Particulate Matter: Perspectives on its Health Effects and Sources

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MANE-VU
Manchester Village, VT
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The Health Effects Institute

30 years of providing impartial, high-quality science on health effects of air pollution

Broad Funding Base

- Joint core funding from
  - Government (U.S. EPA)
  - Industry (Worldwide Vehicle and Engine Manufacturers)
- Expanded partnerships with:
  - Oil, Chemical, other industries
  - DOE, FHWA, WHO, California, other agencies
  - USAID, ADB, Hewlett Foundation

Balanced Structure and Approach

- Independent Board and Expert Science Committees
  - Not affiliated with sponsors
  - ***Research Committee*** selects all research competitively
  - Separate ***Review Committee*** intensively peer reviews all results
- All results and data – both positive and negative – reported
- **Does *not* take policy positions**

A sample of our work…

- ***Targeted research***
  - Over 250 studies on ozone, carbon monoxide, particulate matter, diesel exhaust, benzene, butadiene, MTBE, others
- ***Re-Analysis***
  - e.g. Harvard Six Cities and American Cancer Society Studies on PM; 30 revised “time-series” PM studies
- ***Major Reviews***
  - Traffic Health Effects, MTBE, Diesel Exhaust Epidemiology, Air Toxics
  - Air pollution and Health in Asia

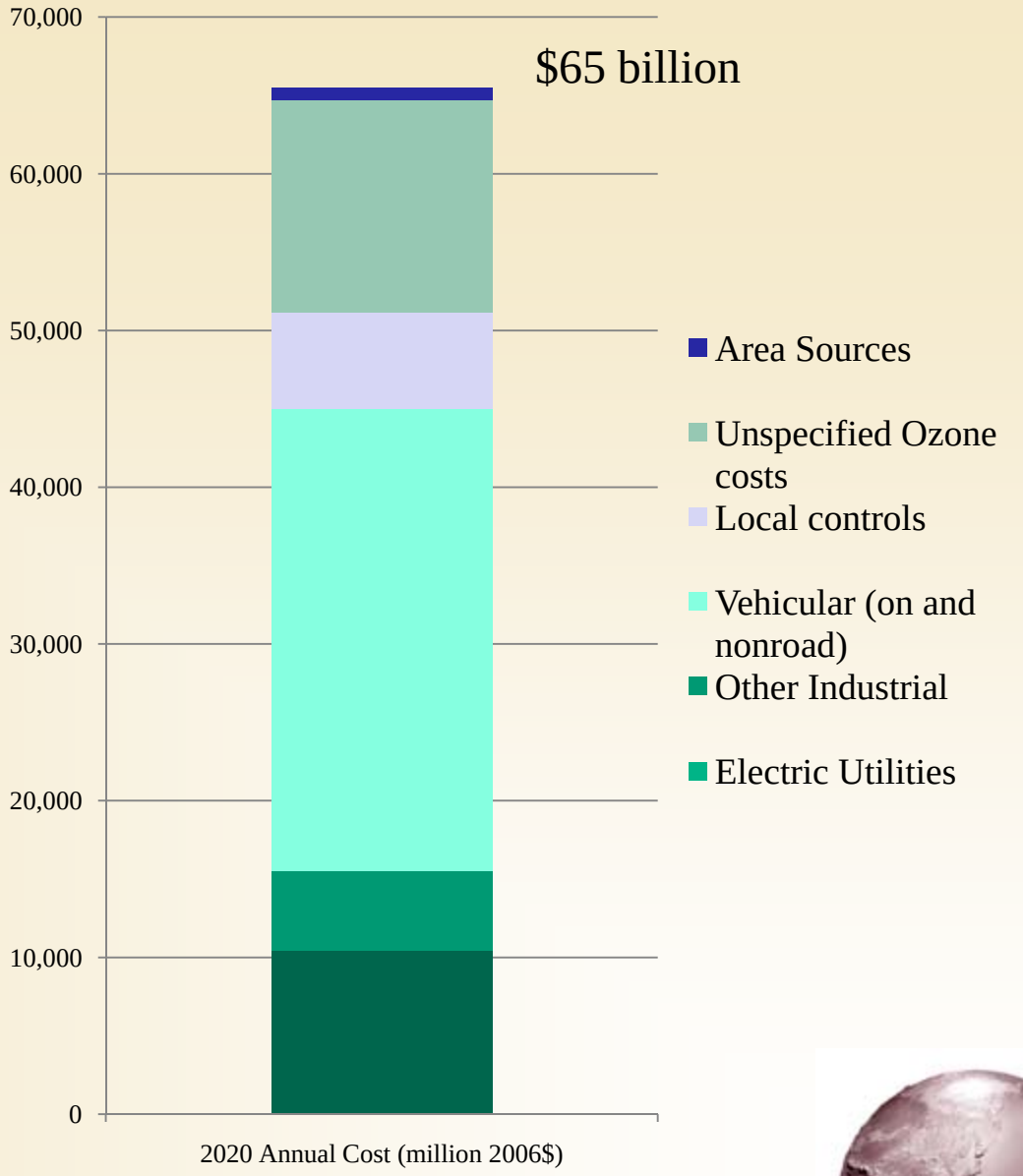
Overview

- Why the focus on PM?
- What constitutes good evidence on health effects?
- Do we have it?
- Can we identify what sources are responsible for PM and its effects?

“Environmental regulations are costly”

Measuring Clean Air Act Progress:

Costs
1990 – 2020
(Section 812 Prospective Analysis)



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But, Estimated Benefits Substantially Exceed Costs

- Primarily estimated **mortality** benefits
  - ~$1,800 billion for **PM alone**
- Even without mortality benefits,
  - morbidity, visibility and other benefits also exceed costs
- Significant unquantifiable benefits

Measuring Clean Air Act Progress:

Costs vs. Benefits

1990 – 2020

(Section 812 Prospective Analysis)

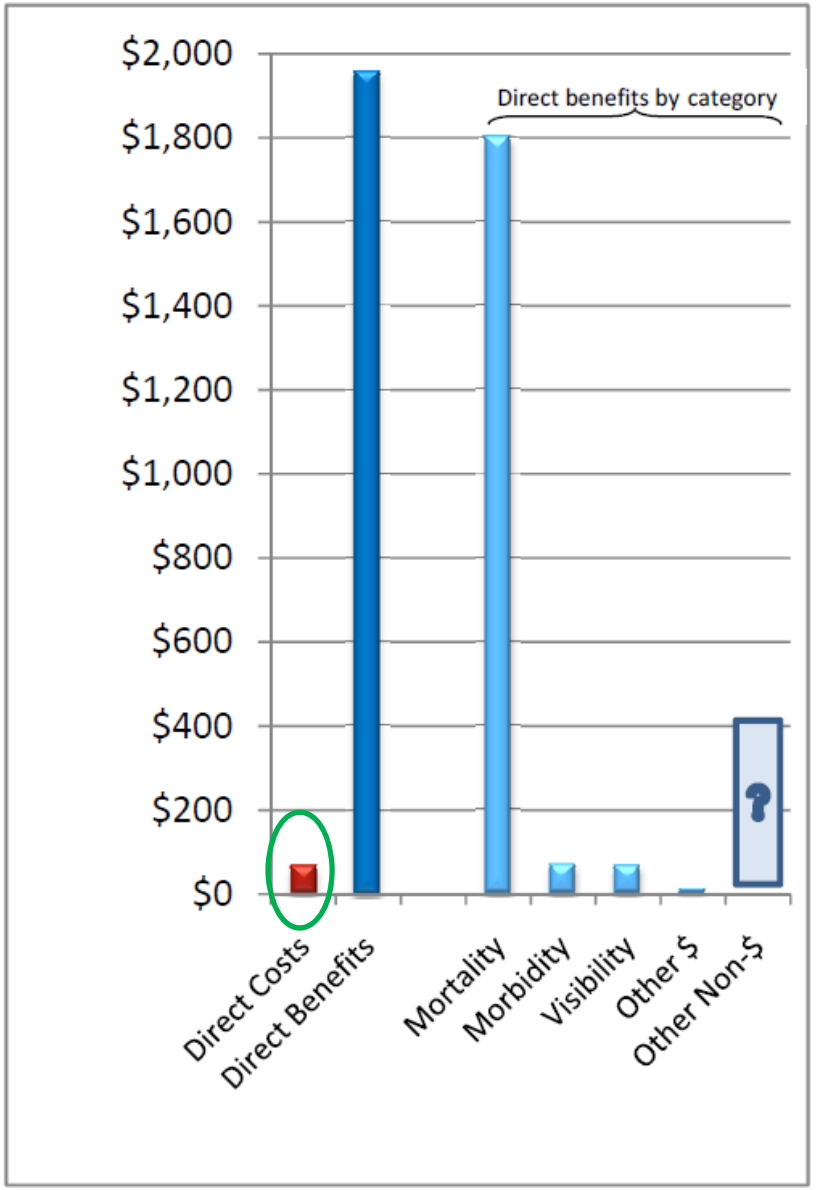


Exhibit 13. Year 2020 Primary Central Estimates of direct costs and direct benefits with breakdown of benefits by effect category. (In billions of year 2006 dollars).

PM mortality – from what?

- All-natural causes
  - Cardiovascular disease
  - Ischemic heart disease
  - Cerebrovascular disease
  - Respiratory disease
  - Lung cancer

Quantitative relationships estimated from epidemiologic studies

How do we know if these relationships are 'real'?

- Do we find similar findings in studies of different design?
- Are findings consistent in studies done different places by different people?
- Do we see evidence of a concentration response relationship?
- Is there some plausible biological mechanism for the effects we see?
- Have we adequately explored alternative explanations?

Alternative explanations

- Investigator and/or publication bias?
- Artifact of the methodology?
- Inadequate control for confounding factors?
- Exposure estimates are wrong?
- There's something about the components or sources that's different?
- Other unknown reasons?

Health Effects of Fine Particulate Air Pollution: Lines that Connect

C. Arden Pope and Douglas Dockery (2006)

Studies of Short term exposure (hours or days)

- Episodes
- Time-series studies
- Panel-based acute exposures
- Case-crossover

Studies of long-term exposure (years to decades)

- Cross sectional studies
- Cohort based mortality studies
- Cohort and panel-based morbidity
- Case control studies

Studies of regulatory interventions or “natural experiments”

Controlled experimental animal studies

The London Smog Episode December 1952

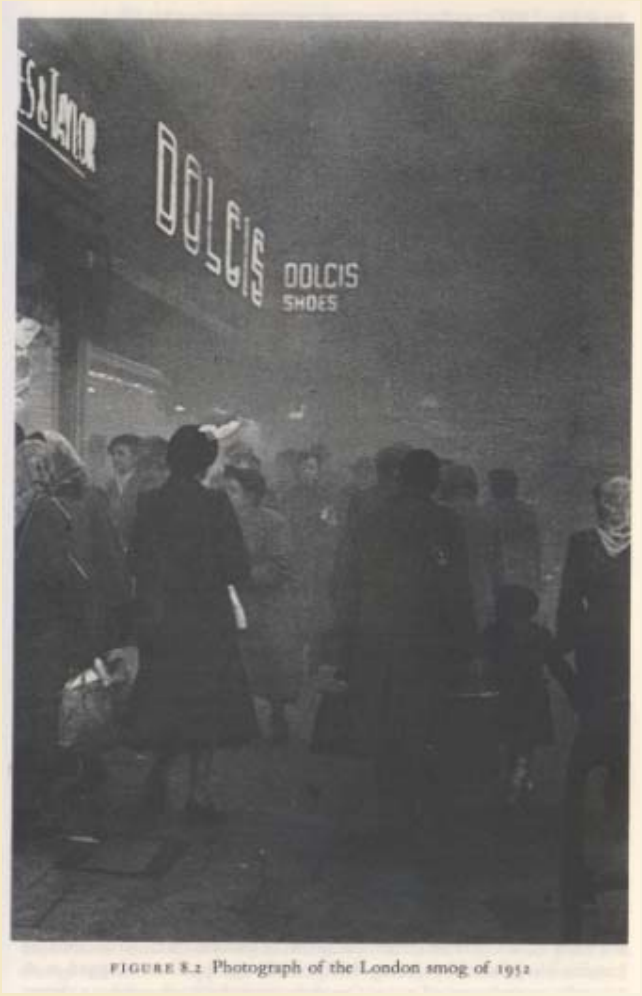
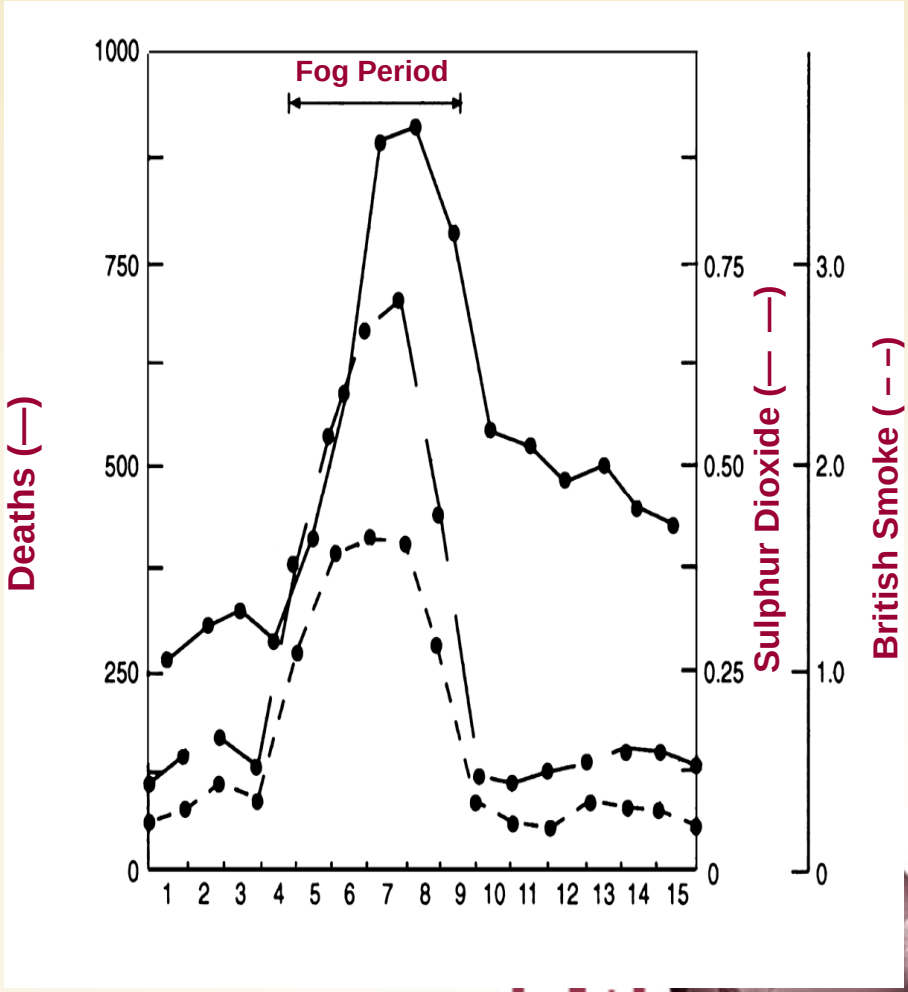
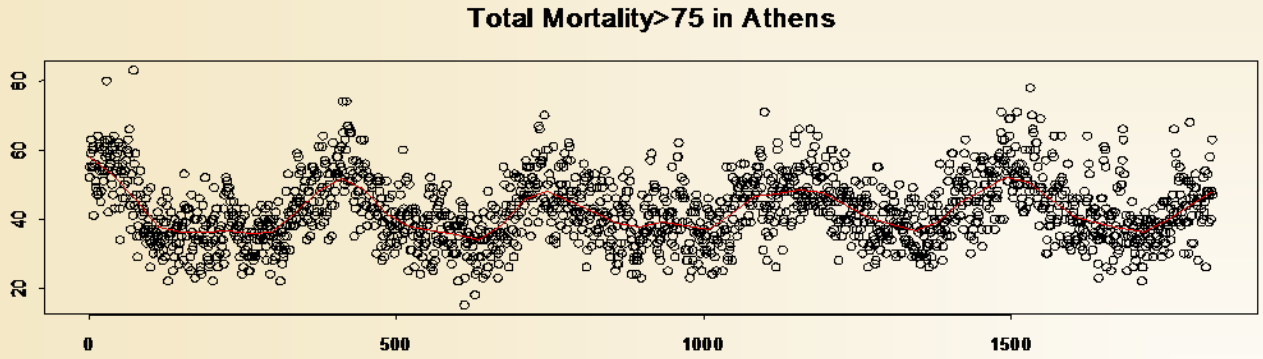


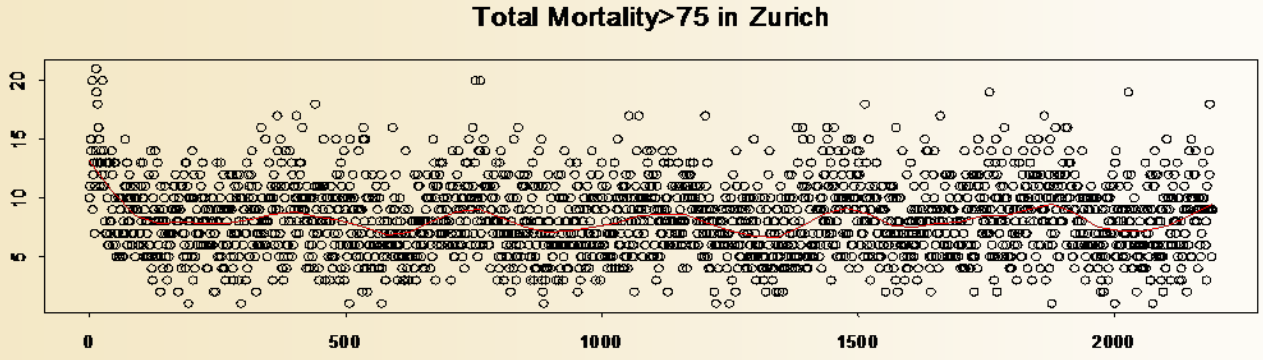
FIGURE 8.2 Photograph of the London smog of 1952



Time-series studies

- **Exploit *temporal* differences in exposure**
- **Estimate the association between *daily mortality or morbidity rates* and the level of air pollution *shortly* before death**
- **Results expressed as the % change in mortality or morbidity per 10 µg/m3**





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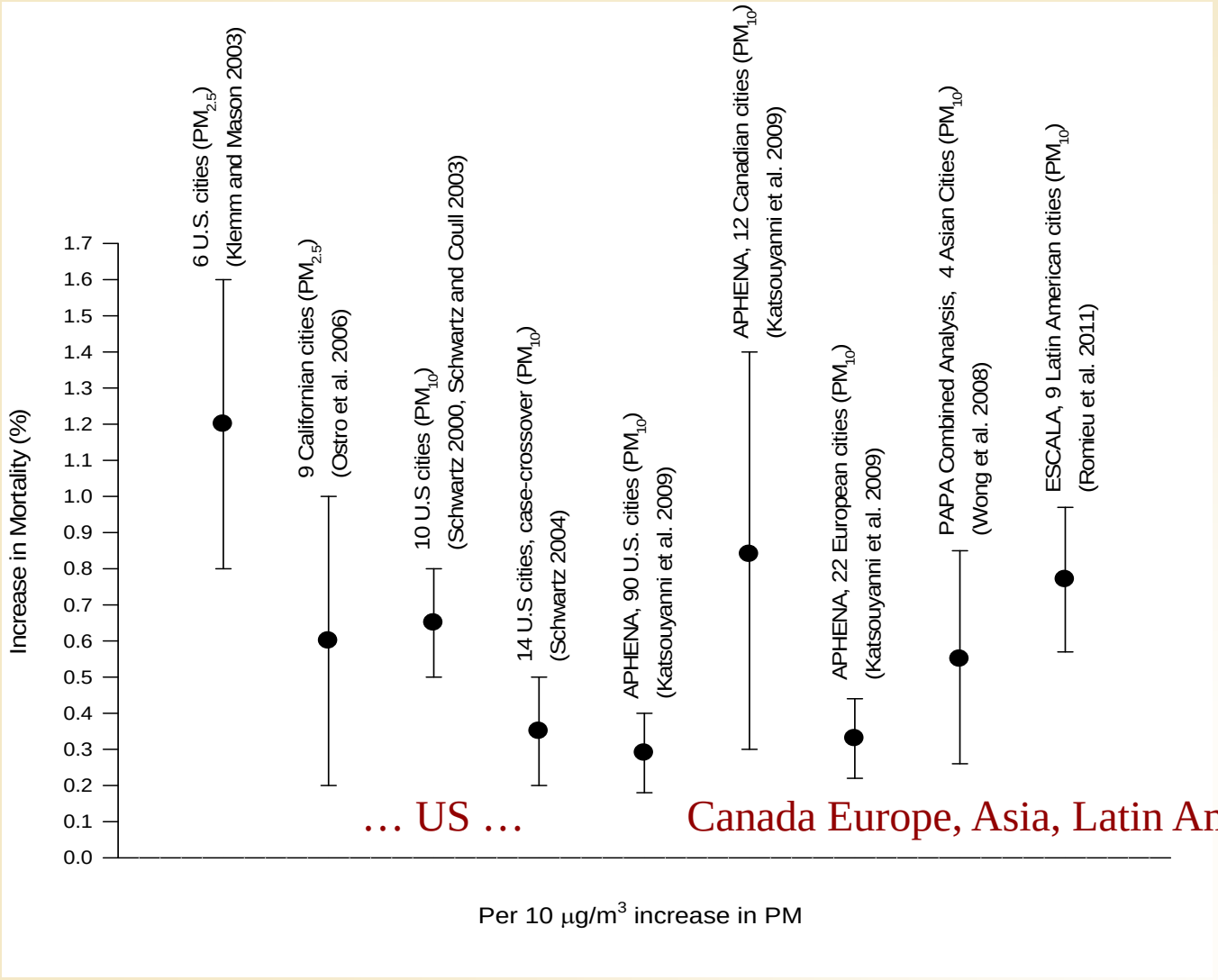
Time-series studies – a history of meeting methodologic challenges

- Proliferation of 100's of time-series studies of individual cities
  - Numerous questions about bias arising from modeling choices, publication preferences, among other issues
- Methodological 'shake-out' – Multi-city studies
  - National Morbidity and Mortality Air Pollution Study (**NMMAPS**) - 90 US cities
  - Air Pollution and Health: a European and North American Perspective (**APHENA**)

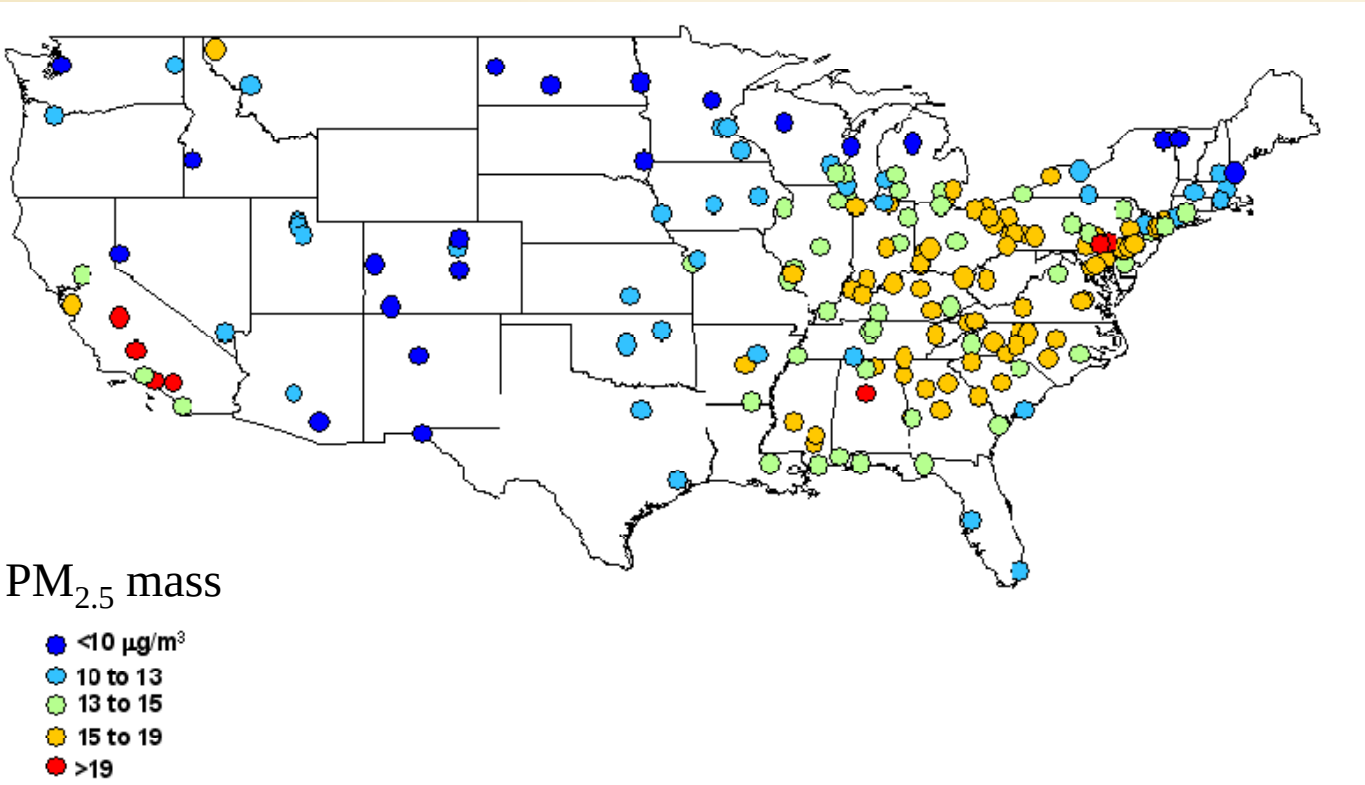
Multi-city time-series studies across the world



All-natural cause mortality

Studies of long term exposures (years or decades)

- **Exploit *spatial* differences in exposure**
- **Estimate the association between *long –term average mortality rates* and *long-term* exposure to air pollution**



Bell et al., 2008

Relatively fewer cohort studies

- Earliest
  - Harvard Six City Study (1993)
  - American Cancer Society Cohort Study (1995)
- More recent
  - Women's Health Initiative
  - Nurses Health study
  - California Teachers Study..
  - And more…

An Association Between Air Pollution and Mortality in Six U.S. Cities (1993)

The NEW ENGLAND JOURNAL of MEDICINE

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE.

Methods:

- 14-16 yr prospective follow-up of 8,111 adults living in **six** U.S. cities. Portage WI >>Steubenville, OH
- Monitoring of TSP PM_{10}, $PM_{2.5}$, SO_4, NH_+, SO_2, NO_2, O_3 .
- Data analyzed using survival analysis, including Cox Proportional Hazards Models.
- Controlled for individual differences in: age, sex, smoking, BMI, education, occupational exposure.

From: A. Pope

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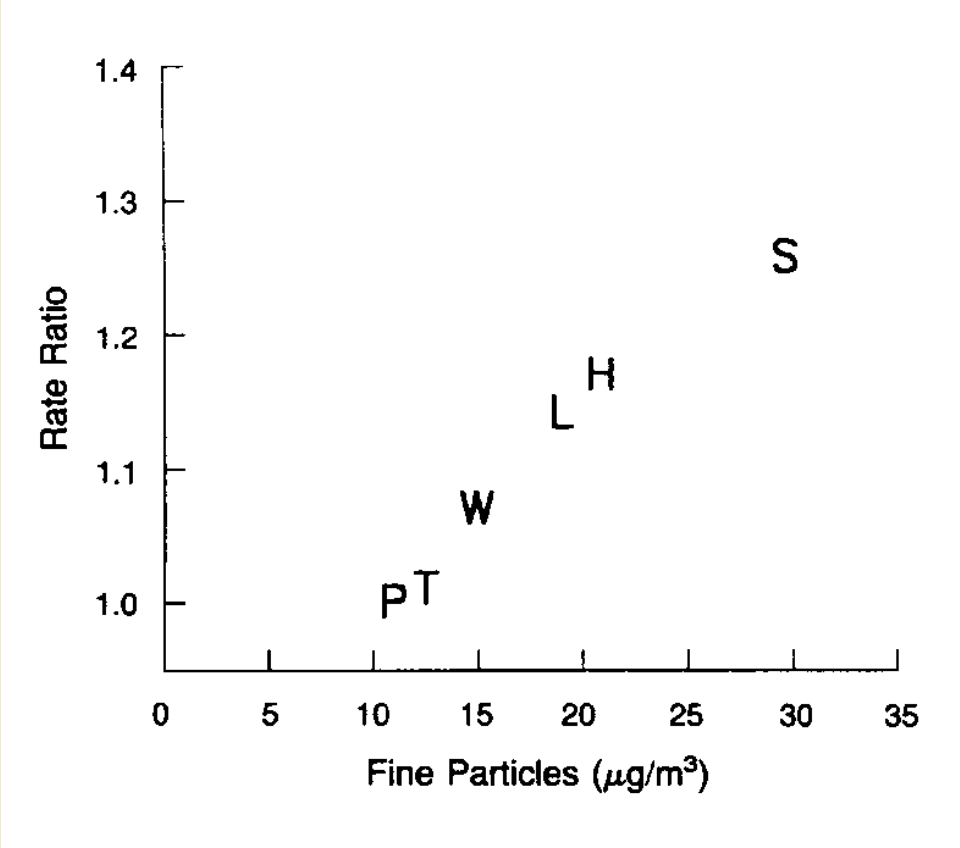
Found clear pollution related differences among cities



- S – Steubenville OH
- H – Kingston/Harriman TN
- L – St. Louis MO
- W – Watertown MA
- T – Topeka KS
- P – Portage WI

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The American Cancer Society Cohort (ACS) Studies

Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults

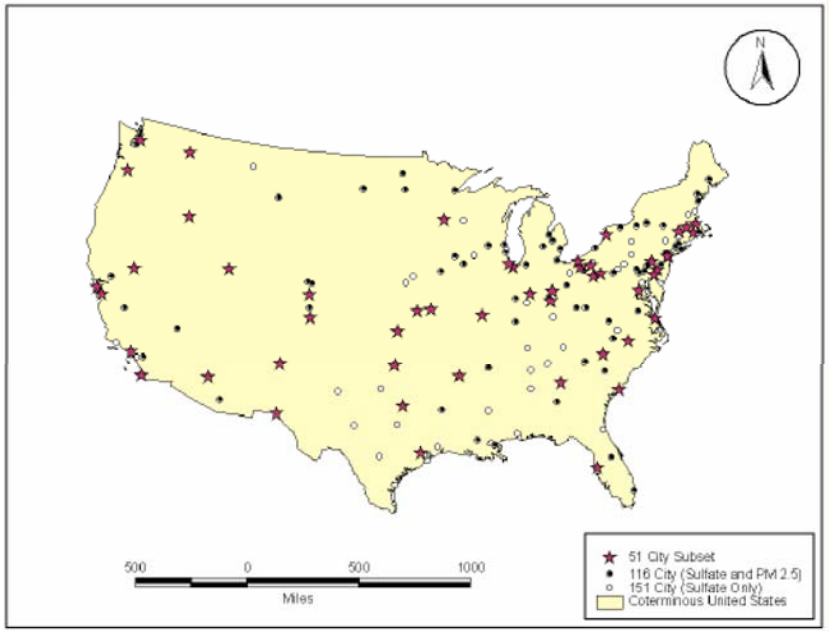

Michael Thun

Pope CA III, Thun MJ, Namboodiri MM,
Dockery DW, Evans JS, Speizer FE, Heath CW Jr.

AMERICAN JOURNAL OF
Respiratory and
Critical Care Medicine®
1995


Clark Heath

Methods: Linked and analyzed ambient air pollution data from 51-151 U.S. metro areas with risk factor data for over 500,000 adults enrolled in the ACS-CPSII cohort.



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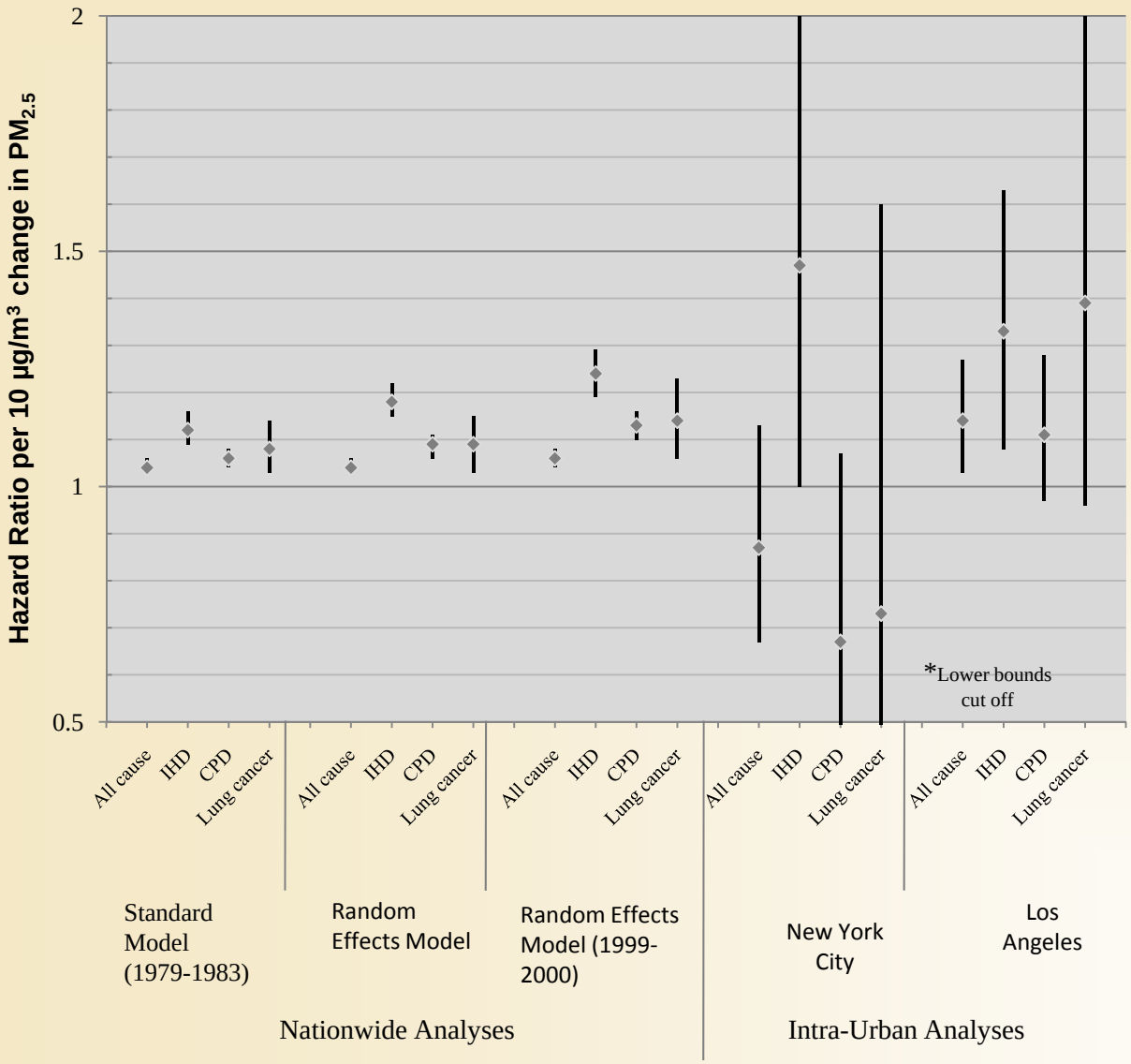

Figure 1. Increase in Mortality from Various Causes for Selected Nationwide and Intra-Urban Analyses of the ACS Cohort (Krewski et al. 2009, HEI Report 142)

Extended Analysis of the ACS cohort

- Various models
- Increased control for confounders
- Longer follow-up
- New exposure estimation on smaller scales

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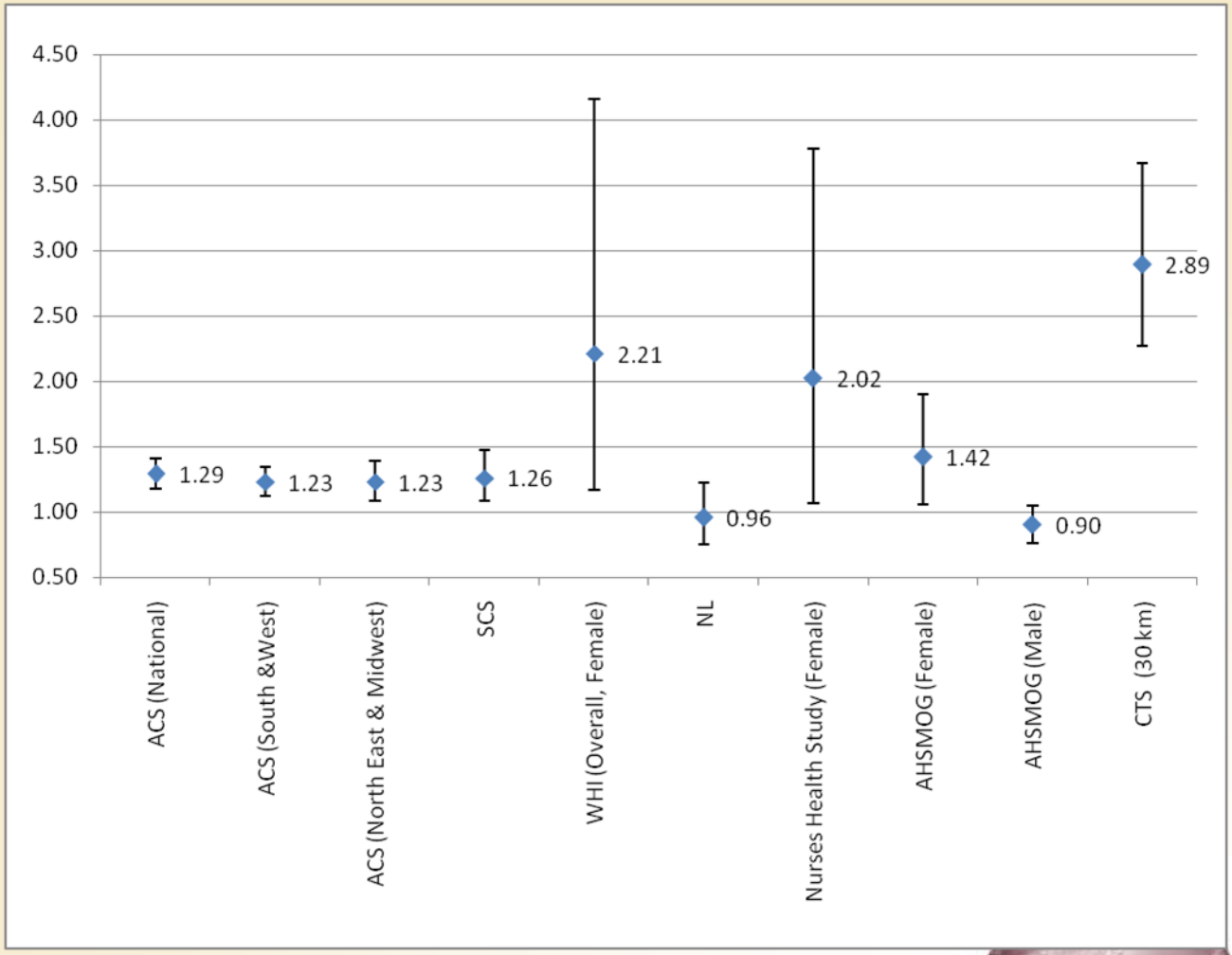
Expert Group Review of Adult Mortality Studies - GBD

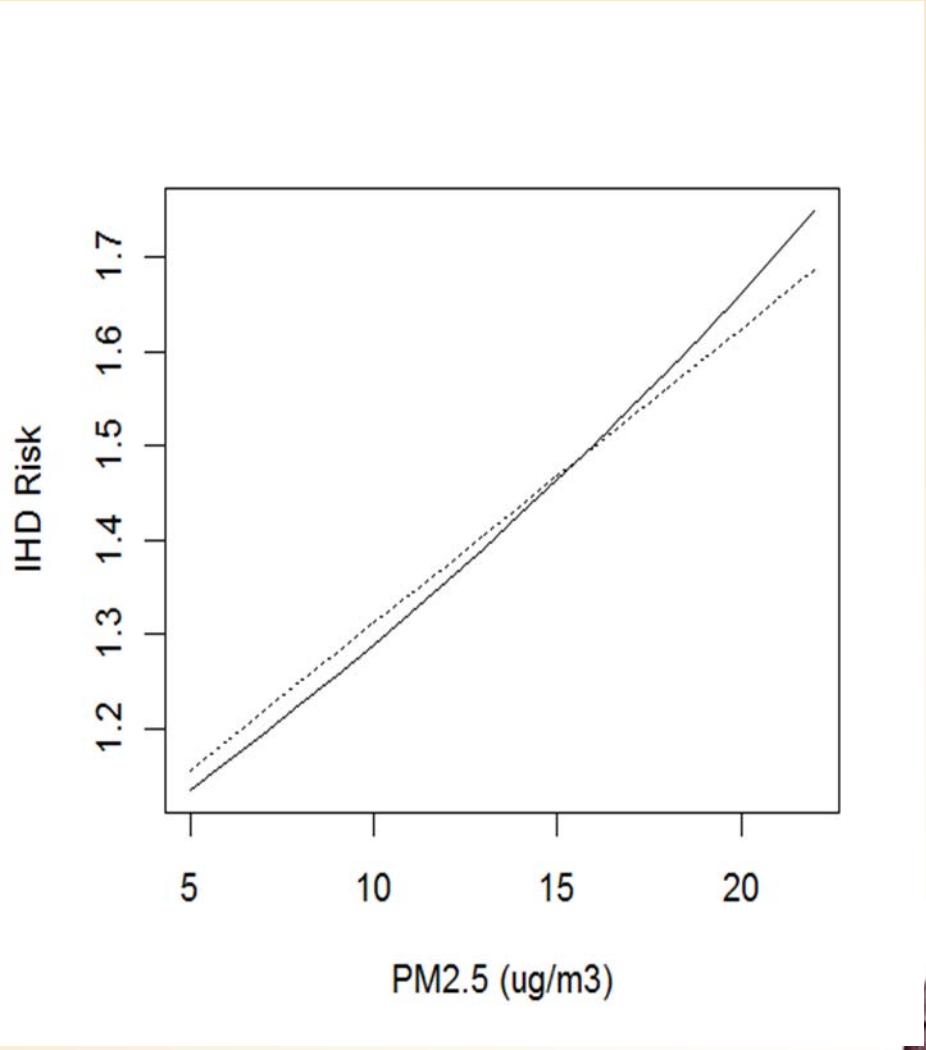
- Evidence is most consistent with a causal effect of long-term exposure on cardiovascular and respiratory disease and lung cancer, based on the lack of competing explanations (e.g., confounding or other bias), broad consistency with evidence from other, related, exposures and evidence of biologic mechanisms
- The shape of the $PM_{2.5}$ C-R functions appear linear from ~4-30 $\mu g/m^3$
- The estimates from different cohort studies vary



RR of death from IHD for a 10 $\mu g/m^3$ change in $PM_{2.5}$ (95% confidence intervals)

Concentration response? Long-term exposure and mortality from chronic disease

- Most monotonically increasing C-R functions predict risk equally well within the range of exposure in the existing cohort studies
- IHD RRs per 10 $\mu g/m^3$ range from 1.3 (ACS) to 2.2 (WHI)
- All direct evidence from cohort studies in US /Western Europe where levels of $PM_{2.5}$ are relatively low



IHD Mortality Risk in ACS Cohort : exposure on linear (-) and log (···)scales Krewski et al. 2009

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION
American Heart Association
Learn and Live

Cardiovascular Mortality and Exposure to Airborne Fine Particulate Matter and Cigarette Smoke

Shape of the Exposure-Response Relationship

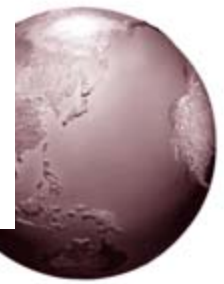
C. Arden Pope III, PhD; Richard T. Burnett, PhD; Daniel Krewski, PhD; Michael Jerrett, PhD; Yuanli Shi, MD; Eugenia E. Calle, PhD; Michael J. Thun, MD

Background—Fine particulate matter exposure from both ambient air pollution and secondhand cigarette smoke has been associated with larger risks of cardiovascular mortality than would be expected on the basis of linear extrapolations of the relative risks from active smoking. This study directly assessed the shape of the exposure-response relationship between cardiovascular mortality and fine particulates from cigarette smoke and ambient air pollution.

Methods and Results—Prospective cohort data for >1 million adults were collected by the American Cancer Society as part of the Cancer Prevention Study II in 1982. Cox proportional hazards regression models that included variables for increments of cigarette smoking and variables to control for education, marital status, body mass, alcohol consumption, occupational exposures, and diet were used to describe the mortality experience of the cohort. Adjusted relative risks of mortality were plotted against estimated average daily dose of fine particulate matter from cigarette smoke along with comparison estimates for secondhand cigarette smoke and air pollution. There were substantially increased cardiovascular mortality risks at very low levels of active cigarette smoking and smaller but significant excess risks even at the much lower exposure levels associated with secondhand cigarette smoke and ambient air pollution.

Conclusions—Relatively low levels of fine particulate exposure from either air pollution or secondhand cigarette smoke are sufficient to induce adverse biological responses increasing the risk of cardiovascular disease mortality. The exposure-response relationship between cardiovascular disease mortality and fine particulate matter is relatively steep at low levels of exposure and flattens out at higher exposures. (***Circulation.*** **2009;120:941-948.)**

Key Words: air pollution ■ cardiovascular diseases ■ mortality ■ tobacco smoke pollution ■ smoking



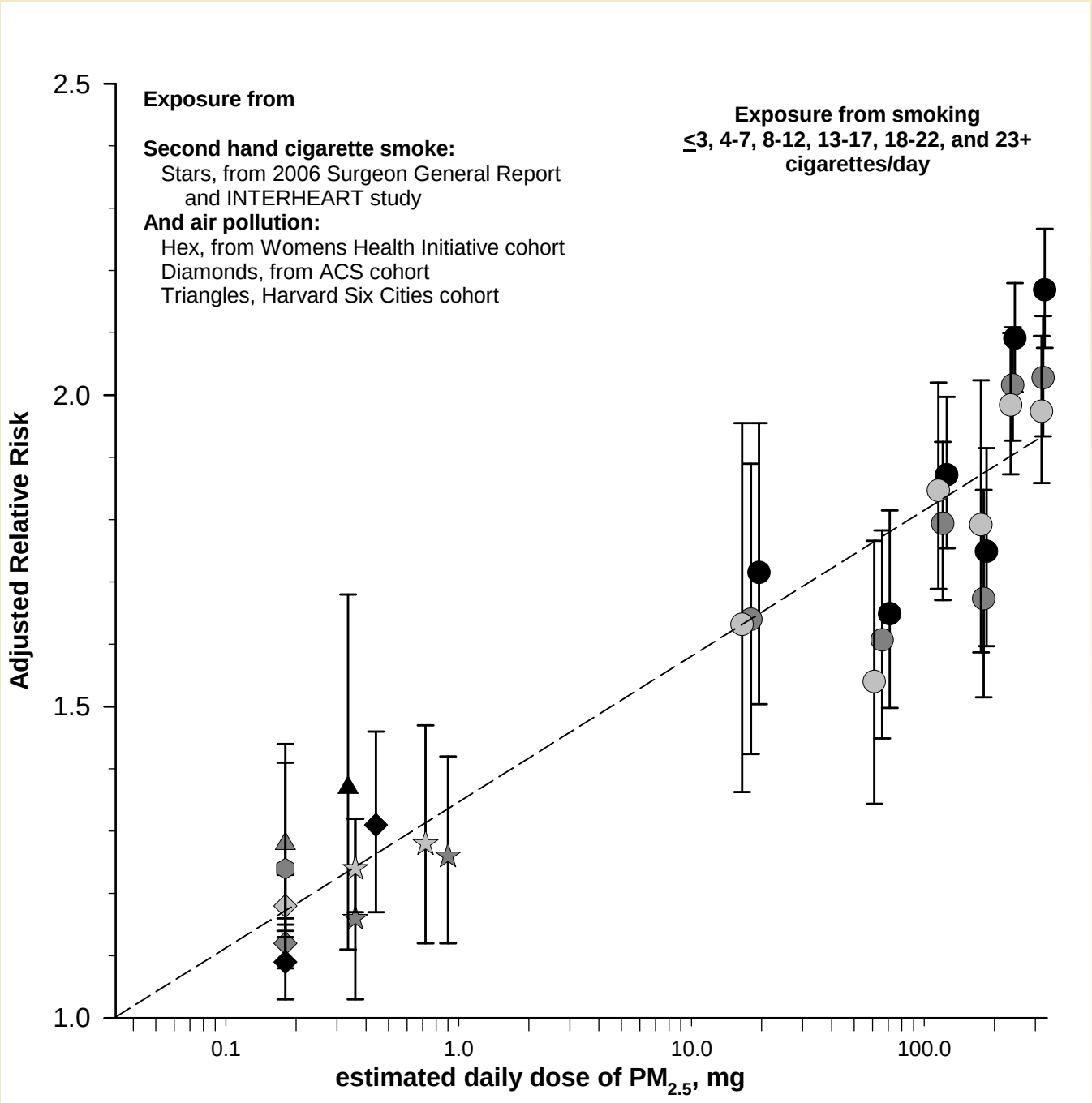


Figure 2. Adjusted relative risks (and 95% CIs) of ischemic heart disease (light gray), cardiovascular (dark gray), and cardiopulmonary (black) mortality plotted over baseline estimated daily dose (using a log scale) of $PM_{2.5}$ from current cigarette smoking (relative to never smokers), SHS, and air pollution.

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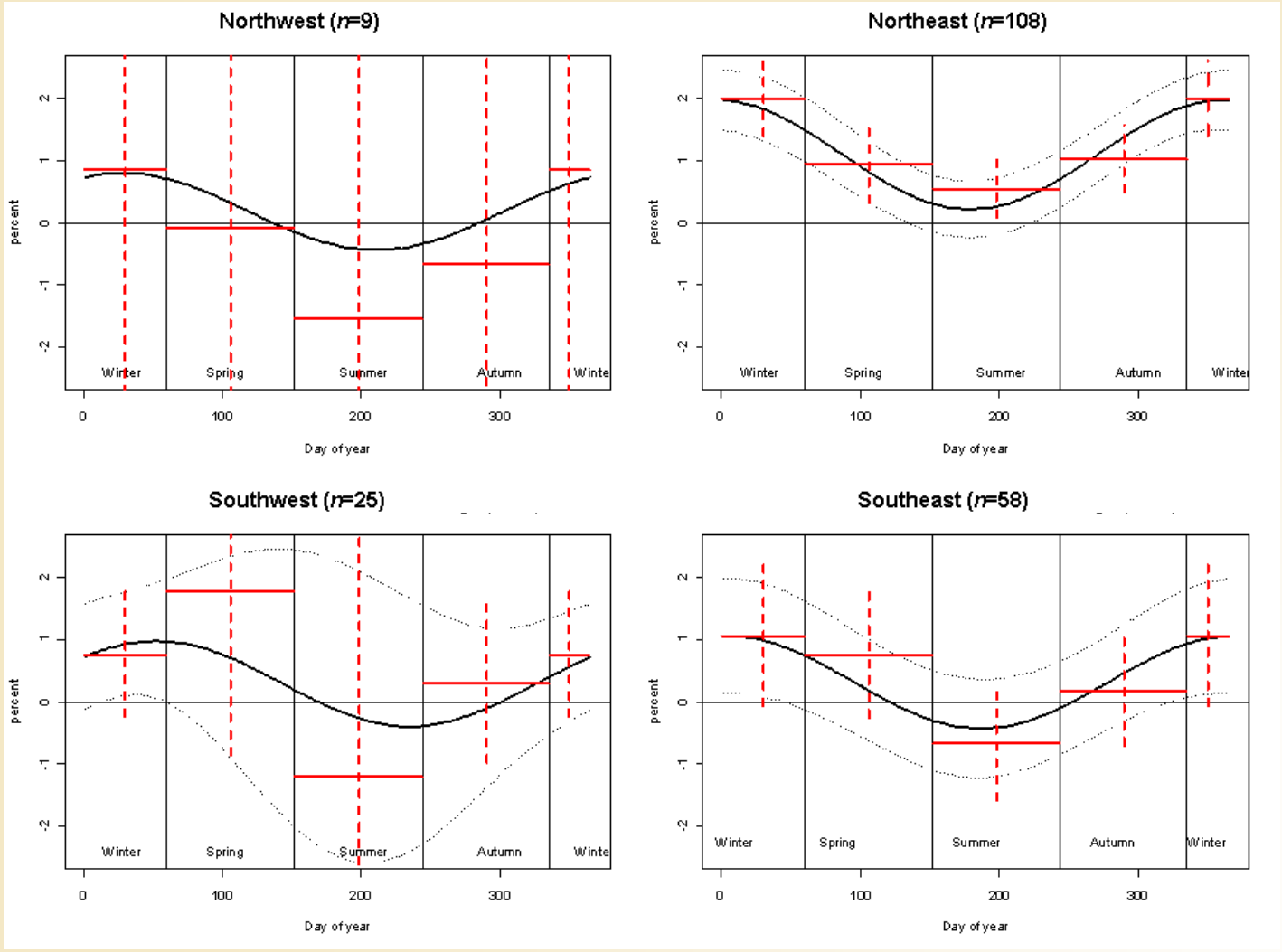
So, where are we? Are these relationships 'real'?

- Do we find similar findings in studies of different design?
- Are findings consistent in studies done different places by different people?
- Do we see evidence of a concentration response relationship?
- Is there some plausible biological mechanism for the effects we see?
- Have we adequately explored alternative explanations?

But we do see regional and seasonal differences we don't understand…



Variations in rates of cardiovascular hospital admissions per 10µg/m^3 $PM_{2.5}$

Bell et al. 2008

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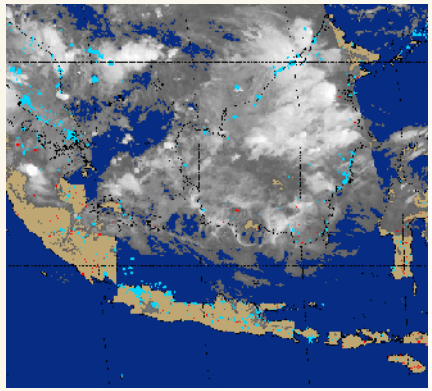
Air Pollution: Many Sources



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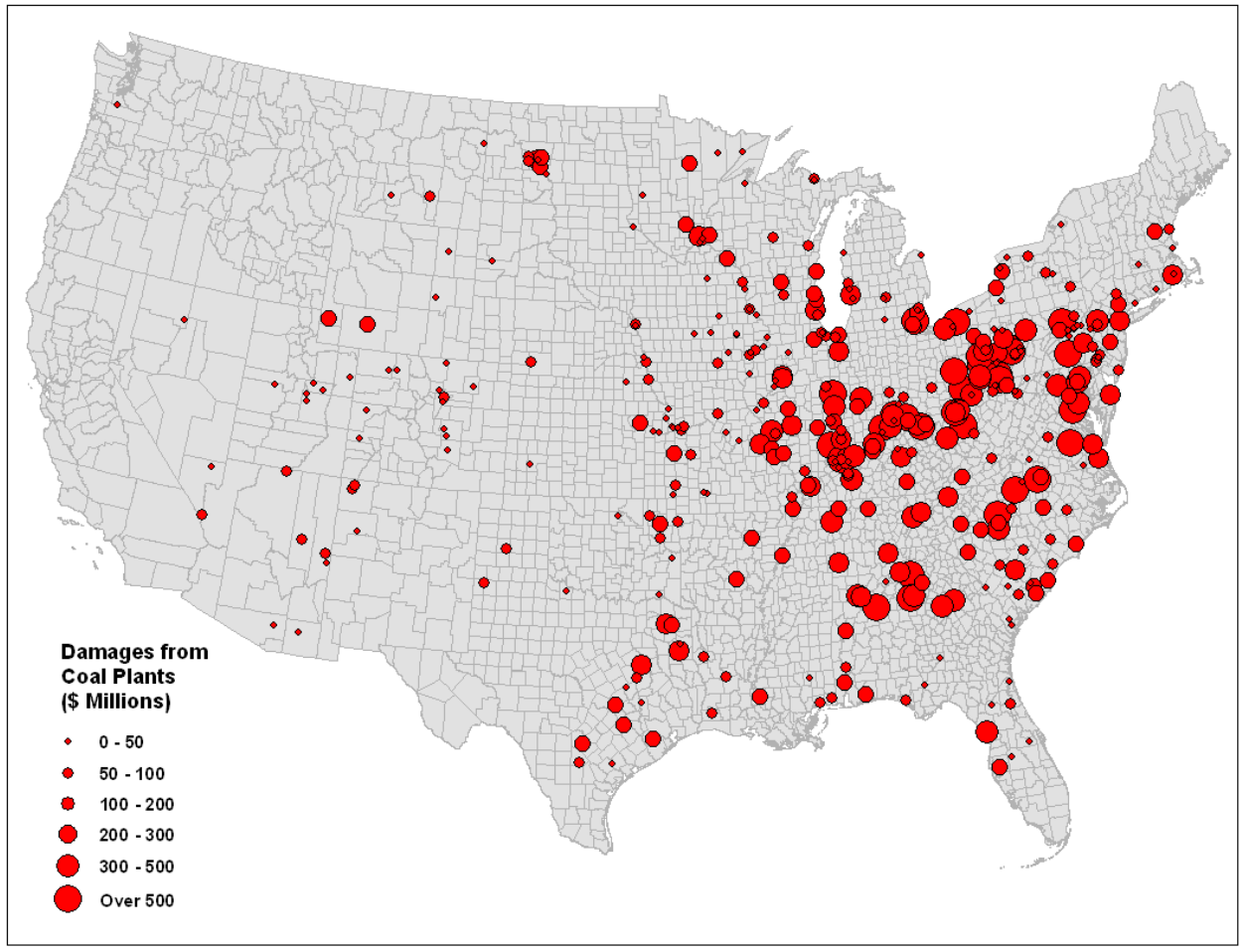
Electricity: Coal

$62 Billion of Health and Other Non-Climate Damages in 2005

Damage Estimates based on SO_2, NO_x, and PM emissions



- Air Pollution Damages from Coal Generation for 406 plants, 2005
  - **3.2 cents/kWh**
- With control, damages lower in 2030
  - **1.7 cents/kWh**
- Damages related to climate-change effects are not included

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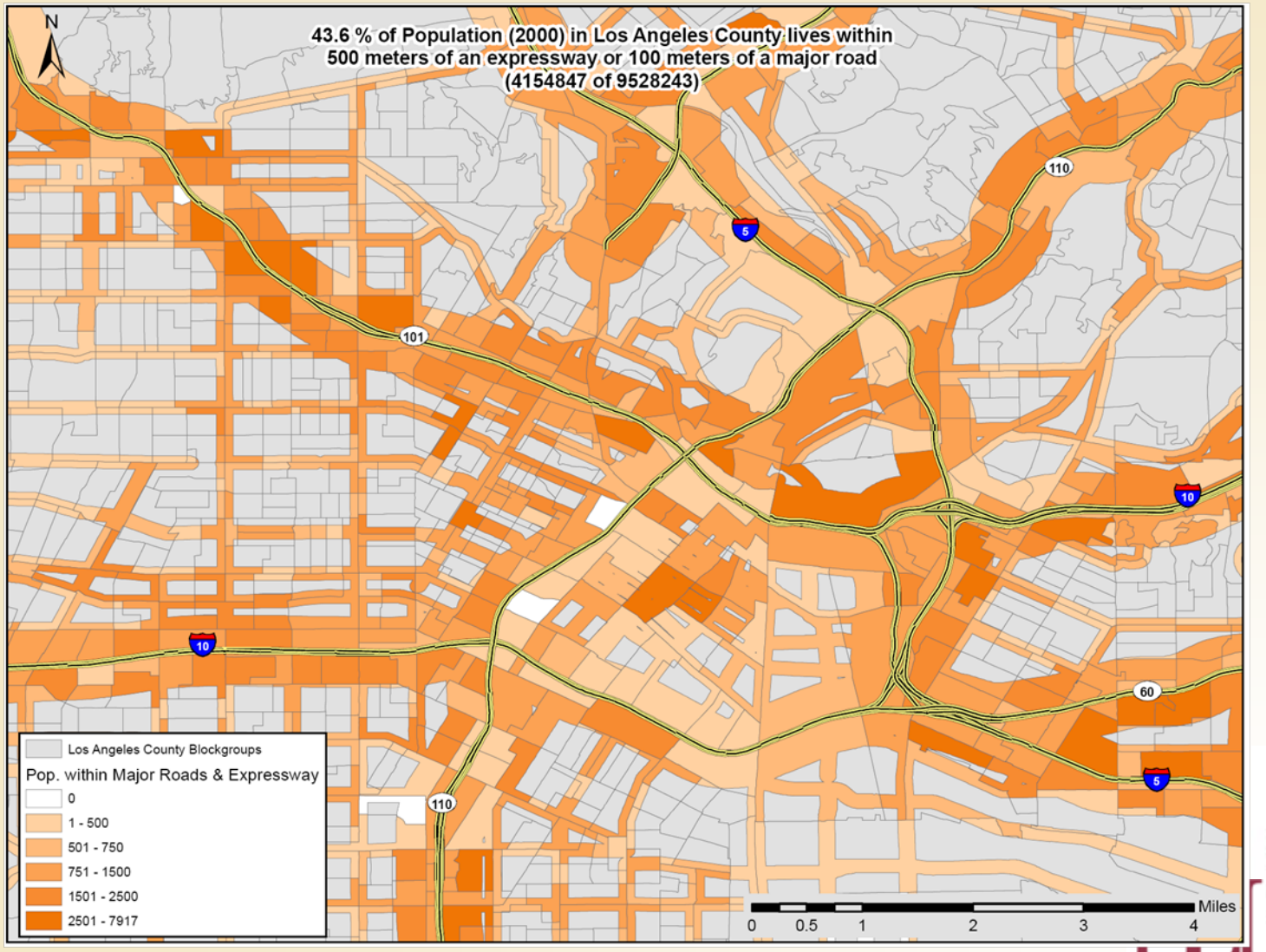
In Los Angeles, 44% of population live in the maximum zone of impact of major roads

HEI Special Report 17



(within 500 meters of an expressway; 100 meters of a major road)

Overall Traffic Conclusions

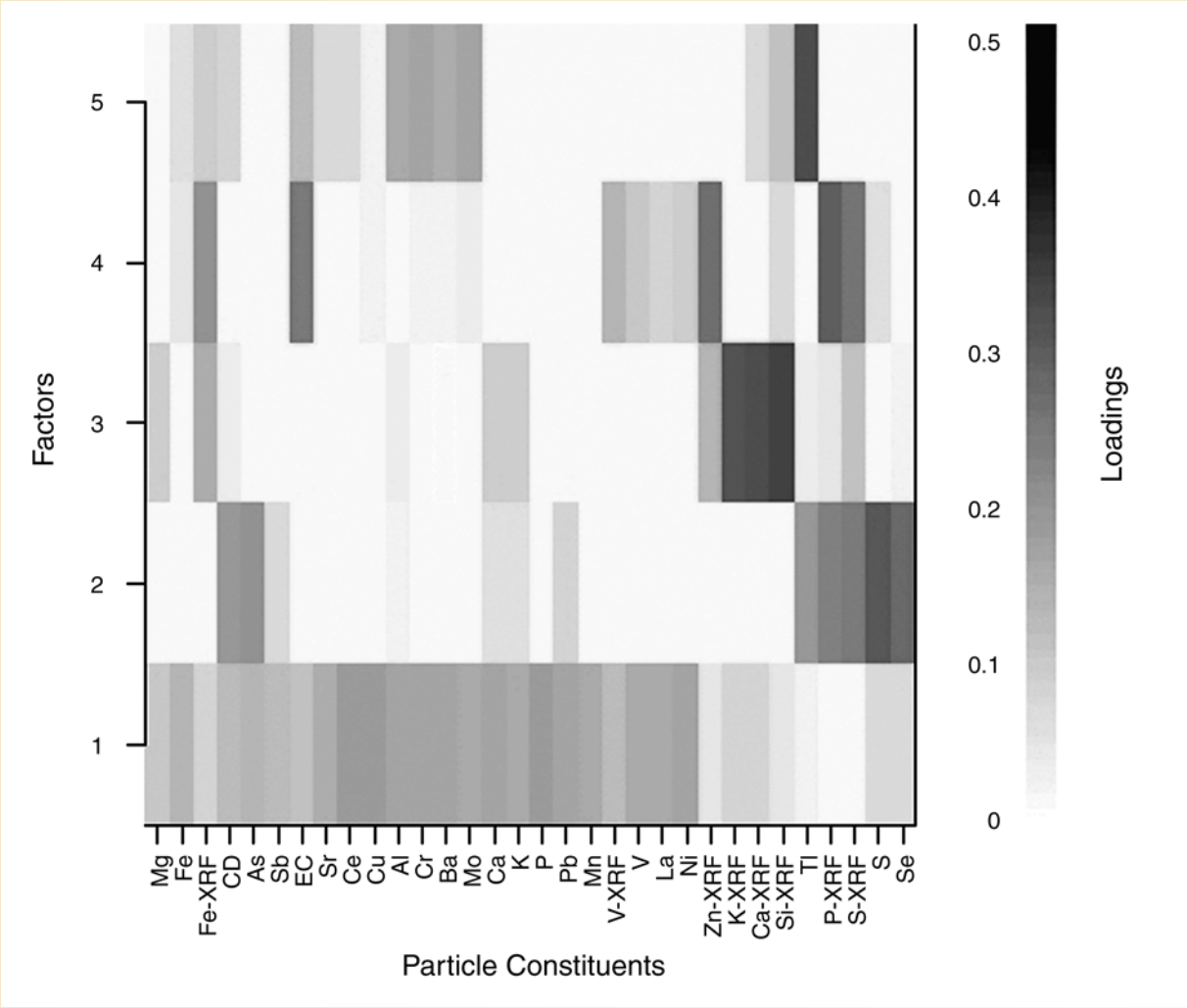
- **The data are incomplete on emissions, their transformations, and exposure assessment**
- **There were enough studies to find:**
  - ***Sufficient* evidence that exposure to traffic can cause exacerbation of asthma, especially in children**
  - ***Suggestive* evidence for other health effects (premature mortality, lung function, respiratory symptoms, and others)**
  - **But only *limited evidence* of effects for: Adult onset asthma; Health care utilization; COPD; Non-asthmatic allergy; Birth outcomes; Cancers**

HEI Special Report 17: Critical Review of Air Pollution from Traffic

Other approaches to identifying impacts of sources

- Source apportionment…
  - Factor analysis
  - Absolute principal components analysis
  - Positive matrix factorization
  - And others…

Attributing factors to particular sources is tricky



Factor loading matrix for CAPs samples using a five-factor model.

Levy et al, 2010
HEI Report 152

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Can we attribute the health effects of PM to different sources?

- Not yet…
  - Tricky statistics, identifying and disentangling source ‘signatures’
- A lot of work going on at HEI and elsewhere
  - HEI National Particle Component Toxicity (NPACT) – Multi-year, multi-center, integrated toxicology and epidemiologic studies
  - HEI Statistical Methods research program
  - EPA Multipollutant initiatives…

Conclusions

- A substantial body of evidence exists showing that PM has “real” adverse effects on health, particularly on exacerbation of CV disease and increased mortality
- We don’t yet know how much to attribute to particular attributes of PM (components, sources, and size) or to differences in the air pollution mixture.

LA then- 1953

We've come a long way....

...and now

"And it was so typically brilliant of you to have invited an epidemiologist."

The New Yorker

Thank-You!

www.healtheffects.org

HEI