Bay Area Air Quality Management District
939 Ellis Street
San Francisco, California 94109

APPROVED MINUTES

Advisory Council Technical Committee
9:00 a.m., Monday, April 16, 2007

1. **Call to Order – Roll Call.** Chairperson Sam Altshuler called the meeting to other at 9:05 a.m. Present: Sam Altshuler, P.E., Chairperson, Louise Bedsworth, Ph.D, John Holtzclaw, Ph.D., Kraig Kurucz, William Hanna, (9:10 a.m.), Robert Bornstein Ph.D., (9:20 a.m.).

2. **Public Comment Period.** There were no public comments.

3. **Approval of February 28, 2007 Minutes.** The minutes carried unanimously.

4. **Overview of the South Coast Air Quality Management District's (SCAQMD) MATES III Program and the Air District's CARE program:** The Committee Members compared and contrasted the MATES III program and the CARE program. The following Matrix was developed. A draft of the matrix was sent to the SCAQMD for review.

COMPARISON OF PROGRAMS

MATES	CARE
General Information	
MATES I 1987	CARE Phase I: Sp 2004 – F 2006
MATES II 1988-1999	CARE Phase II: F 2006 – Sp 2008
MATES III 2004 - 2006	CARE Phase III: Sp 2008 – F 2009
District Population: 16 Million	District Population: 7 million
Program Cost: $2 million per year	Program Cost: $1 million per year
Focus: Marine Ports, Airports, Freeways, Freeways, Regional	Focus: Marine Ports, Construction, Freeways, Regional
20 Member Technical Advisory Group	15 Member Technical/Community Task Force
Compounds Monitored	
Metals (e.g. CrVI, Ni, Cd), PAHs, VOCs, (e.g. benzene, 1,3-butadiene, perc), carbonyls (e.g., Formaldehyde) , elemental carbon, acrolein being considered	Metals (3 sites only), PAHs, VOCs, carbonyls, elemental carbon, acrolein-begun
PM sizing	No PM sizing
Wood smoke from wildfires	Special study markers for wood smoke – residential wood-burning, carbon dating

No lube oil measurements	No lube oil measurements
10 fixed sites; 6 microscale sites	23 fixed sites: metals @ 4 sites; VOC @ 22 sites; PAHs, carbonyls @ 3 Sites; elemental carbon @ 6 sites
3 temporary sites	No temporary sites
No mobile sampling	No mobile sampling
Estimated Risks**	
Results: 1998/1999 cancer risk – 1,400 per million from air toxics	Results: 2000 cancer risk – 700 per million from air toxics
Diesel PM cause 71% of cancer, 8% from 1,3-butadiene, 7% from benzene 3% from carbonyls	Diesel PM cause 81% of cancer risks, 6% from 1,3-butadiene, 7% from benzene, 1% from formaldehyde
No estimates of non-cancer risks	Chronic and acute non-cancer risks: acrolein represents 48% of chronic and 94% of acute non-cancer risk. Important sources are mobile and aircraft.
Toxics Trends	
Benzene is decreasing in South Coast Area	Benzene is decreasing in Bay Area
Perc is decreasing in South Coast Area	Perc is decreasing in Bay Area
Formaldehyde and acetaldehyde show no trend	Formaldehyde and acetaldehyde are decreasing slowly (2-4% per year)
Insufficient data to establish acrolein trend	Insufficient data to establish acrolein trend
Modeling	
Modeling: Some regional and local scale modeling, plans to update	Modeling: Plans to conduct regional and local scale modeling
Grid: 4/2 km	Grid: 2 km

* Note: This comparison is based on our current understanding of the MATES program. As we continue to gather information, there may be future updates to this table.
** CARE risks are based on the ARB's California Almanac of Emissions and Air Quality (2006 Edition) and CARE toxicity-weighted emissions estimates. These estimates may change as the CARE program progresses.

The Committee held discussions regarding chronic and acute health effects as it relates to the MATES/CARE programs. The MATES/CARE programs focus on chronic long term health effects; AQS focuses on acute effects. PM, NO_3 has very low impact to health while Zinc (from lube oil?) has a relatively high impact on public health, pound for pound. The Committee recommends staff consider adding sizing of PM as well as include measurements of lube oil within CARE in as much as possible.

Topics for future discussion: Lube oil, PM sizing, Acrolein trend.

5. **Presentation on "Health Effects of Fine PM Species in Daily Mortality and Morbidity in California":** Dr. Bart Ostro Ph.D., Chief Air Pollution Epidemiology Unit, Office of Environmental Health Hazard Assessment (OEHHA), California Environmental Protection Agency (Cal EPA); delivered to the Committee a presentation on the "Health Effect of Fine PM Species on Daily Mortality and Morbidity in California" which he co-authored. Dr. Ostro's presentation included the following topics:

    - Introduction – Background on PM2.5
    - Previous Epidemiologic results on PM2.5 and its components
    - Mortality Study
    - Findings on Susceptible Subgroups (preliminary)
    - Findings on Morbidity (preliminary)
    - Biologic Mechanisms
    - Summary
    - Future Work

    Dr. Ostro stated he is with the California Office of Environmental Health Hazard Assessment (Cal OEHHA) which is part of Cal EPA. His official responsibility is to recommend state air quality status to the Air Resource Board (ARB). Dr. Ostro did a great deal of research with regards to issues relating to Criteria Air Quality; his presentation focused on the issues published a month or two ago on Mortality. The Committee heard the first public presentation on Morbidity. Dr. Ostro states that he has worked on sensitive populations to see which population is particularly sensitive to some of the elements of the study that will be enumerated later. Most of the morbidity epidemiology discussion is based on the conditions, respirations and data. Bio-monitoring is a medium with which to see chemical analysis in the body; it captures chemicals that people have in their bodies that are higher than the official standard helped achieve result in this study. At this point, Mr. Altshuler noted that Richard Jackson from CDC gave a presentation of the subject to the Advisory Council about a year ago. Dr. Ostro added that findings show that people have much chemical in their body; about 100 times the normal amount.

    Dr. Ostro explained the components of PM2.5 as a heterogeneous mixture of solid and liquid from multiple sources, which can be gas to particle conversion or directly emitted particles. He added that to identify the components and sources of PM2.5 could help target its control and strategy. Several epidemiology studies link PM2.5 with mortality and these include:

    1. Short Term exposure and daily mortality
        - Six United State cities (Schwartz et al. 1996, 2003)
        - Eight Canadian cities (Burnett et al. 2003)
        - Nine counties (Ostro et al. 2006)
    2. Long term exposure and mortality
        - Dockery et al. 1993; Laden et al. 2006
        - Pope et al. 2006
        - Krewski et al. 2000

    Dr. Ostro noted a crucial question "what is the relative toxicity of PM2.5 components?" He also stated that one criticism is of control strategy, we think about high cost and things that are

toxic. With all PM2.5 components, be it toxic or diesel, factory, restaurant, or from dwellings, the most important question are its health effects and its source. NAS and WHO recommended that determining the toxicity of different particle characteristics and sources is a research priority because: (1) very few epidemiologic studies have examined components or sources; (2) it could help target pollution control efforts and reduce overall abatement costs; (3) it could improve health impact assessments; and (4) it may help explain heterogeneity in multi-city studies.

Dr. Dave Fairley asked if any research has taken these multi-city studies and estimated the range of vaults to see the difference? In his response, Dr. Ostro said that John Hopkins and his group are looking into the variations. Dr. Ostro also added that in some hypotheses, there are some generic responses due to generic particles and generic depositors in different counties and countries. The one for California is different due to the toxicity. The results and studies of components or sources on mortality include: (a) Mar et al. 2000 showed that EC/OC generated from motor vehicle exhaust is related to mortality in Phoenix; (b) Laden et al. did studies in six US cities and showed markers for motor vehicles and residual oil sulfates, but not from crusty materials relate to death; (c) Burnett et al. 2000 also did a study in Canada and found that sulfates, zinc, nickel and iron relate to death. However, NO3, EC, and OC in relation to mortality were not measured.

In California, PM2.5 studies are different from that typically studied; the source mix and chemistry are quite different with regards to PM2.5 in California and Southern California in particular. The study shows that Nitrate is a greater share of PM2.5, but different in the east and many other parts of the world. Dr. Ostro also added that the winter concentration is higher than in summer. At this point, Dr. Bornstein asked the reason why the winter is higher. Dr. Ostro further explained that many pollutants come into play in different ways; the biomass, nature of gas constituent, adequate chemistry and other combination theory that change all the time. Dr. Ostro added that the data collected will depend on the country. Dr. Bornstein further clarified that ozone produces a lot of Nitrate particles in the summer and Nitrate is higher because it does not pull until it is colder and thus wood burning in winter along with the higher concentration of PM2.5. Other reasons why PM2.5 in California is typically different according to Dr. Ostro's study is greater indoor penetration and people spending more time outdoors.

Methodologies in this study included:

Methodology I, Time-series regression analysis used follows that of Ostro et al. (2006) and many others (HEI 2003) linking PM2.5 to mortality. Daily counts of mortality that involve hospital admits modeled as a Poisson distribution, conditional on time-varying covariates of time, weather, and day of week, were also used. The use of smoothing splines to control for time, temperature and humidity was also part of the methodology used. (Splines are non-linear data-driven functions that smooth the relation of mortality and time).

Dr. Ostro pointed out the All-cause mortality in Sacramento County for 2000-2003 and emphasized the differences between the Mortality and Time without Smooth versus the Mortality and Time with Smooth on the presentation. He noted that the Smoothing made the control variation for seasonality more effective.

Methodology II comprised formulae for Log(Mt), as well as examining single-day pollutant lags of 0 to 4 confounders like Smoking, Occupational exposure, and Indoor pollution that were taken into consideration.

Methodology III involved the random effects meta-analysis used to combine individual county results. Sensitive analysis like varying degree of freedom for time and weather, penalized spline, treatment of missing data and seasonal-specifics of cool season (October to March), were also used.

Results of the findings are as follows:

PM2.5 in the California Study of 2000-2003 showed that some counties have higher concentrations than others. The highest concentration is found in Riverside County (27.1 units), followed by Orange County (21.5) mean daily PM2.5 per microgram. Los Angeles came third (20.8), Kern (19.5), Fresno (17.5), Santa Clara (13.9) while Contra Costa and Sacramento had 12.8 and 12.6 respectively and San Diego came with the least amount of concentration of 15.3 mean daily PM2.5.

The components of PM2.5 studied in six California Counties where mean PM2.5 =19.3 ug/m3; resulted in OC having the highest of 7.1 mass (ug/m3) followed by NO3 with 5.5 mass (ug/m3); SO4 came out with 1.9; EC resulted in 1.00; S was .5 while CU+Fe+Zn, K, Si and Cl were at the barest minimum i.e., a little above zero. However, some components noted as Other on the graph had the PM2.5 components of approximately 2.7 mass (ug/m3).

With regards to the Temporal Correlations of PM2.5 and Components, the presentation table showed the moderation of the chemicals overtime; with NO3 being the highest with 0.65 correlation. Also sulfate is seen to be higher in the summer.
The selective summary of meta-analytic associations for alternative lags is color-coded (red = $p<0.05$; green = $p<0.10$). Red denotes the most significant chemicals with health related problems. The chemicals that are most prominent with cardiovascular health issues are PM2.5 (3), NO3 (3); denoted in green, SO4 (3), Zn (3), EC (2), Fe (2), K (2) also denoted in green. These chemicals; PM2.5, EC, OC, NO3, SO4, Cu, Fe, K, according to the findings do not show mortality caused by respiratory problem except for Zn that rate at 1 ($p<0.10$). Mortality at ages above 65 is seen in PM2.5 (3), Zn (3), and EC (2) all denoted with green is (equivalent to $p<0.5$) while NO3 is (0) denoted in red. At this point, Phil Martien commented that it is surprising that not much respiratory death existed in the findings.

The Cardiovascular Mortality 3 knots/year and 4 knots/year graph show the range of distribution possibilities of Excess Risk per Inter Quartile Range (IQR) and Species and Lag Days of 75^{th} to 25^{th} concentration risk of pollution per year differential. Knots were used to default the smoothing to see which is smoother. The graph shows which chemicals are at significant 5 point level; these are PM2.5, SO4, and Zn while NO3 is at 10 point level whereas above zero percent is the normal range.

Selective summary of meta-analytic associations for Winter showed the cardiovascular related mortality traced the following chemicals; PM2.5 (3), NO3 (3), SO4 (3), Zn (3), all denoted in red ($p<0.05$) and EC (2), Fe (2), K (2), Zn (2), denoted in green ($P<0.10$). Respiratory related

mortality was SO4 (3). Chemicals related to death at age above 65 that were significant included PM2.5, Fe, K and Zn.

Excess risks per microgram (ug/m3) for Cardiovascular Mortality of pollutants were tabularized with corresponding lags and percent change per microgram. The pollutants (PM2.5, EC, OC, NO3, SO4, K, Fe, Zn,) all have lags of three (3). Fe has the highest percent per microgram of 8.38 followed by K with 7.51, EC has 2.38, SO4 has 1.22 while PM2.5, OC, NO3, have 0.18, 0.34 and 0.36 respectively. However, Zn has overwhelmingly 194.9 and Sam Altshuler commented if Zn lined very well; that is if Zn is actually 194.6 or 1.946. Dr. Ostro responded that these numbers are not to be taken seriously and that 2.2% is the low estimate considering difference in measurement error and problems of measurement.

The Effect Modification and Mortality was examined with regards to gender, race and education. Cardiovascular mortality by education showed that non-high school graduates have about 10% while high school graduates is 46% of mortality related to EC, OC, Nitrate , Zn and Iron. Dr Ostro added that education is a proxy for a whole bunch of lag but possibility includes exposure study shows that lower income, lack of medical care and lack of exercise and smoking may be prime factors.

Future Work for the study will be based on the following areas:

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

6. **Committee Member Comments/Other Business:** Chairperson Altshuler stated that Tom Cahill, Professor Emeritus, University of California Davis will be at the next meeting. The Committee thanked Dr. Ostro for his presentation and presented him with a token of appreciation from the Air District.

7. **Time and Place of Next Meeting**. The next meeting will be at 9:00 a.m., June 11, 2007, 939 Ellis Street, San Francisco CA 94109.

8. **Adjournment**. 12:11p.m.

/s/ Chioma Dimude
Chioma Dimude
Acting Executive Secretary

AGENDA: 3

Advisory Council Technical Committee

April 16, 2007

Comparison of SCAQMD MATES and BAAQMD CARE Programs*

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PM sizing	No PM sizing
Wood smoke from wildfires	Special study markers for wood smoke – residential wood-burning, carbon dating
No lube oil measurements	No lube oil measurements
10 fixed sites; 6 microscale sites	23 fixed sites: metals @ 4 sites; VOC @ 22 sites; PAHs, carbonyls @ 3 Sites; elemental carbon @ 6 sites
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Estimated Risks**	
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No estimates of non-cancer risks	Chronic and acute non-cancer risks: acrolein represents 48% of chronic and 94% of acute non-cancer risk. Important sources are mobile and aircraft.
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Topics for future discussion: Lube oil, PM sizing, Acrolein trend.

The Effects of Fine Particle Species on Daily Mortality and Morbidity in California

Bart Ostro, Ph.D., Chief
Air Pollution Epidemiology Unit
Office of Environmental Health Hazard Assessment (OEHHA)
CalEPA

Acknowledgments

OEHHA

Rachel Broadwin
Shelley Green
Brian Malig
Lindsey Roth
Janice Kim
Rupa Basu
Melanie Marty

UC Davis

Wen-Ying Feng

DHS

Michael Lipsett

I. Introduction – background on PM2.5

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

III. Mortality study

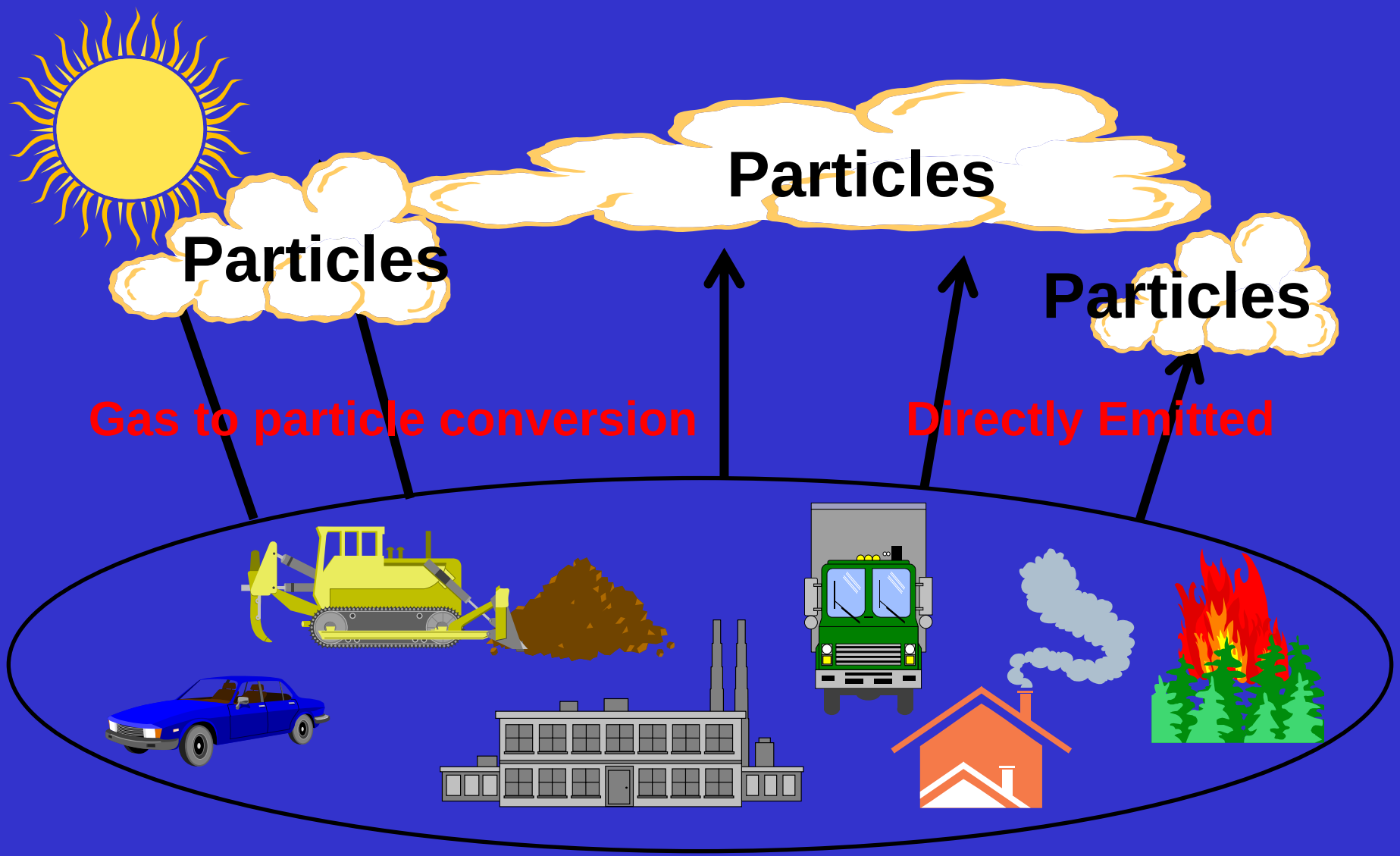
IV. Findings on susceptible subgroups (prelim)

V. Findings on morbidity (prelim)

VI. Biologic mechanisms

VII. Summary

PM2.5 is a heterogeneous mixture of solids and liquids from multiple sources



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
  - Improve estimates of health impact assessment
  - May help explain heterogeneity in multi-city studies

Results of studies of 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 motor vehicles and residual oil-sulfates
- Burnett et al 2000 (Canada): sulfates, zinc, nickel and iron (NO3, EC, OC not measured)

PM2.5 in California different from that typically studied

- 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 certain components of PM2.5 associated with greater risks?**

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, NO3, SO4, 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:
   - o all-cause, cardiovascular, respiratory, and age > 65
   - o male/female
   - o race/ethnicity (White, Black, Hispanic)
   - o educational attainment (High school grads versus non-HSG)
4. Analysis restricted to counties with 180+ observations (total obs = 1870)

Data III

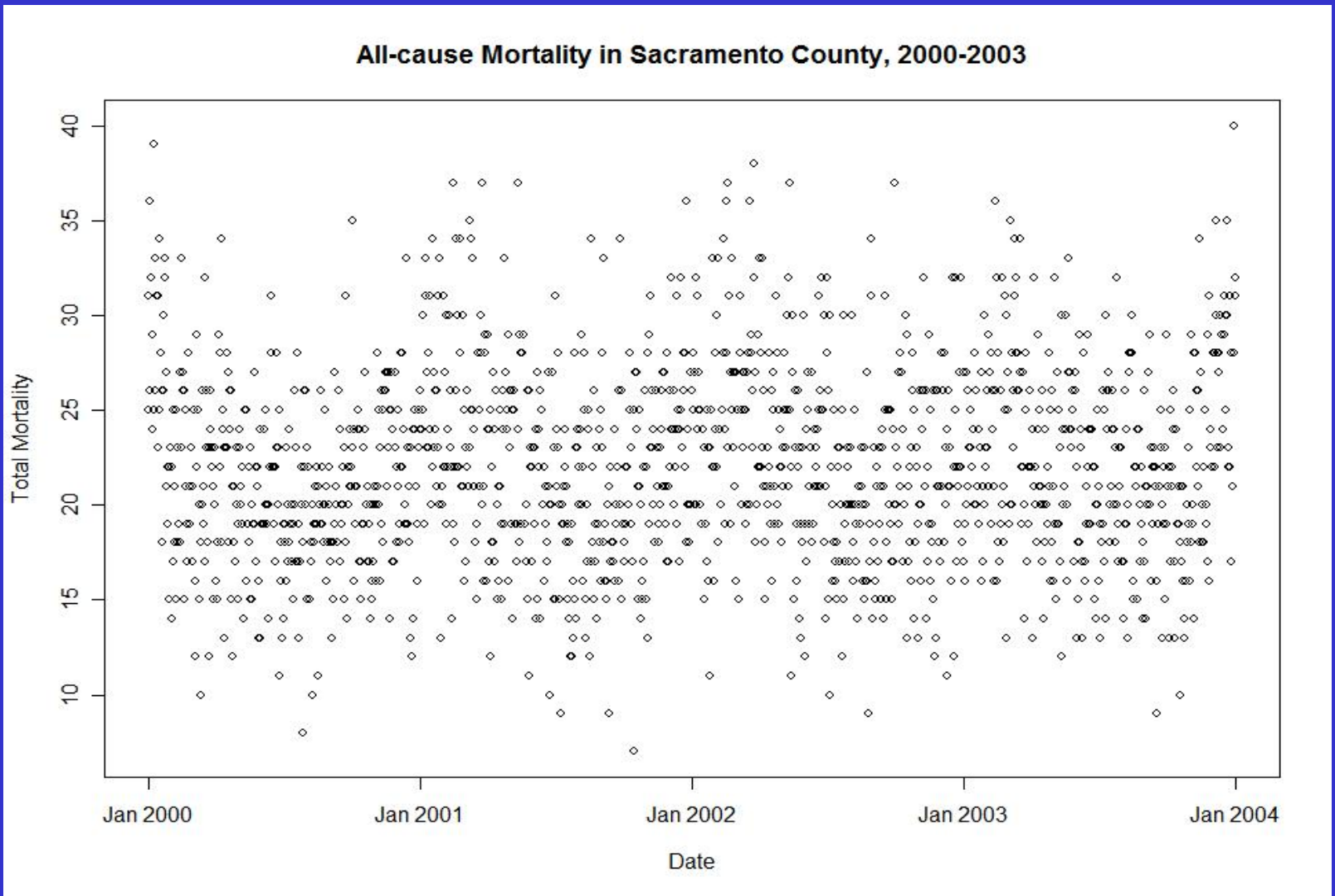
5. Daily hospital admissions: 9 California Counties (9.5 million admits) for 2000-2003 for the following outcomes:
   - Respiratory Disease
     - Asthma, Bronchitis, Pneumonia
     - By age
   - Cardiovascular Disease
     - Myocardial Infarction, Heart Failure, Dysrhythmia, Stroke
     - By race/gender

Methodology I

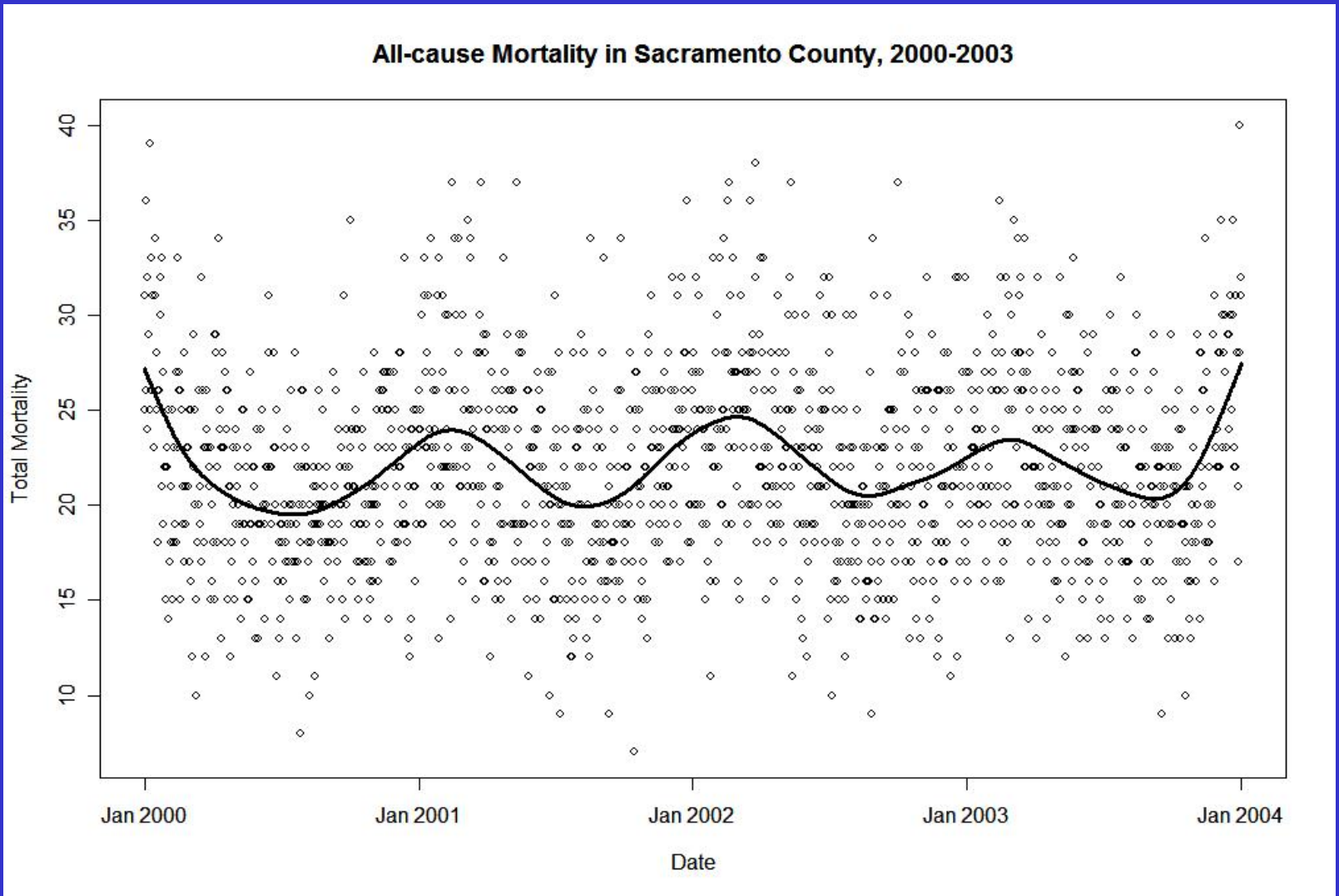
- Time-series regresssion analysis follows that of Ostro et al. (2006) linking PM2.5 to mortality, and many others (HEI, 2003)
- Daily counts of mortality (hospital admits) modeled as Poisson, conditional on time-varying covariates (time, weather, day of week)
- Use smoothing splines to control for time, temperature and humidity

  (splines = non-linear data-driven functions that smooth the relation of mortality and time)

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
- Note non-confounders:
  - Smoking
  - Occupational exposure
  - Indoor pollution

Methodology III

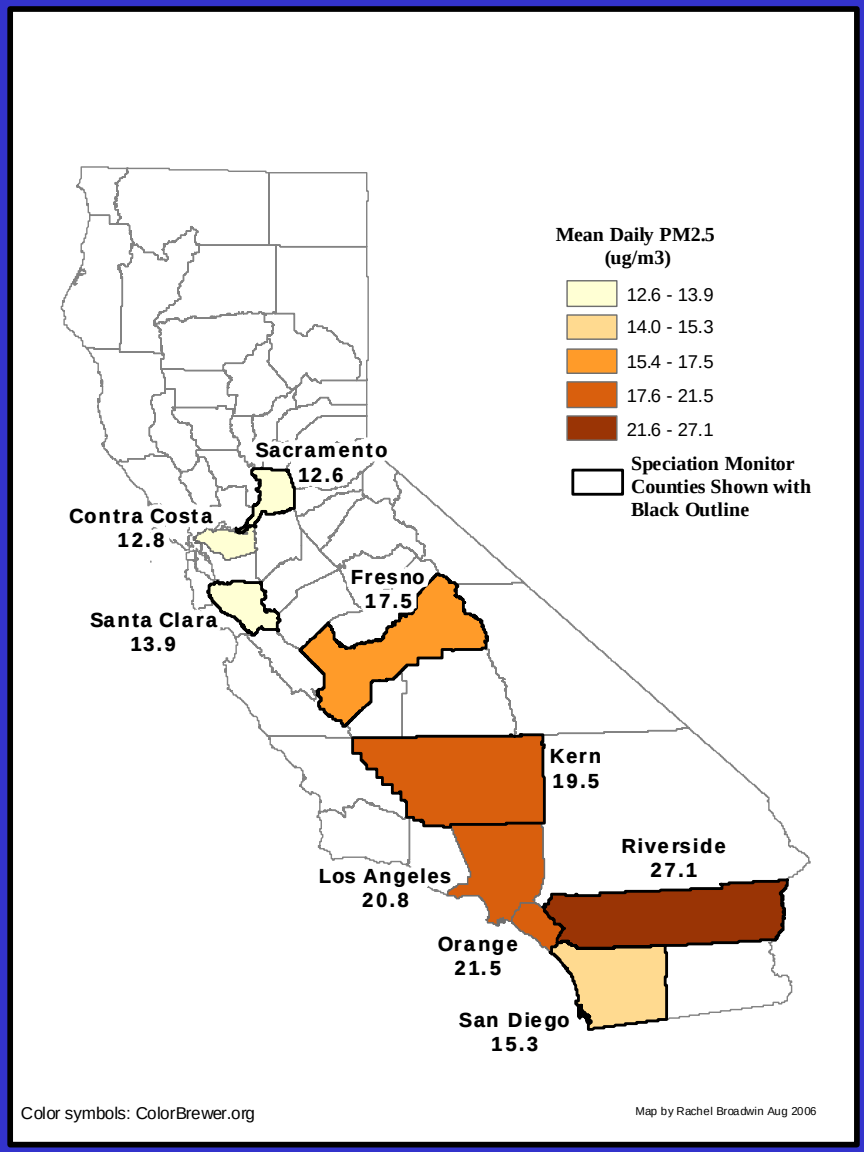
- Random effects meta-analysis used to combine individual county results
- Sensitivity analyses:
  - Varying df for time, weather
  - Penalized splines
  - Treatment of missing data
  - Season-specific (cool season = Oct – Mar)

Results

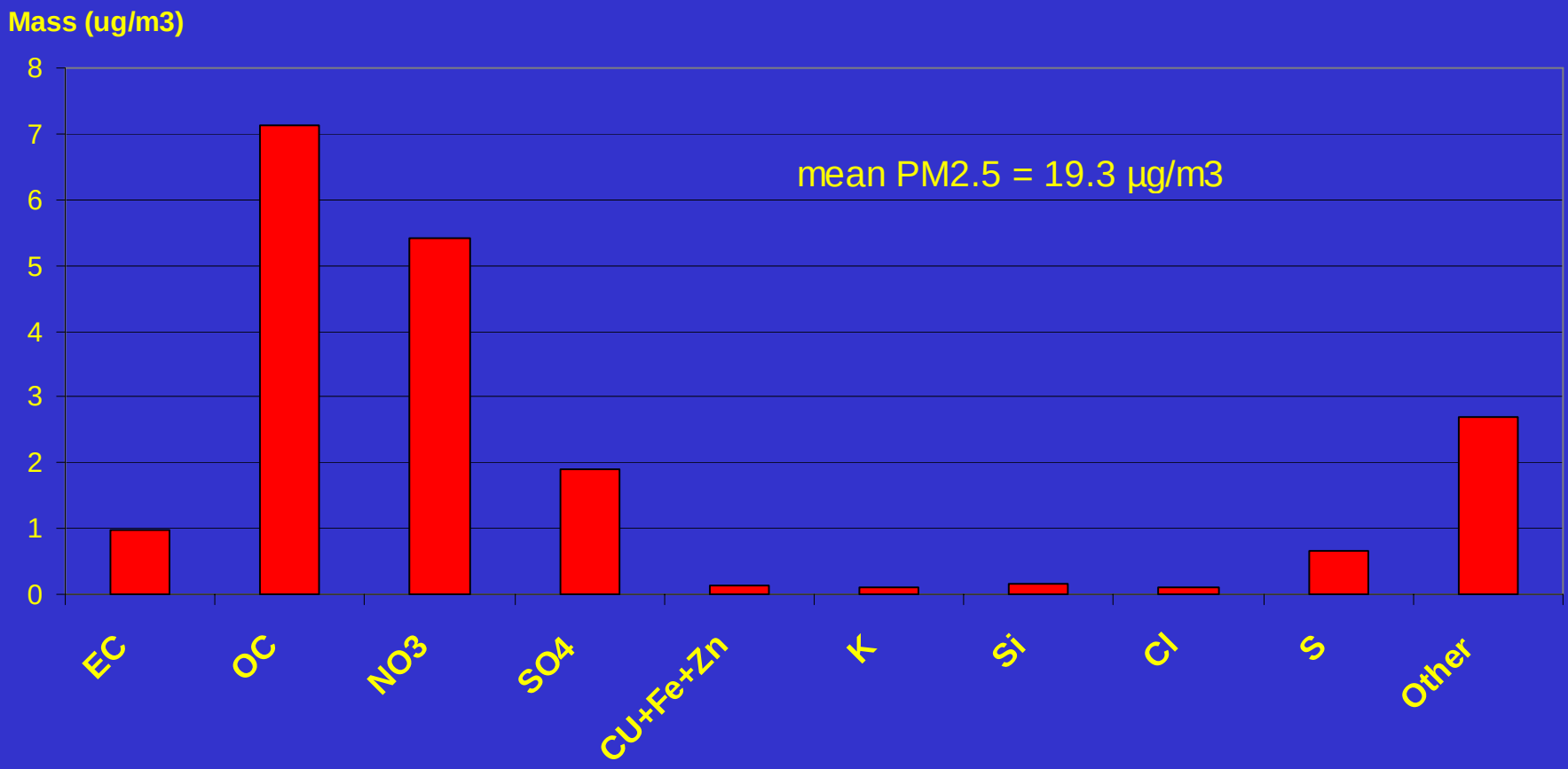
www.ehponline.com

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



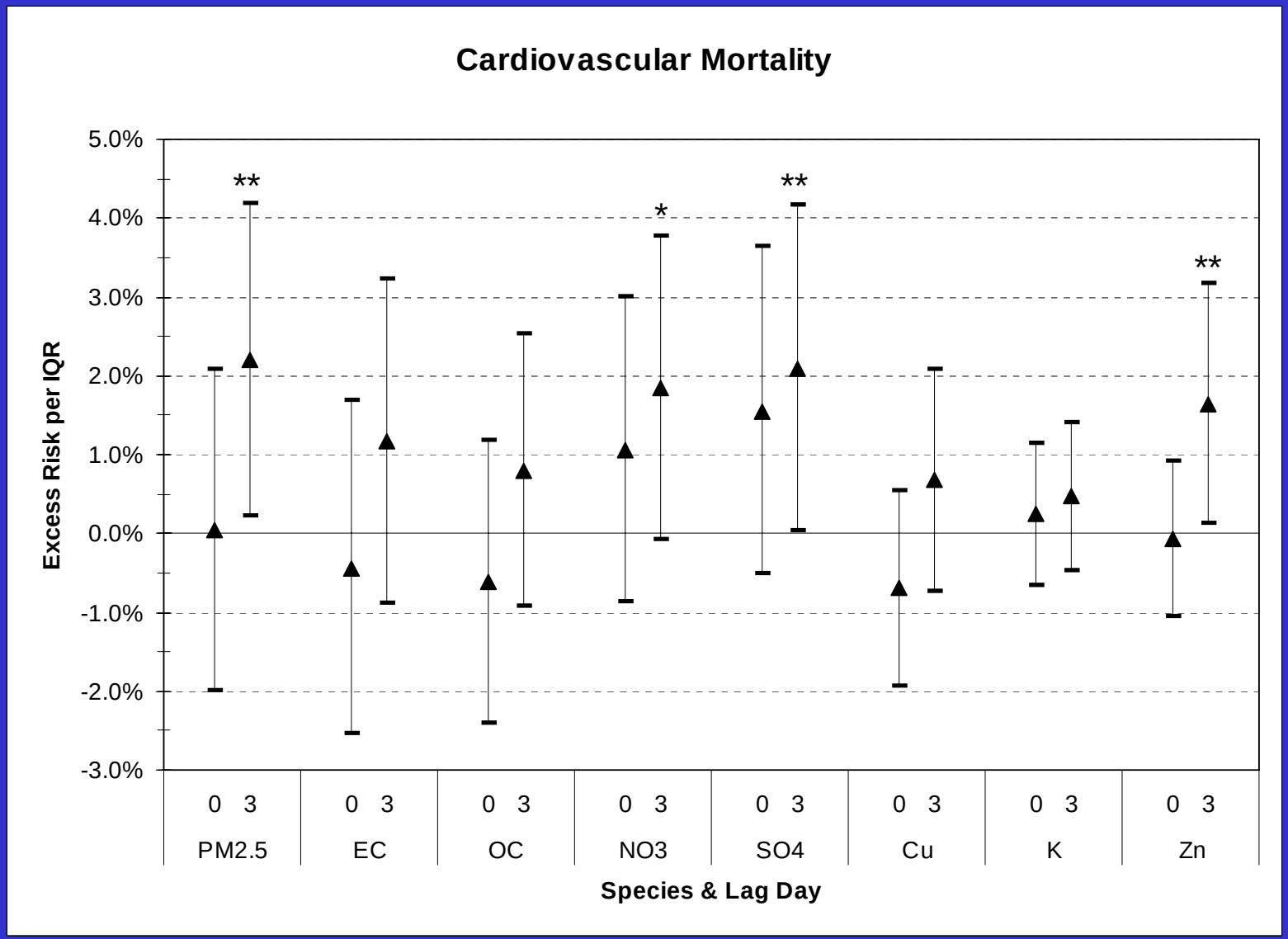
Temporal Correlations of PM2.5 and Components

	PM2.5	EC	OC	NO3	SO4	Cu	Fe	K
PM2.5	1							
EC	0.53	1						
OC	0.62	0.61	1					
NO3	0.65	0.41	0.44	1				
SO4	0.32	0.05	0.12	0.35	1			
Cu	0.23	0.29	0.26	0.15	0.10	1		
Fe	0.38	0.48	0.39	0.23	0.16	0.32	1	
K	0.52	0.48	0.57	0.34	0.09	0.26	0.41	1
Zn	0.51	0.53	0.50	0.45	0.11	0.23	0.37	0.45

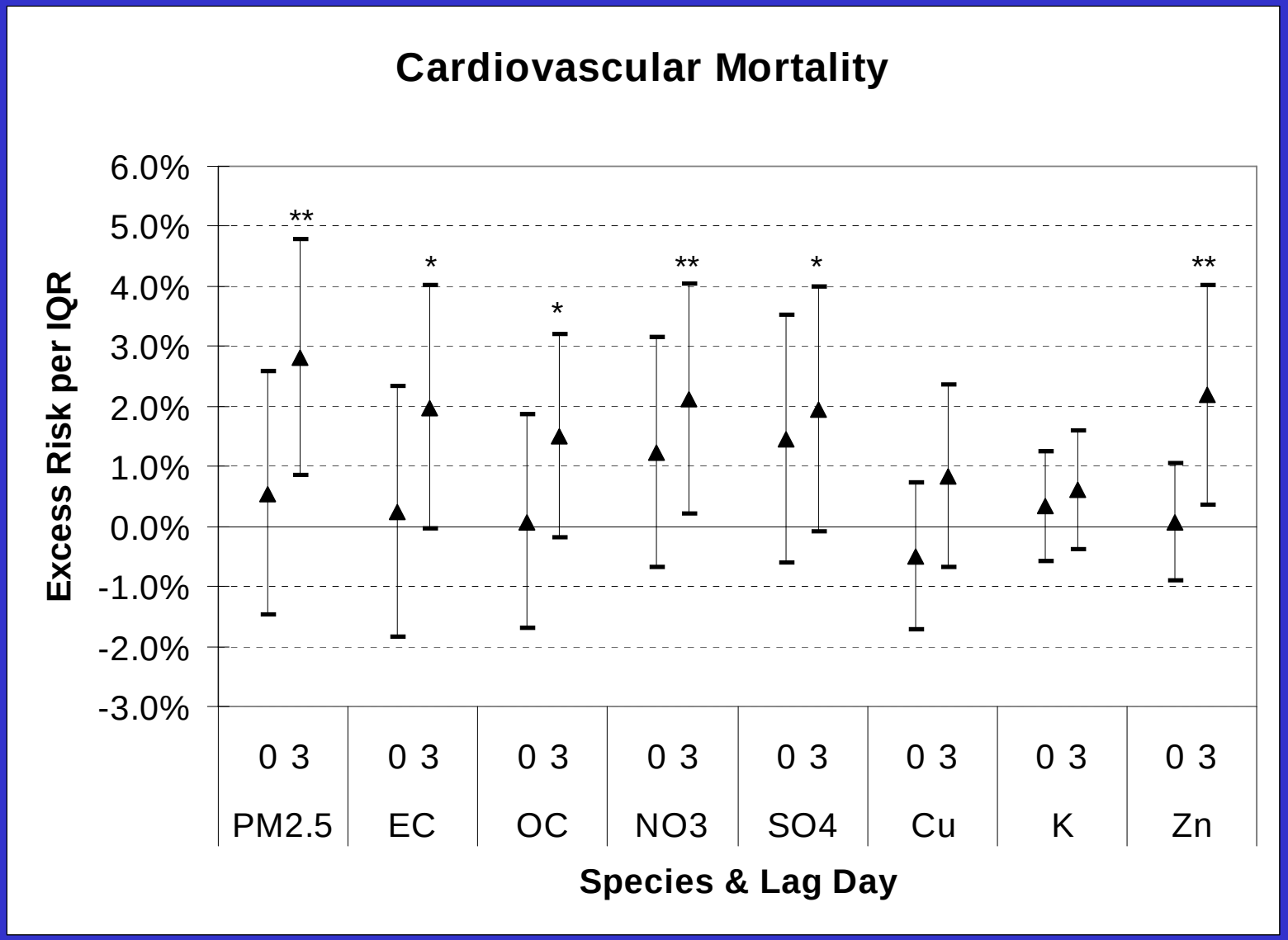
Selective summary of meta-analytic associations for alternative lags (red = p<0.05; green = p<0.10)

	All-cause	Cardiovascular	Respiratory	Age > 65
PM2.5		3		3
EC		2		2
OC				
NO3	0	3		0
SO4		3		
Cu				
Fe		2		
K		2		
Zn		3	1	3

CV Mort, 4 knots/yr



CV Mort, 3 knots/yr



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

	All-cause	Cardiovascular	Respiratory	Age> 65
PM2.5		3		3
EC		2		
OC				
NO3		3		0
SO4		3	3	
Cu				
Fe		2		2
K	2	2		2,3
Zn		2,3		3

Excess Risks Per μg/m^3 for Cardiovascular Mortality

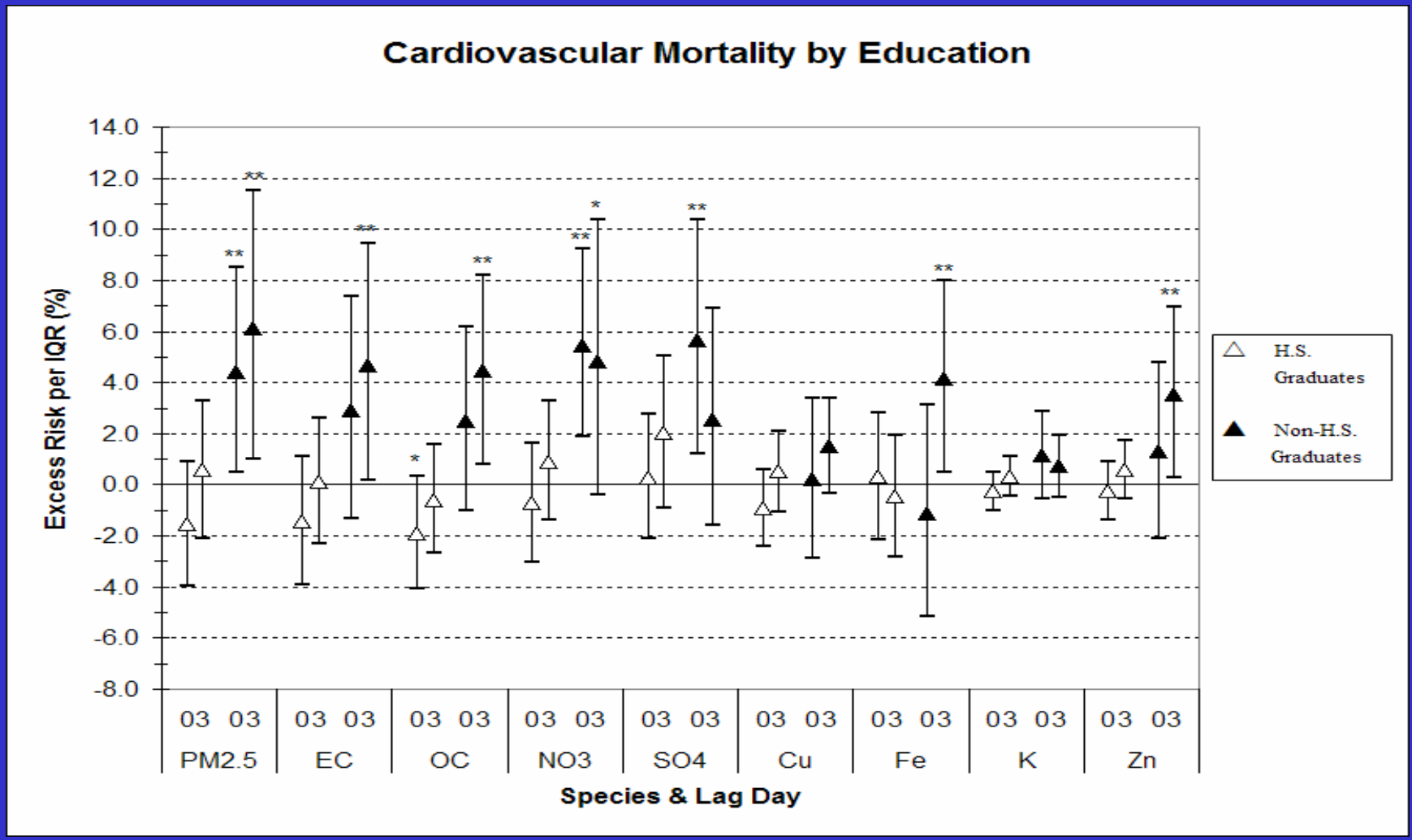
Pollutant	Lag	%change per ug/m3
PM2.5	3	0.18
EC	3	2.38
OC	3	0.34
NO3	3	0.36
SO4	3	1.22
K	3	7.51
Fe	3	8.38
Zn	3	194.6

Effect Modification and Mortality

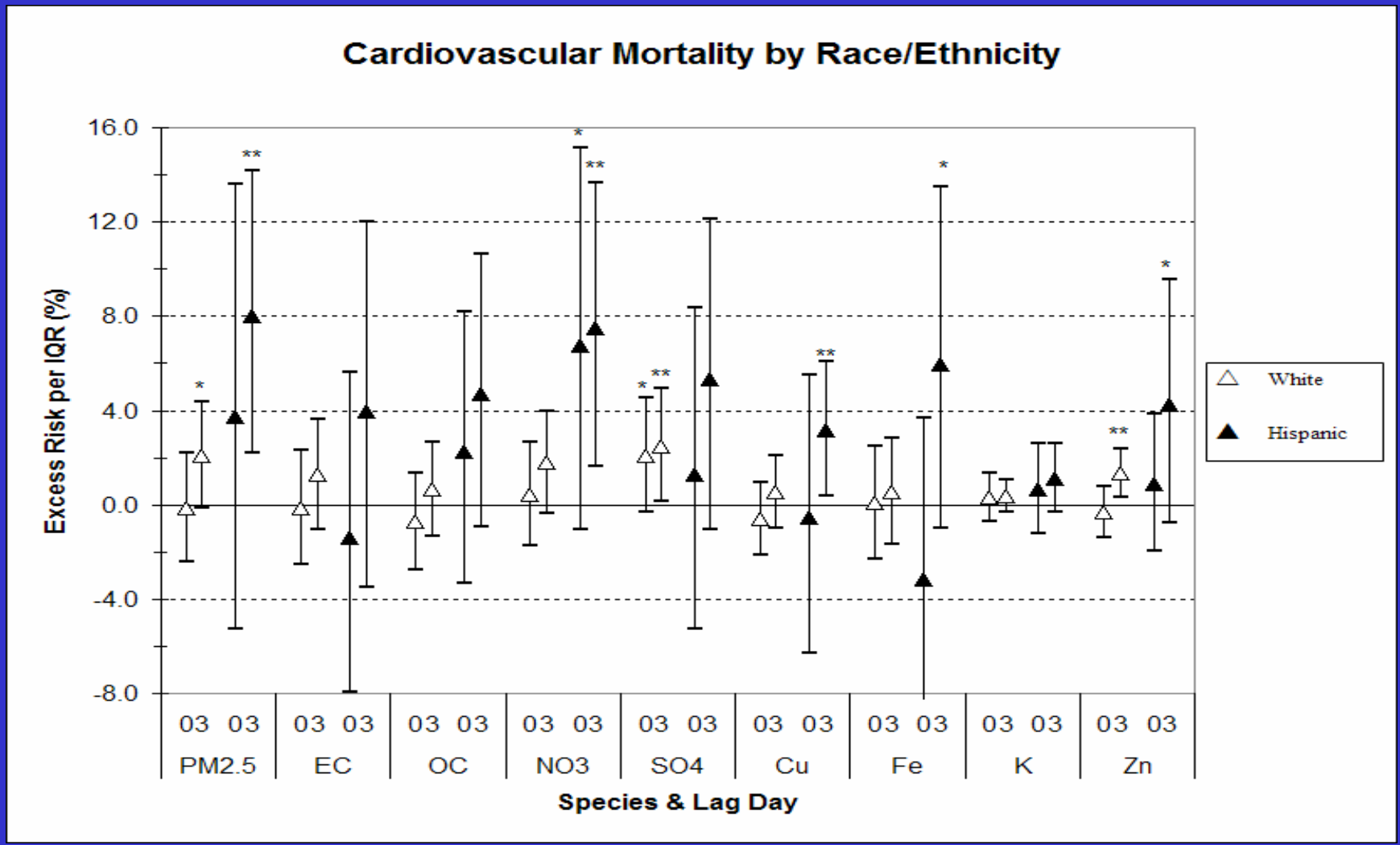
Are there subgroups that are particularly susceptible to the components of PM2.5?

Examined:
Gender/Race/Education

Cardiovascular Mortality by Education



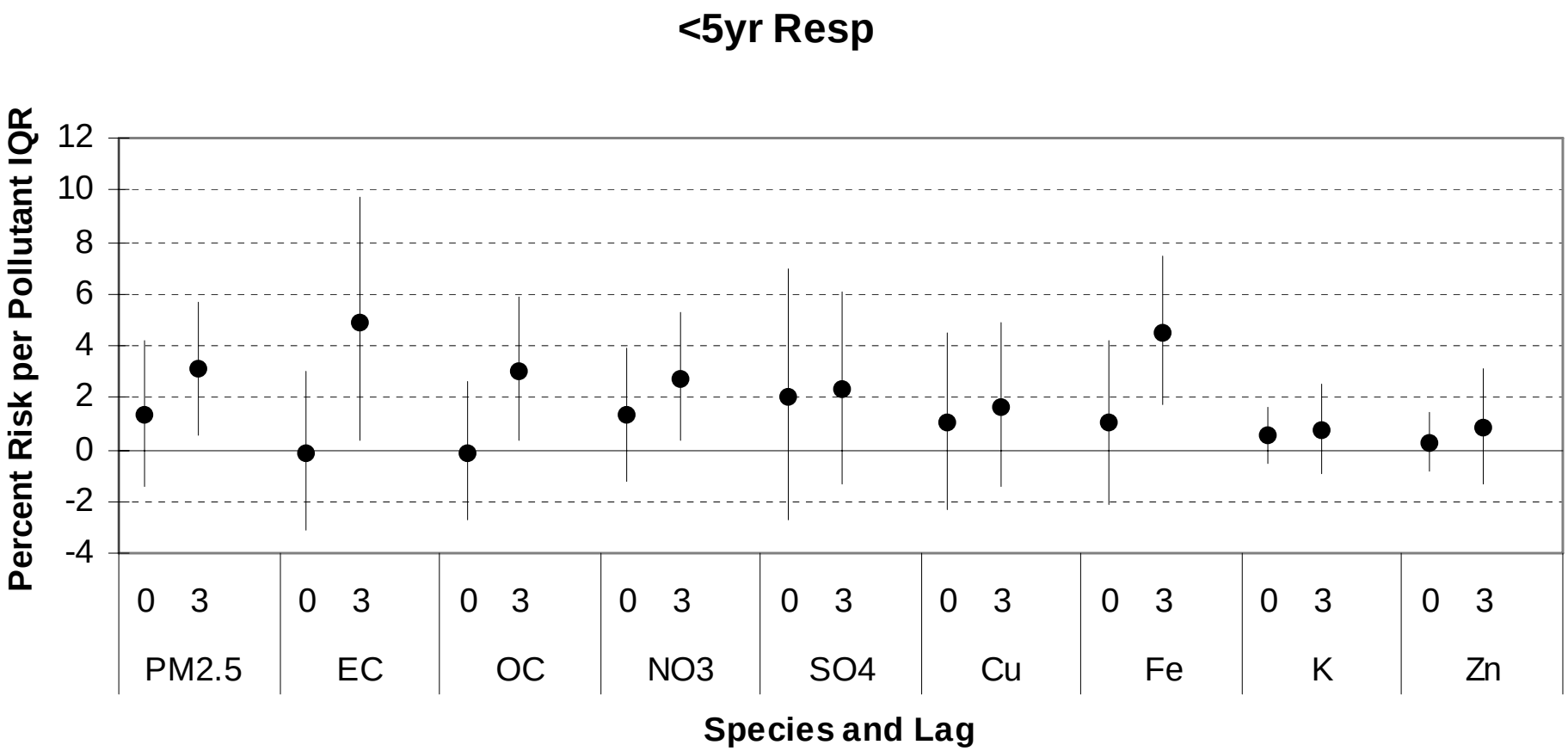
Cardiovascular Mortality by Race/Ethnicity

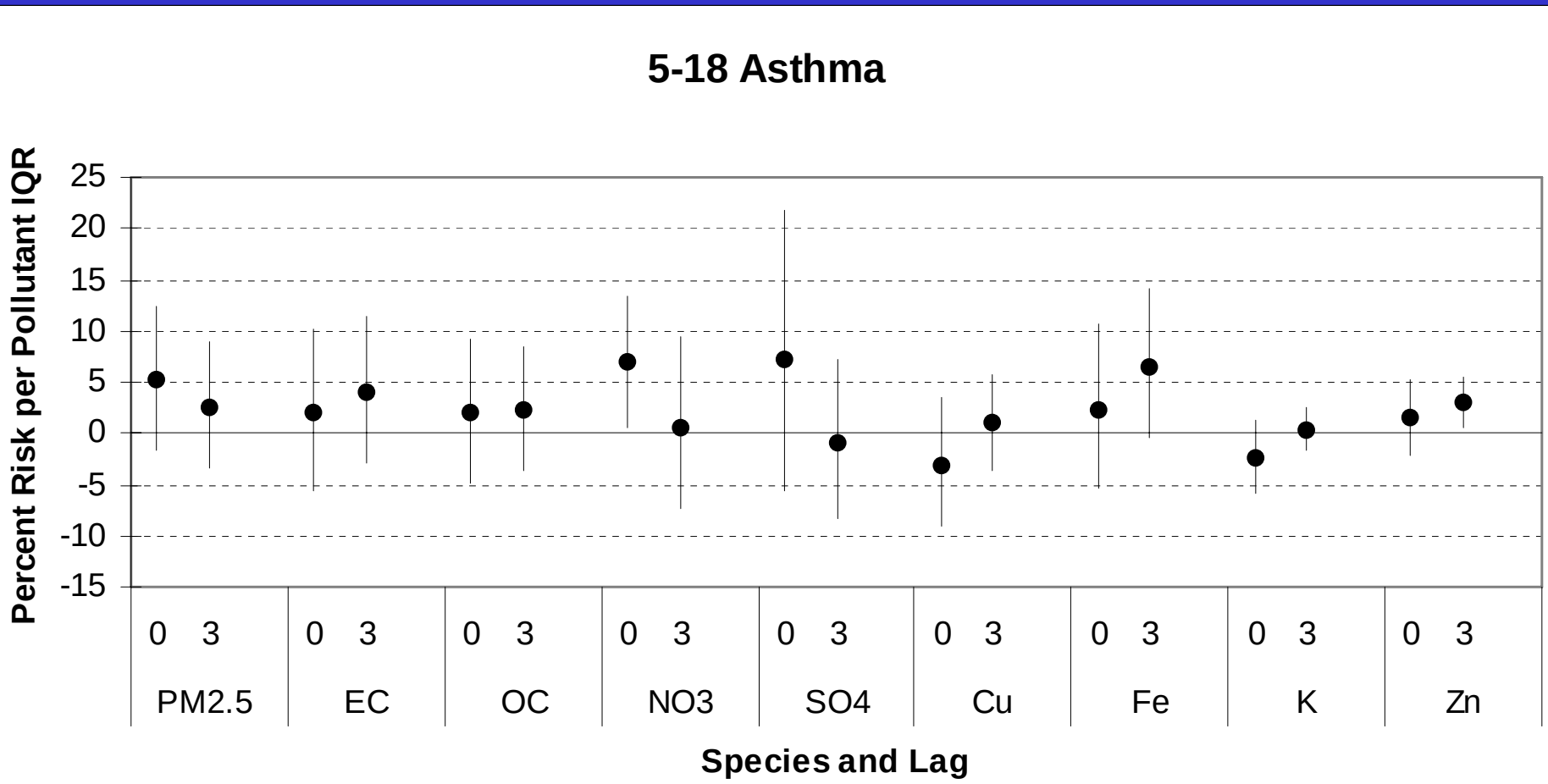


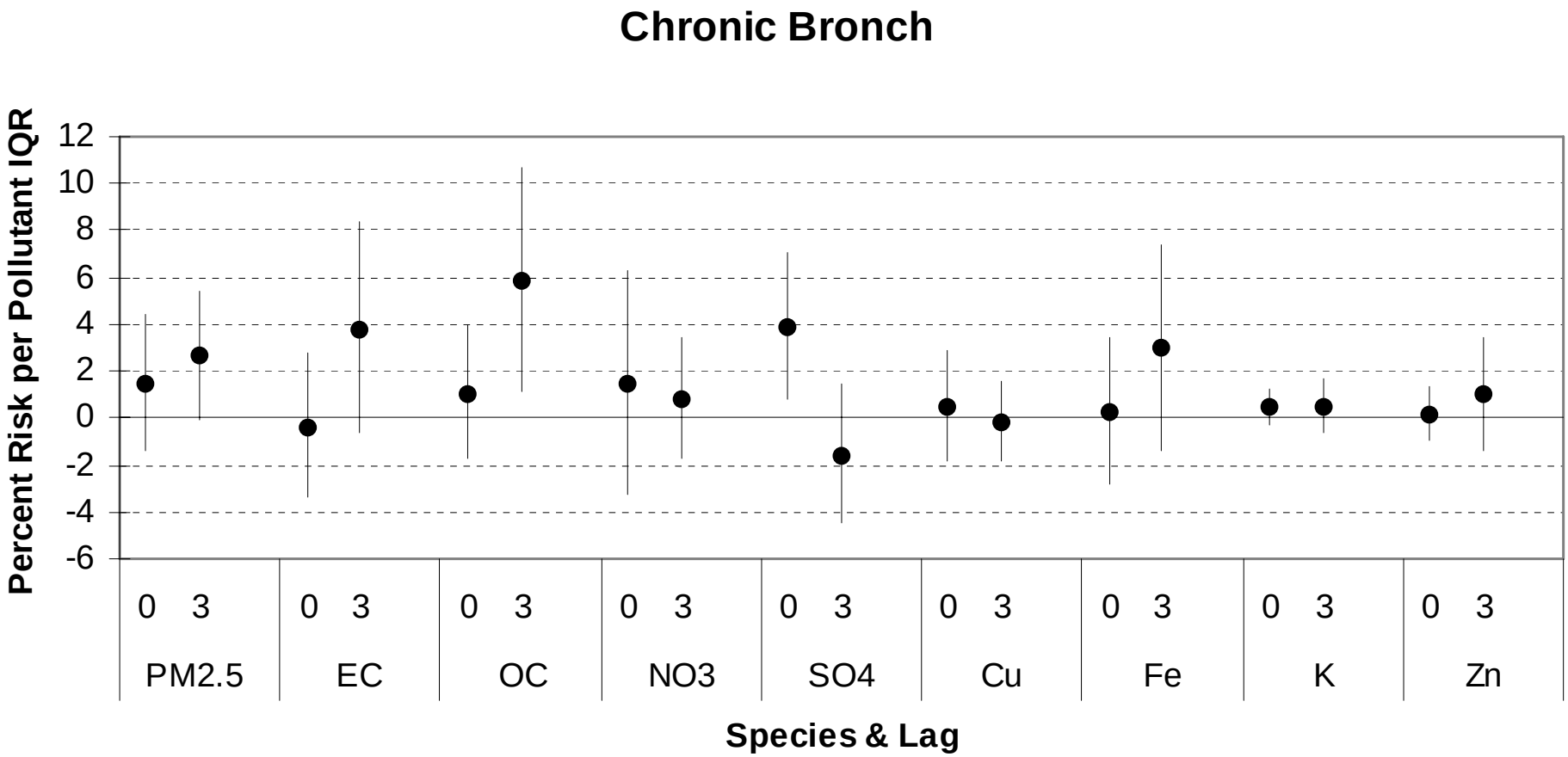
Results for Daily Hospital Admissions

Respiratory Diagnoses Results

- All Respiratory Conditions, age < 5
- Asthma, ages 5-18
- Asthma, age 19-45
- Chronic bronchitis

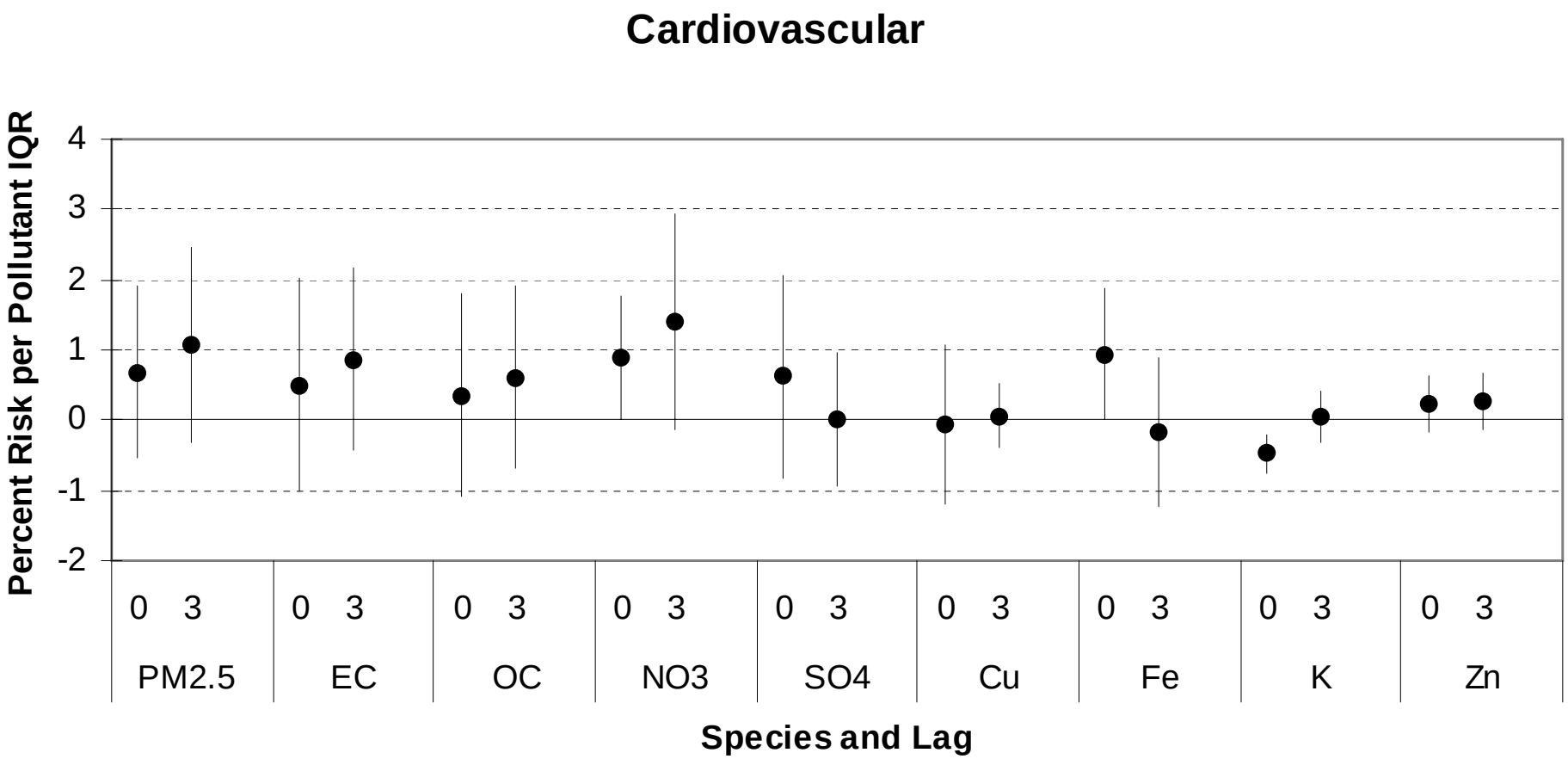

<5yr Resp
Percent Risk per Pollutant IQR
12
10
8
6
4
2
0
-2
-4
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag


5-18 Asthma
Percent Risk per Pollutant IQR
25
20
15
10
5
0
-5
-10
-15
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag

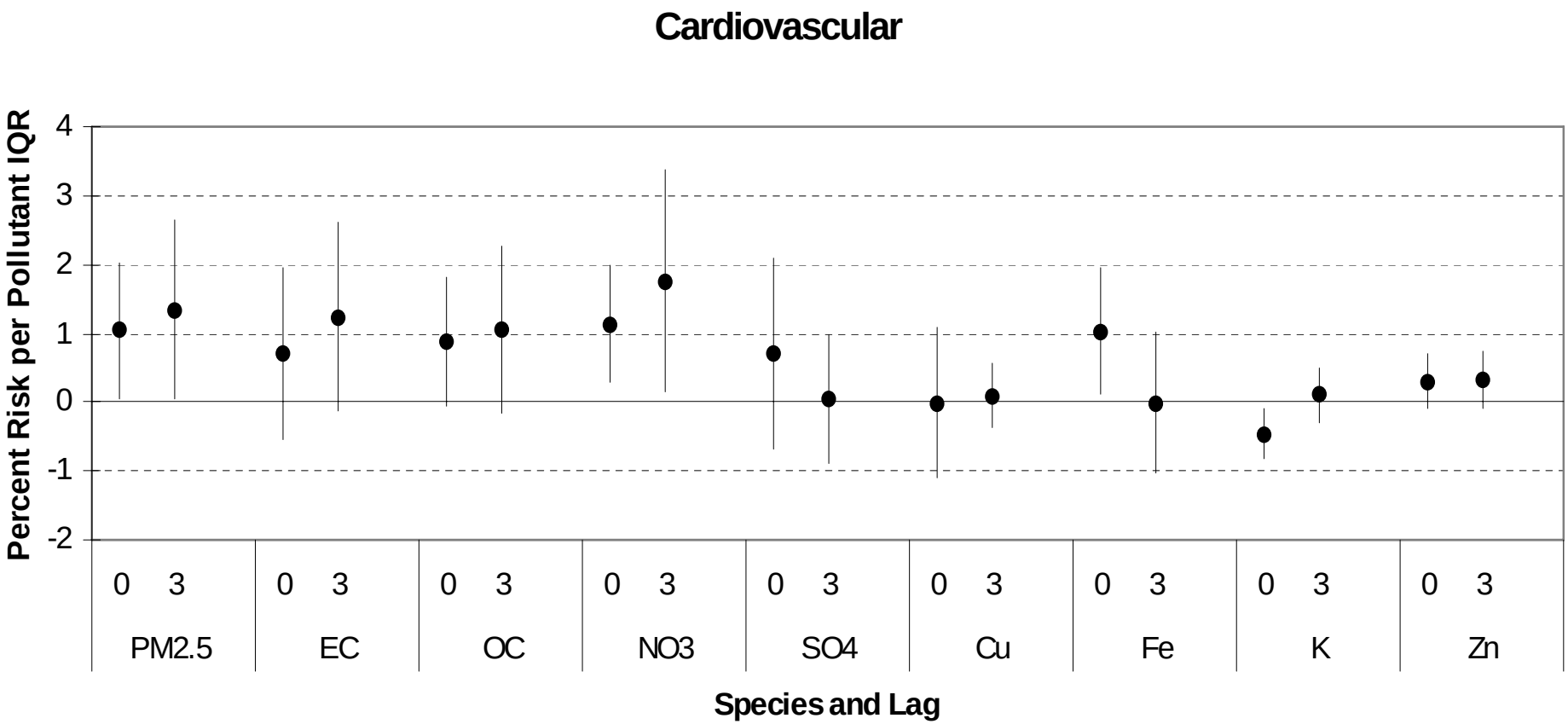

Chronic Bronch
Percent Risk per Pollutant IQR
12
10
8
6
4
2
0
-2
-4
-6
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species & Lag

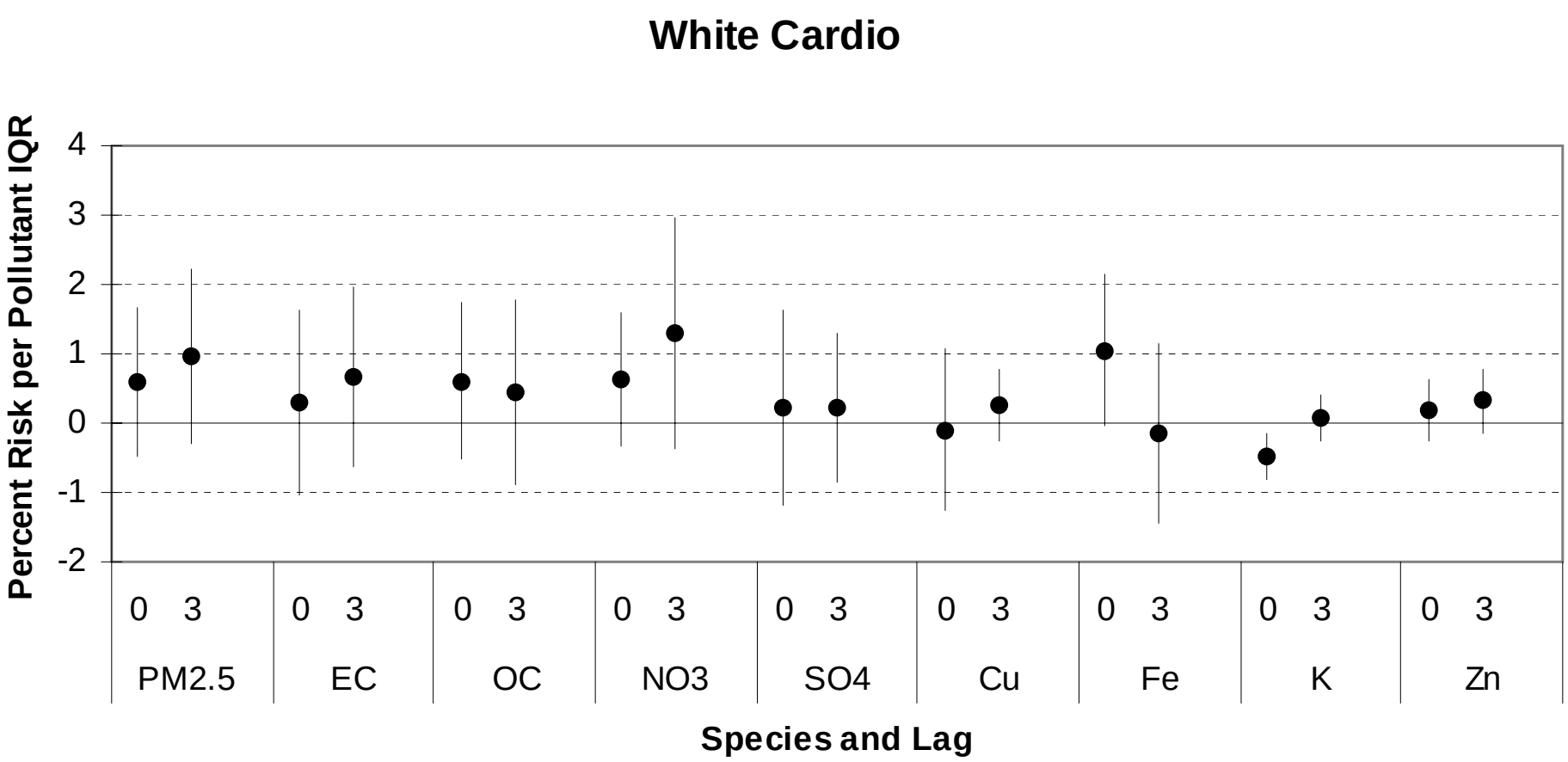
Cardiovascular Diagnoses Results

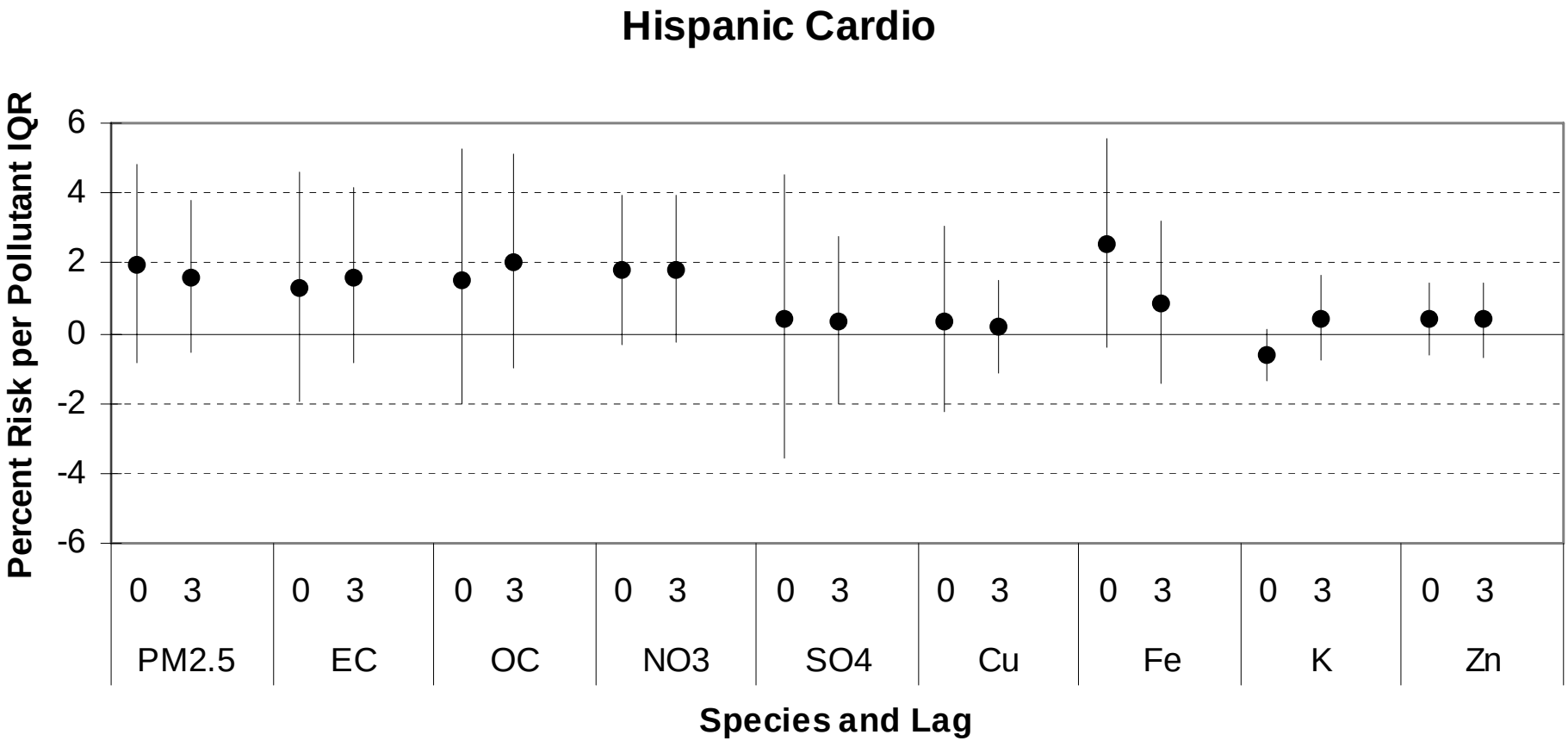
- All Cardiovascular (4 and 3 k/yr)
- White Cardiovascular
- Hispanic Cardiovascular
- Acute MI
- Cardiac Dysrhythmia
- Heart Failure
- Stroke

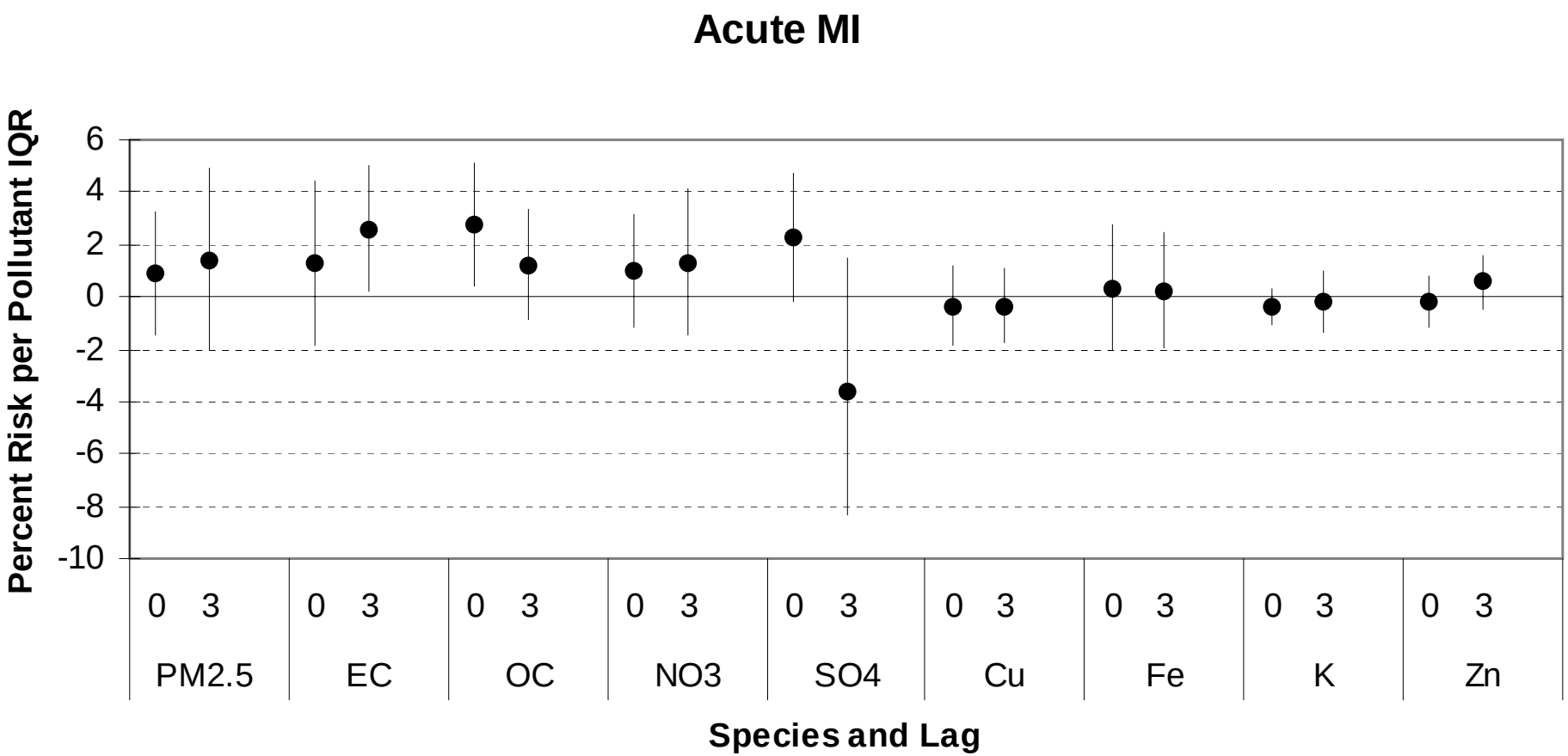

Cardiovascular
Percent Risk per Pollutant IQR
4
3
2
1
0
-1
-2
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag

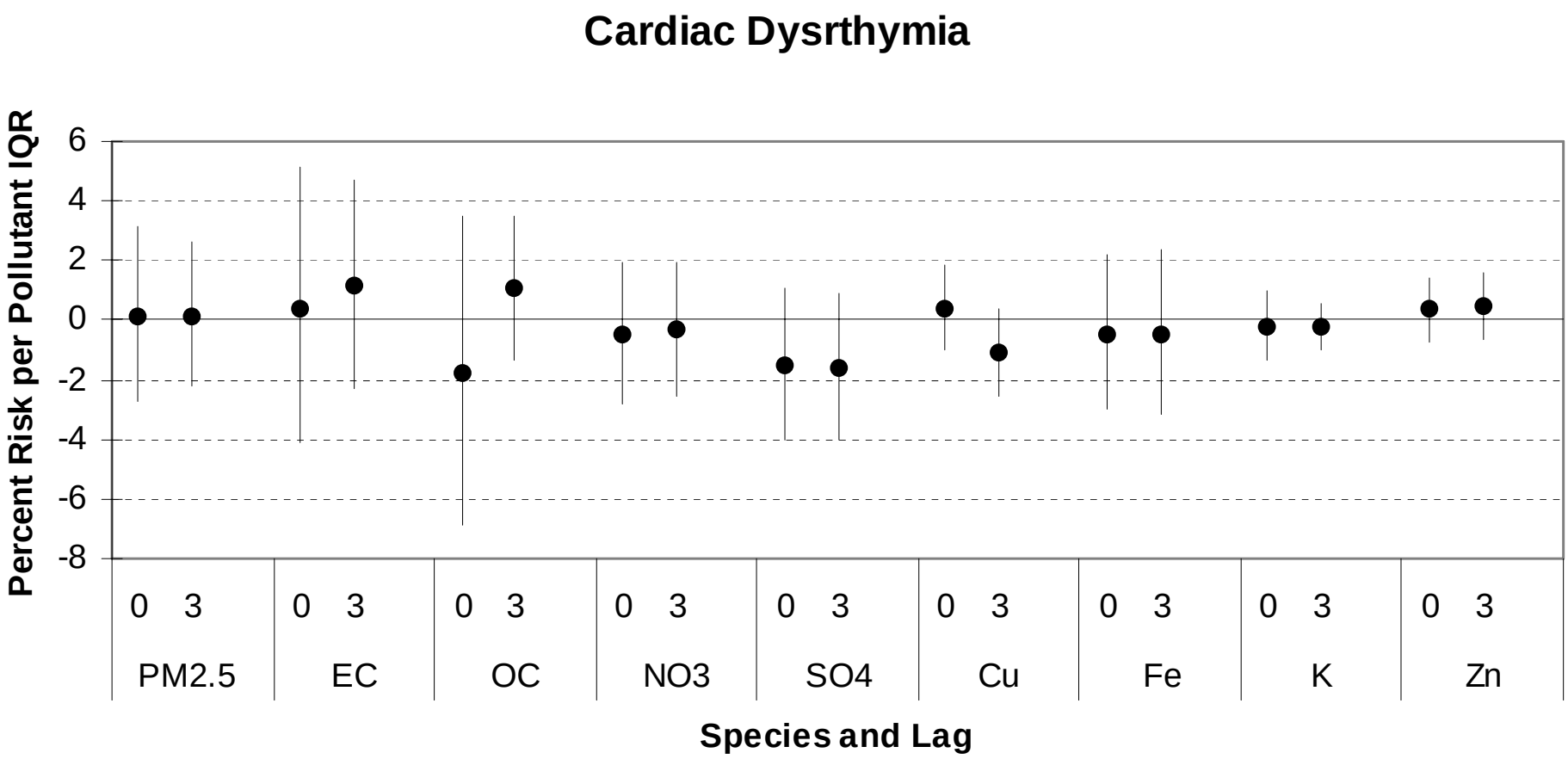
3 knots/year smooth of time



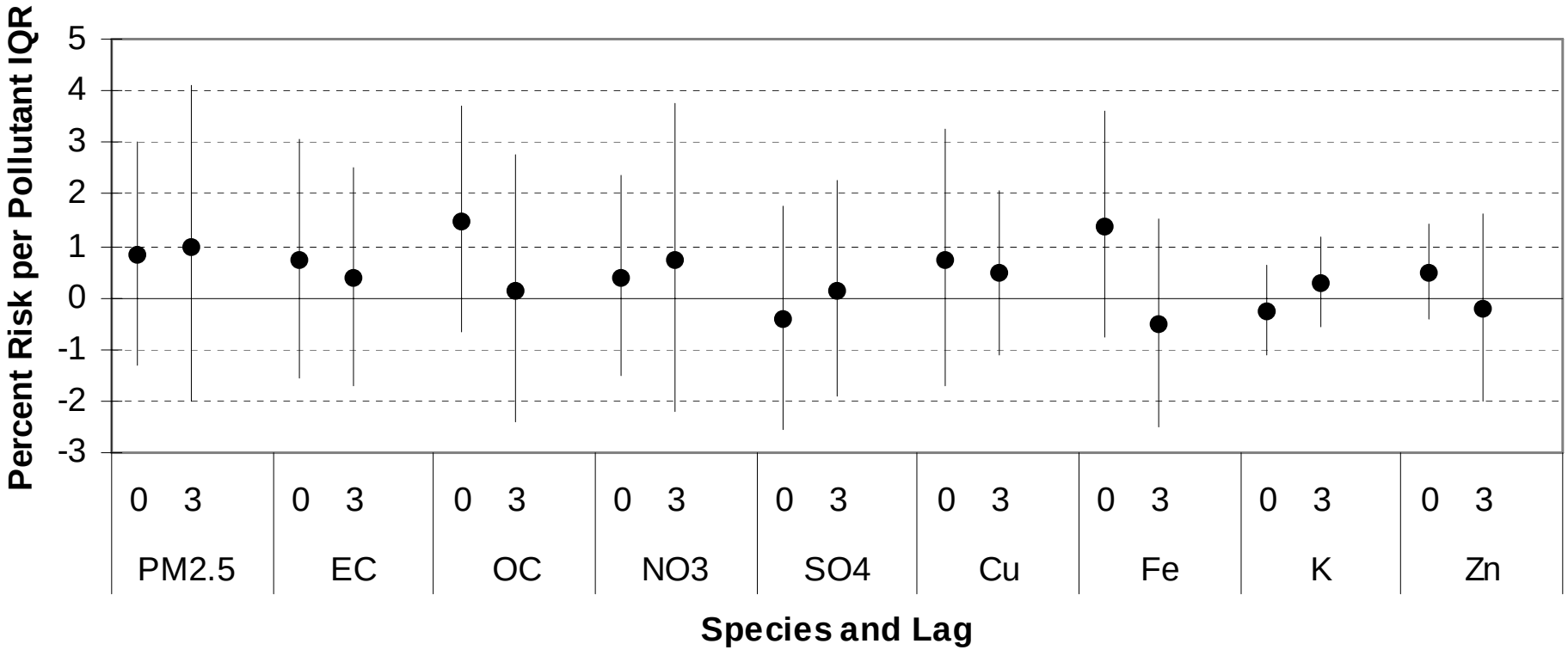

White Cardio
Percent Risk per Pollutant IQR
4
3
2
1
0
-1
-2
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag

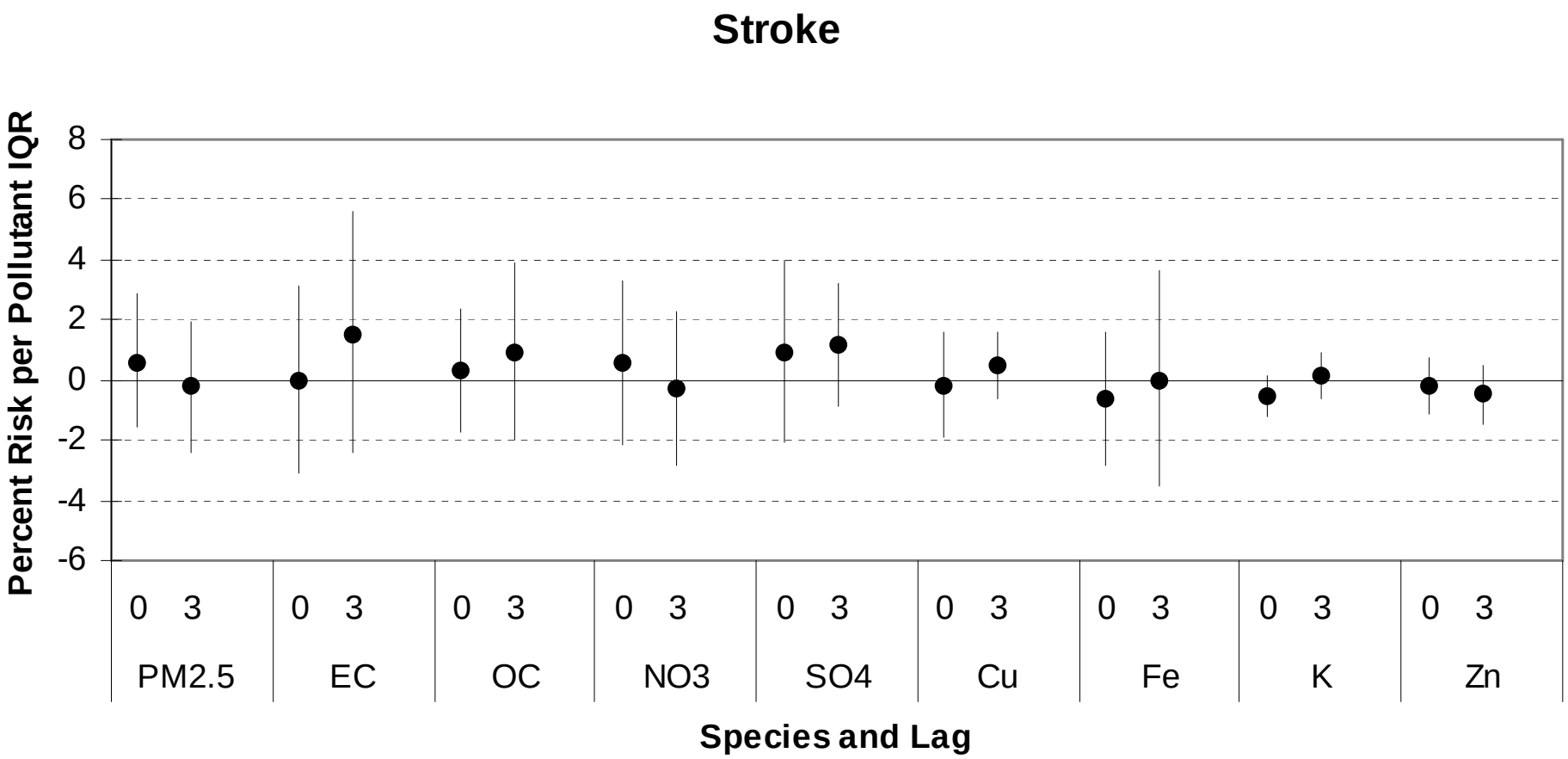

Hispanic Cardio
Percent Risk per Pollutant IQR
6
4
2
0
-2
-4
-6
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag


Acute MI
Percent Risk per Pollutant IQR
6
4
2
0
-2
-4
-6
-8
-10
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag


Cardiac Dysrthymia
Percent Risk per Pollutant IQR
6
4
2
0
-2
-4
-6
-8
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag

Heart Failure




Stroke
Percent Risk per Pollutant IQR
8
6
4
2
0
-2
-4
-6
0 3
PM2.5
EC
OC
NO3
SO4
Cu
Fe
K
Zn
Species and Lag

Biological Mechanisms I

- Evidence from epidemiologic, toxicologic and human clinical studies suggest plausible mechanisms for cardiovascular mortality and morbidity

- Among the components of PM2.5, mechanisms for CV mortality/morbidity and respiratory outcomes have been investigated most extensively for diesel exhaust (EC/OC).

Biological Mechanisms II

- EC/OC associated with oxidative stress (Cho 2005; Serensen 2003), ECG changes (Henneberger 2005), vascular reactivty (Urch 2004), ST depression (Lanki 2006), HRV (Schwartz 2005)
- PM2.5 (or traffic) associated with MI (Peters et al., 2004; Zanobetti and Schwartz, 2005; von Klot et al. 2005; Pope et al, 2006)
- Zn/CU/V (fuel combustion, brake wear, lube oil, tire dust) with fibrinogen (Huang 2003) and oxidative stress (Schlesinger et al., 2006; Ghio, 2004)

Summary of Findings I

1. Mortality associated with particles from gasoline and diesels (EC, OC, NO3, Cu, Fe, Zn), wood smoke (EC, OC, K) and other combustion sources
2. Notable effects for cardiovascular disease, especially MI, and for respiratory disease < age 5

Summary of Findings II

3. Apparent effect modification by race/ethnicity and SES

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

4. Excess mortality risks for IQR between 1–2% but 2x greater in susceptible subgroups

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

Summary of Findings III

6. Sample size is small – stronger associations possible as are spurious results
7. Measurement issues
    - o Species might be marker for another correlated pollutant
    - o Differential instrument error
    - o Differential spatial variabililty

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