The Effects of Fine Particle Species on Daily Mortality and Morbidity in Six California Counties: Results from CALFINE

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Slides can be downloaded from:

- http://www.arb.ca.gov/research/seminars/seminars.htm

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I. Introduction – background on PM2.5

II. Previous epidemiologic results on PM2.5 and its components

III. Mortality study results

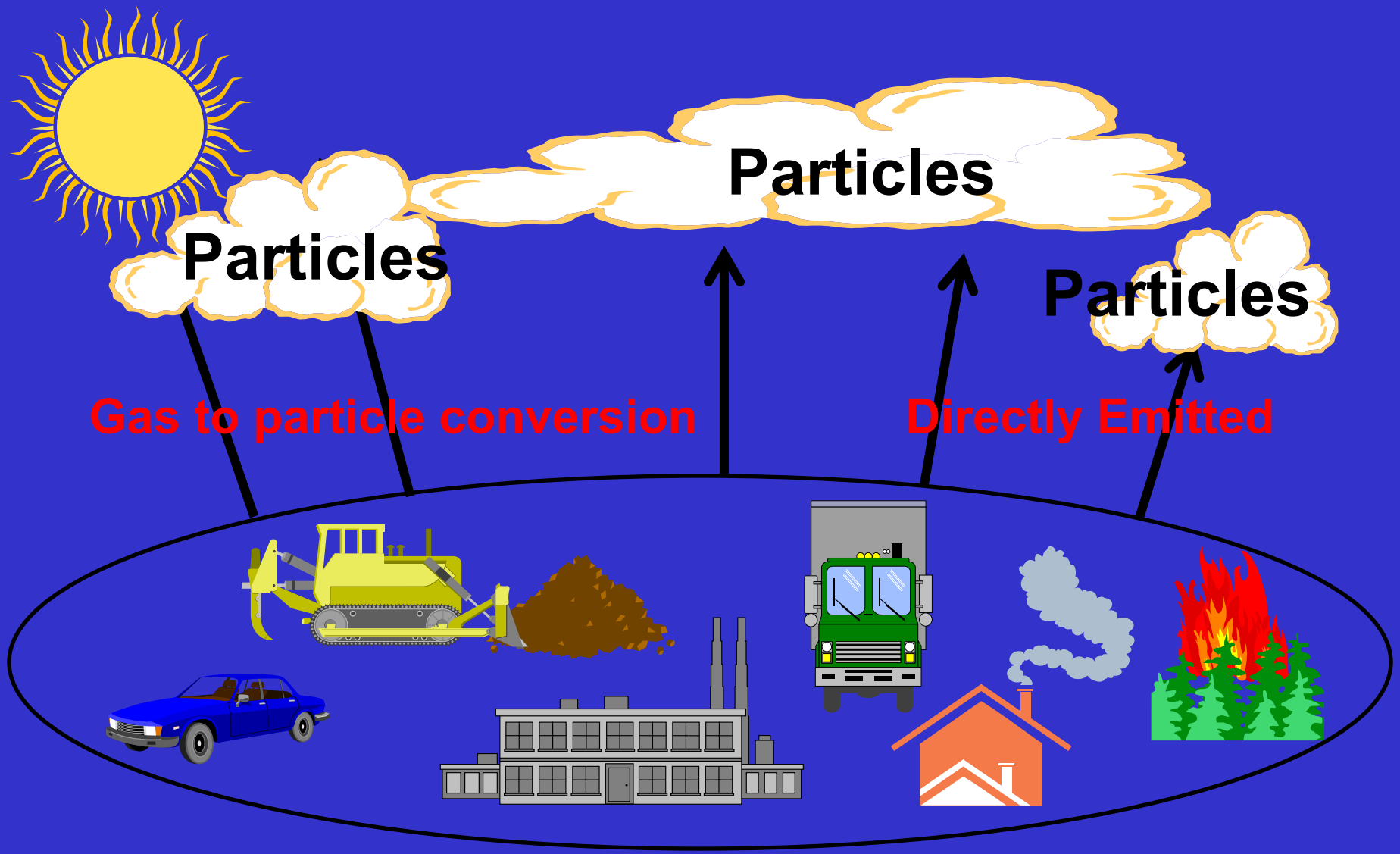
IV. Findings on susceptible subgroups (preliminary)

V. Findings on morbidity (preliminary)

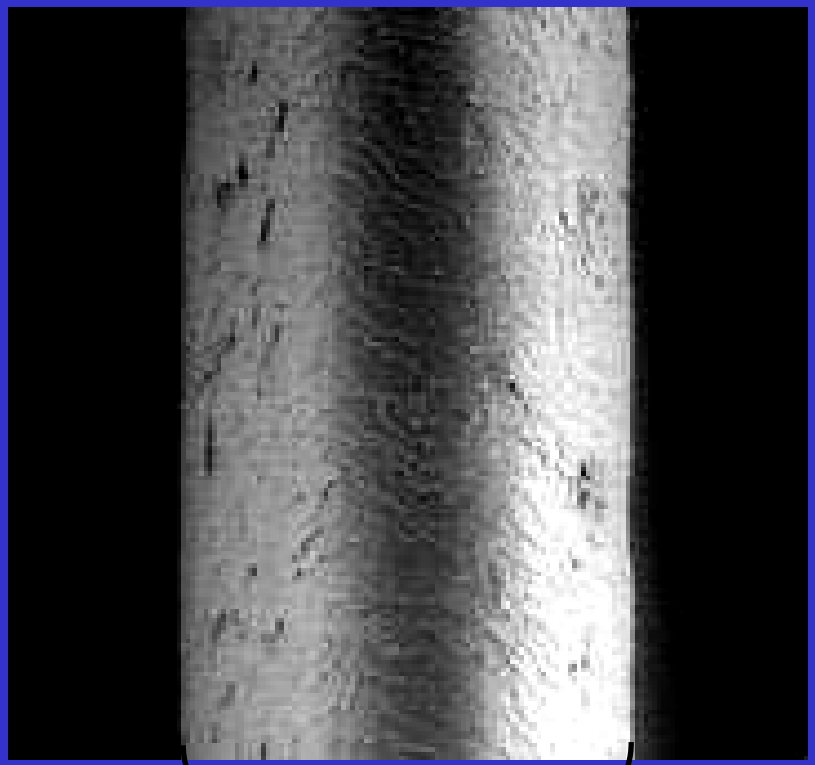
VI. Biological plausibility

VII. Summary

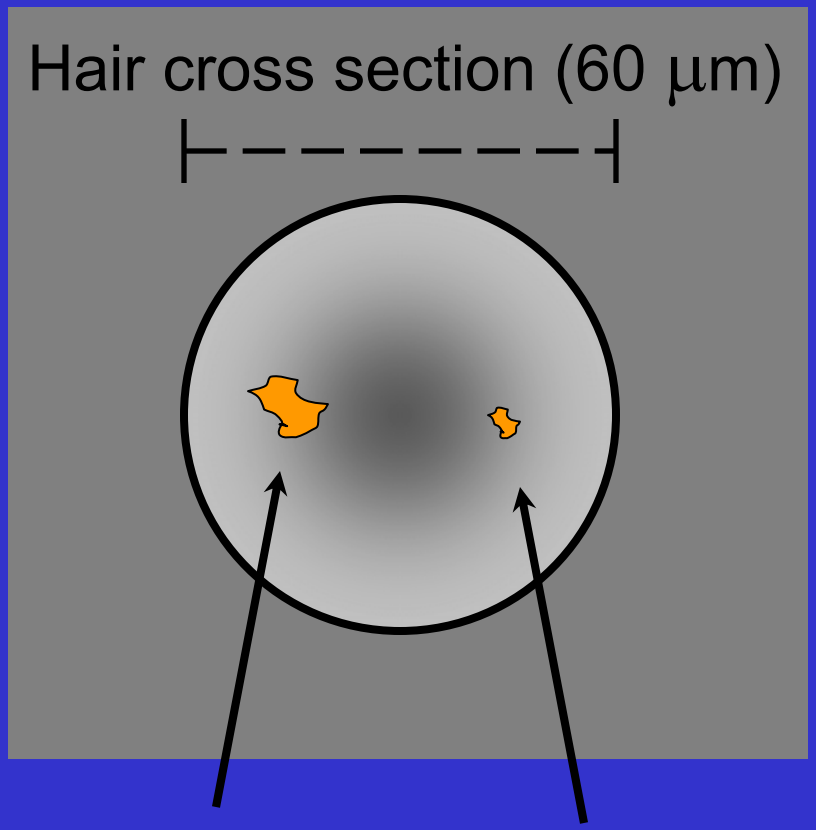
PM2.5 is a heterogeneous mixture of solids and liquids



HOW SMALL IS PM2.5?



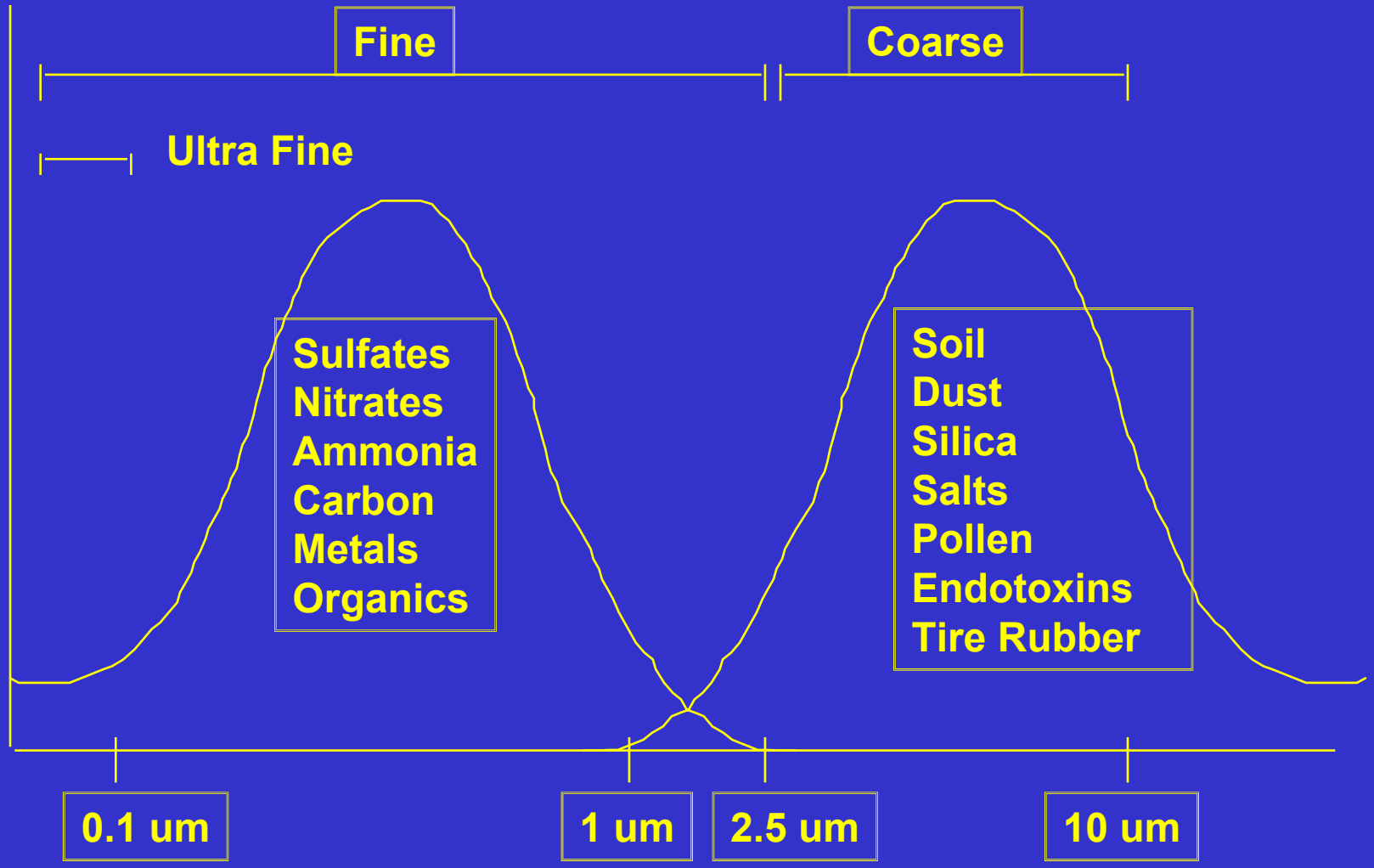
Human Hair
(60 μm diameter)



PM10
(10 μm)

PM2.5
(2.5 μm)

PM10 and PM2.5: Size and Composition



Several Epidemiologic Studies link PM2.5 with Mortality

- Short-term exposure and daily mortality
  - 6 U.S. cities (Schwartz et al. 1996, 2003)
  - 8 Canadian cities (Burnett et al. 2003)
  - 9 CA counties (Ostro et al. 2006)
- Long-term exposure and mortality
  - Dockery et al. 1993; Laden et al. 2006
  - Pope et al. 1995, 2002
  - Krewski et al. 2000

Crucial Question: What is the relative toxicity of PM2.5 components?

- NAS/WHO: Determining toxicity of different particle characteristics and sources is a research priority
  - Very few epidemiologic studies have examined components or sources
  - Could help target pollution control and reduce overall abatement costs
  - Could improve estimates of health impact assessment
  - May help explain heterogeneity in results of multi-city studies

Results of studies of PM2.5 components or sources on mortality

- Mar et al. 2000 (Phoenix): EC/OC and motor vehicle exhaust
- Laden et al. 2000 (6 US cities): markers for mobile sources and coal combustion sources/sulfates
- Burnett et al. 2000 (8 Canadian cities): sulfates, zinc, nickel and iron (NO_3, EC, OC not measured)

PM2.5 composition in California different from most other regions

- Source mix and chemistry different
- Nitrates are a greater share of PM2.5
- Winter concentrations > summer
- Greater indoor penetration
- People spend more time outdoors

Research Questions

1. Are components of PM2.5 associated with adverse health (mortality and morbidity)?
2. If so, are risk estimates associated with components of PM2.5 greater than those associated with total PM2.5 mass?

Data I

1. 24-hr PM2.5 mass and species data from 6 counties (Fresno, Kern, Riverside, Sacramento, San Diego, Santa Clara) for 2000 - 2003 (population ~ 9 million)
   - 13 Components include EC, OC, NO_3, SO_4, Ca, Cl, Cu, Fe, K, S, Si, Ti, Zn
   - 2 monitors in each county with collection every 3rd or 6th day
   - Additional PM2.5 from 3 other counties (PM2.5ext)
2. Weather data (temperature, humidity)

Data II

3. Daily mortality categorized into:
   - all-cause, cardiovascular, respiratory, and age > 65
   - male/female
   - race/ethnicity (White, Black, Hispanic)
   - educational attainment (high school grads versus non-high school grads)
4. Analysis restricted to counties with > 180 observations (total obs = 1870)

Data III

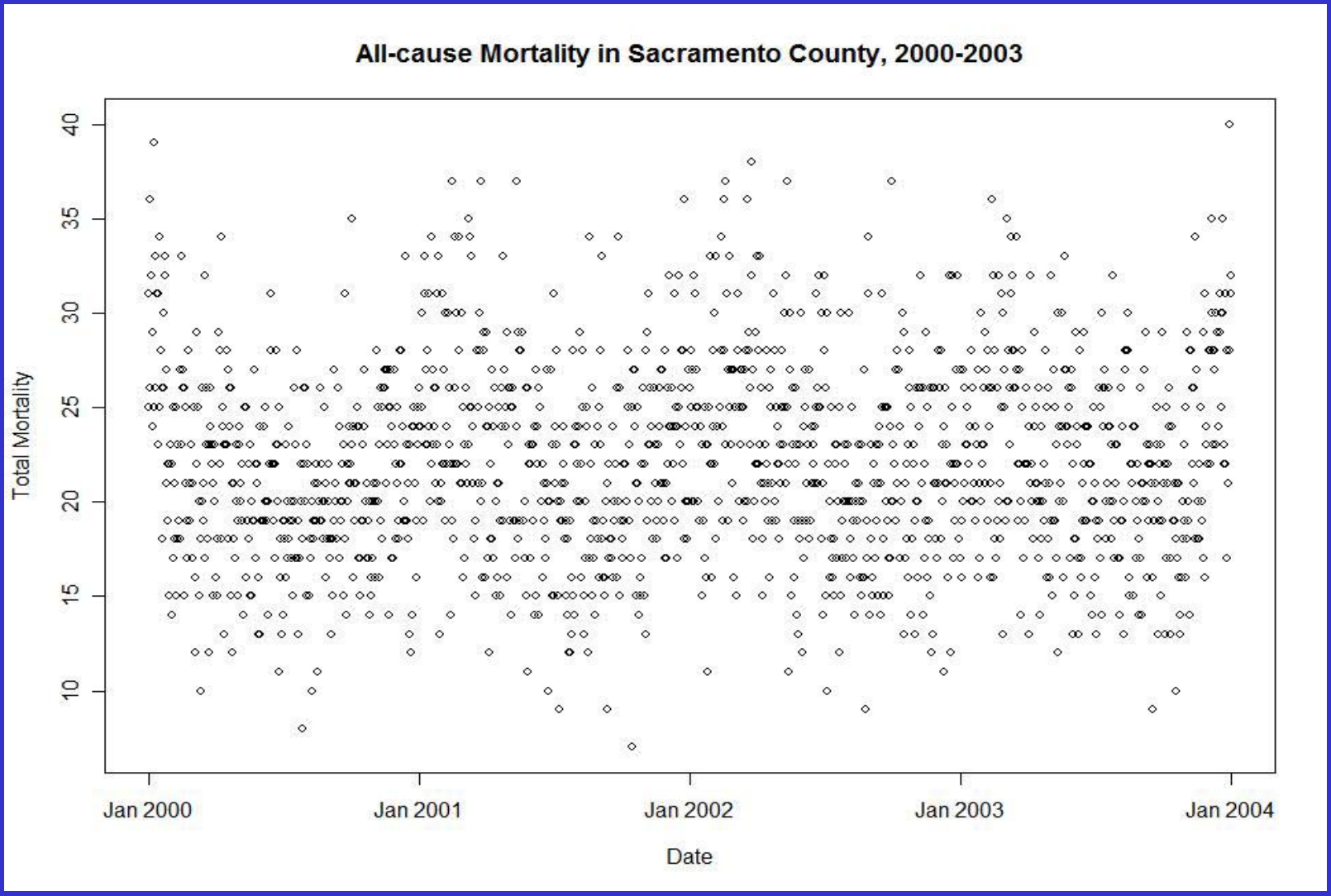
5. Daily hospital admissions: 6 California counties (9.5 million admits) for 2000-2003 for the following outcomes:
   - All respiratory disease
     - Asthma, Bronchitis, Pneumonia, COPD
     - By age
   - All cardiovascular disease
     - Myocardial Infarction, Heart Failure, Stroke
     - By race/gender

Methodology I

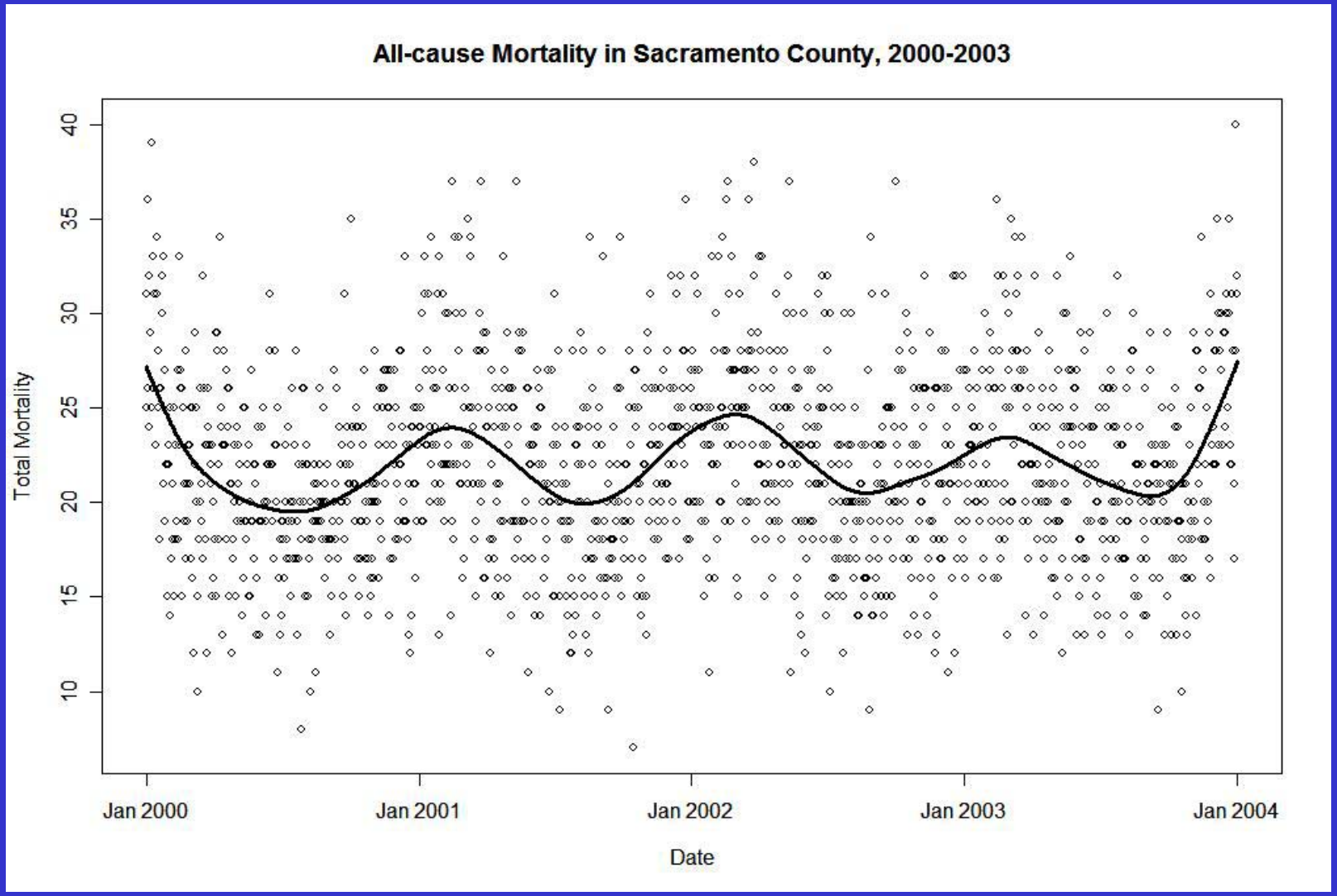
- Time-series regression analysis follows well accepted methodology linking air pollution to mortality and morbidity (HEI, 2003)
- Model daily counts of mortality (hospital admissions) using Poisson regression, controlling for time-varying covariates (time, weather, day of week) and pollution
- Use smoothing splines to model effects of time, temperature and humidity

  (smoothes = flexible, data-driven functions that approximate the relation of mortality and factor of concern. Degrees of freedom determine "bumpiness"

Mortality and Time



Mortality and Time with Smooth



Methodology II

- $Log(M_t) = \beta o + \beta * PM2.5_t$ + day of week+ +s(time, 4df) + $s(temp_{t-1}$, 3df) + $s(humidity_{t-1}$, 3df)
- Examine single-day pollutant lags of 0 to 4 days (focus on 0 and 3)
- Note non-confounders:
  - Smoking
  - Occupational exposure
  - Indoor pollution

Methodology III

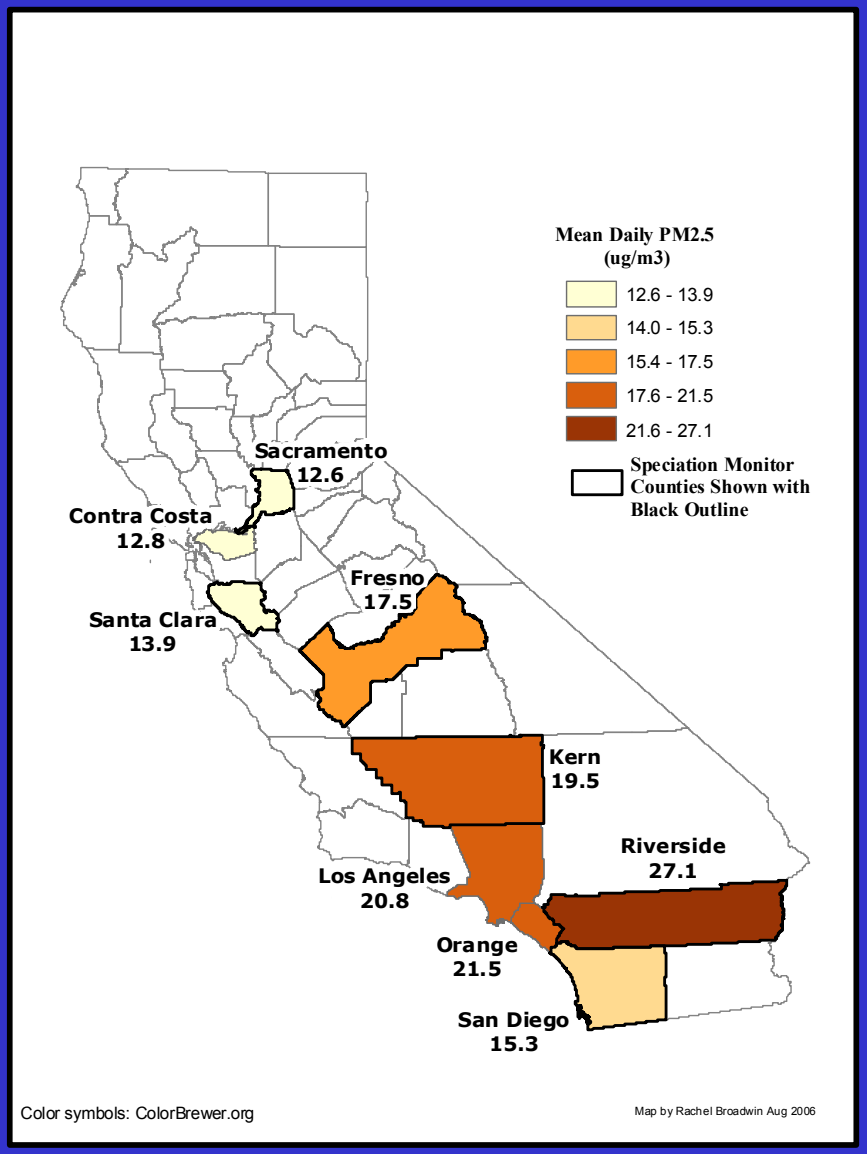
- Combine individual county results using random effects meta-analysis
- Calculate excess risk (RR-1)*100 for interquartile range (IQR = 75% - 25%) of pollutant
- Sensitivity analyses:
  - Examine other types of smoothing functions
  - Treatment of missing data
  - Season-specific (cool season = Oct – Mar)

Mortality Results

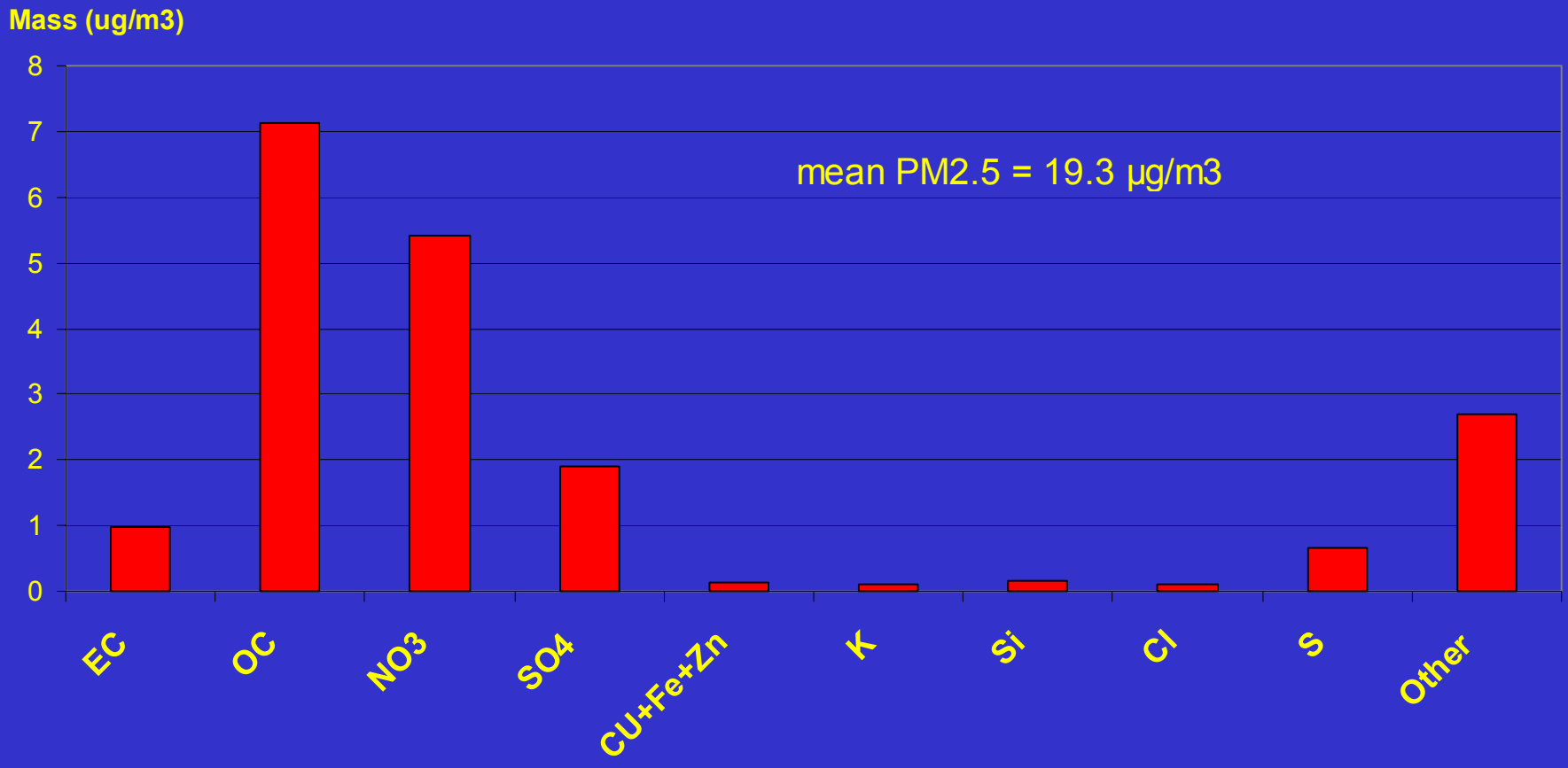
www.ehponline.com

Ostro et al. (2007) Environ Health Perspect 115: 13-19.

PM2.5 in California Study 2000-2003



Components of PM2.5 in Six CA Counties

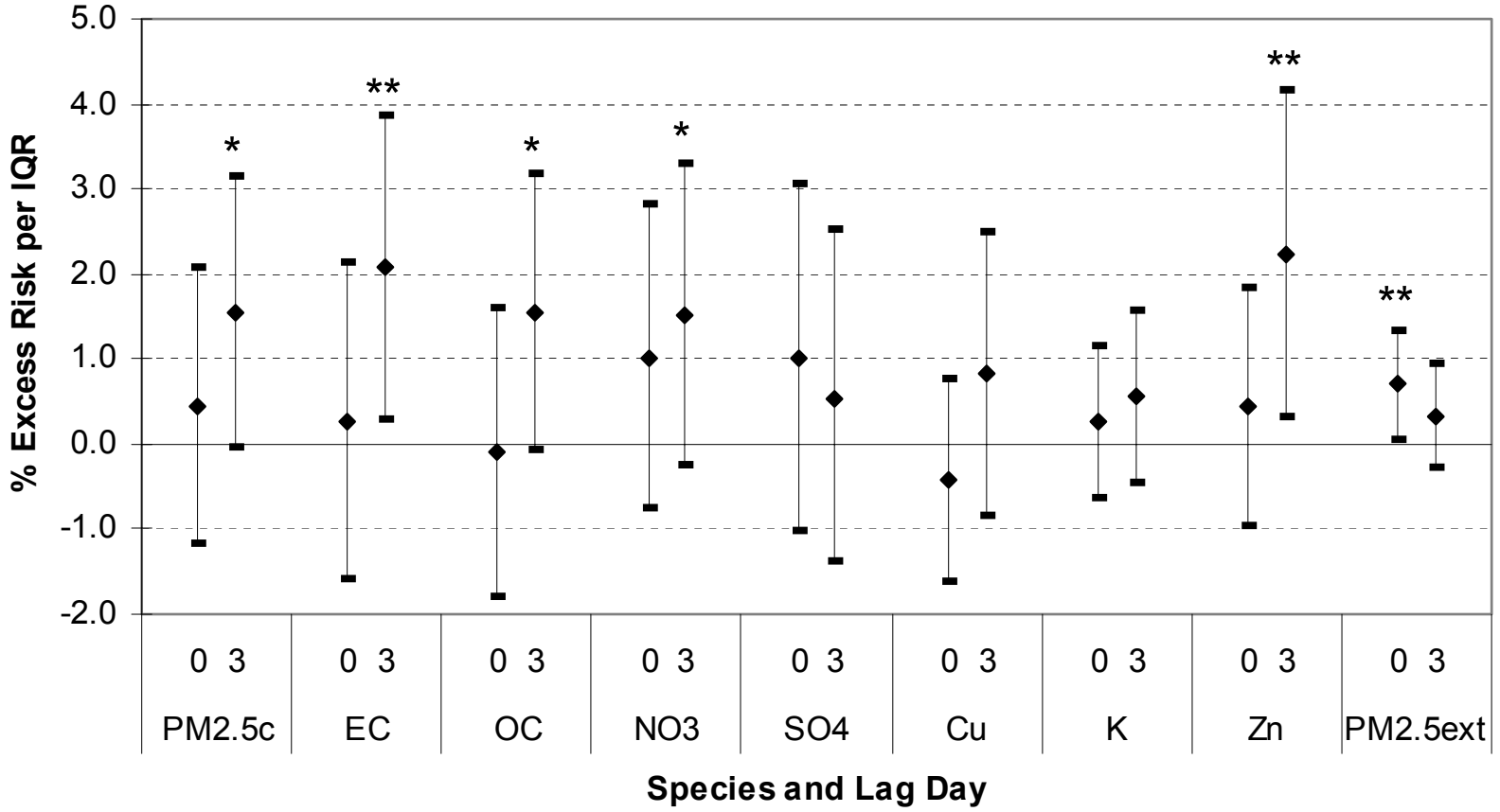


Correlation with PM2.5: EC (.5), OC (.6), NO_3 (.65), SO_4 (.32), K (.5), metals (~.4)

Selective summary of meta-analytic associations for alternative lags (red=p<0.05; green = p< 0.10)

	All-cause	Cardio	Age > 65
PM2.5		1,3	3
EC		3	
OC		3	
NO3	0	3	0
SO4			
Cu	1		
Fe		2	
K		2	2
Zn		3	1,3
PM2.5 (extended)	0,1	0,1	0,1

Cardiovascular Mortality and PM2.5 Components, Full year

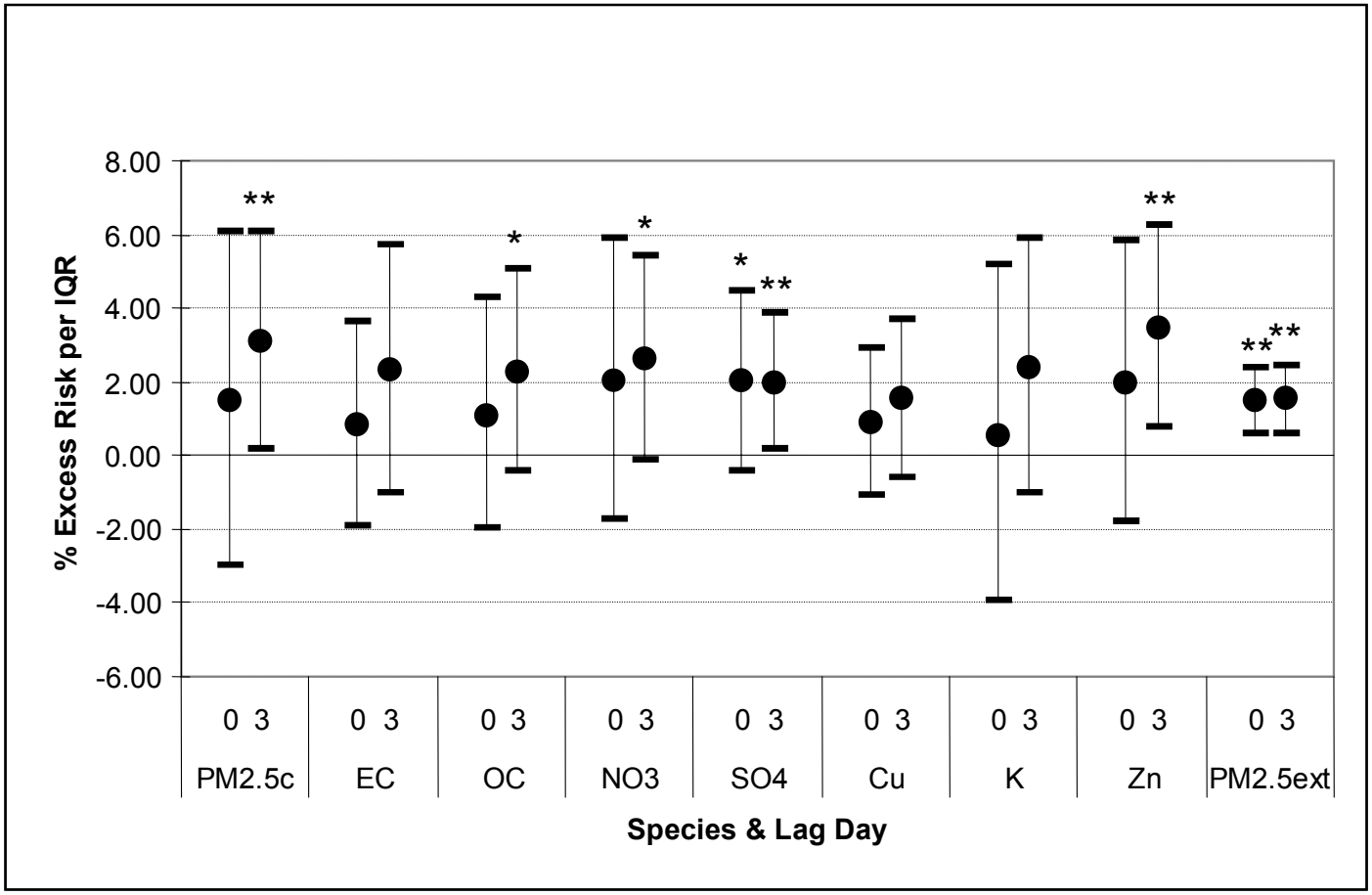


* p < 0.10; ** p < 0.05

Selective summary of meta-analytic associations for Cool Season (red=p<0.05; green = p< 0.10)

	All-cause	Cardio	Age > 65
PM2.5	3	3	3
EC	3		3
OC	3	3	3
NO3		3	0
SO4	0,3	0,3	0
Cu	1	1	
Fe	2,3	3	3
K	3		3
Zn	3	3	3
PM2.5 (ext.)	0,1,2,3	0,1,2,3	0,1,2,3

Cardiovascular Mortality (Cool Season)



Excess and Relative Risks for Cardiovascular Mortality and Selected Pollutants

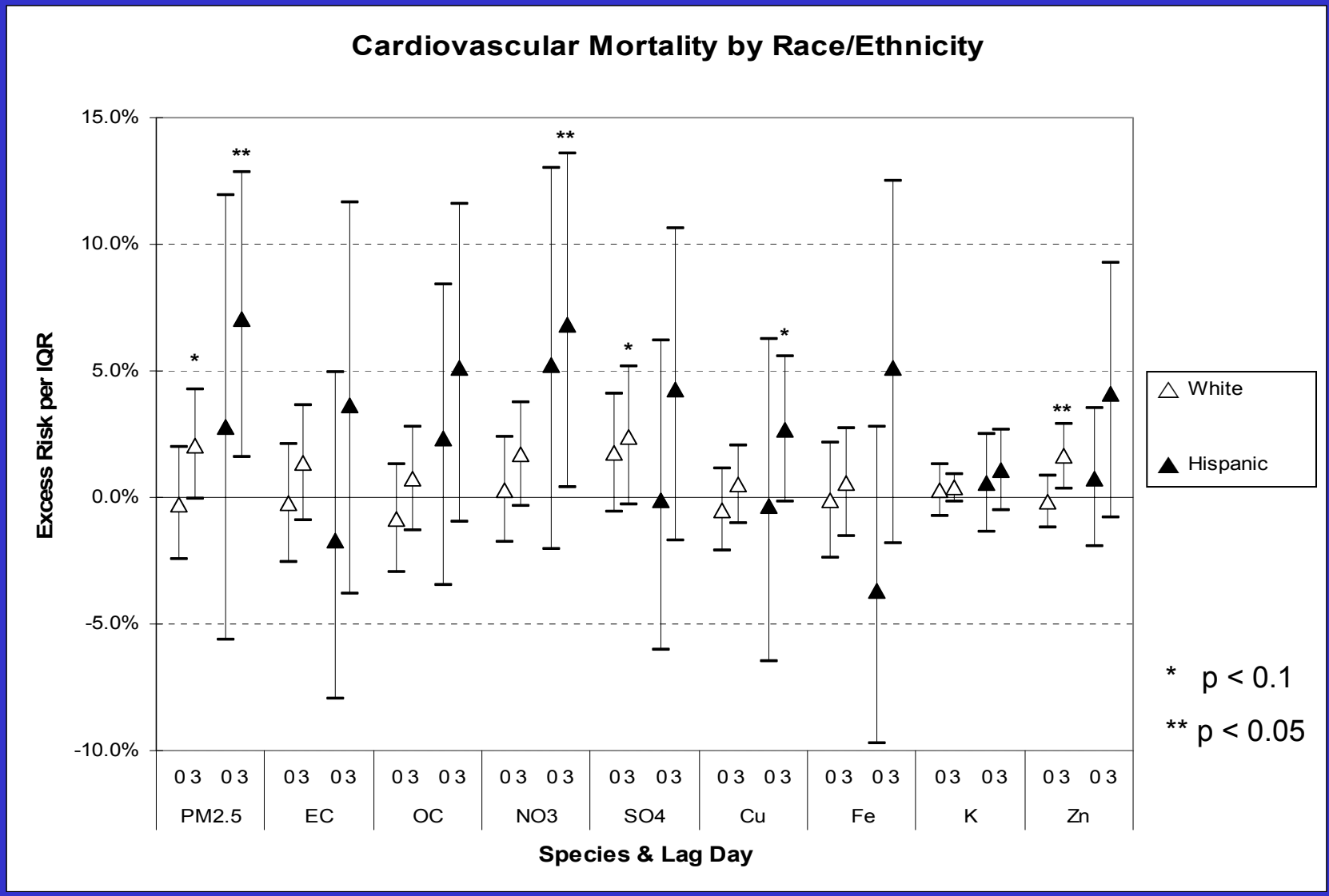
Pollutant	Lag day	beta*100	% change in risk per IQR
PM2.5	3	0.11	1.55
EC	3	2.6	2.07
OC	3	0.33	1.55
NO3	3	0.27	1.52
SO4	3	0.66	1.01
K	3	6.9	0.50

Effect Modification and Mortality

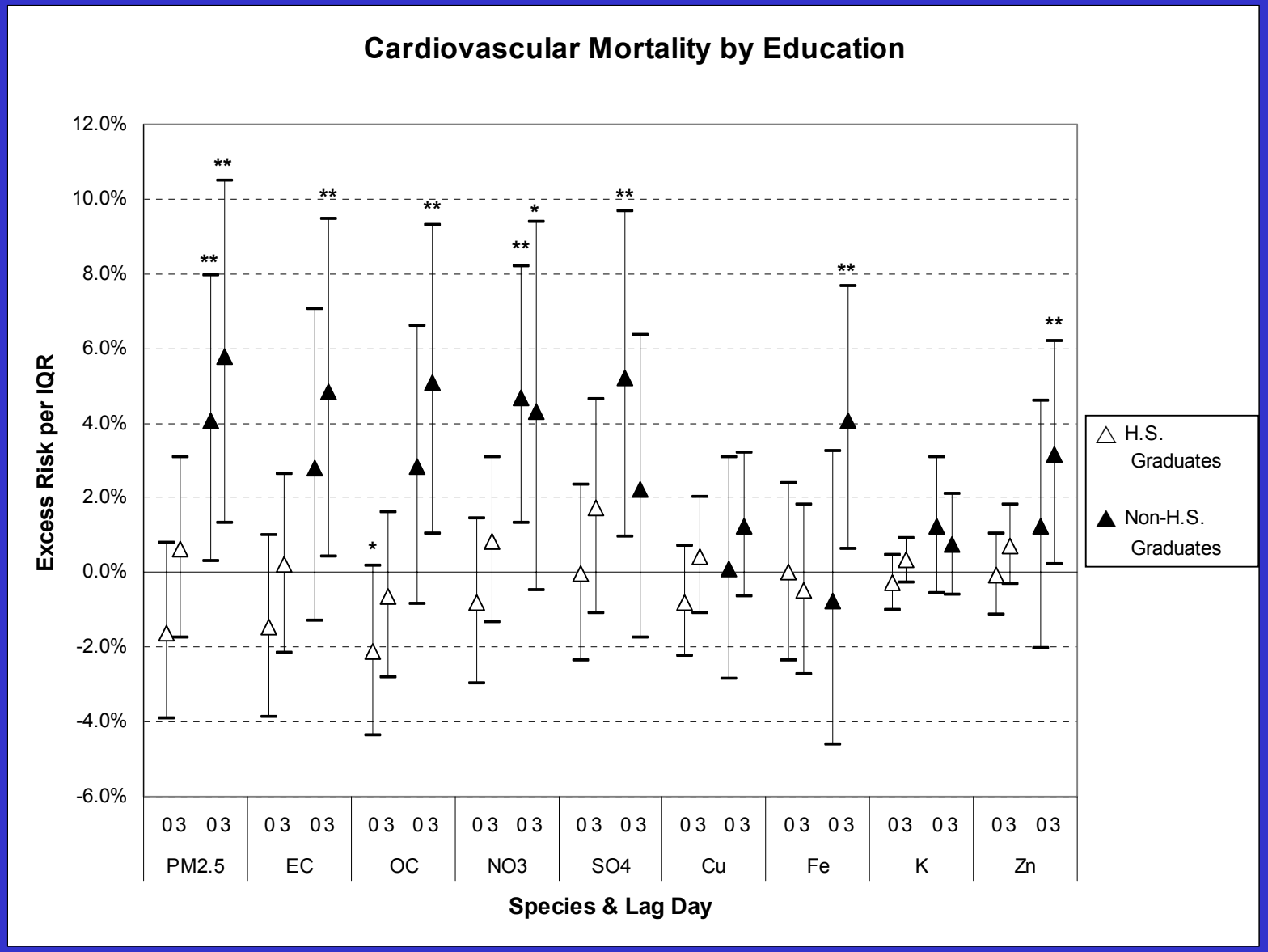
Are there subgroups that are particularly susceptible to the components of PM2.5?

Examined:
Gender/Race/Education

Cardiovascular Mortality by Race/Ethnicity



Cardiovascular Mortality by Education



What is “Education” Measuring?

Possibly:

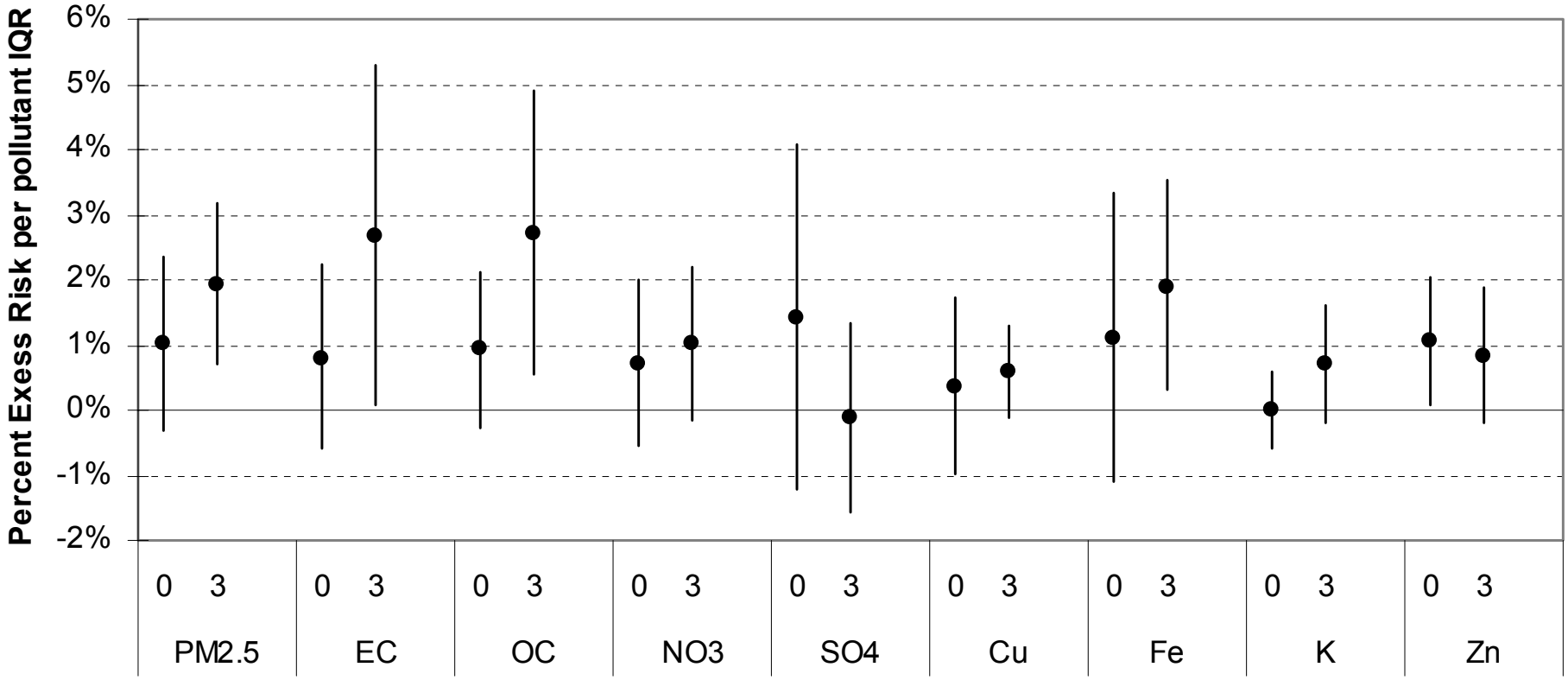
- Greater exposure
- Suite of factors related to lower SES (smoking, diet, exercise, obesity, access to and use of medical care)
- Greater co-morbidity

Results for Daily Hospital Admissions

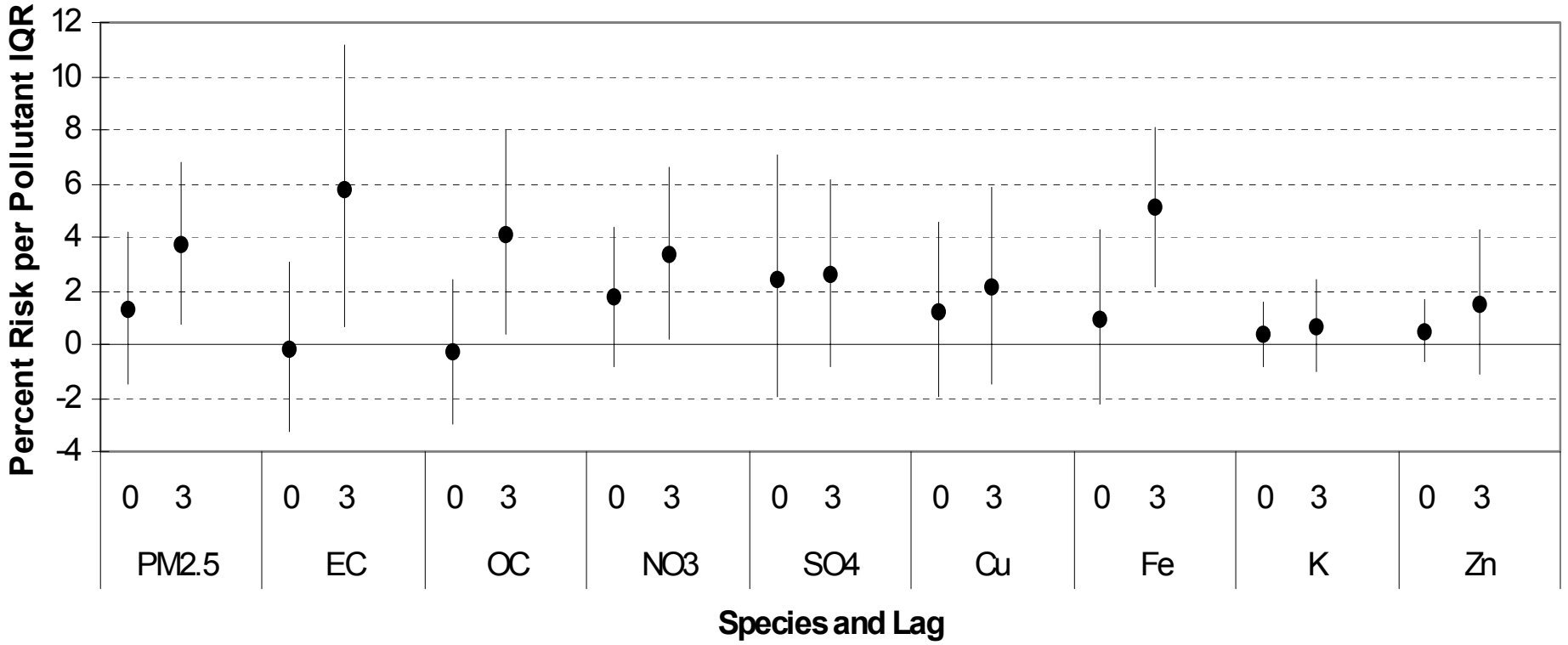
Respiratory Results

- Total respiratory, all ages and age < 5 (includes pneumonia, bronchitis, wheeze)
- Asthma, ages 5-18
- Chronic bronchitis

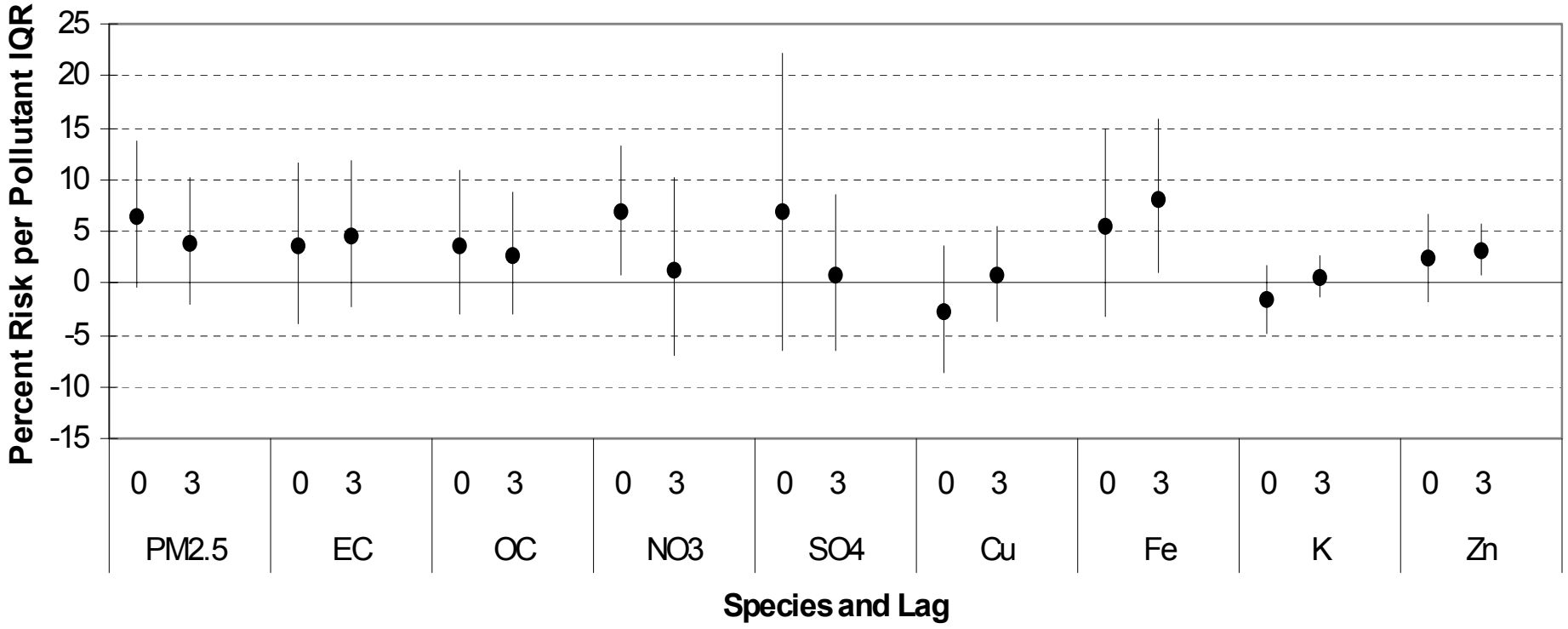
Total respiratory admissions, all ages



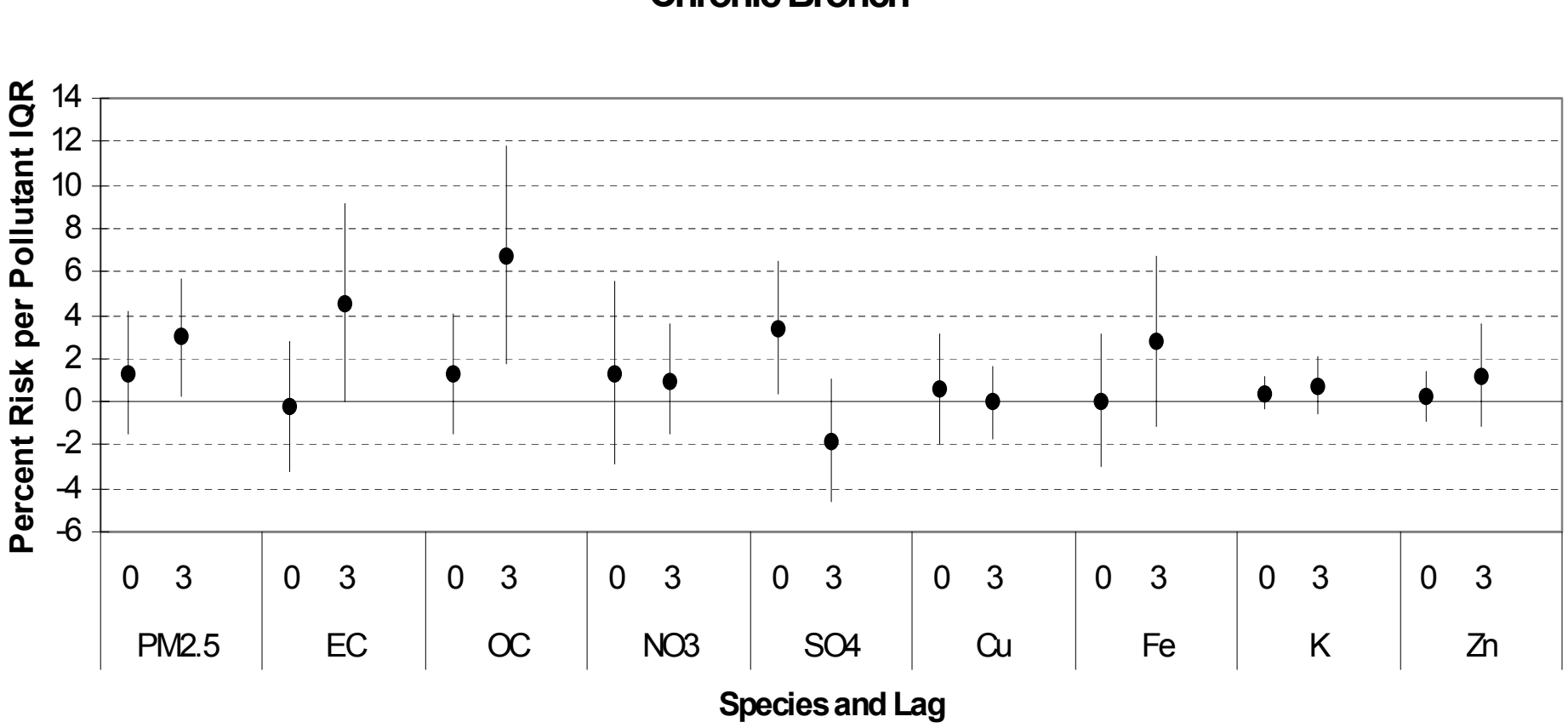
Total respiratory admissions, age < 5



Asthma admissions, ages 5-18



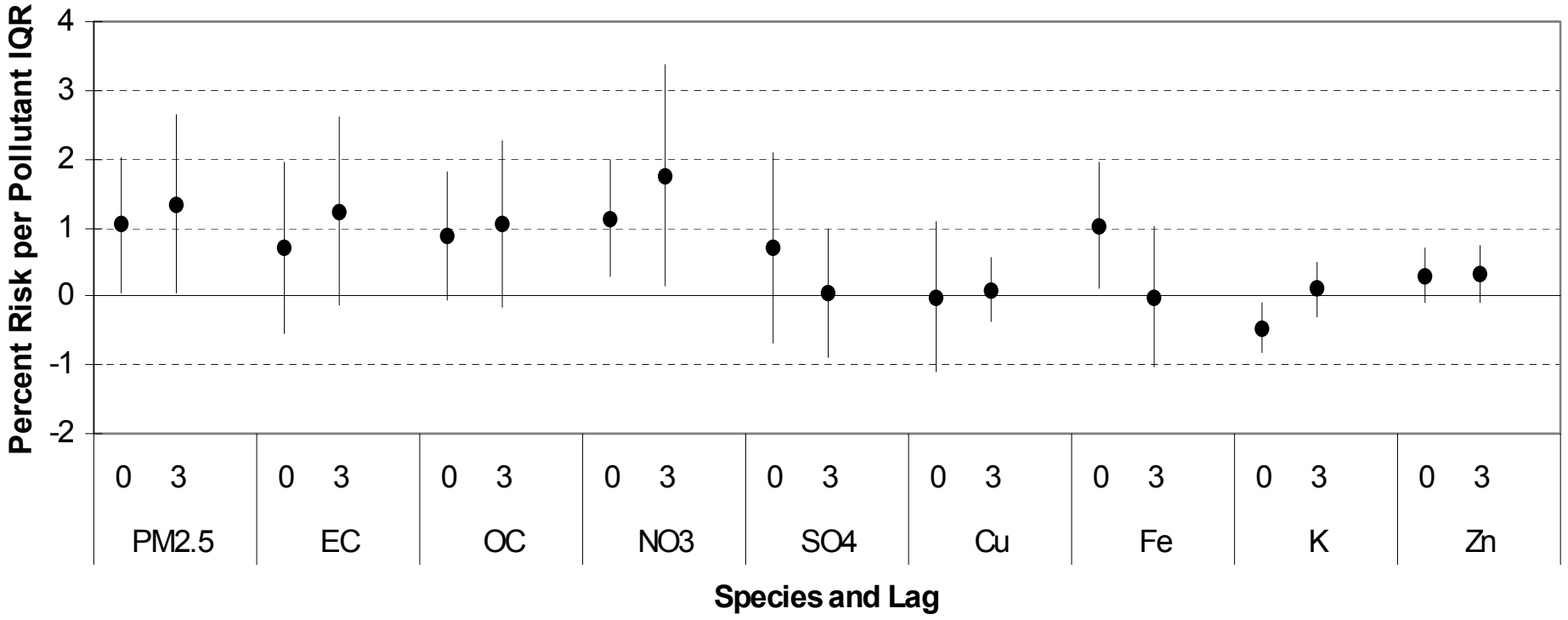
Chronic bronchitis admissions



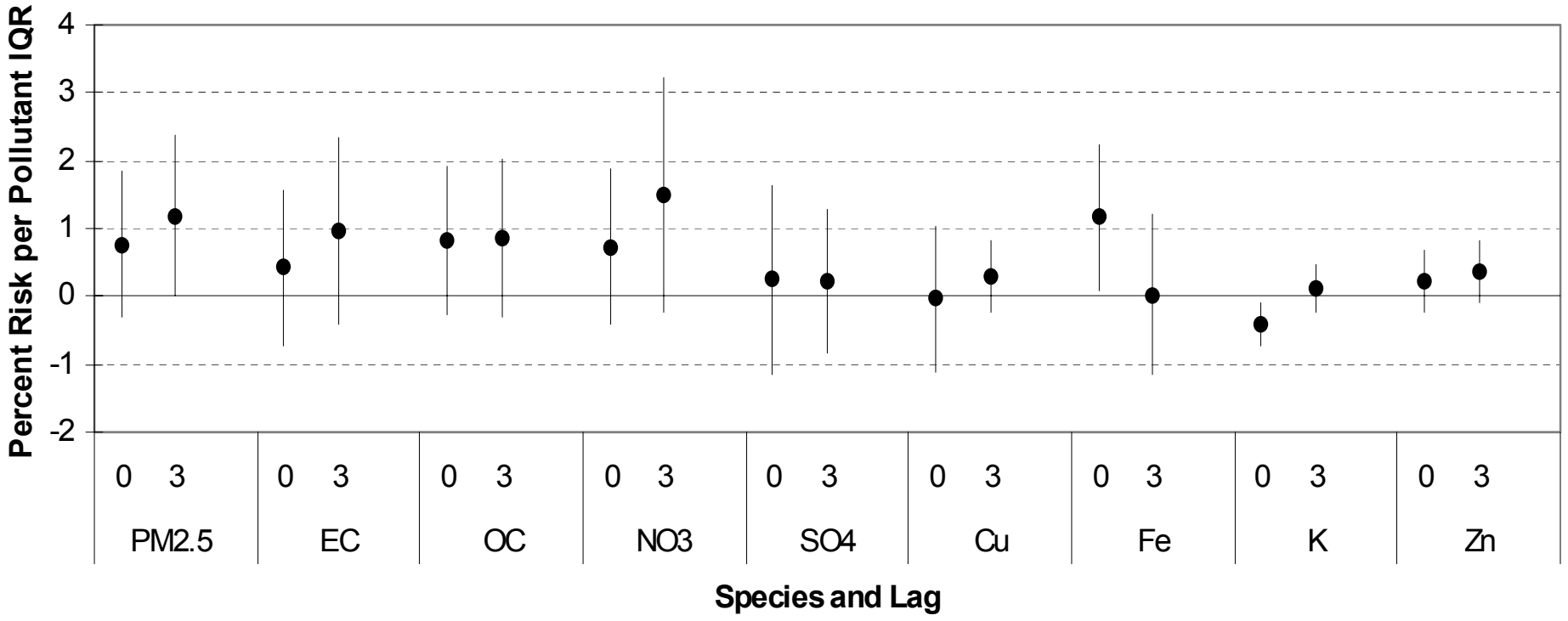
Cardiovascular Results

- All Cardiovascular
- White Cardiovascular
- Hispanic Cardiovascular
- Acute Myocardial Infarction
- Cardiac Dysrhythmia
- Heart Failure
- Stroke

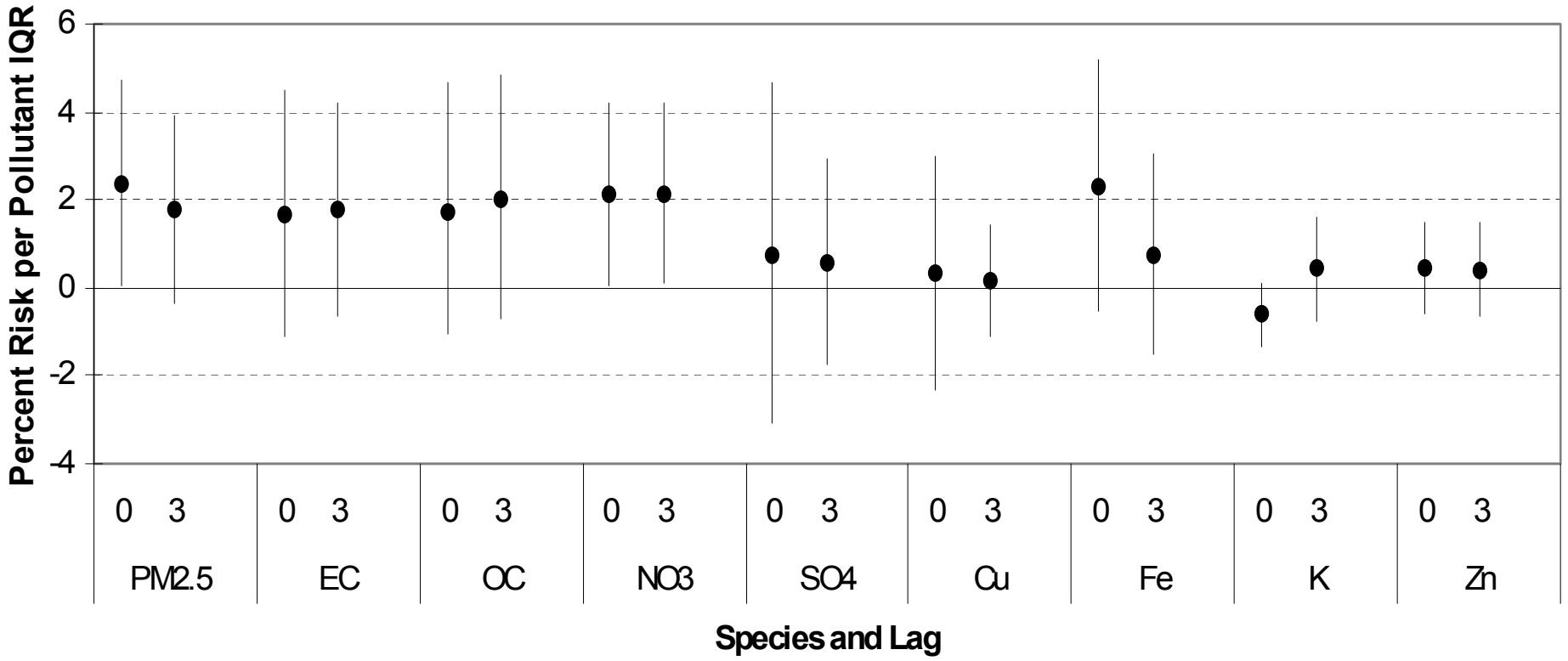
All cardiovascular admissions



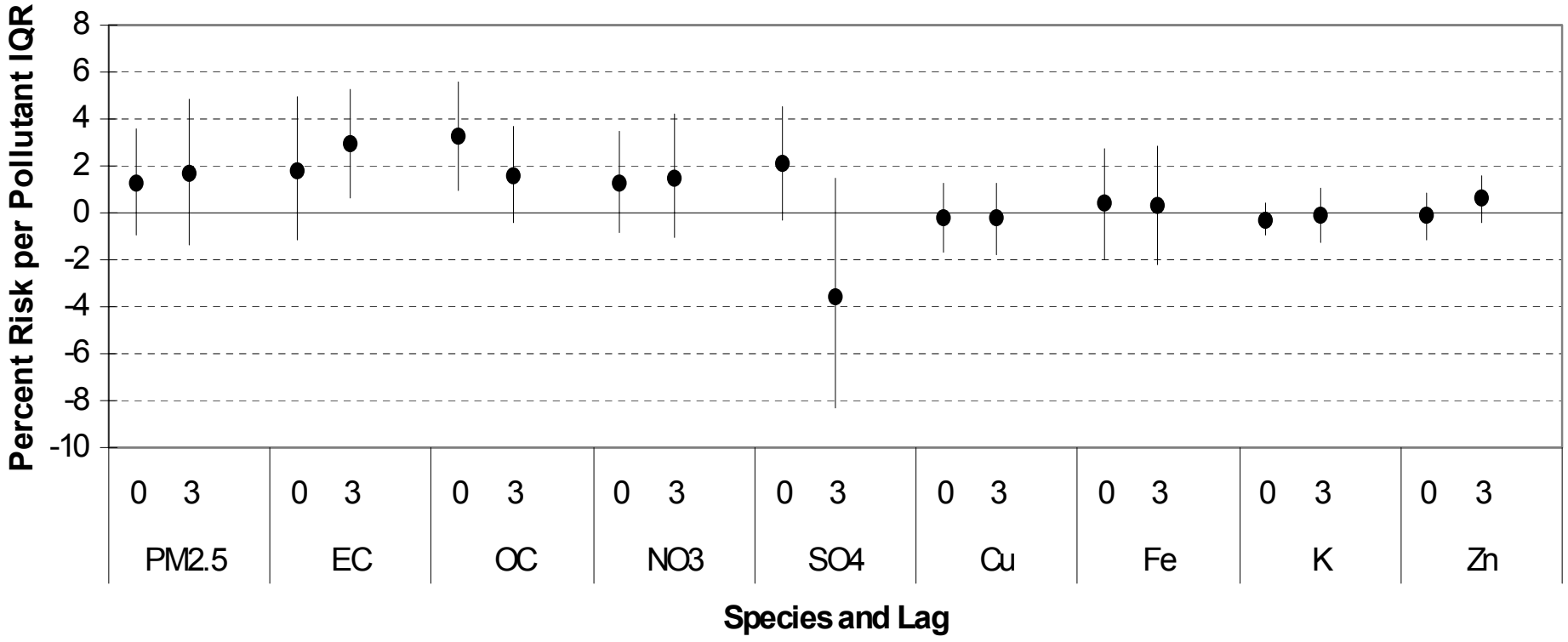
White cardiovascular admissions



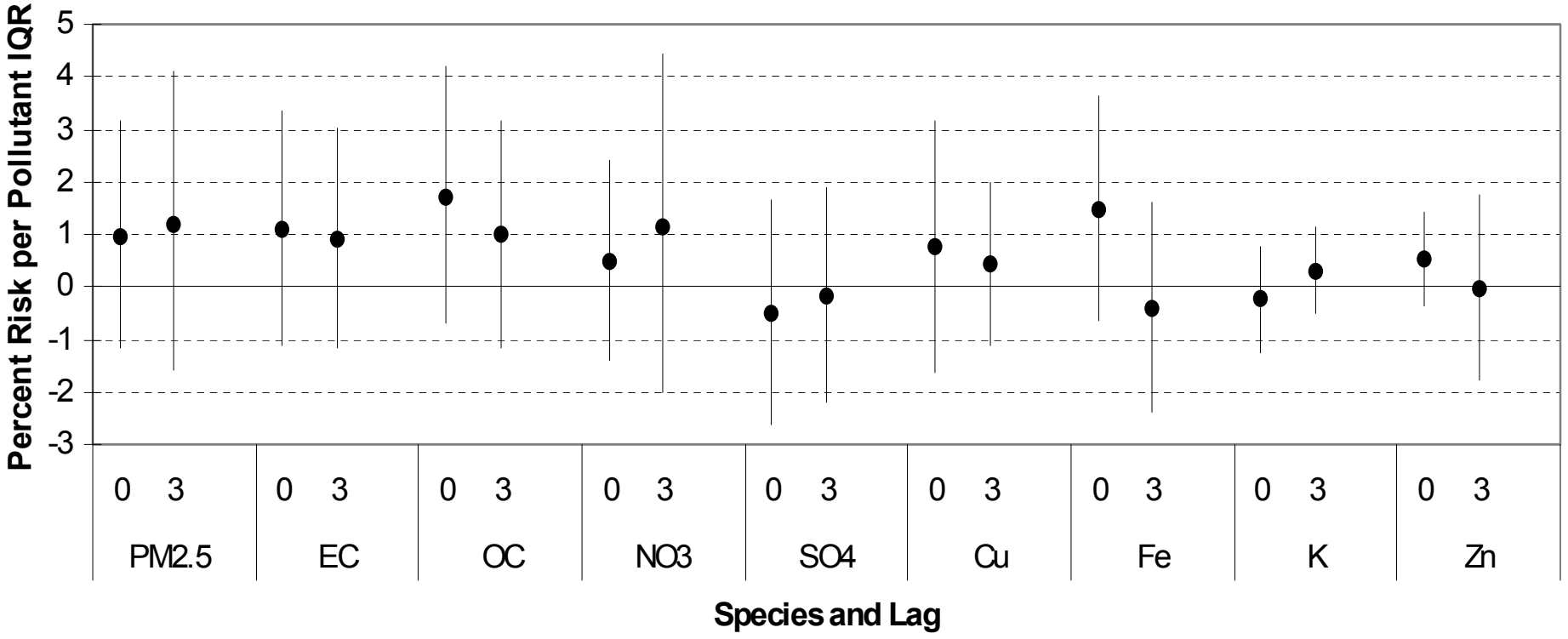
Hispanic cardiovascular admissions



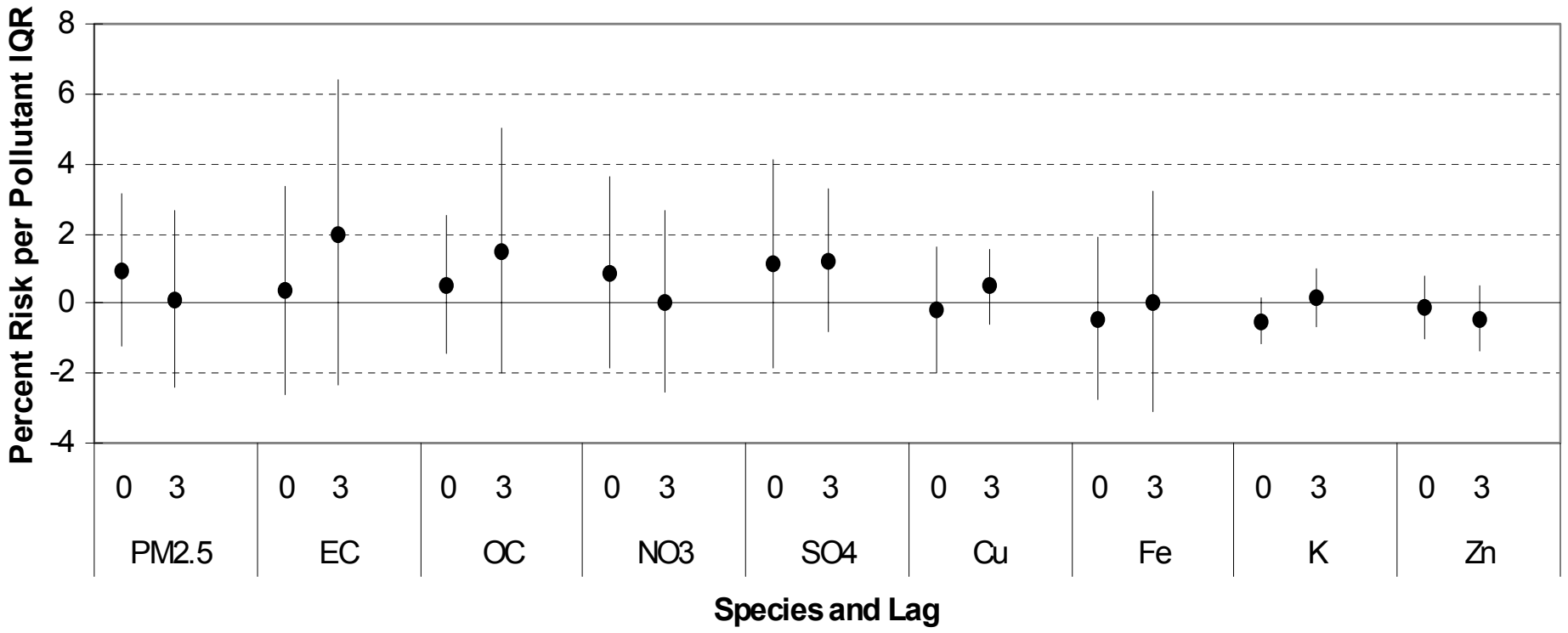
Acute myocardial infarction admissions



Congestive heart failure admissions



Stroke admissions



Biological Plausibility I

- Among the components of PM2.5, biological mechanisms for effects have been investigated most extensively for diesel exhaust, including EC and OC
- Approximately 65-80% of EC in CA from diesel; wood smoke is another major source
- Evidence from epidemiologic, toxicologic and human clinical studies suggest plausible mechanisms for mortality

Biological Plausibility II

- EC/OC associated with oxidative stress (Cho 2005), ECG changes (Henneberger 2005), ST depression (Lanki 2006), HRV (Schwartz 2005)
- PM2.5 (or traffic) associated with myocardial infarction (Peters et al., 2004; Zanobetti and Schwartz, 2005; von Klot et al. 2005; Pope et al, 2006)
- Metals (fuel combustion, brake wear, lube oil, tire dust) may generate reactive oxygen species (Wilson et al. 2002; Ghio 2004; Schlesinger et al. 2006) and are associated with fibrinogen (Huang 2003)

Biological Plausibility III

Respiratory Outcomes:

- Diesel exhaust particles (DEP) induce pulmonary inflammation through the generation of reactive oxygen species
- Human and animal studies suggest that DEP may induce inflammation and symptoms in asthmatics and atopics

Summary of Findings I

1. Mortality and morbidity associated with particles from:
   - Gasoline and diesel engines (EC, OC, NO_3, metals)
   - Wood smoke (EC, OC, K)
   - Other combustion sources
2. Notable associations include:
   - Mortality and morbidity from cardiovascular disease, especially MI and for Hispanics
   - Respiratory admissions for children
3. Winter-time effects (when PM2.5 levels are 2x higher) stronger except for coarse particles which show effect in summer

Summary of Findings II

4. Excess mortality risks for IQR between 1–2% but 2-3 times greater for “susceptible” subgroups
5. Apparent effect modification by race/ethnicity and SES

   - “Hispanics” in current study: 50% non-HSG and 17% below poverty vs 12% and 4% for Whites

6. Some species (EC, metals) have very high unit risks

Summary of Findings III

7. Sample size is small – stronger associations possible as are spurious results
8. Results may be impacted by measurement issues
    - Species might be marker for another correlated pollutant
    - Differential instrument error for species
    - Differential spatial pattern of species

Future Work

1. Repeat study with larger data set
2. Develop Chemical Mass Balance models to estimate effects of sources
3. Estimate independent effects of temperature on mortality and morbidity and determine susceptible subgroups
4. GIS-based analysis to examine exposure misclassification