

Assessing Low Levels of Air Pollution: A Historical Perspective

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Health Effects Institute
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Early “**Killer smog**” episodes demonstrated that air pollution at extreme levels can contribute to respiratory and cardiovascular disease and death



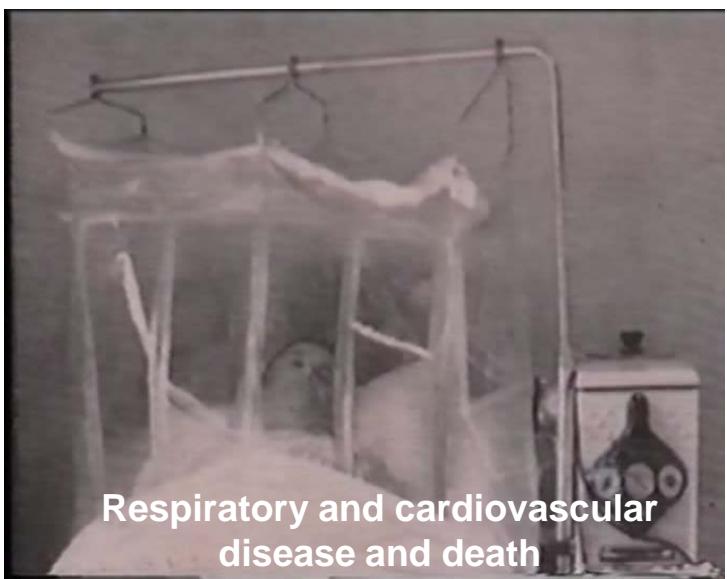
Dec. 1-5, 1930: Meuse Valley, *Belgium*
60 deaths (10x expected)



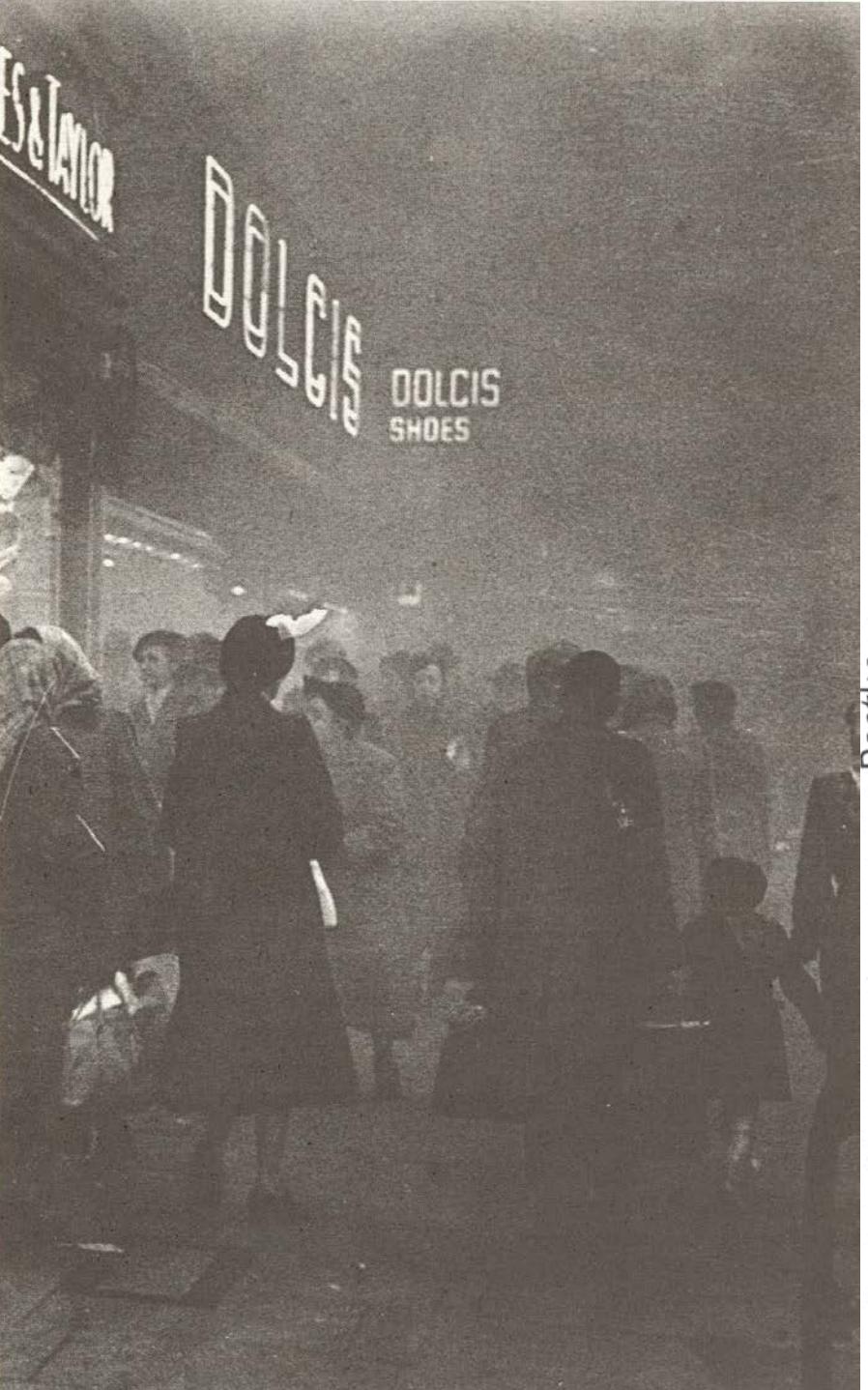
Oct. 27-31, 1948: Donora, PA
20 deaths, $\frac{1}{2}$ the town's population fell ill



Dec. 5-9, 1952: London--1000's of excess deaths

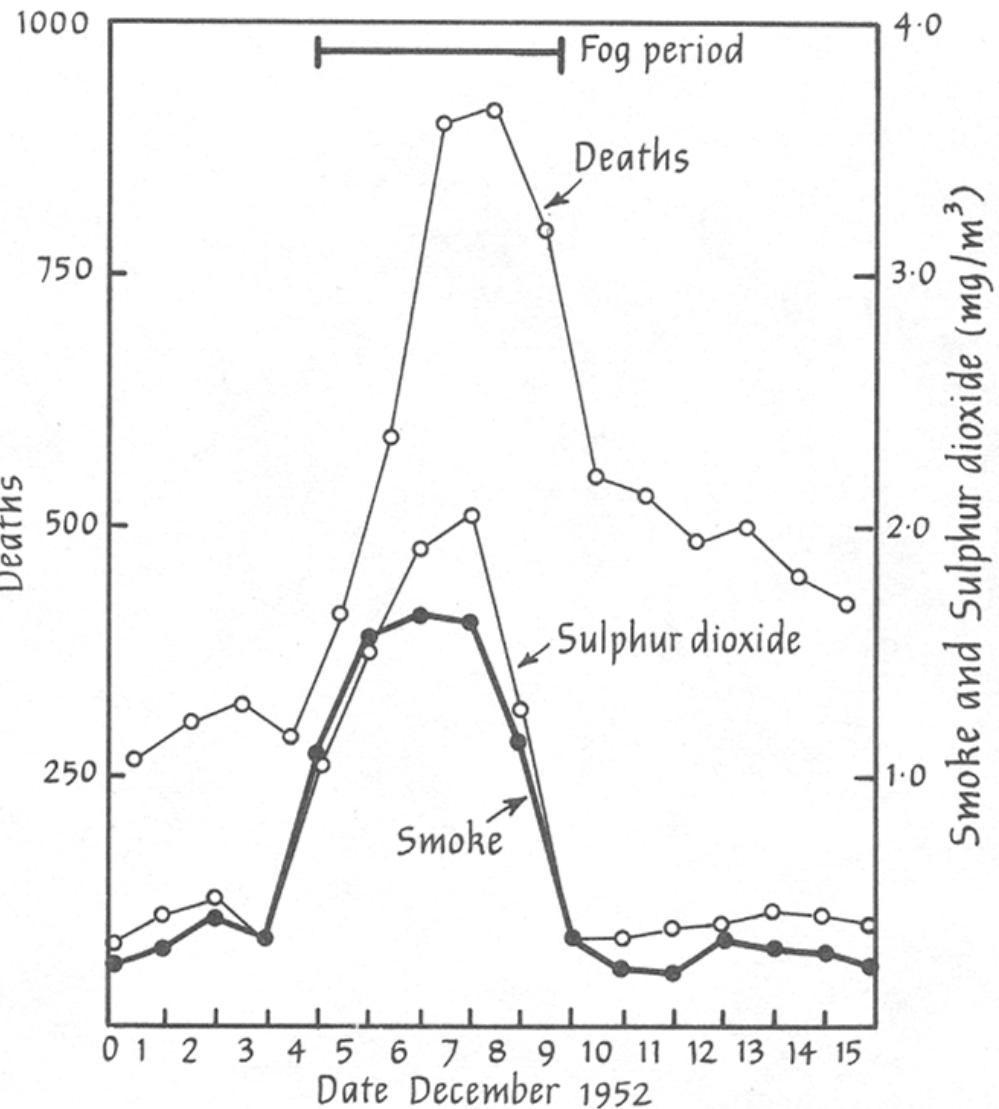


Respiratory and cardiovascular
disease and death



London Fog Episode, Dec. 1952

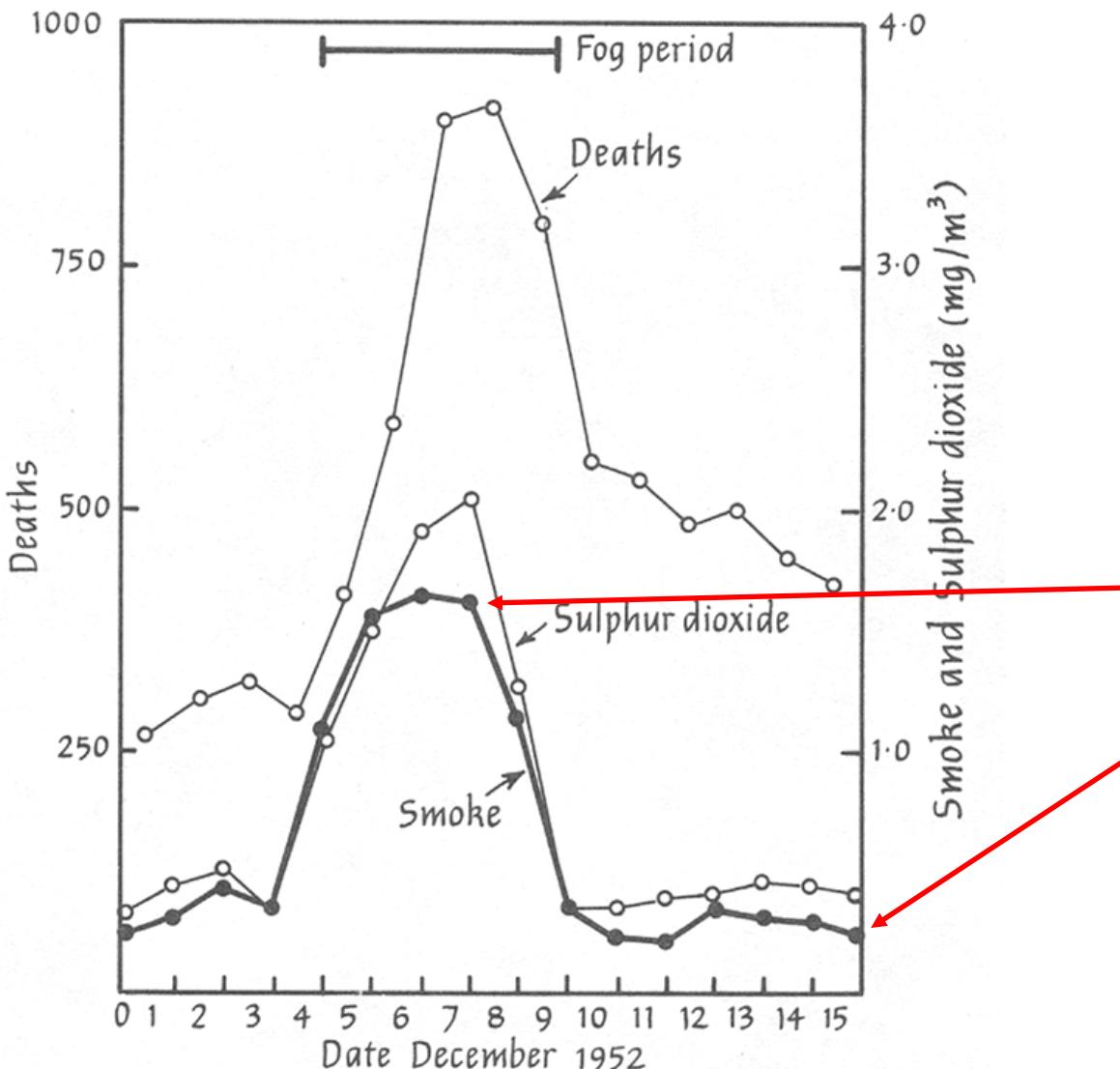
THE BIG SMOKE



From: Brimblecombe P. The Big Smoke, Methuen 1987

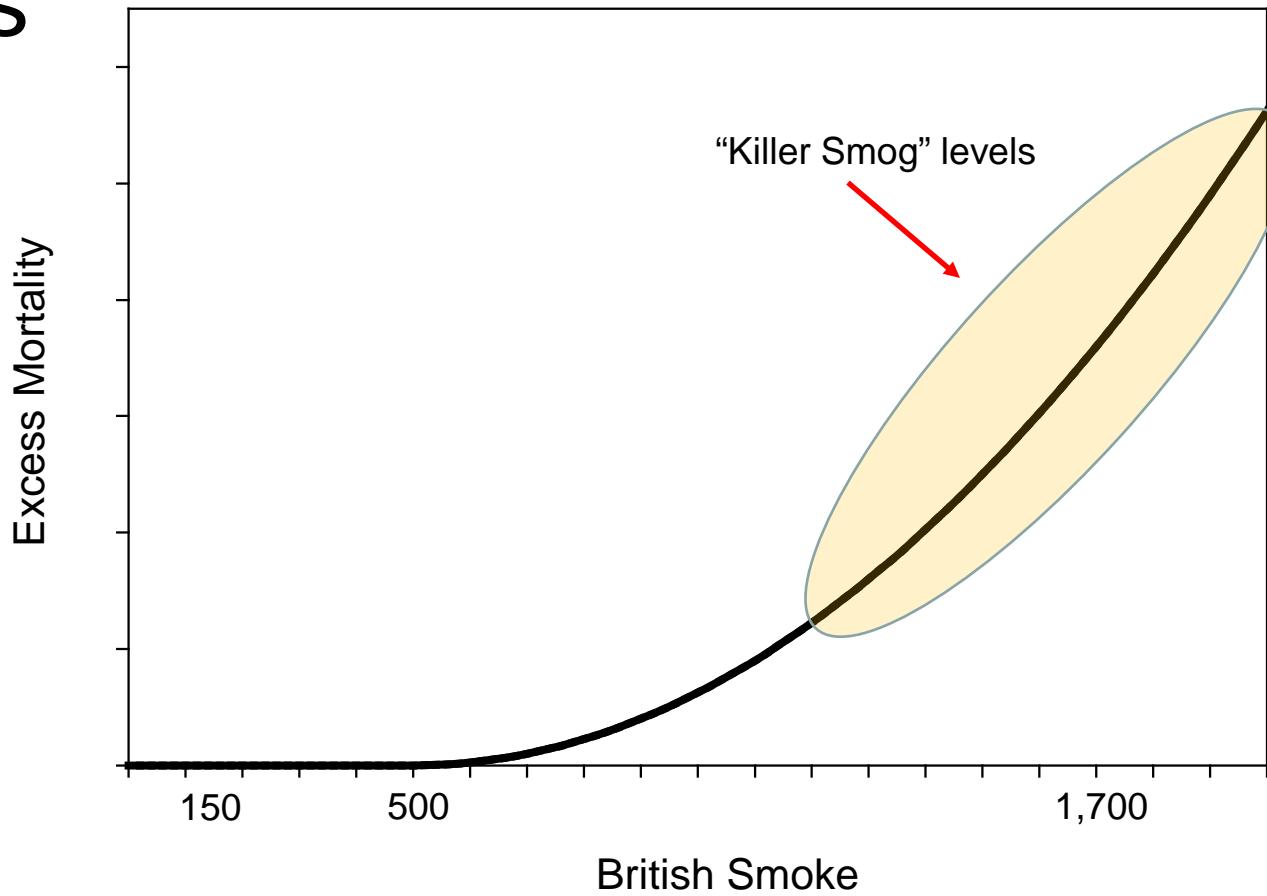
London Fog Episode, Dec. 1952

THE BIG SMOKE



Note the concentrations of PM or BS (British Smoke):
High concentrations about 1.7 milligrams (mg/m^3) or 1,700 micrograms ($\mu\text{g}/\text{m}^3$)
Low concentrations about 150 $\mu\text{g}/\text{m}^3$

1970's



The Clean Air Act and NAAQSs basically assumed “threshold” exposure-response relationships between adverse health effects and “criteria” pollutants. Debate was largely about **“where is the threshold”** and **“what is an adequate margin of safety.”**

A Search for a Threshold in the Relationship of Air Pollution to Mortality: A Reanalysis of Data on London Winters

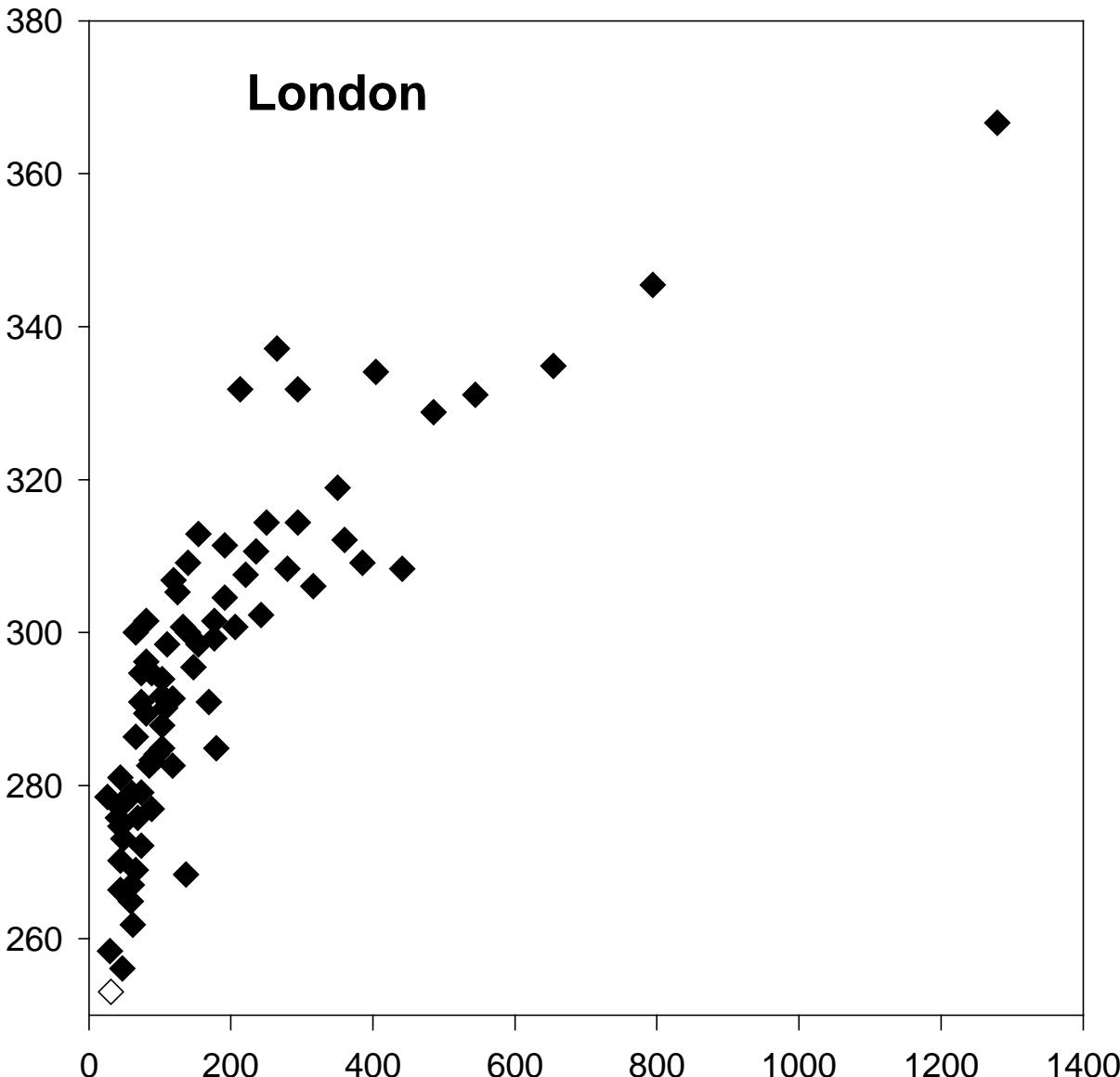
by Bart Ostro*

The relationship between air pollution and increased risk of mortality has been explored previously using data on 14 winters in London. The results of these analyses have been used to determine a no-observable-effects level. This reanalysis of the data finds no evidence to support the existence of a no-effects level. Further, the reanalysis suggests that the estimated pollution-mortality relationship exists even in nonepisodic winters, when British Smoke readings were less than $500 \mu\text{g}/\text{m}^3$.



Our perspective about what consists of “low-level air pollution has changed over the last several decades





1990

Early analyses of concentration-response function.

Used London mortality data for 14 winters

C-R function less steep at higher concentrations

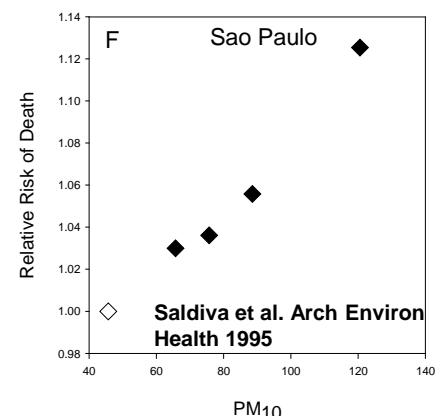
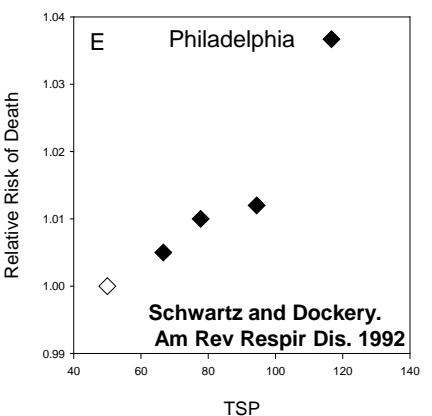
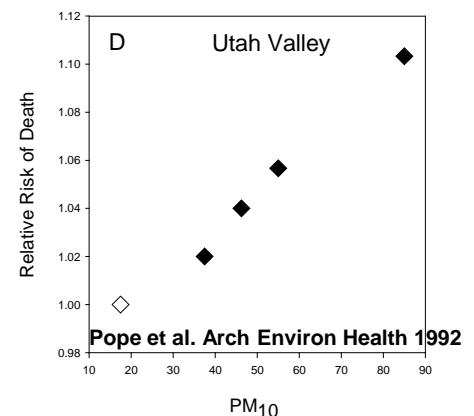
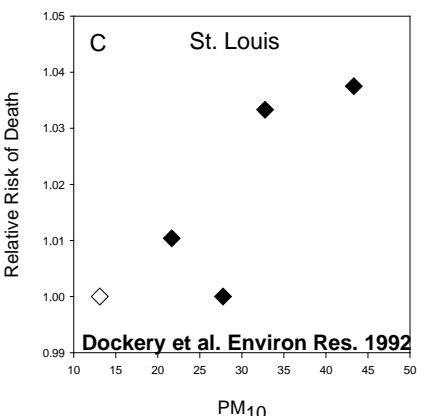
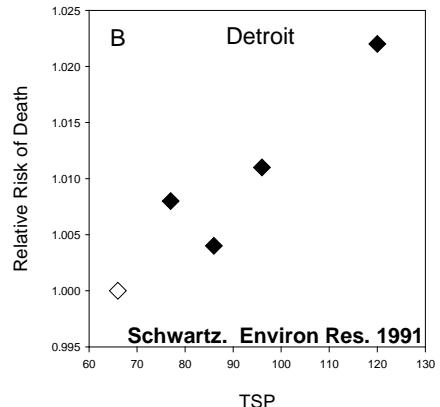
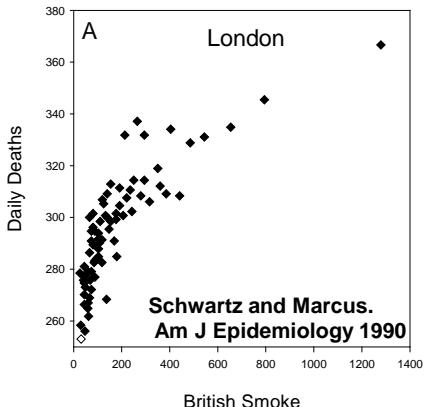
No evidence of a threshold



Early 1990's

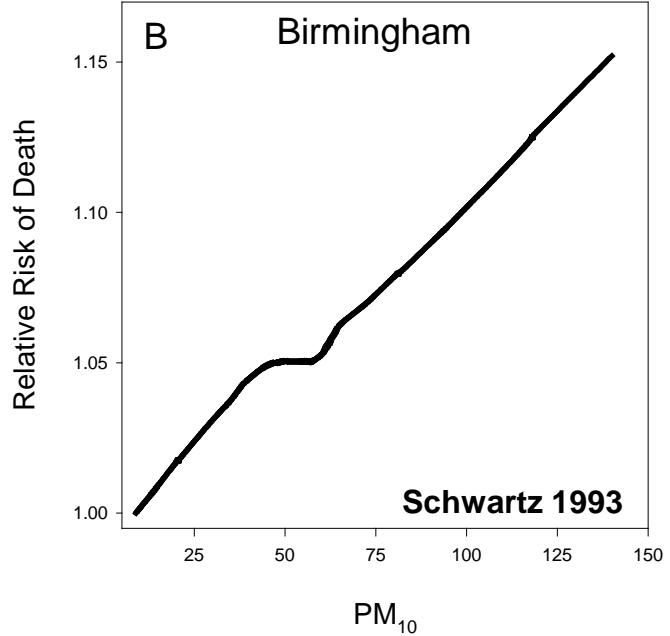
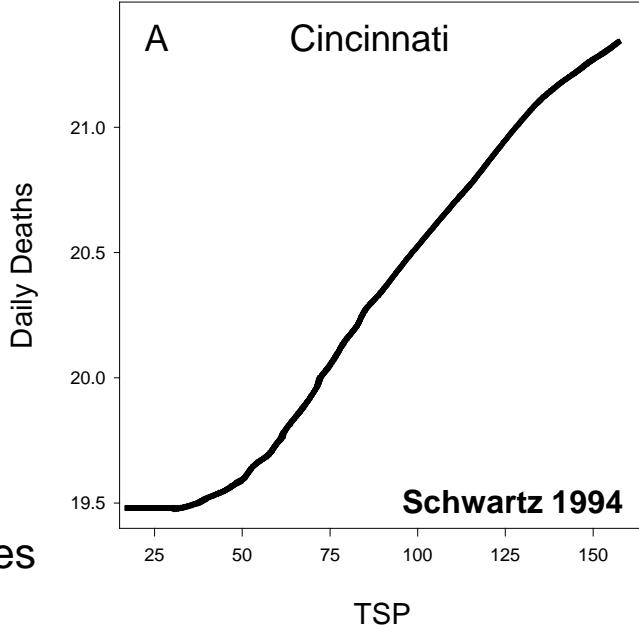
To explore for effects at low pollution levels, many early single-city time-series Studies used quintile (or quartile) Analysis where indicator variables for Different ranges of air pollution were Included in the time-series regression models.

Examples

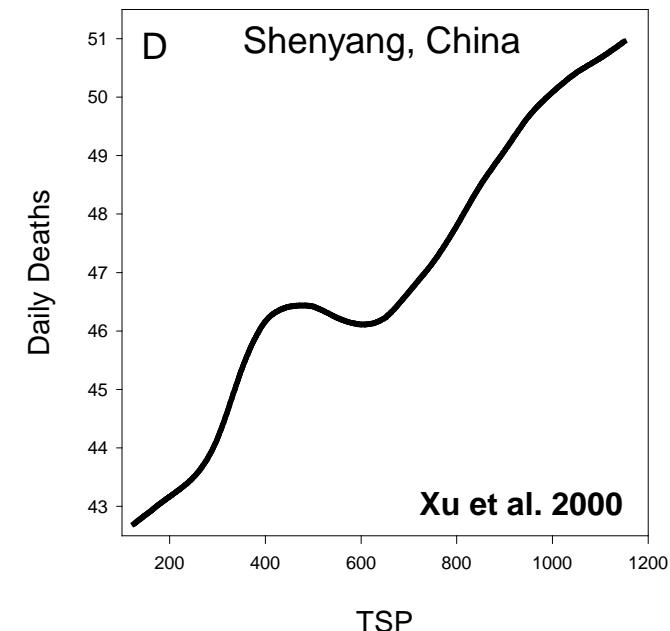
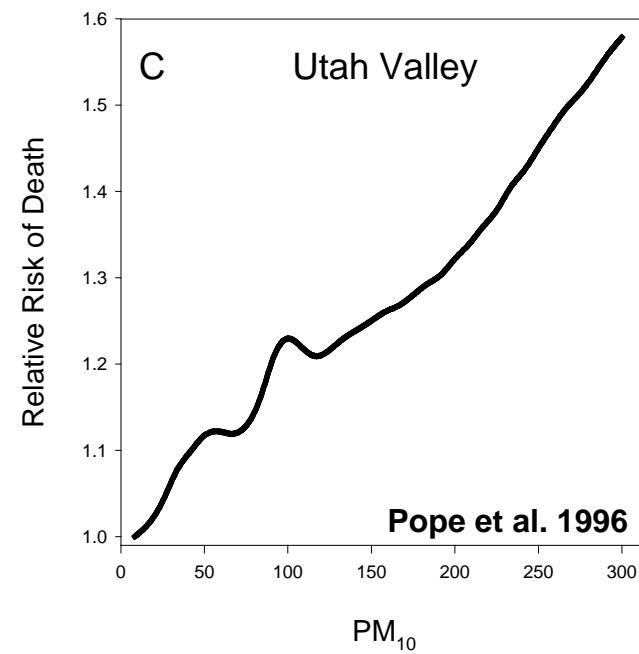


Mid to late 1990's

Various parametric
(spline functions)
or non-parametric
smoothing approaches
to allow for flexible
fitting of C-R.



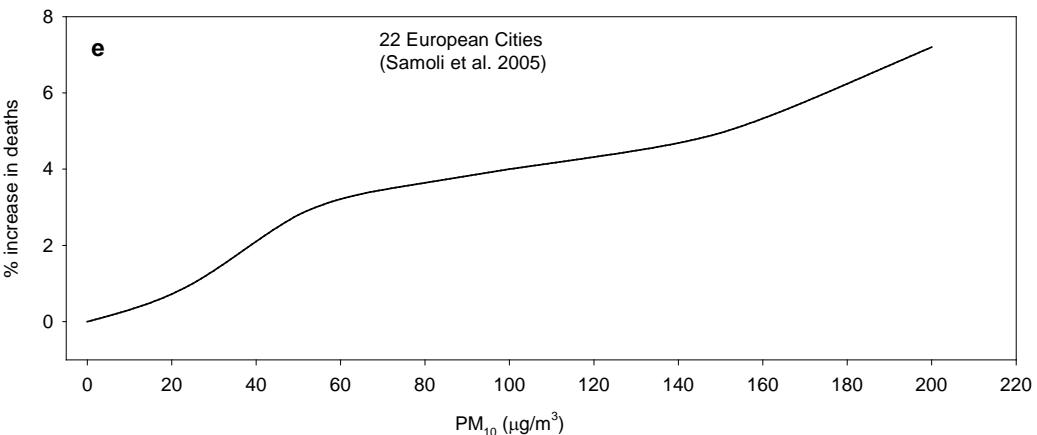
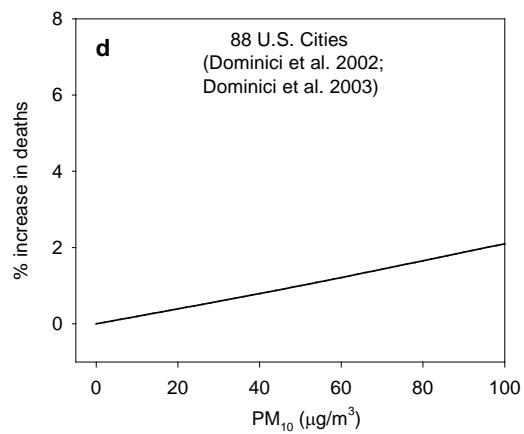
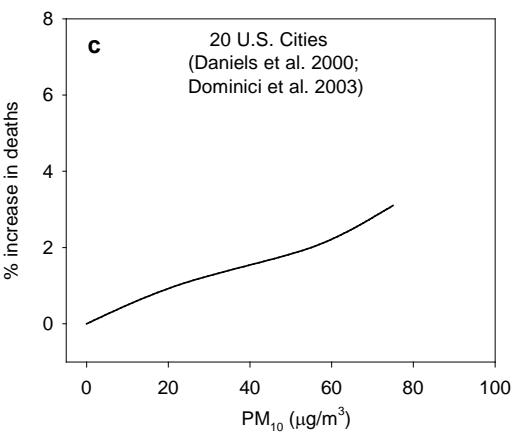
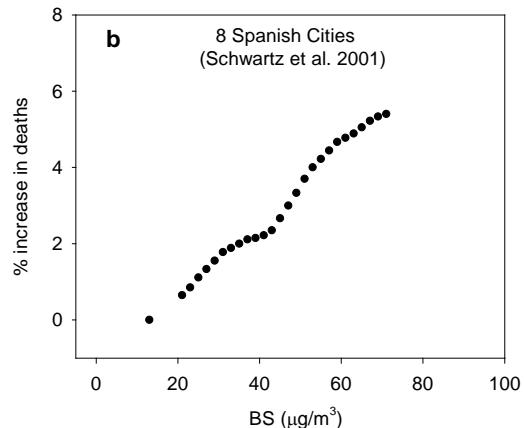
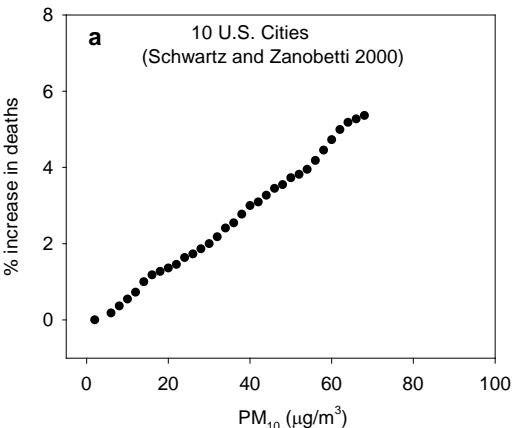
Examples:



Early to mid 2000's

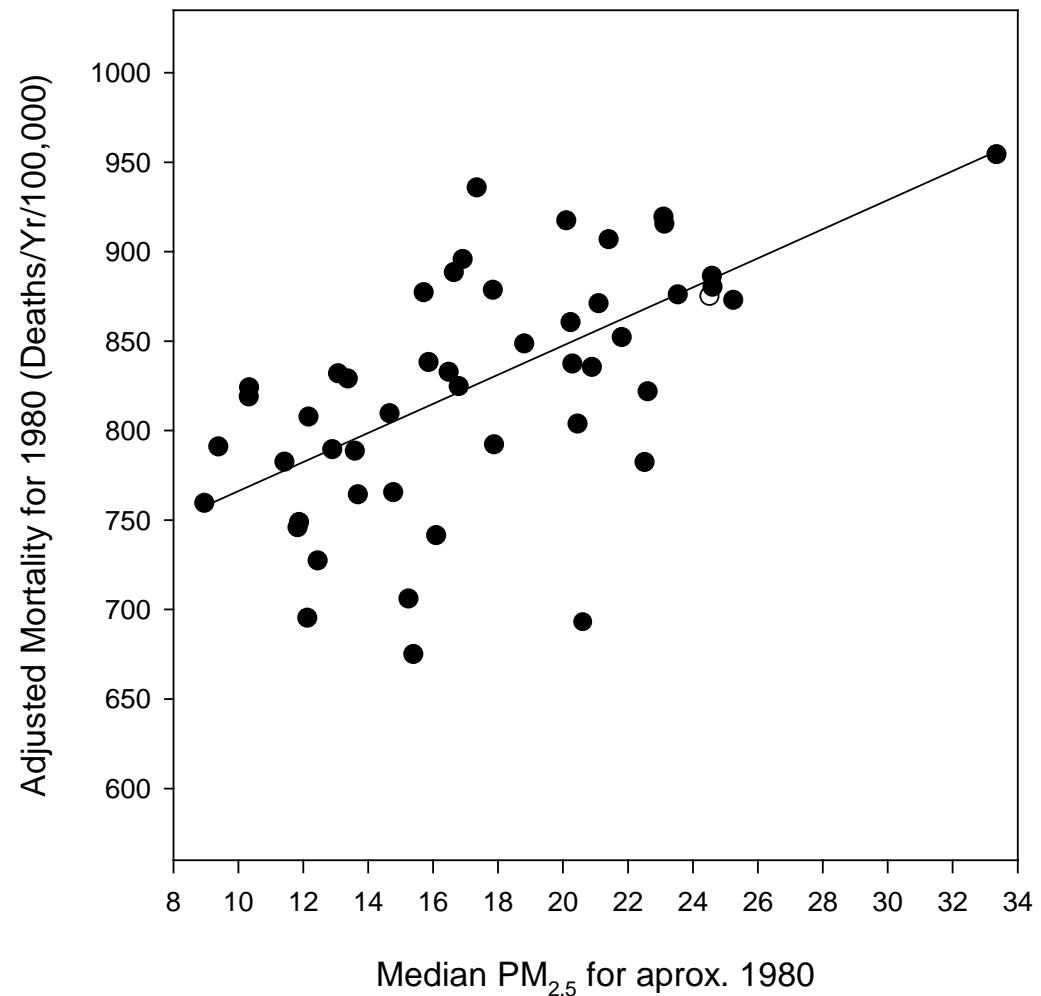
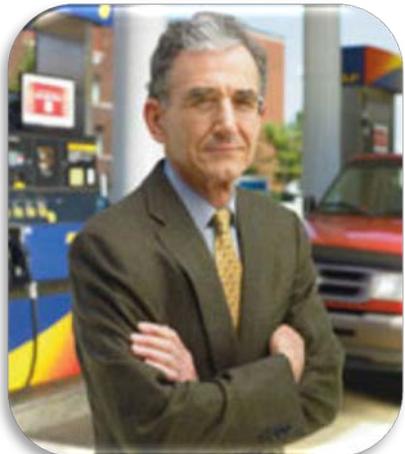
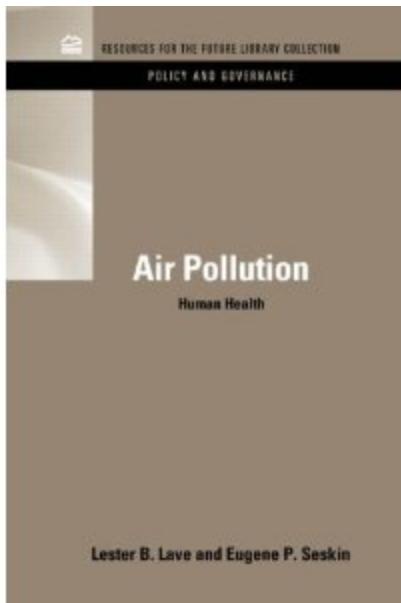
Combined or “meta-smoothed”
C-R function estimated using
multiple cities—enhancing
statistical power and
generalizability.

Examples:



1970's and 1980s

Population-based mortality rates studies
rates in U.S. cities.

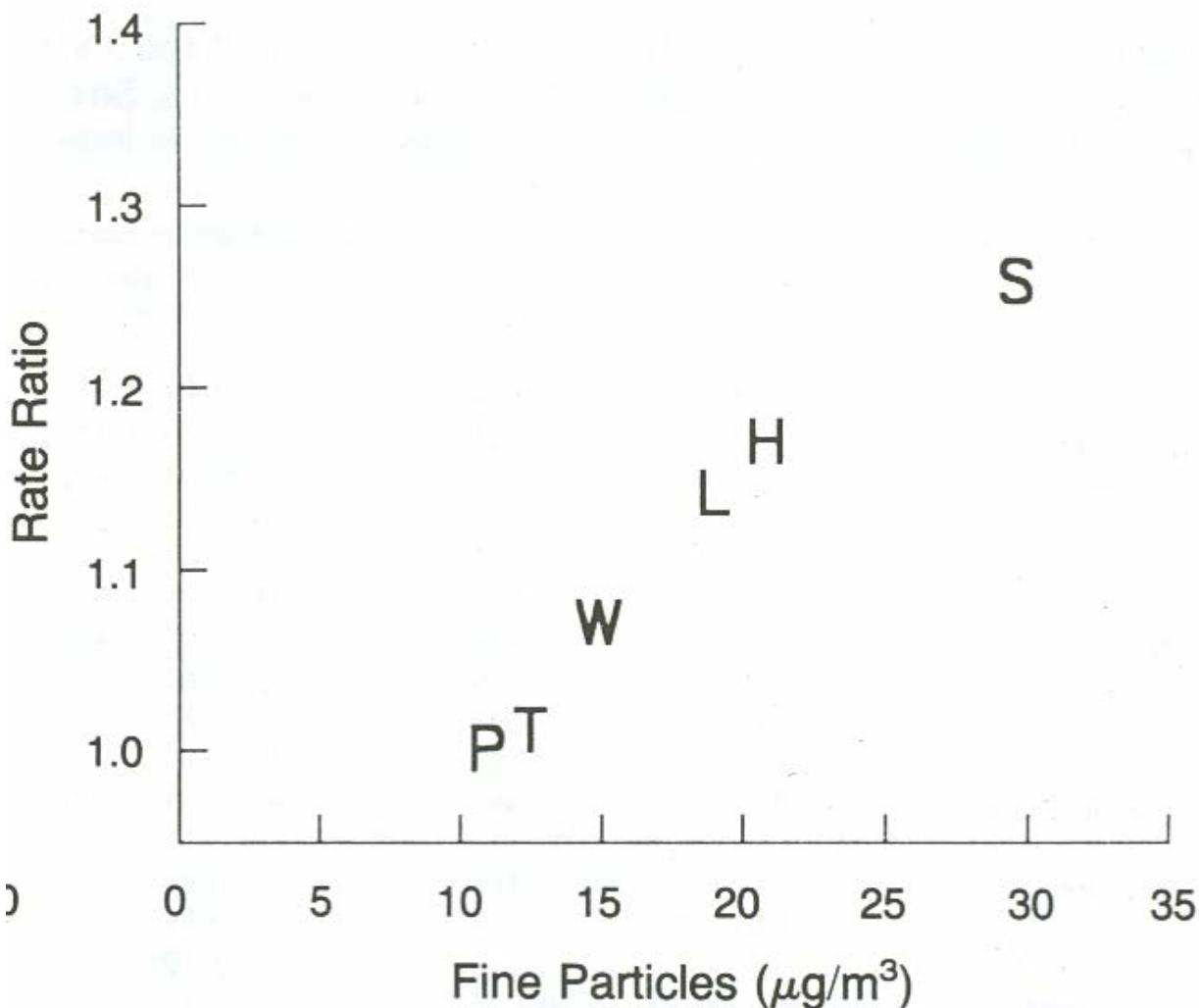


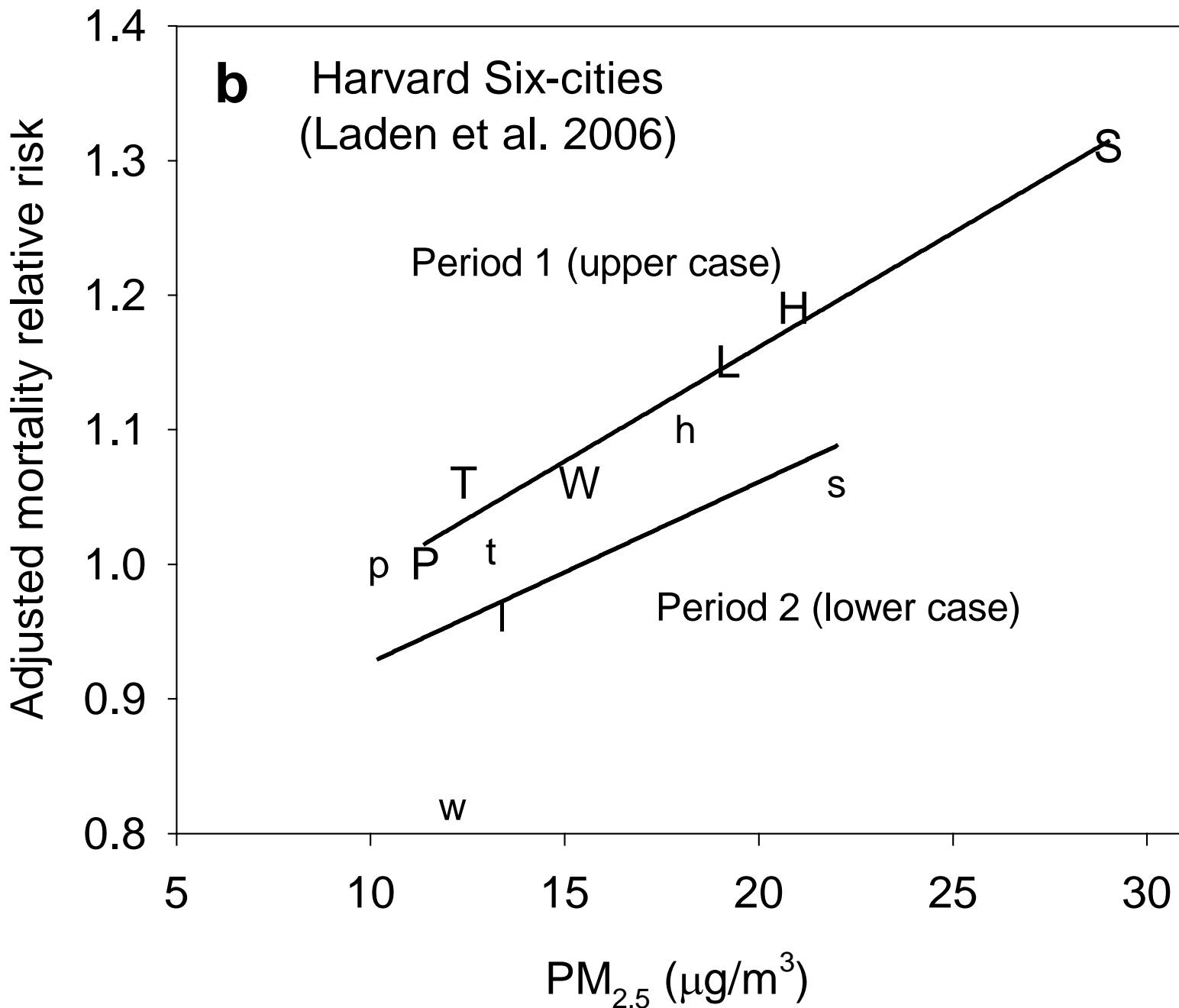
1993 + Cohort Mortality studies



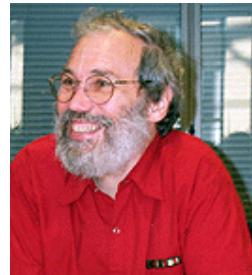
The NEW ENGLAND
JOURNAL of MEDICINE

Harvard Six Cities Study,
Dockery et al. 1993.



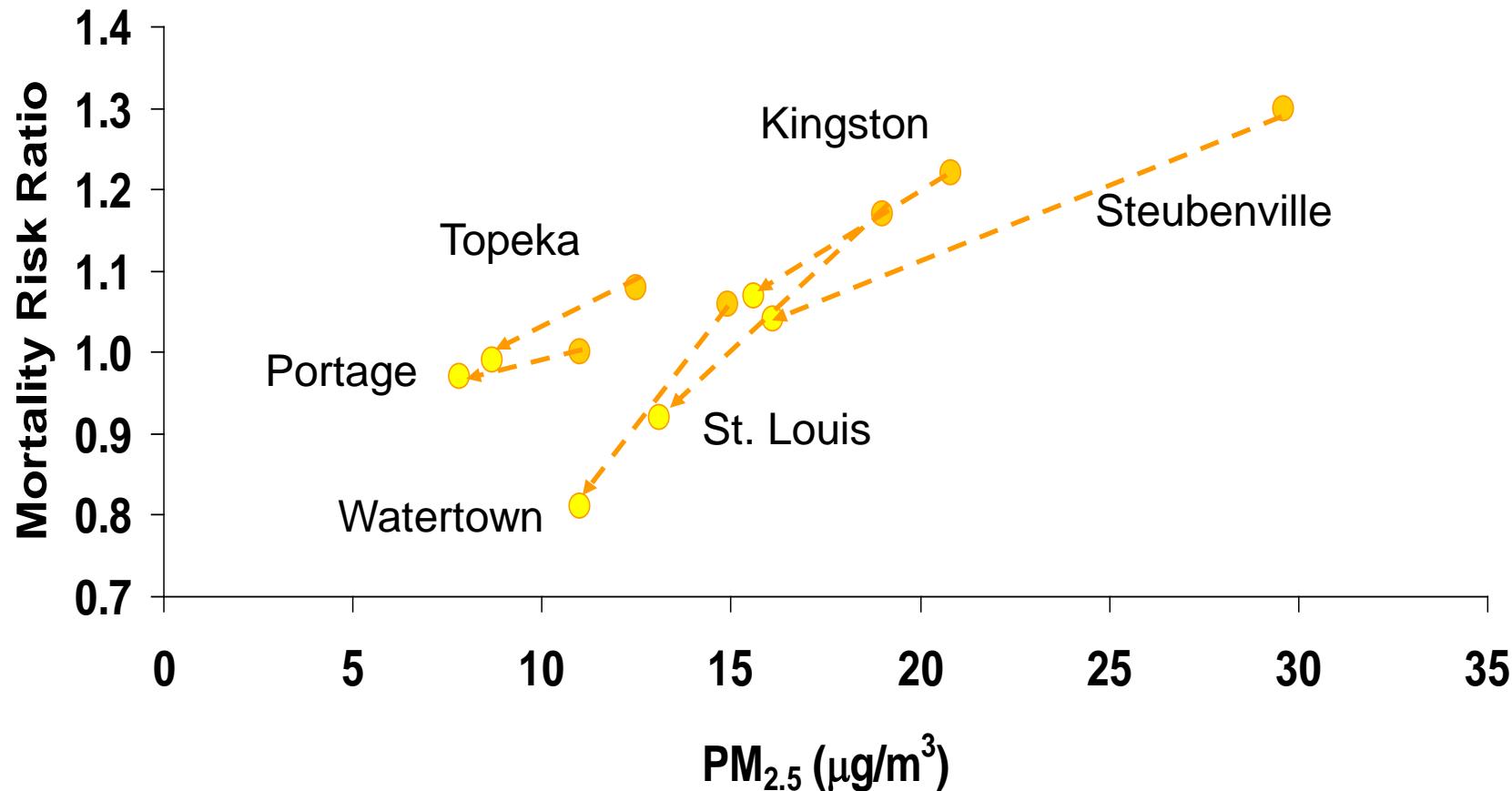


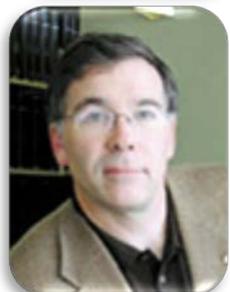
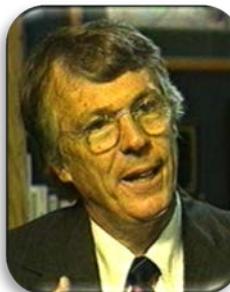
Reduction in fine particulate air pollution: Extended follow-up of the Harvard Six Cities Study (Laden, Schwartz, Speizer, Dockery. AJRCCM 2006)



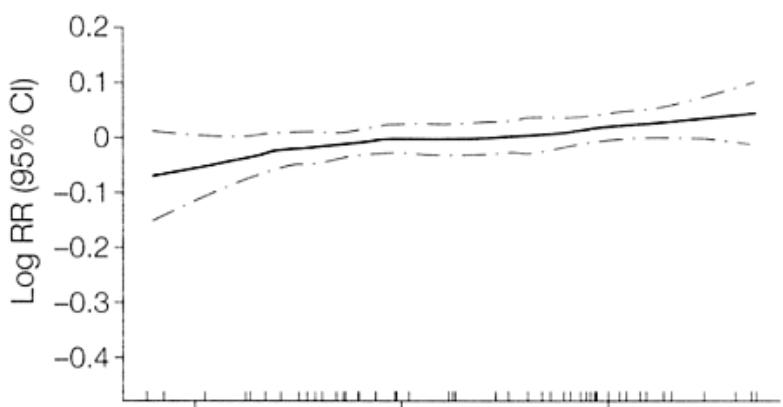
Francine Laden

Joel Schwartz

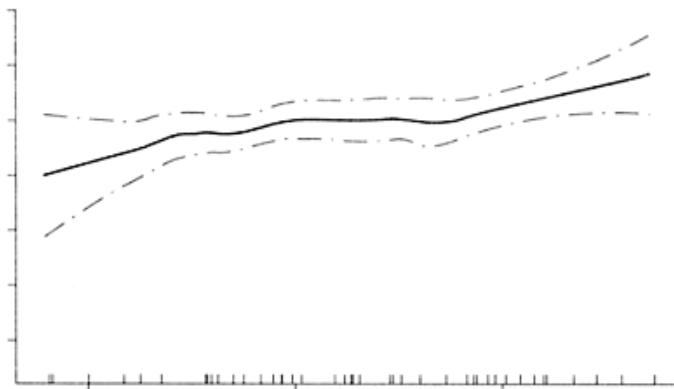




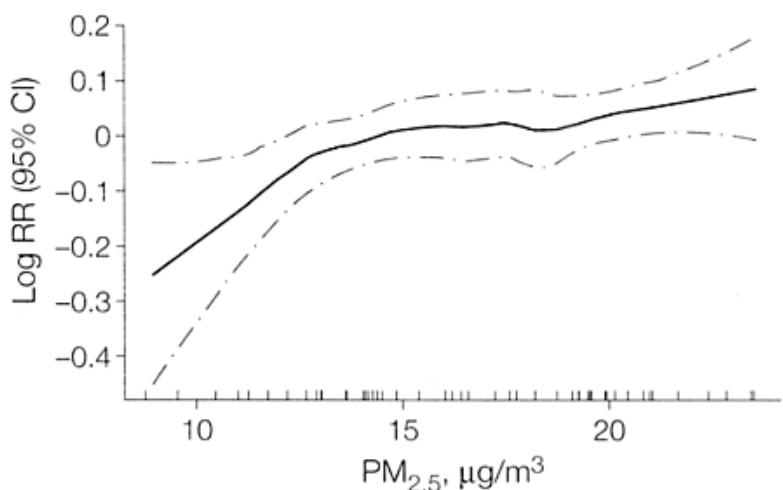
A All-Cause Mortality



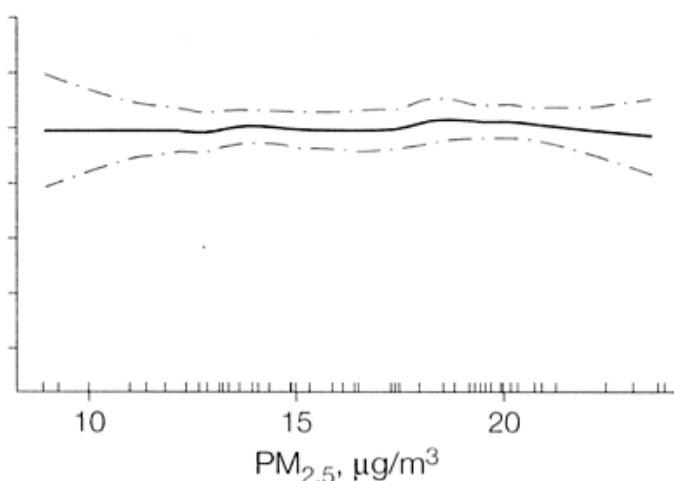
B Cardiopulmonary Mortality



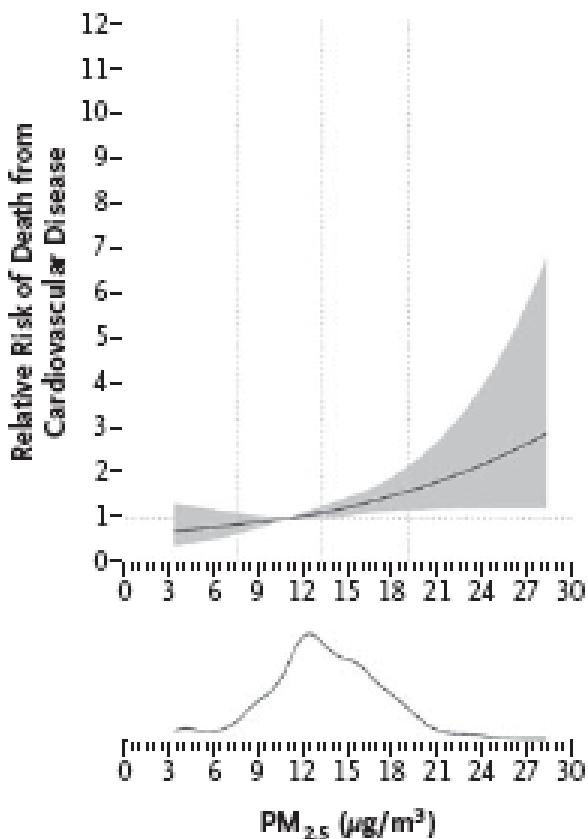
C Lung Cancer Mortality



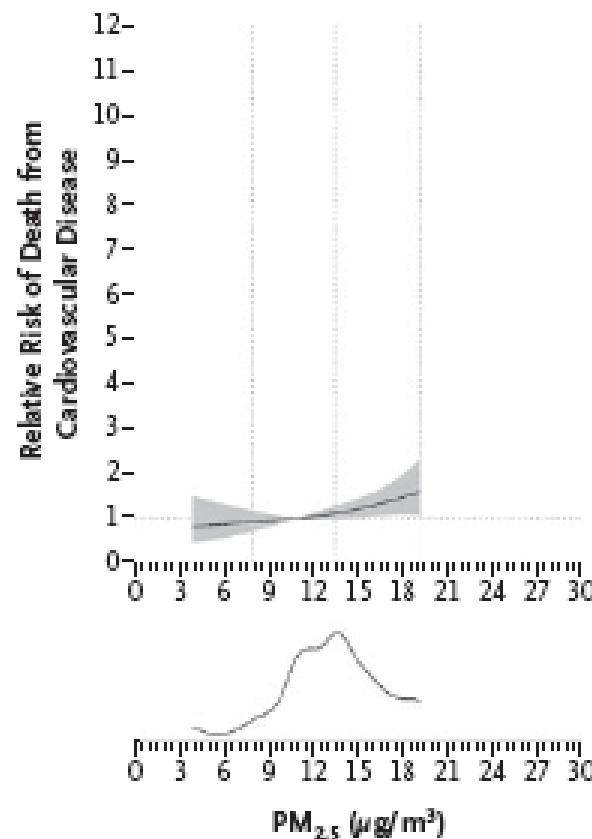
D All Other Cause Mortality



A Overall Effect



B Between-City Effect



C Within-City Effect

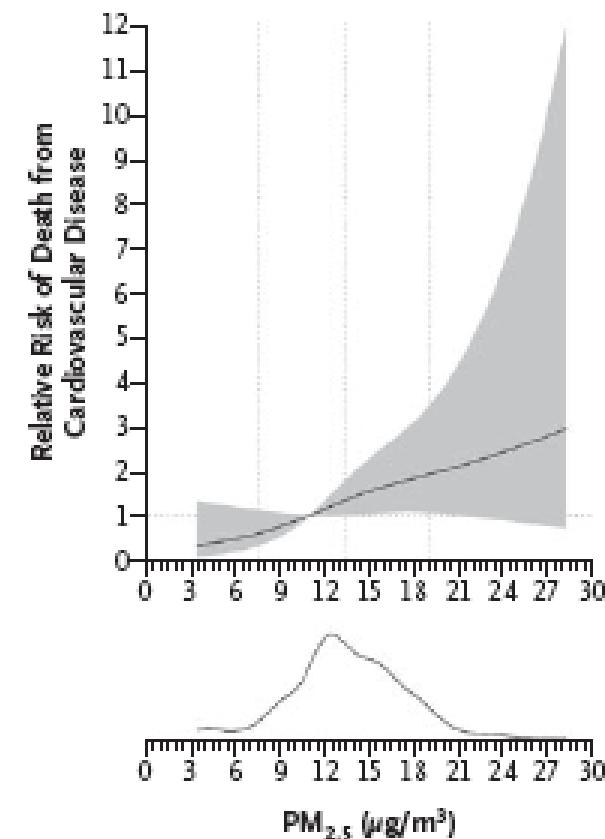


Figure 1. Level of Exposure to Fine Particulate Matter and the Risk of Death from Cardiovascular Causes in Women.

Miller et al. Long-Term exposure to Air Pollution and risk of Cardiovascular death Women. NEJM 2007

Risk of Nonaccidental and Cardiovascular Mortality in Relation to Long-term Exposure to Low Concentrations of Fine Particulate Matter: A Canadian National-Level Cohort Study

Dan L. Crouse,¹ Paul A. Peters,² Aaron van Donkelaar,³ Mark S. Goldberg,⁴ Paul J. Villeneuve,^{1,5} Orly Brion,¹ Saeeda Khan,² Dominic Odwa Atari,² Michael Jerrett,⁶ C. Arden Pope III,⁷ Michael Brauer,⁸ Jeffrey R. Brook,^{5,9} Randall V. Martin,^{3,10} David Stieb,¹ and Richard T. Burnett¹

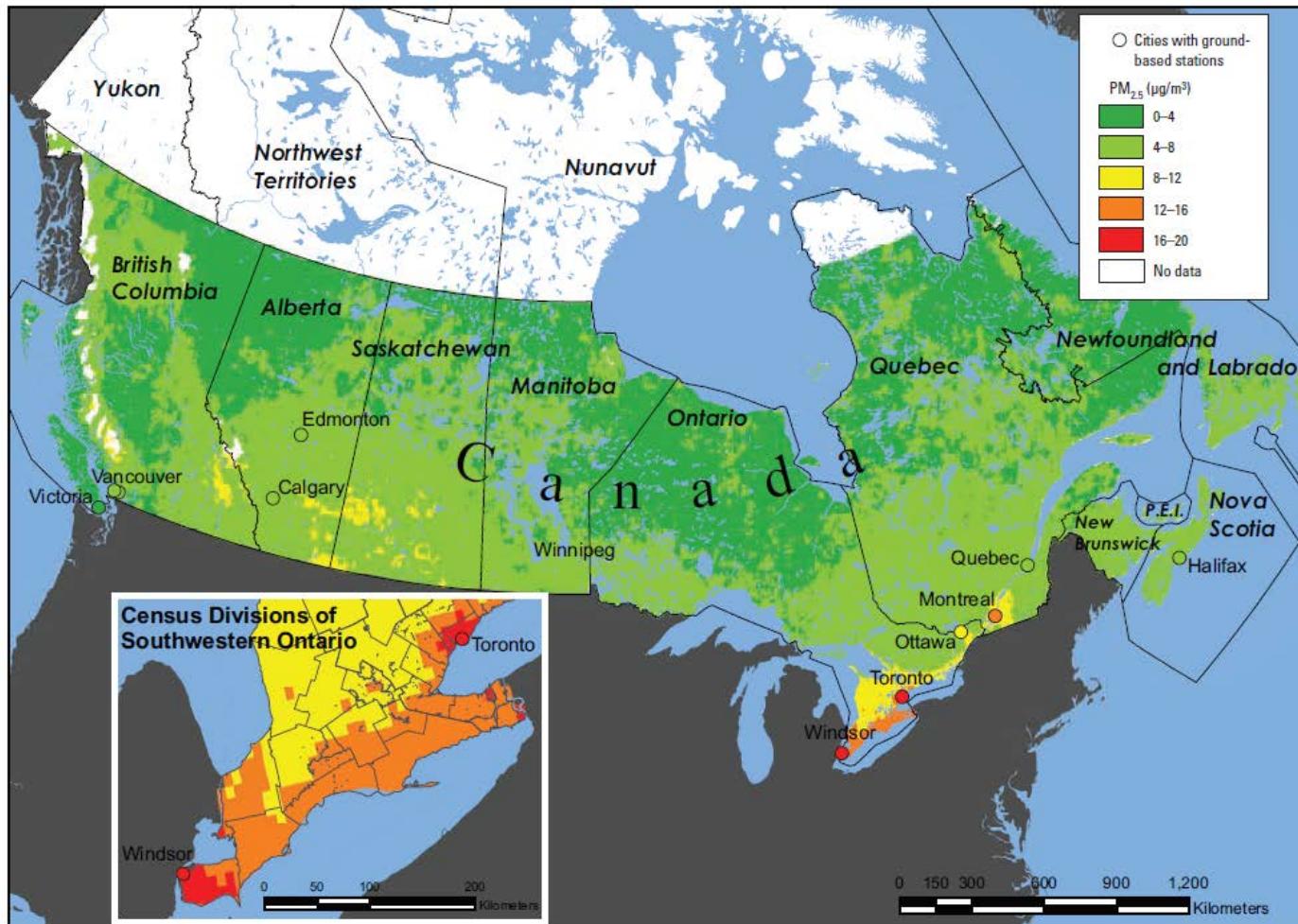


Figure 1. Mean satellite-derived estimates of PM_{2.5} across Canada, 2001–2006, and the mean concentrations in the 11 cities included in our subcohort analysis. P.E.I., Prince Edward Island.

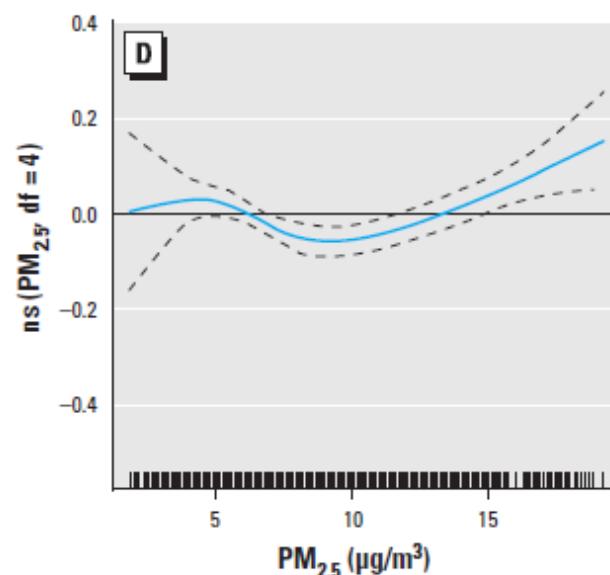
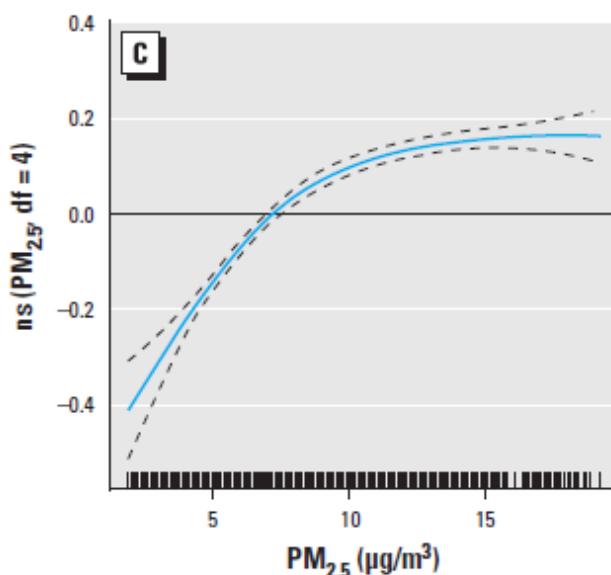
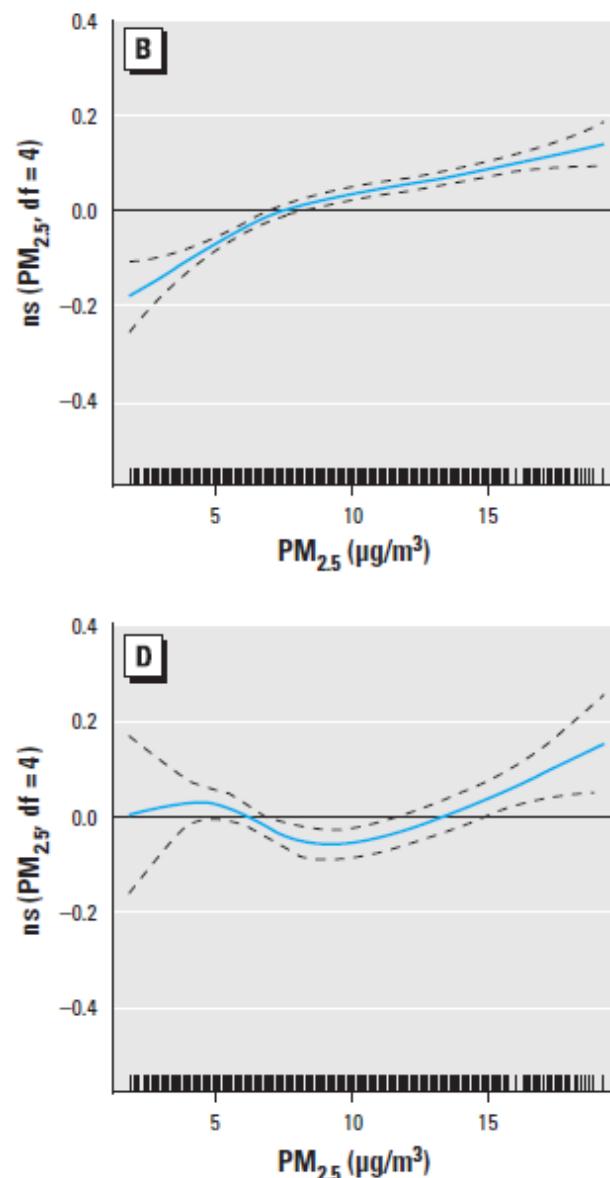
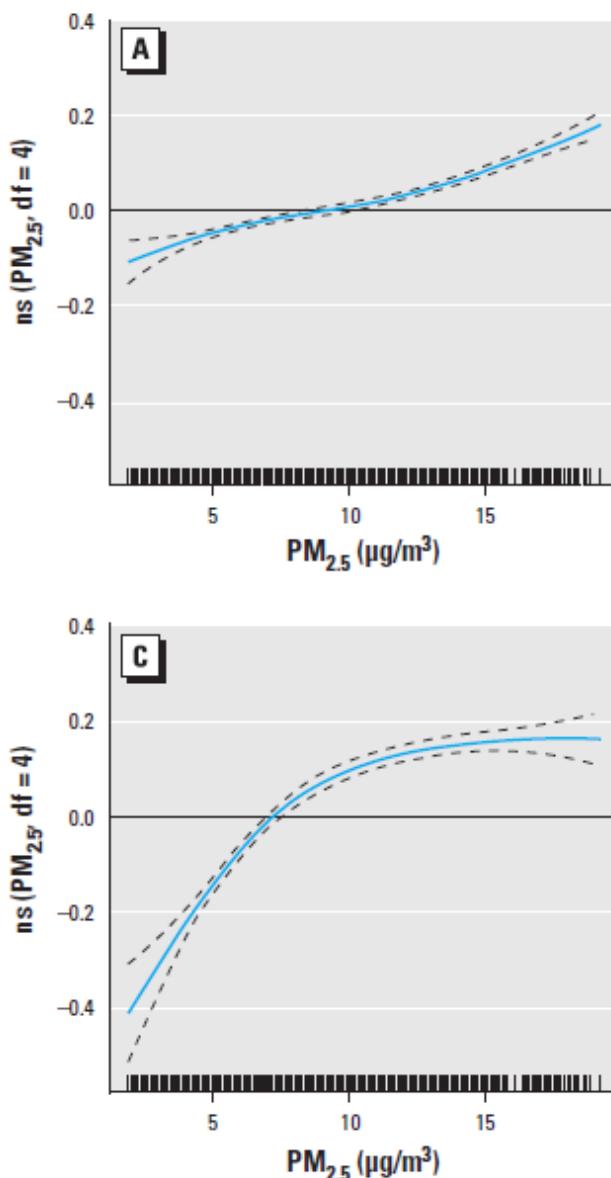
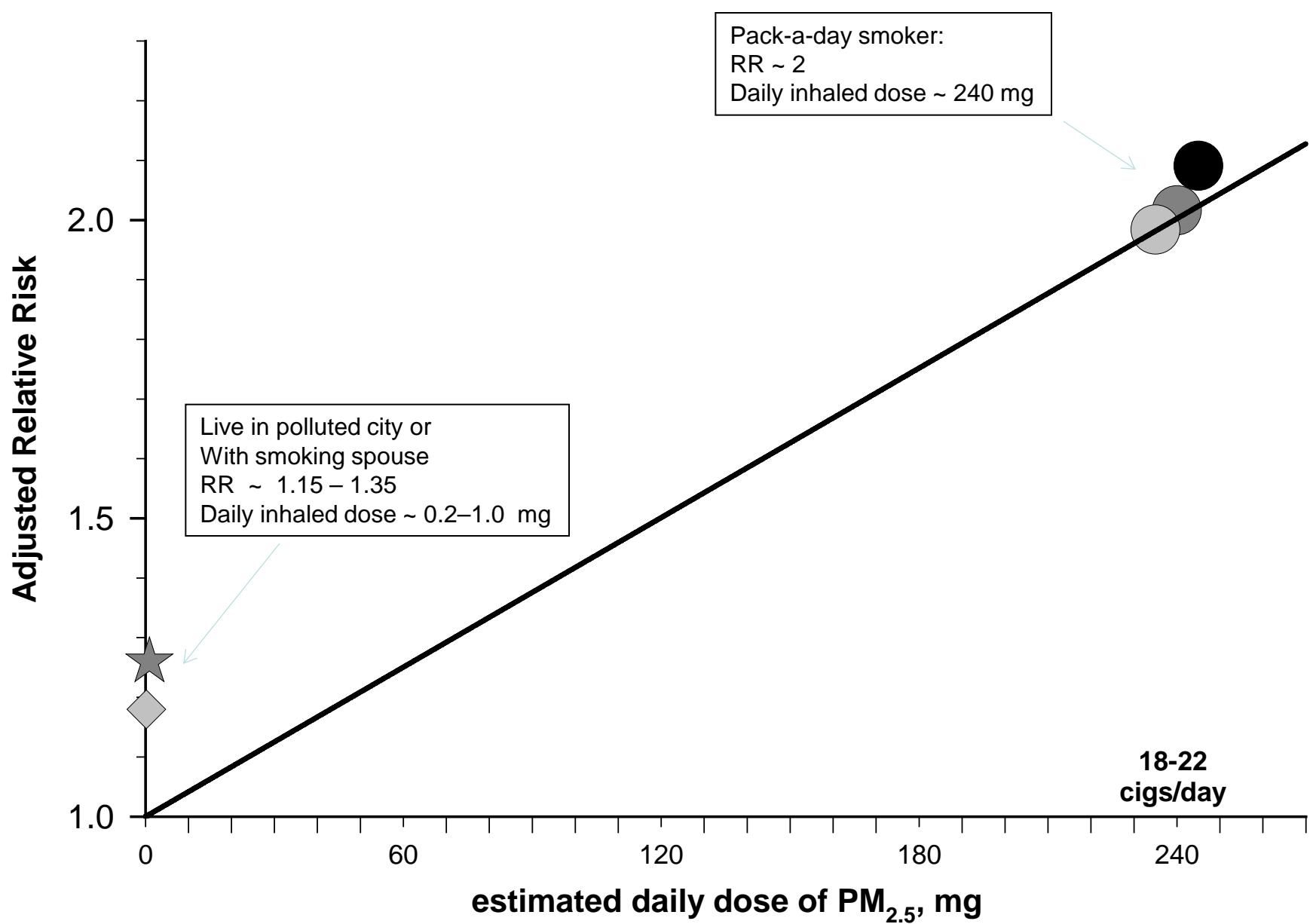


Figure 2. Concentration-response curves (solid lines) and 95% CIs (dashed lines) based on natural spline (ns) models with 4 df, standard Cox models stratified by age and sex, adjusted for all individual-level covariates, urban/rural indicator, and ecological covariates. (A) Nonaccidental causes. (B) Cardiovascular disease. (C) Ischemic heart disease. (D) Cerebrovascular disease. The tick marks on the x-axis identify the location of the PM_{2.5} concentrations.



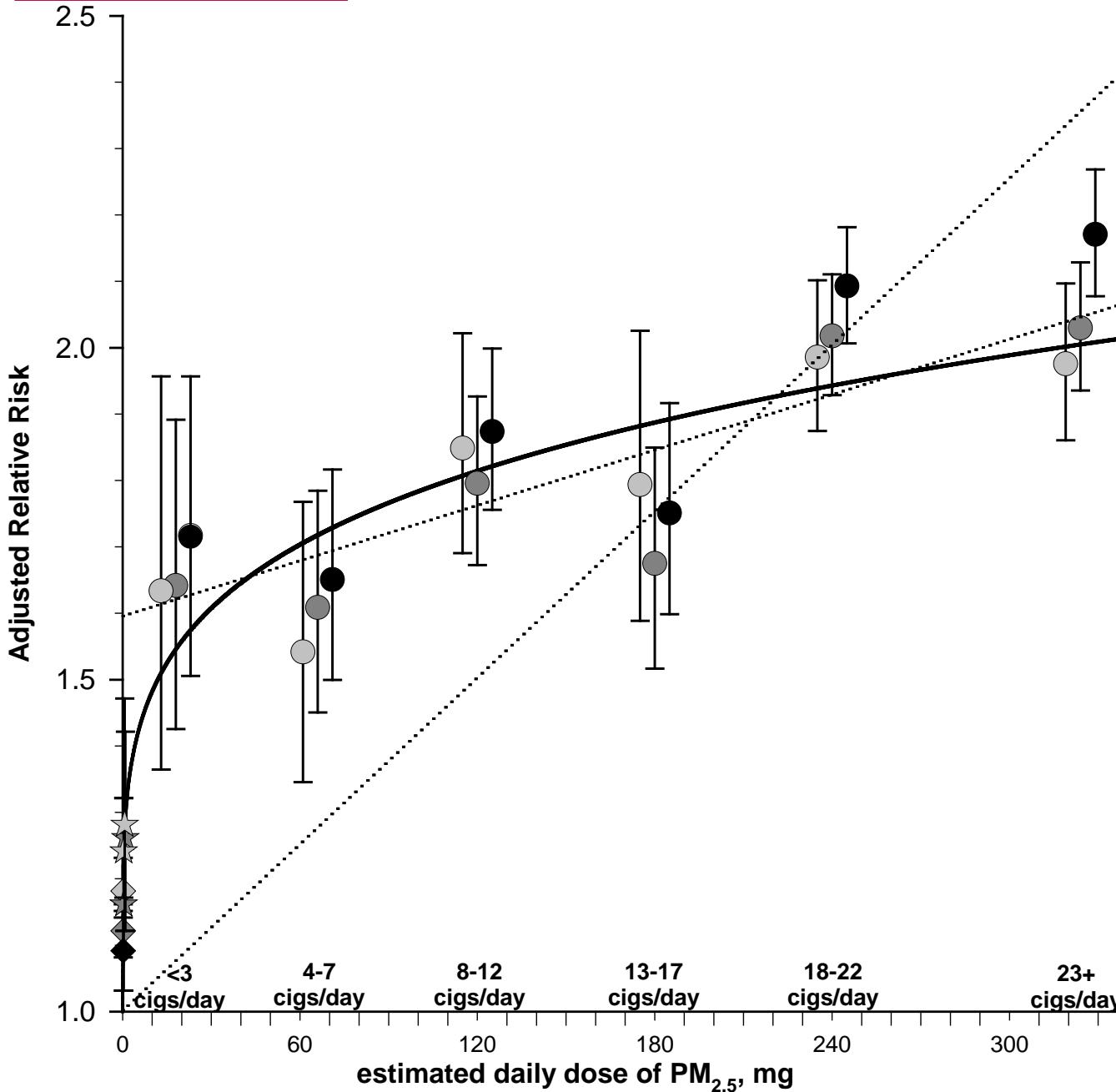


Figure 1. Adjusted relative risks (and 95% CIs) of IHD (light gray), CVD (dark gray), and CPD (black) mortality plotted over estimated daily dose of $\text{PM}_{2.5}$ from different increments of current cigarette smoking. Diamonds represent comparable mortality risk estimates for $\text{PM}_{2.5}$ from air pollution. Stars represent comparable pooled relative risk estimates associated with SHS exposure from the 2006 Surgeon General's report and from the INTERHEART study.

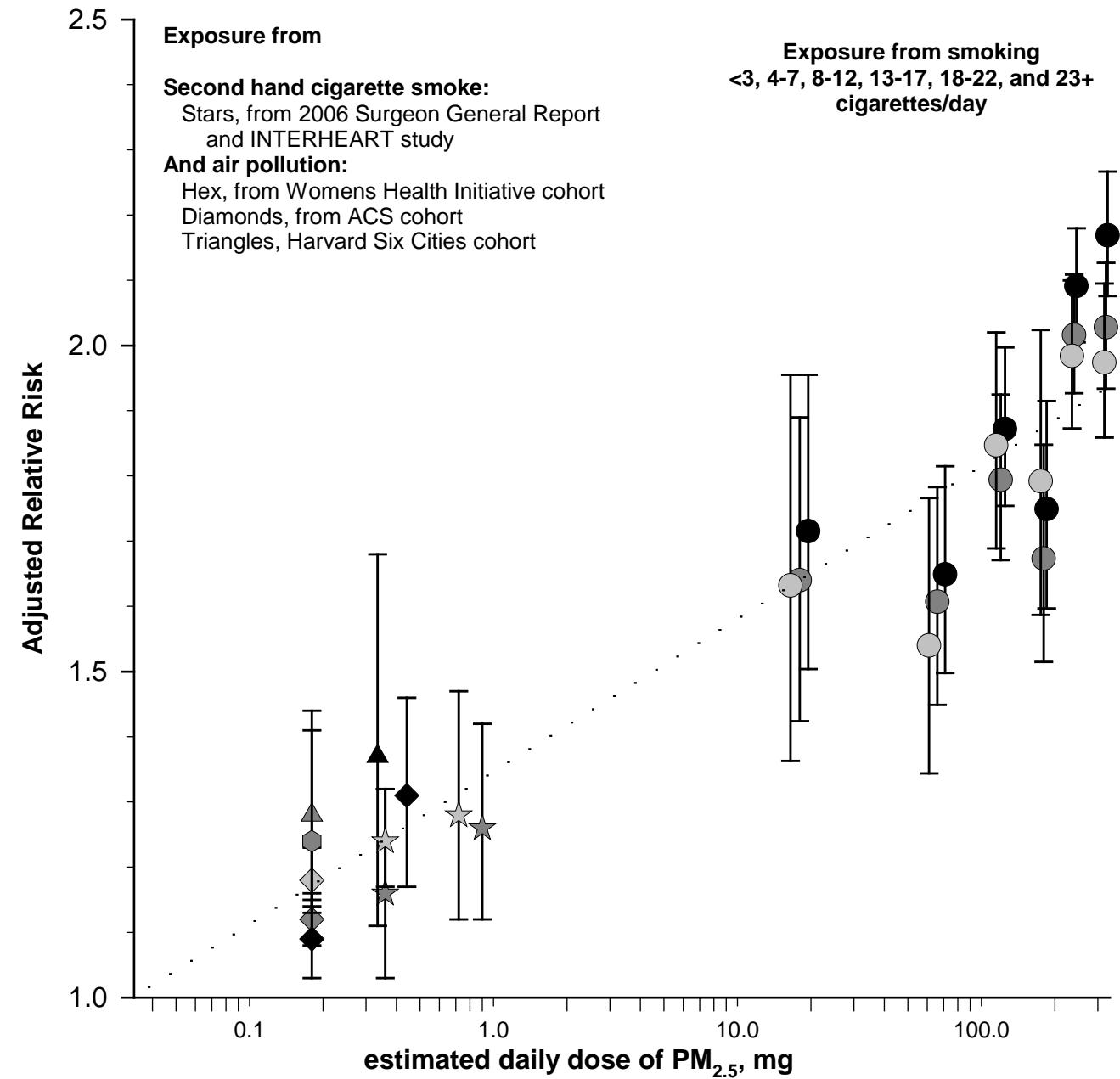
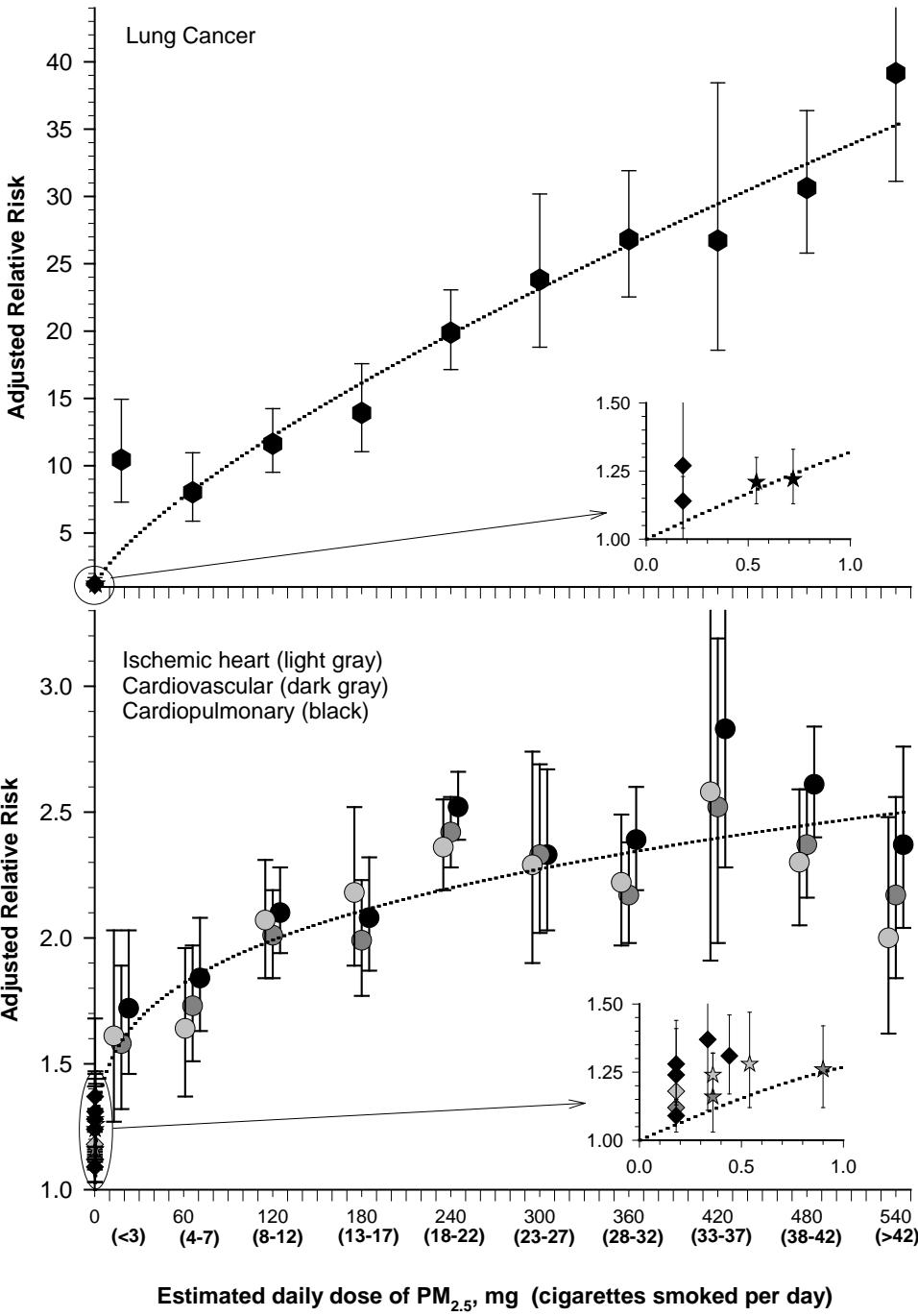
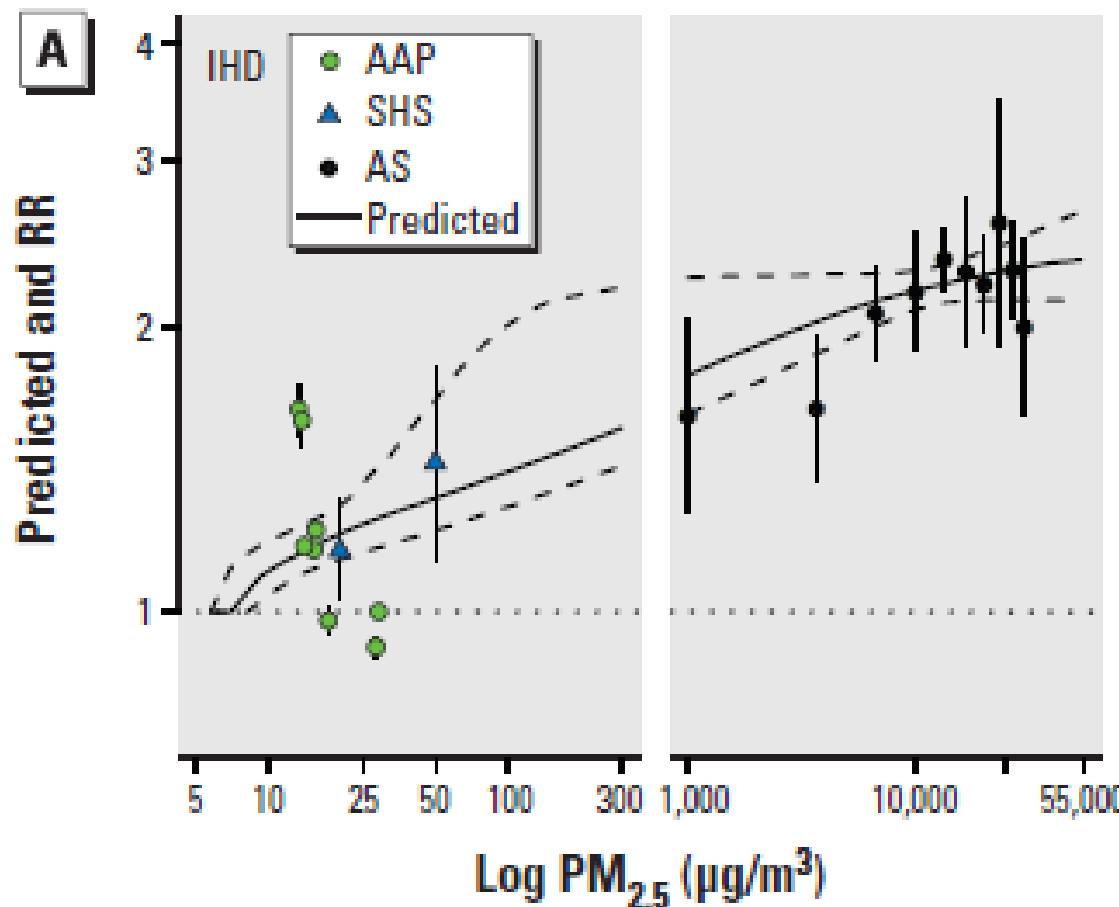


Figure 2. Adjusted relative risks (and 95% CIs) of ischemic heart disease (light gray), cardiovascular (dark gray), and cardiopulmonary (black) mortality plotted over baseline estimated daily dose (using a log scale) of $\text{PM}_{2.5}$ from current cigarette smoking (relative to never smokers), SHS,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



An Integrated Risk Function for Estimating the Global Burden of Disease Attributable to Ambient Fine Particulate Matter Exposure

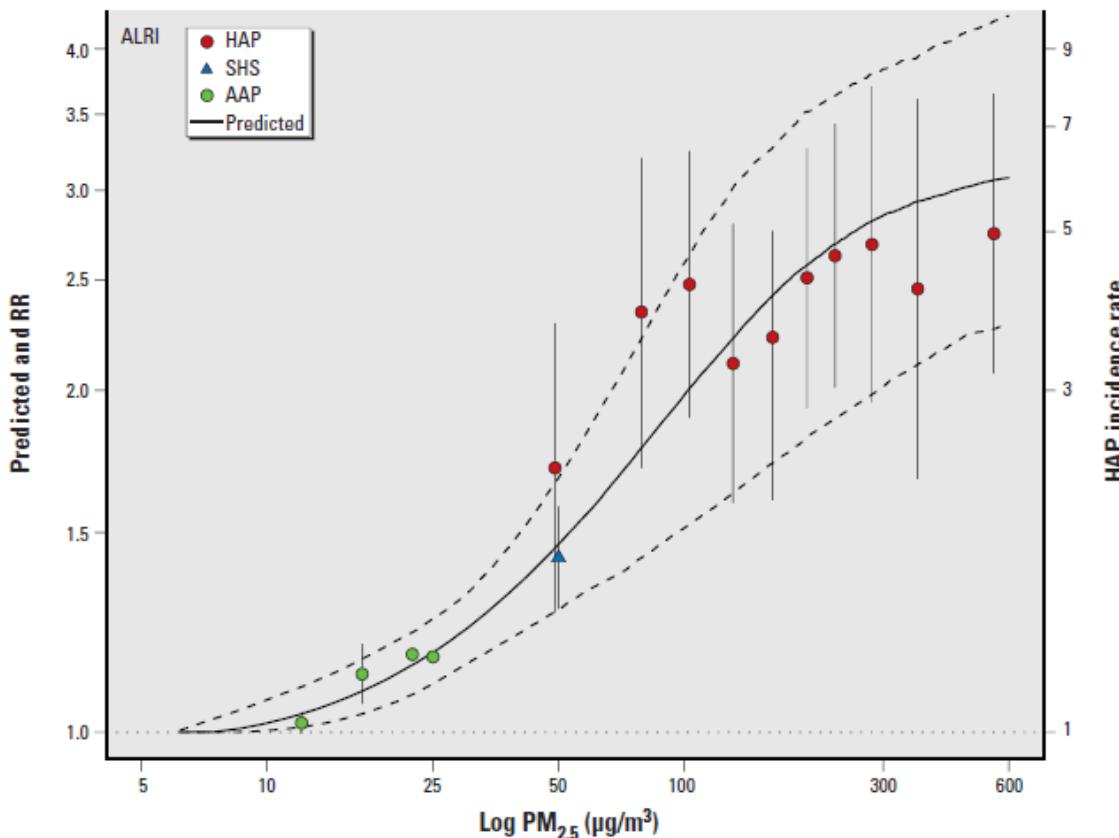
Richard T. Burnett,¹ C. Arden Pope III,² Majid Ezzati,³ Casey Olives,⁴ Stephen S. Lim,⁵ Sumi Mehta,⁶ Hwashin H. Shin,¹ Gitanjali Singh,⁷ Bryan Hubbell,⁸ Michael Brauer,⁹ H. Ross Anderson,¹⁰ Kirk R. Smith,¹¹ John R. Balmes,^{12,13} Nigel G. Bruce,¹⁴ Haidong Kan,¹⁵ Francine Laden,¹⁶ Annette Prüss-Ustün,¹⁷ Michelle C. Turner,¹⁸ Susan M. Gapstur,¹⁹ W. Ryan Diver,¹⁹ and Aaron Cohen^{20*}



Integrated Exposure-Response (IER) model for ischemic heart disease integrating information from studies of ambient air pollution (AAP), second hand smoke (SHS), and active smoking.

An Integrated Risk Function for Estimating the Global Burden of Disease Attributable to Ambient Fine Particulate Matter Exposure

Richard T. Burnett,¹ C. Arden Pope III,² Majid Ezzati,³ Casey Olives,⁴ Stephen S. Lim,⁵ Sumi Mehta,⁶ Hwashin H. Shin,¹ Gitanjali Singh,⁷ Bryan Hubbell,⁸ Michael Brauer,⁹ H. Ross Anderson,¹⁰ Kirk R. Smith,¹¹ John R. Balmes,^{12,13} Nigel G. Bruce,¹⁴ Haidong Kan,¹⁵ Francine Laden,¹⁶ Annette Prüss-Ustün,¹⁷ Michelle C. Turner,¹⁸ Susan M. Gapstur,¹⁹ W. Ryan Diver,¹⁹ and Aaron Cohen^{20*}



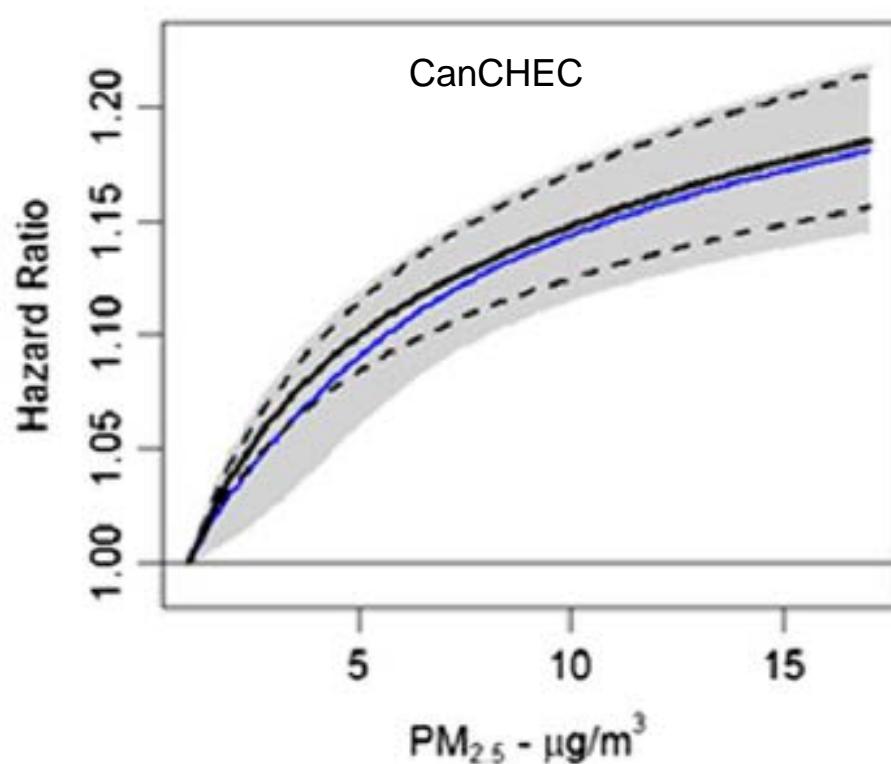
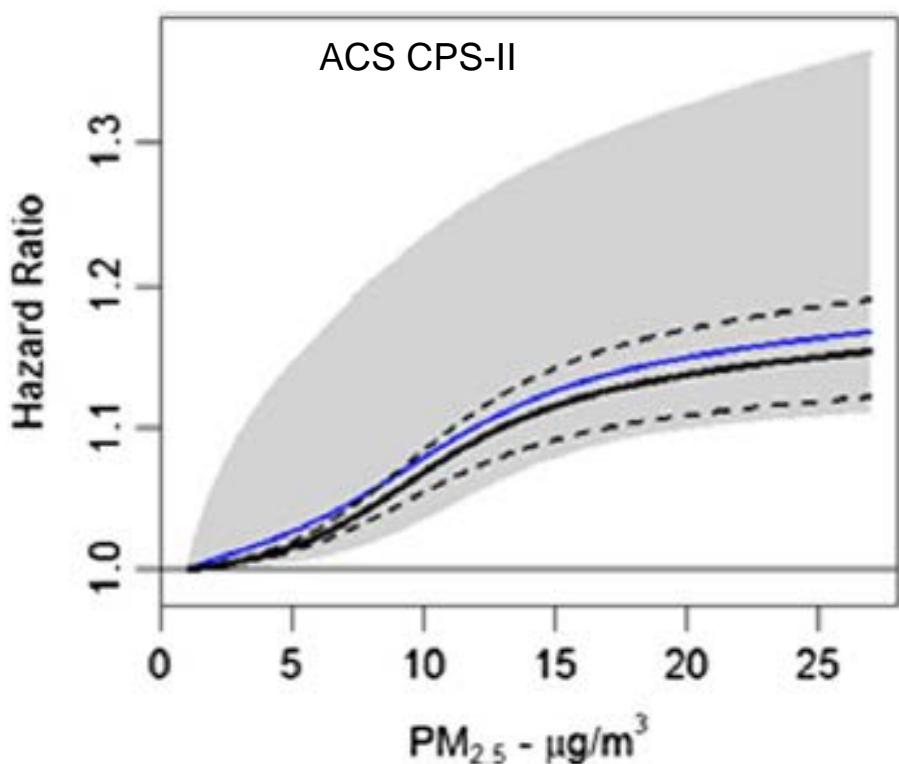
Integrated Exposure-Response (IER) model for acute lower respiratory infection in infants integrating information from studies of household air pollution (HAP), ambient air pollution (AAP), and second hand smoke (SHS).

Figure 2. Predicted values of IER model (solid line) and 95% CIs (dashed line) and type-specific RRs (points) and 95% CIs (error bars) for ALRI in infants.

A class of non-linear exposure-response models suitable for health impact assessment applicable to large cohort studies of ambient air pollution

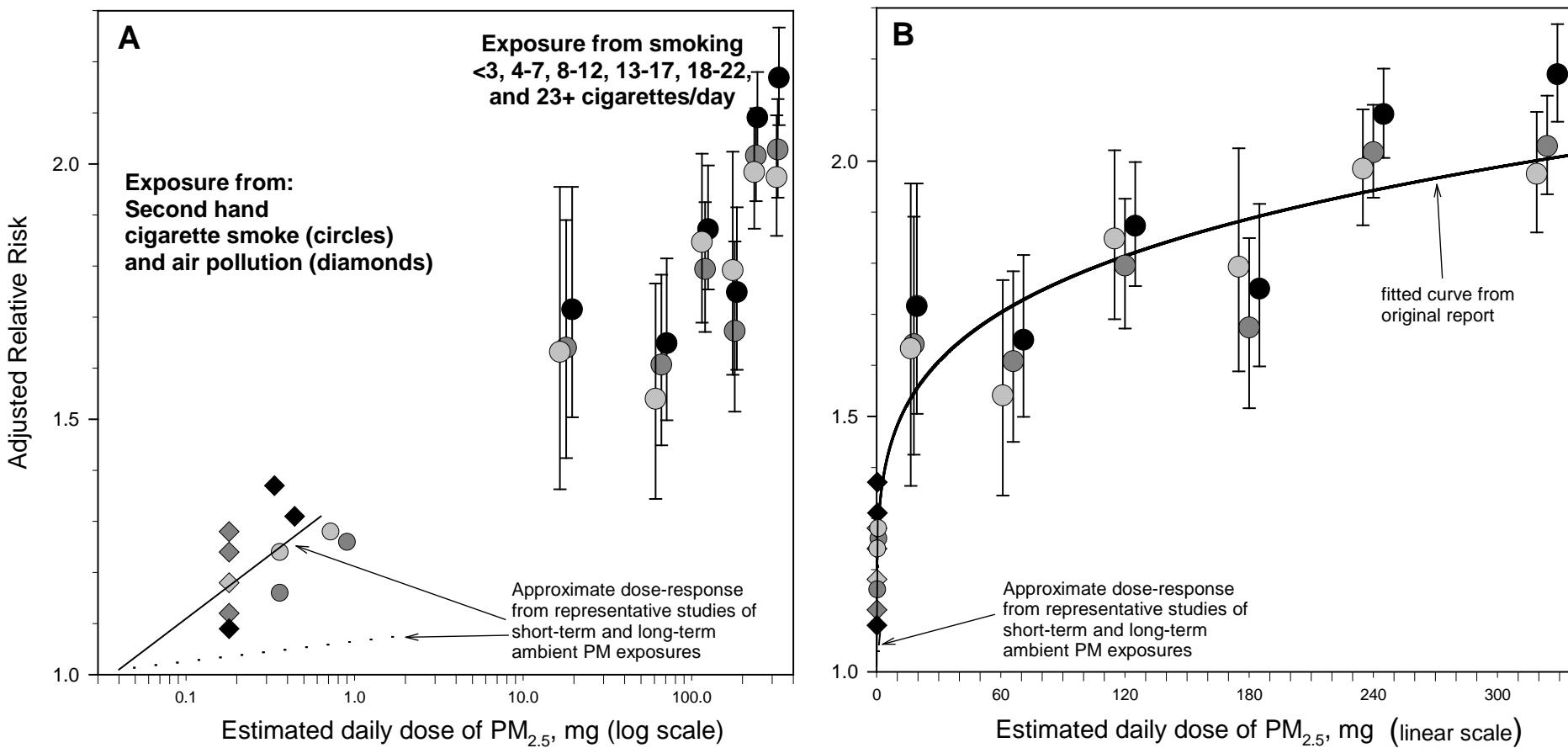
Air Qual Atmos Health 2016

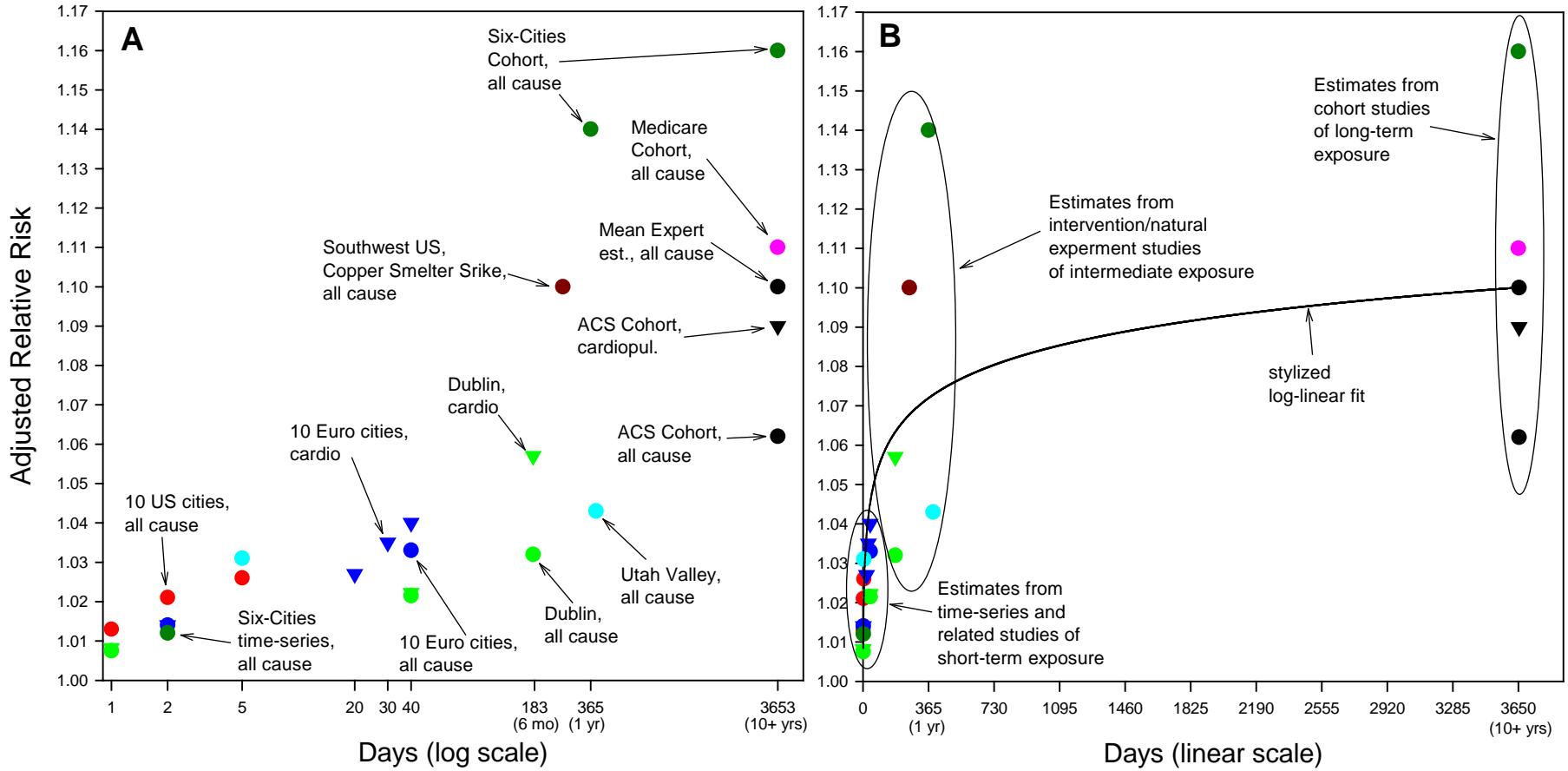
Masoud M. Nasari¹ • Mieczysław Szyszkowicz¹ • Hong Chen² • Daniel Crouse¹ •
Michelle C. Turner^{3,4,5,6} • Michael Jerrett⁷ • C. Arden Pope III⁸ • Bryan Hubbell⁹ •
Neal Fann⁹ • Aaron Cohen¹⁰ • Susan M. Gapstur¹¹ • W. Ryan Diver¹¹ • David Stieb¹ •
Mohammad H. Forouzanfar¹² • Sun-Young Kim¹³ • Casey Olives¹⁴ • Daniel Krewski³ •
Richard T. Burnett¹



How is cardiovascular disease mortality risk affected by duration and intensity of fine particulate matter exposure? An integration of the epidemiologic evidence

C. Arden Pope III • Robert D. Brook •
Richard T. Burnett • Douglas W. Dockery





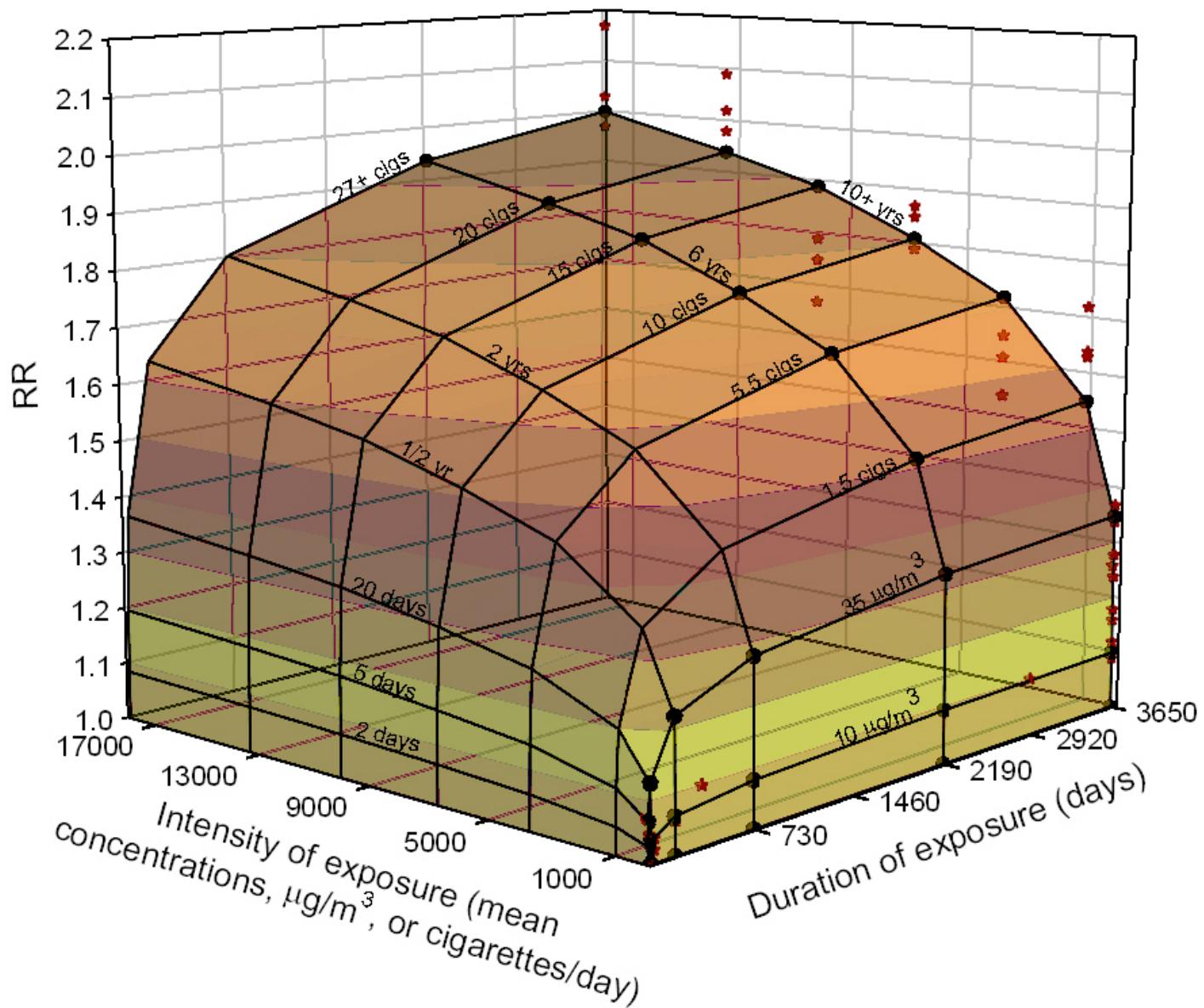
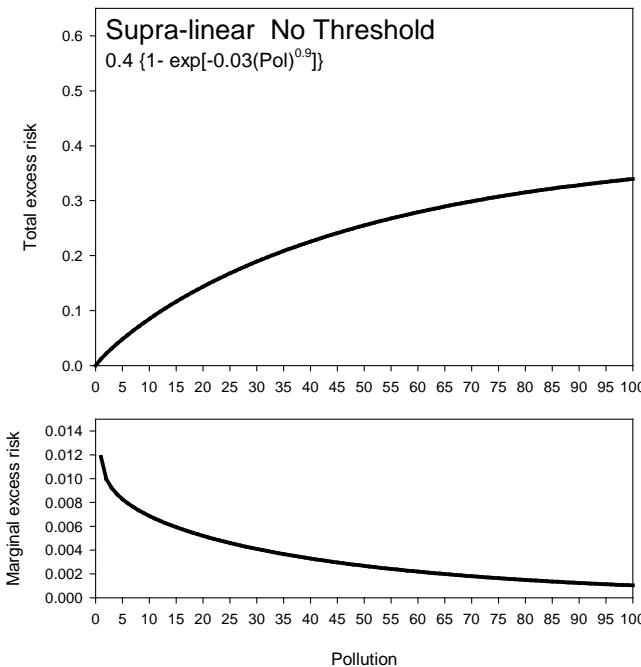
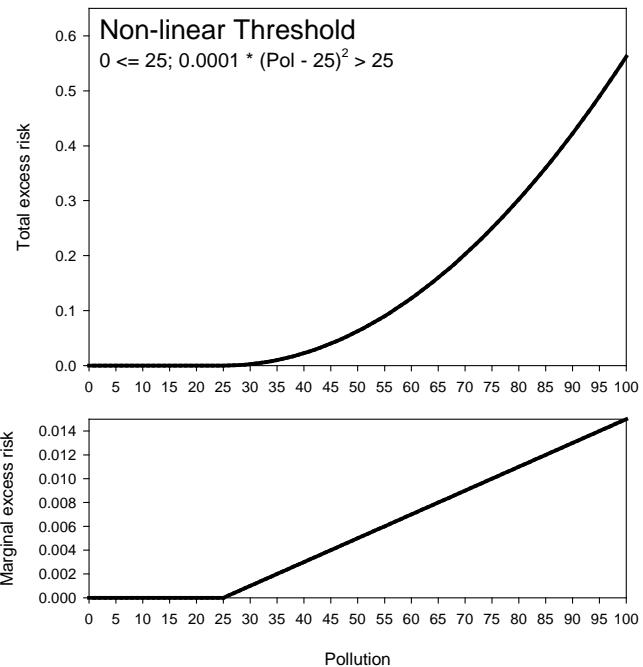
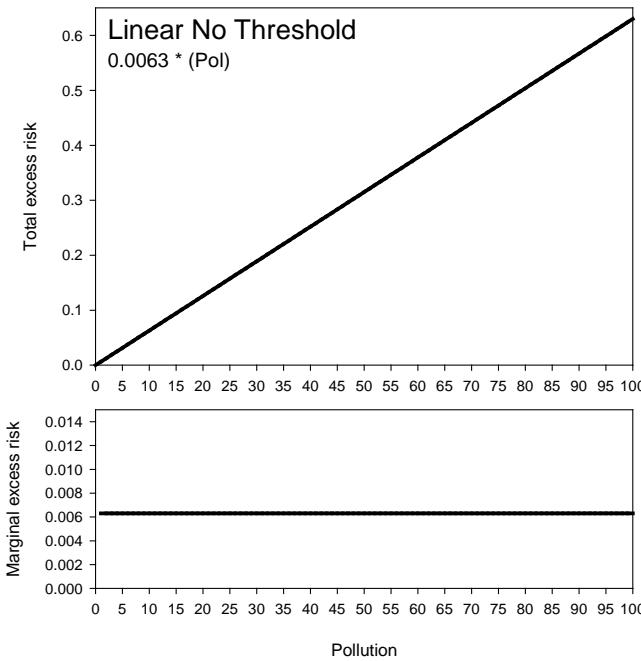
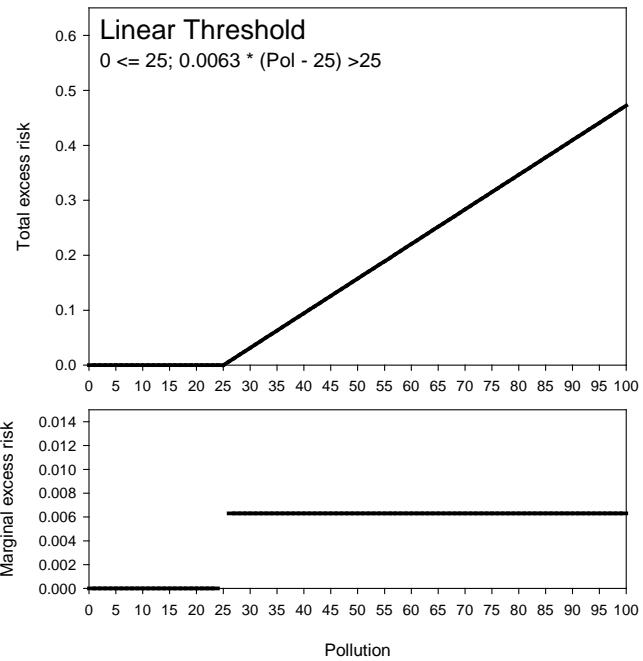
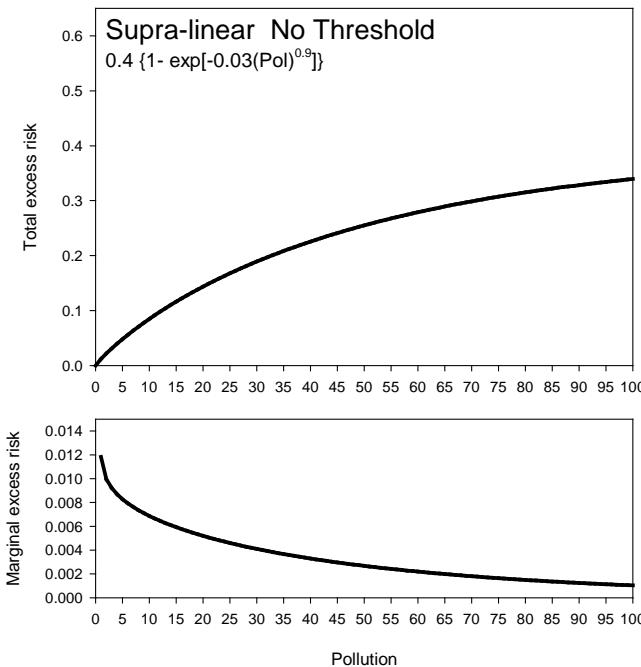
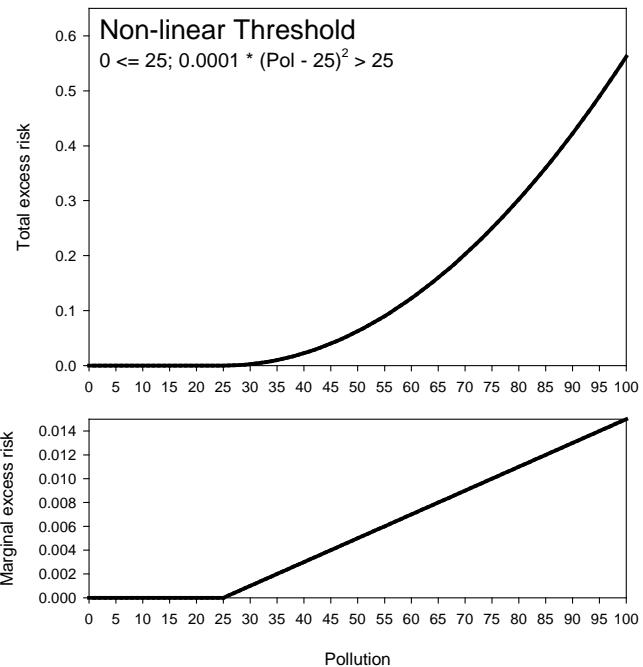
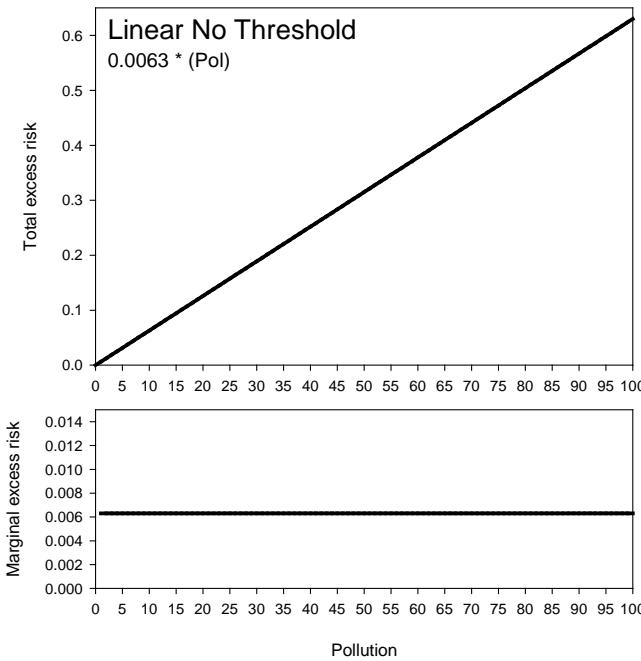
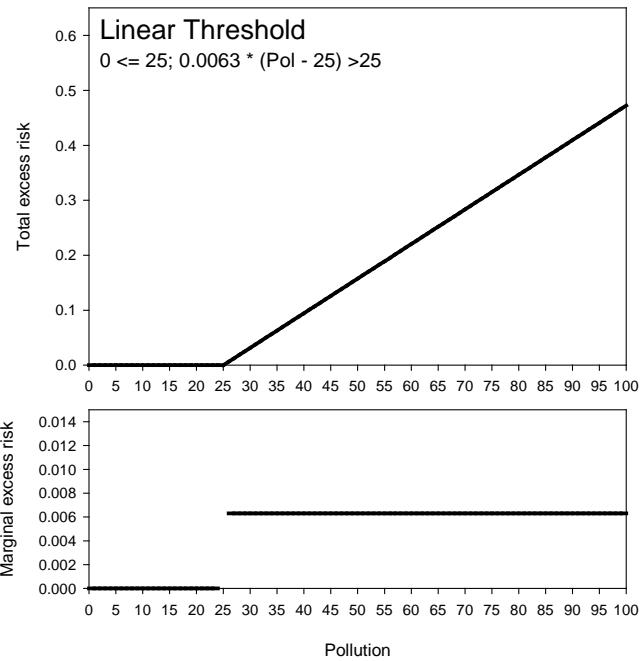
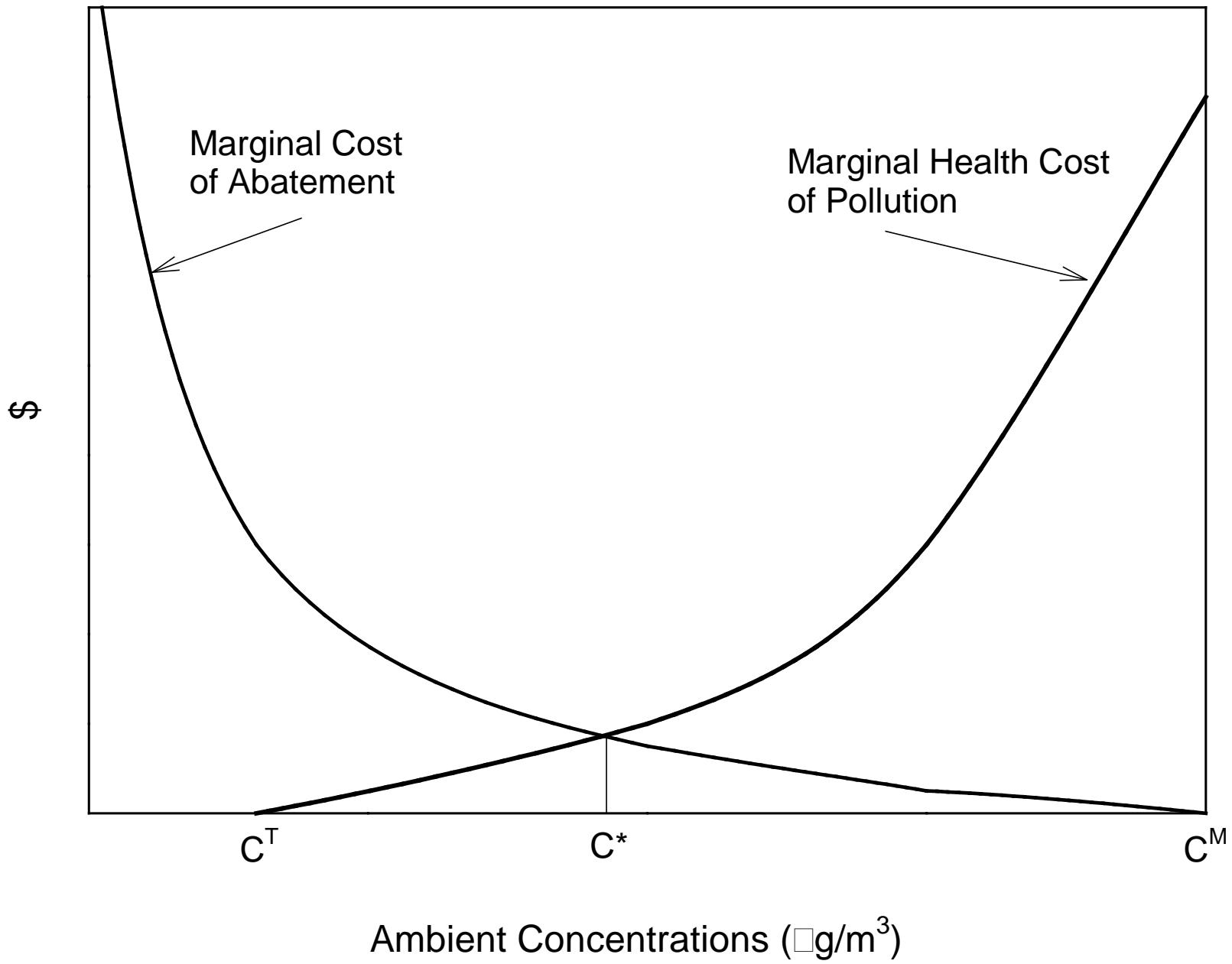
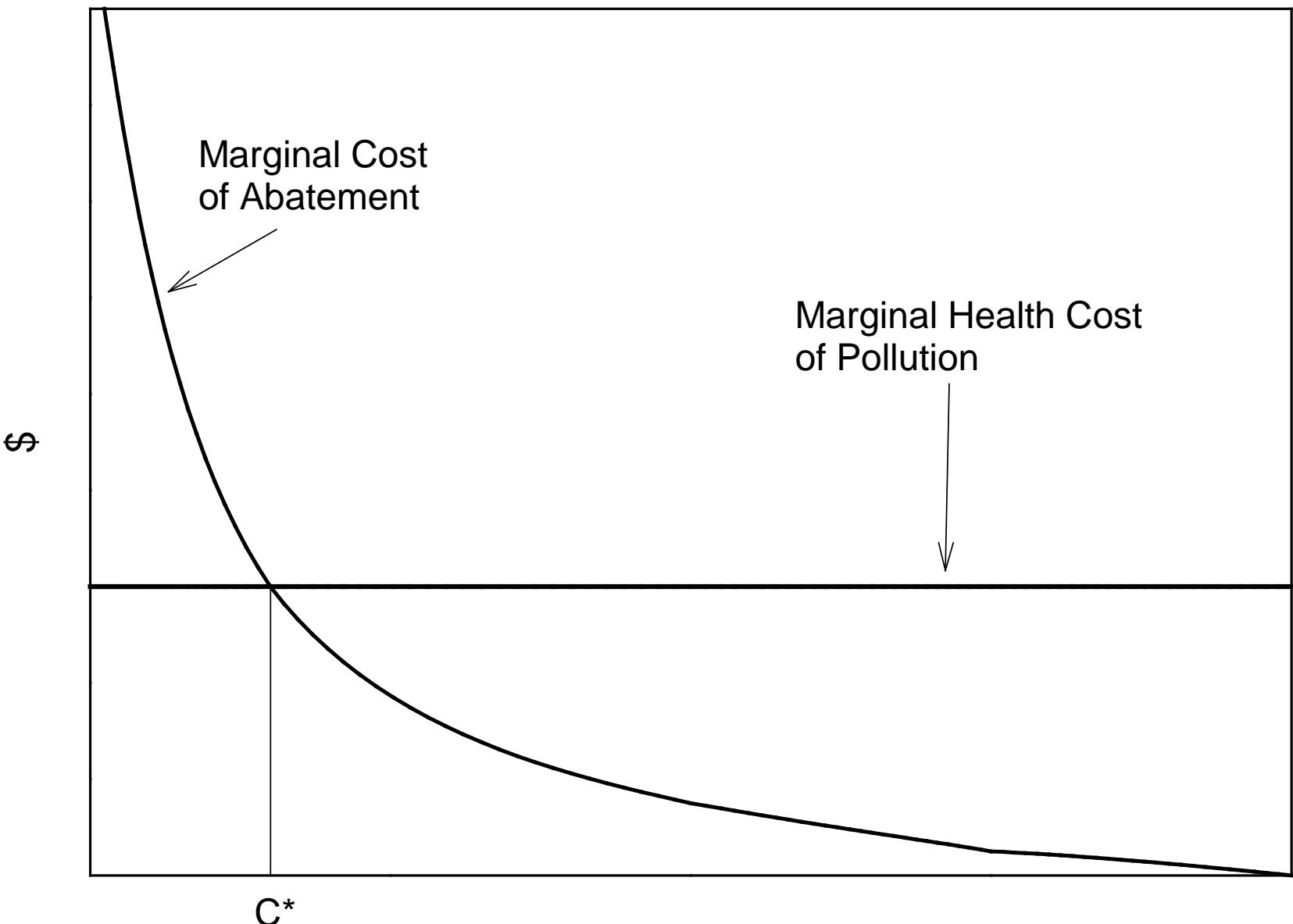


Figure 1. Stylized representation of the risk-response relationship between cardiopulmonary mortality and two primary dimensions of cumulative exposure to $\text{PM}_{2.5}$ (intensity and duration).





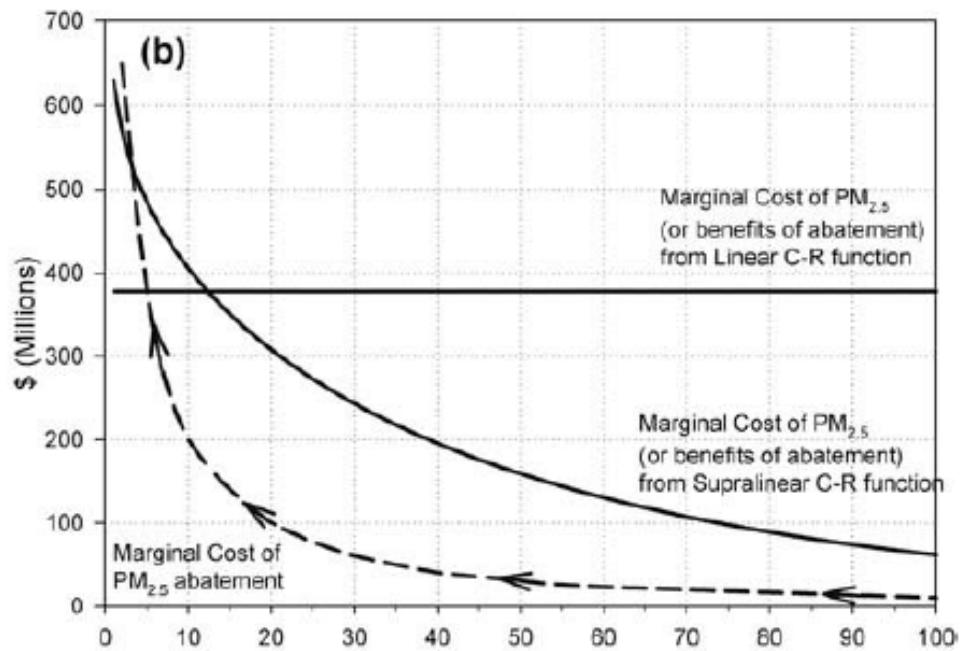
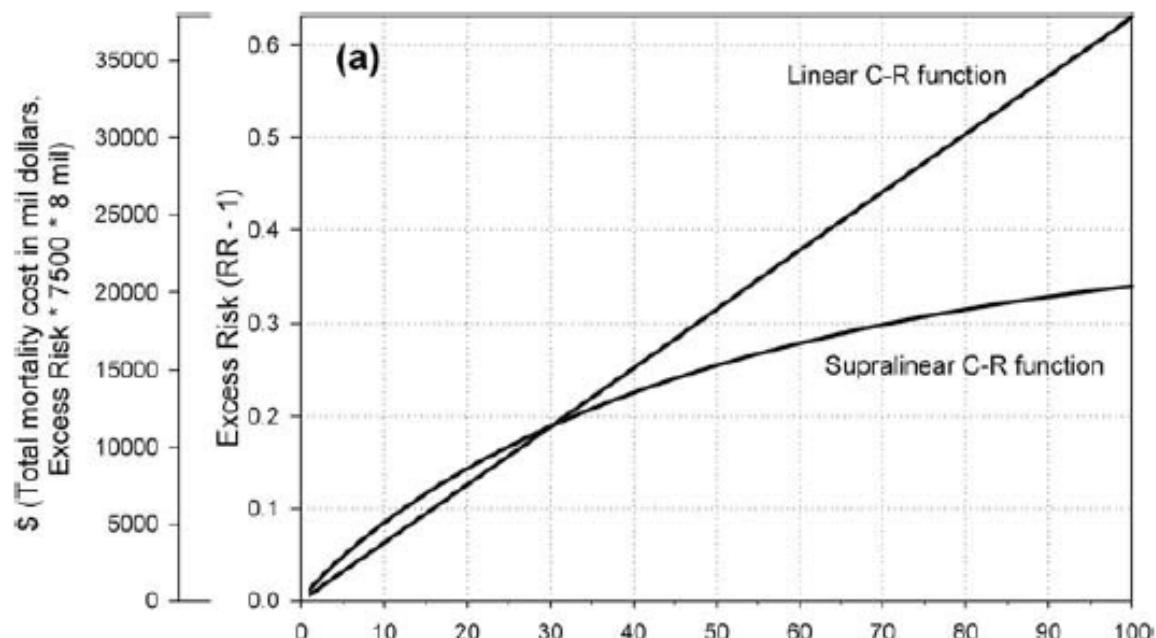




Ambient Concentrations ($\mu\text{g}/\text{m}^3$)



2015



2017 and beyond— or how low can we go?

Great HEI Initiative and Teams

Michael Brauer

University of British Columbia



Bert Brunekreef

Utrecht University, Netherlands



Francesca Dominici

Harvard University

