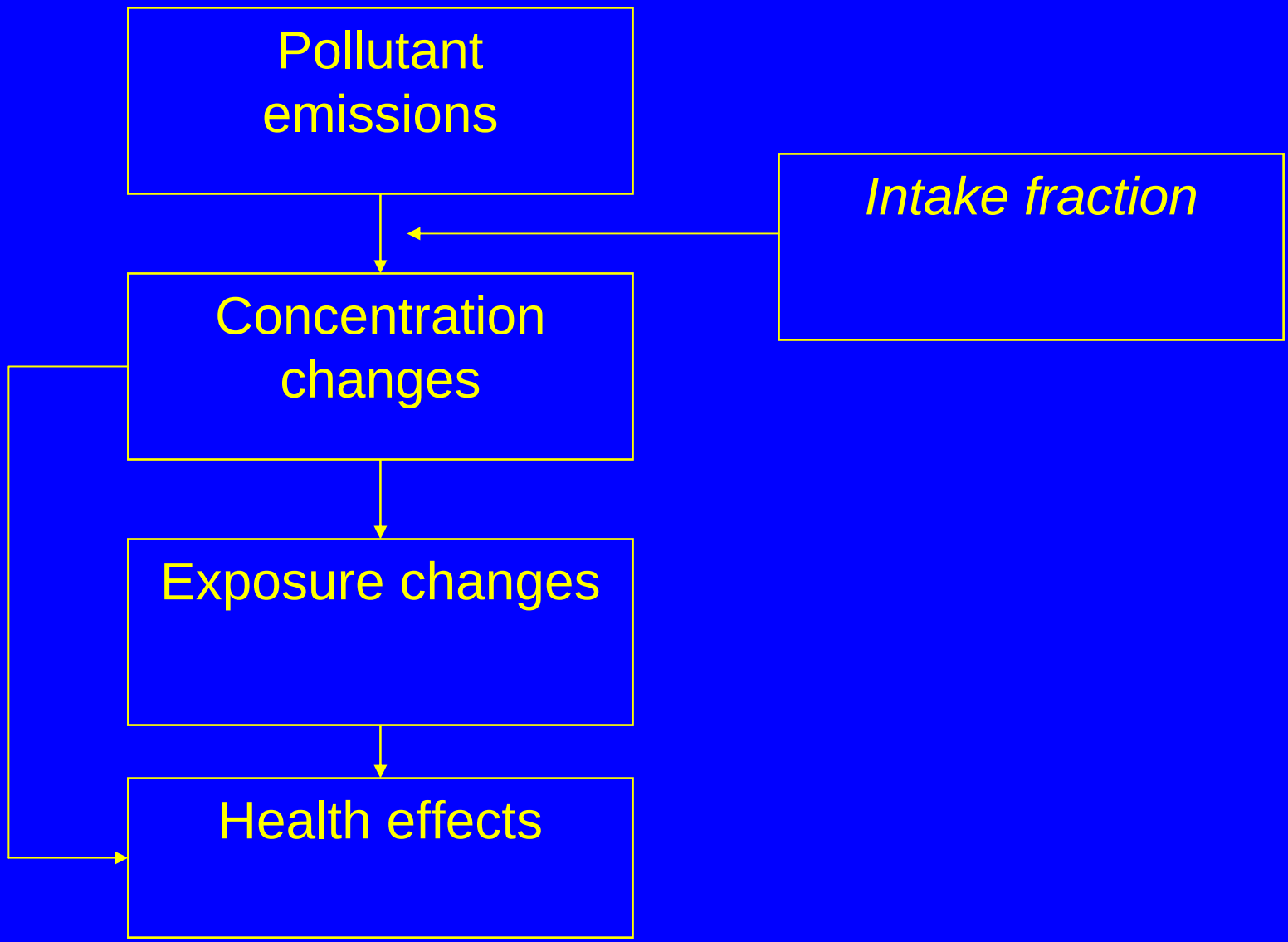
- Relative lack of daily ambient concentration data
- Significantly associated with mortality in CA and Netherlands; not significant in preliminary GA findings
- Differential toxicity question largely unanswered to date

Toxicological evidence

- Results mixed to date for most PM constituents
  - CAPs, ROFA have shown respiratory/cardiovascular effects in some (but not all studies)
    - Difficult to isolate influence of AN, AS
  - Acid aerosols have had limited effects in controlled experiments, but with artificial particle composition

Influence of ammonia controls on PM health effects

- Atmospheric chemistry of sulfate-nitrate-ammonia system already discussed in detail
- Key questions:
  - How do the health benefits of SO_2 or NOx control depend on ambient NH_3?
  - What are the implications of NH_3 control (from power plants) for population exposure to PM?
- Findings taken from:
  - Wilson AM, Hammitt JK, Levy JI. Reduced-form characterization of fine PM exposure due to US power plant emissions.


Pollutant emissions
Intake fraction
Concentration changes
Exposure changes
Health effects

What is intake fraction?

- Fraction of material or its precursor released from a source that is eventually inhaled or ingested
  - Dimensionless term
  - Also called exposure efficiency, dose fraction, etc.
  - Function of how the pollutant disperses in the atmosphere and where the population is located

Why calculate iF?

- Directly relevant to risk/benefit calculations
- Summarizes the total “exposure” per unit emissions from different sources/constituents, helping inform control decisions
- Extrapolation to other settings (useful when data limited, numerous sources)
- Supports consideration of model uncertainty in an appropriate framework for risk assessment

Estimating iF

- $\sum(Pop_i * Conc_i * BR)/Q$
- For a given source:
  - Atmospheric dispersion model used to estimate incremental concentration, *Conc*, at a number of locations *i* with affected populations, *Pop*
  - Breathing rate (BR) assumed constant
  - Emission rate (Q) of pollutant or precursor known and constant over defined averaging time

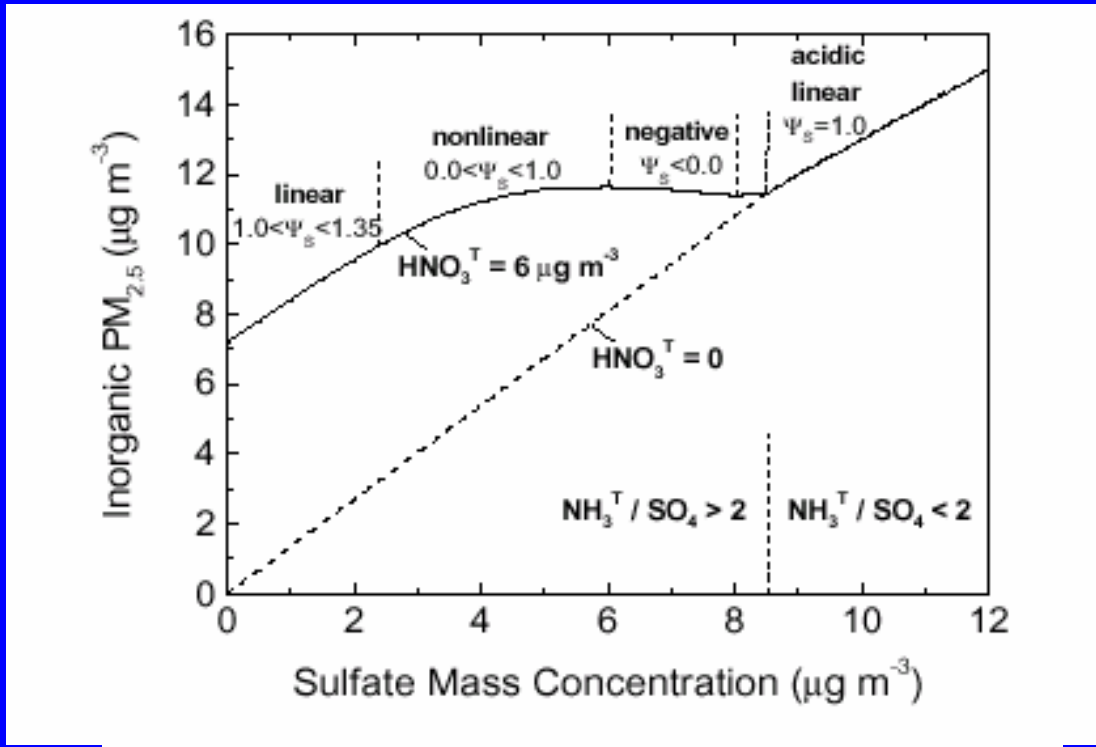
Some terminology

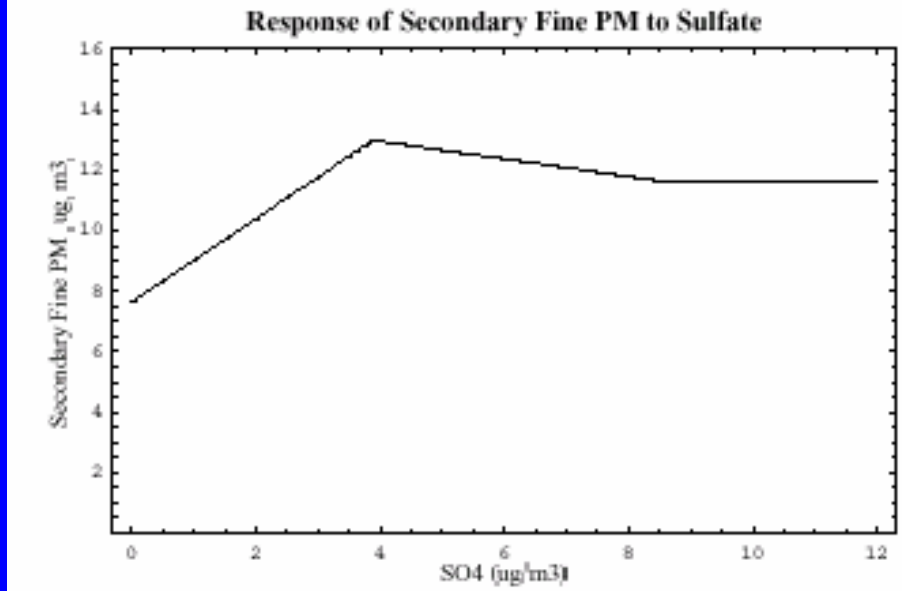
Abbreviation	Exposure pollutant	Emission pollutant
iF(p)	Primary fine PM	Primary fine PM
iF(as,SO_2)	Ammonium sulfate	Sulfur dioxide
iF(an,NOx)	Ammonium nitrate	Nitrogen oxides
iF(an,SO_2)	Ammonium nitrate	Sulfur dioxide
iF(an, NH_3)	Ammonium nitrate	Ammonia

Note: iF represents partial derivative which must be evaluated under specific conditions (e.g., SO_2, NOx, NH_3 concs)

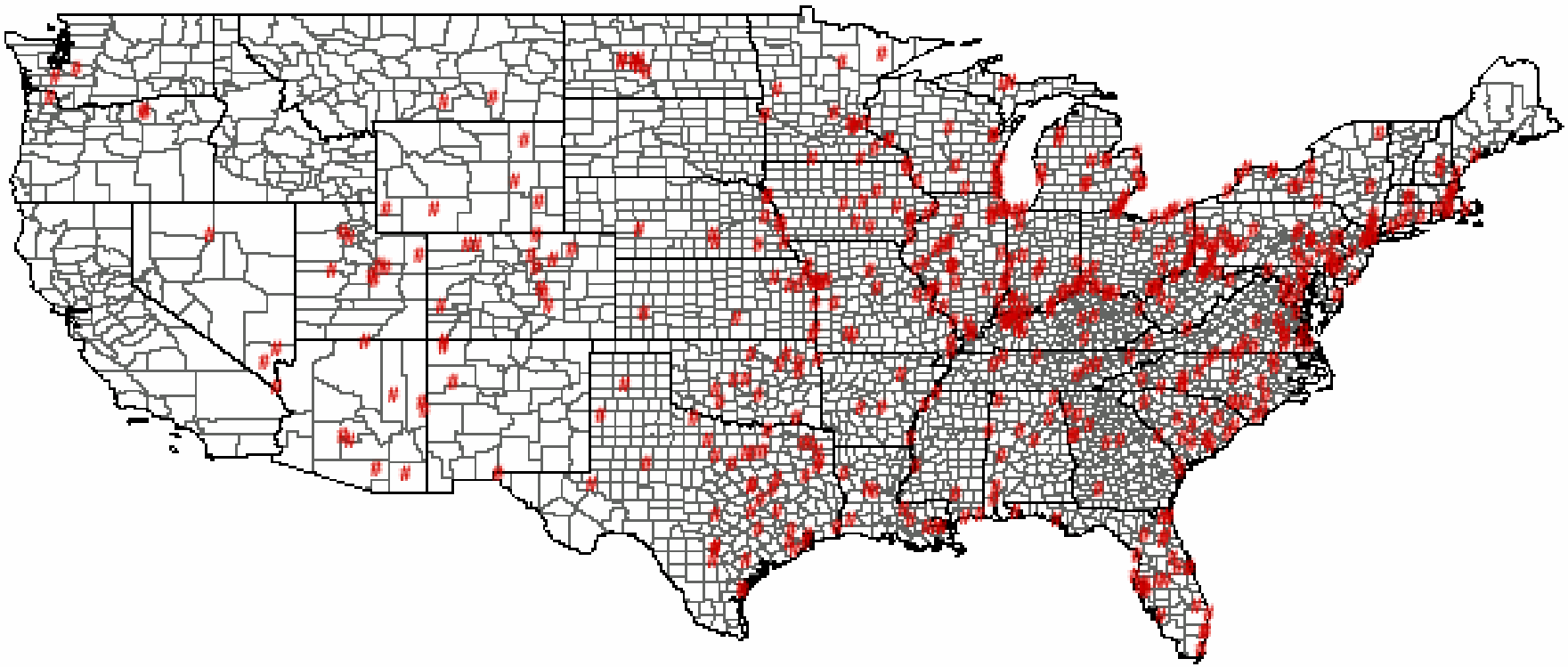
Dispersion model applied

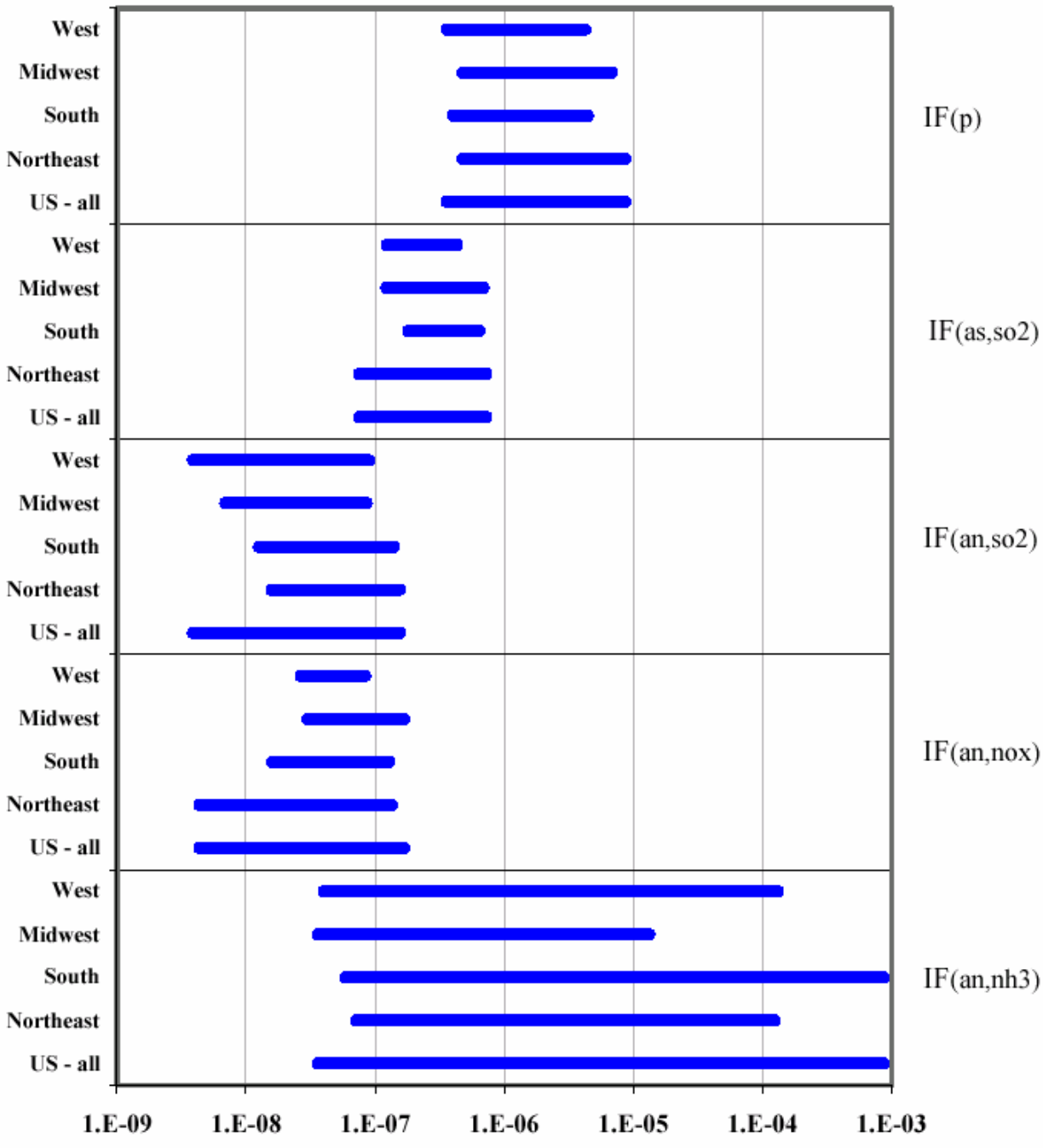
- S-R matrix
  - Simplified source-receptor matrix used in past regulatory impact analyses
  - Yields similar iF estimates as more complex models
  - Captures sulfate-nitrate-ammonia system reasonably (e.g., sulfate vs. PM curve looks similar to curve in West et al.)


Inorganic $PM_{2.5}$ ($\mu g\ m^{-3}$)
Sulfate Mass Concentration ($\mu g\ m^{-3}$)
linear
$1.0<\Psi_S<1.35$
nonlinear
$0.0<\Psi_S<1.0$
negative
$\Psi_S<0.0$
acidic
linear
$\Psi_S=1.0$
$HNO_3^T = 6\ \mu g\ m^{-3}$
$HNO_3^T = 0$
$NH_3^T / SO_4 > 2$
$NH_3^T / SO_4 < 2$


Response of Secondary Fine PM to Sulfate
Secondary Fine PM
SO4 (µg/m3)

507 power plants in S-R matrix




West
Midwest
South
Northeast
US - all
IF(p)
West
Midwest
South
Northeast
US - all
IF(as,so2)
West
Midwest
South
Northeast
US - all
IF(an,so2)
West
Midwest
South
Northeast
US - all
IF(an,nox)
West
Midwest
South
Northeast
US - all
IF(an,nh3)
1.E-09
1.E-08
1.E-07
1.E-06
1.E-05
1.E-04
1.E-03

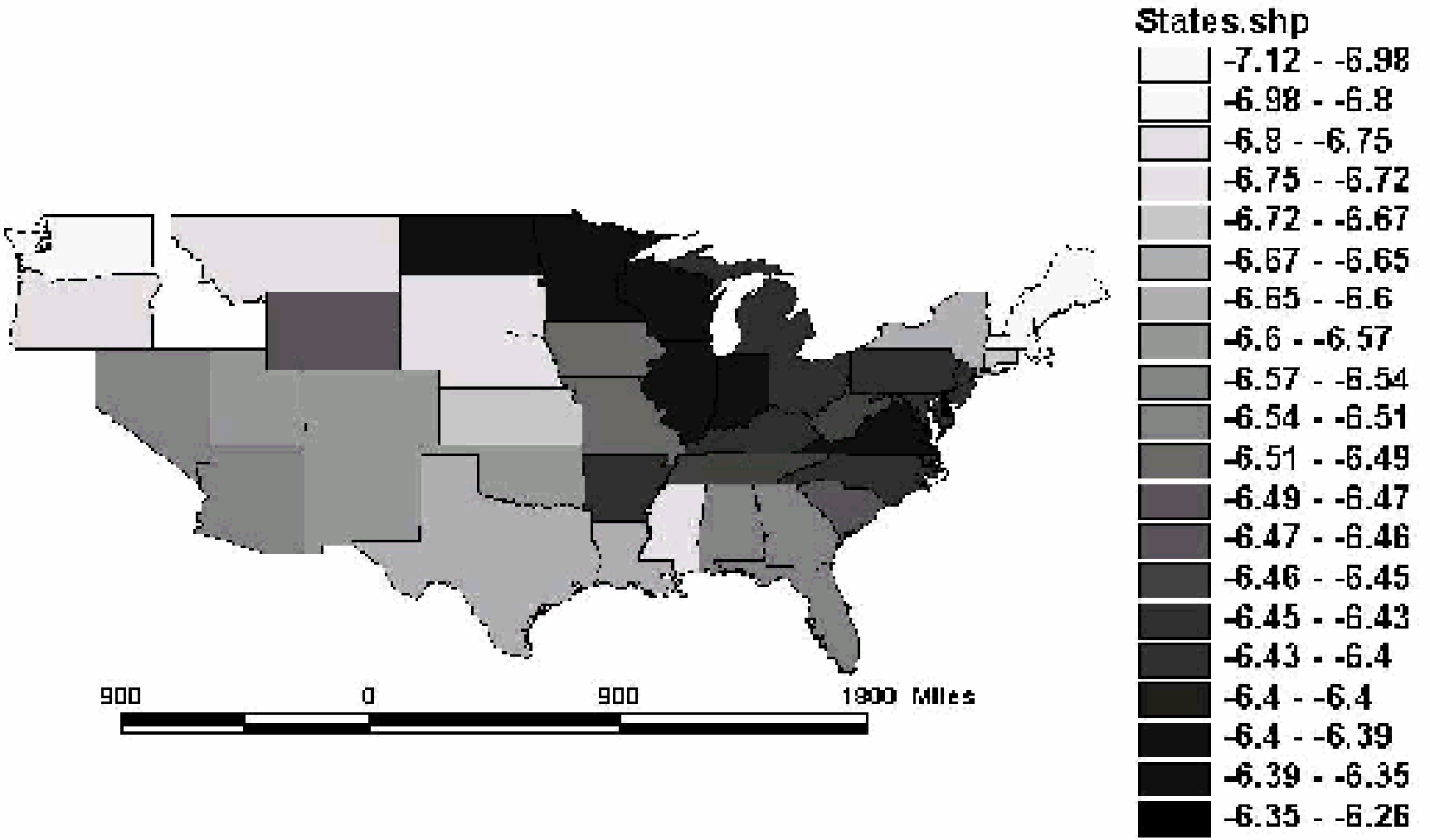


Figure 4. Map of mean iF(as,SO2) estimates for US power plants, by state (log10 scale).

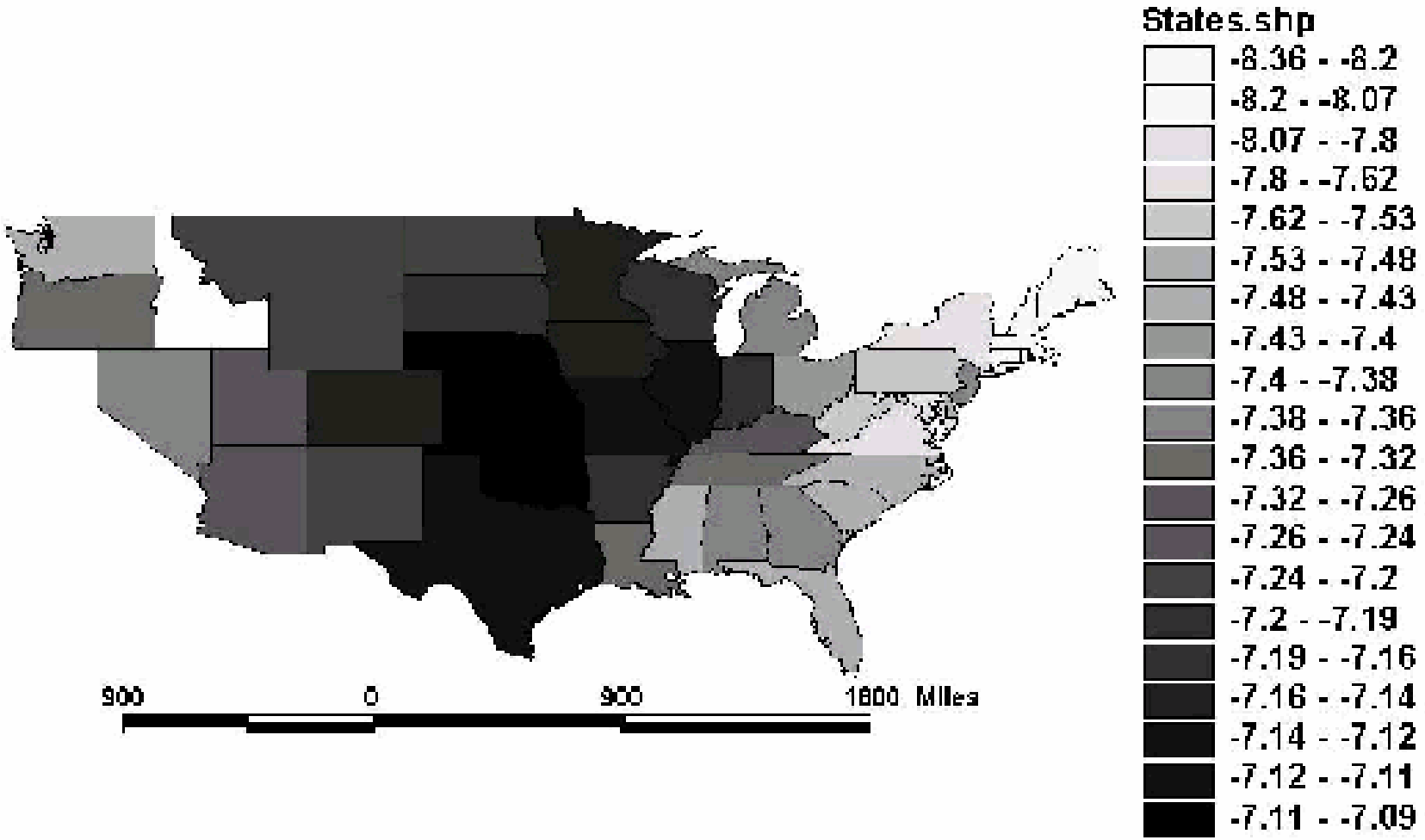


Figure 5. Map of mean iF(an,nox) estimates for US power plants, by state (log10 scale).

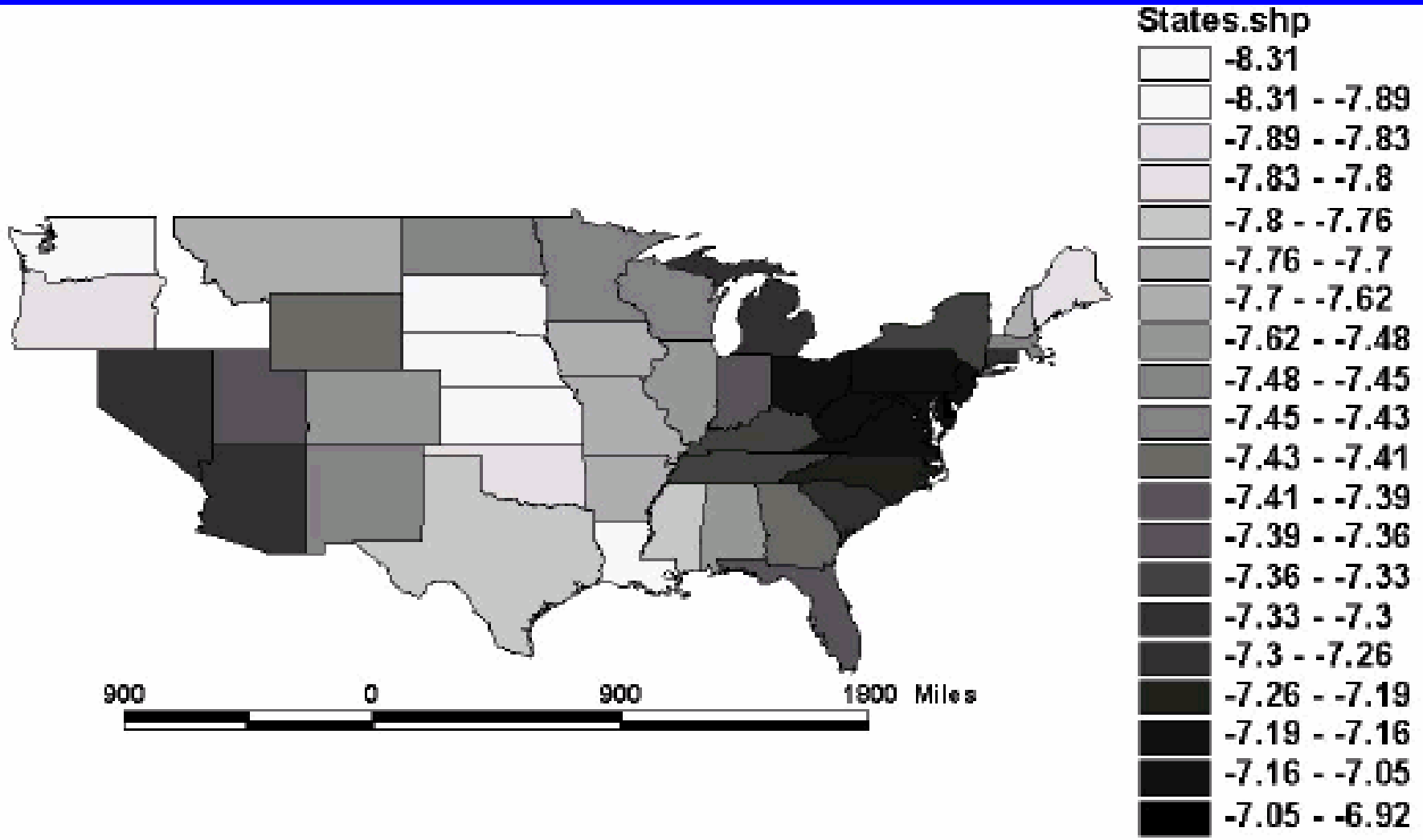


Figure 6. Map of mean iF(an,SO2) magnitudes for US power plants, by state (log10 scale, negative sign removed to take log).

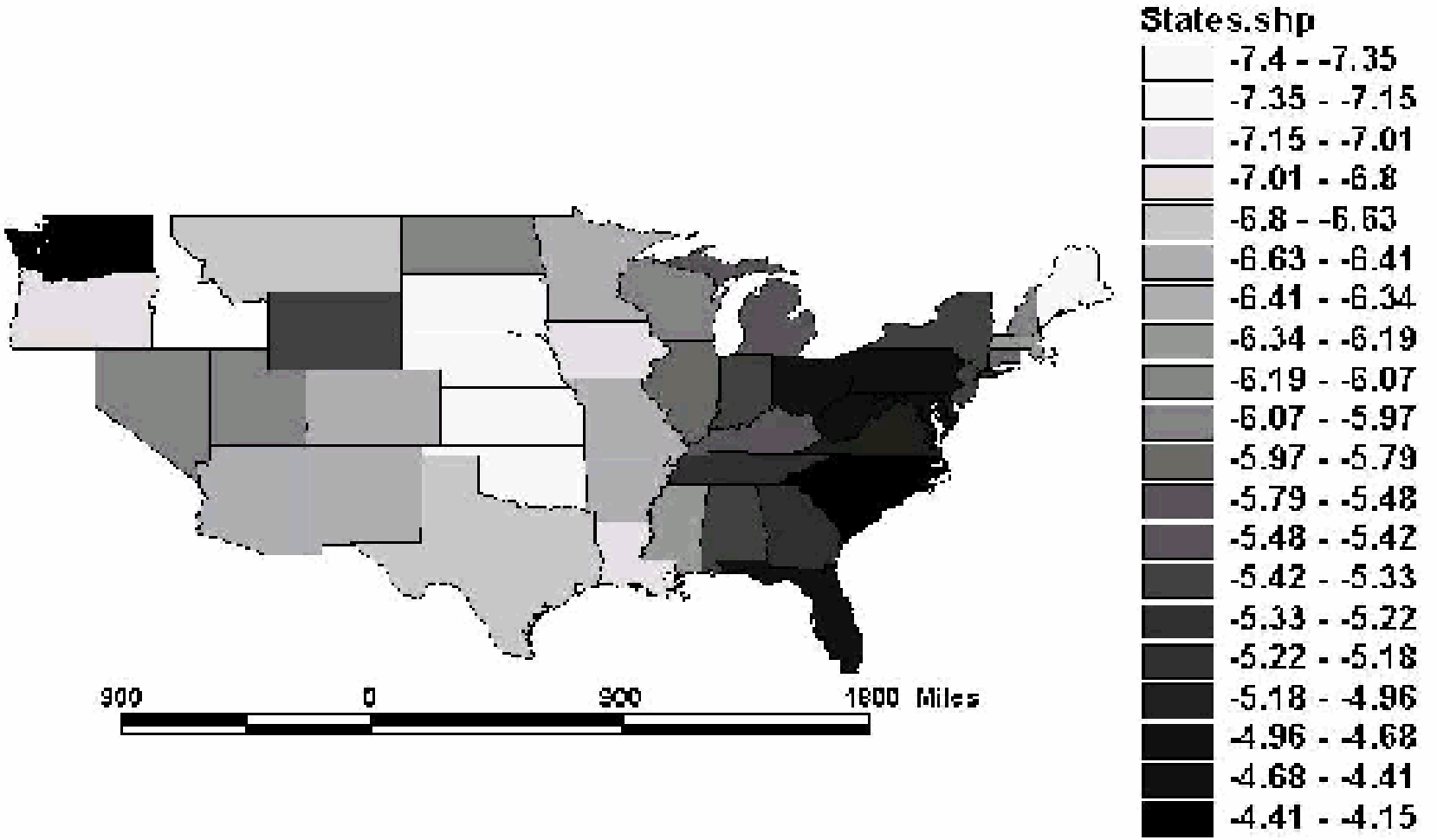


Figure 7. Map of mean iF(an,NH_3) estimates for US power plants, by state (log10 scale).

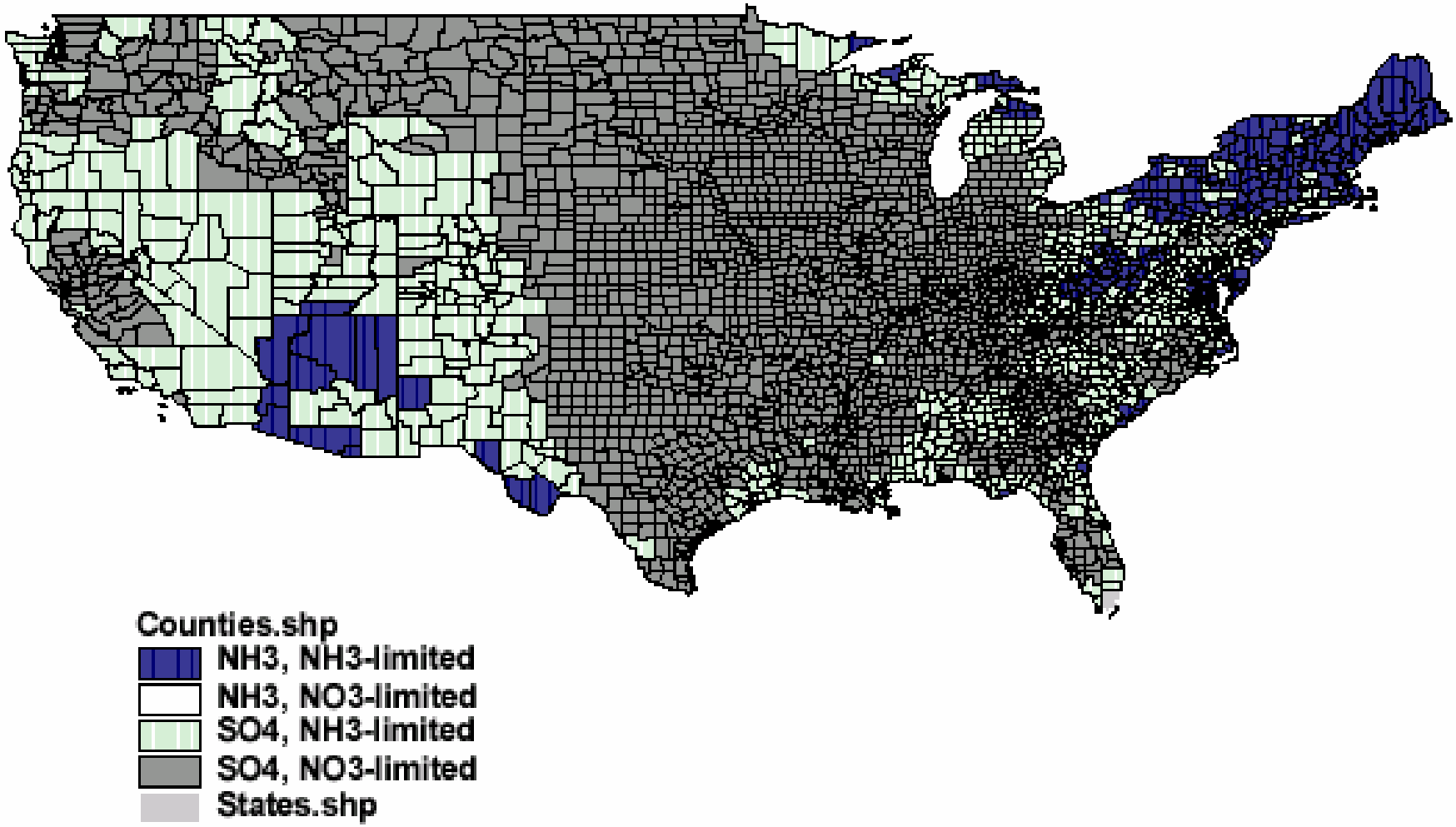


Figure 11. Rate-limiting Regions in S-R Matrix.

Conclusions

- Ambient NH_3 unlikely to have significant direct health impacts, will contribute to PM formation
- Ambient PM strongly linked with cardiopulmonary mortality/morbidity, but limited evidence on differential toxicity
- Ammonia concentrations/control will have influence on AN and AS formation that will vary significantly by site
- Important to keep long-range transport, potential risk tradeoffs in mind when thinking about ammonia control
  - Acidity vs. particle/gas tradeoff