Health Effects of Air Pollution

C. Arden Pope III

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National Association of Clean Air Agencies

Spring Membership Meetings

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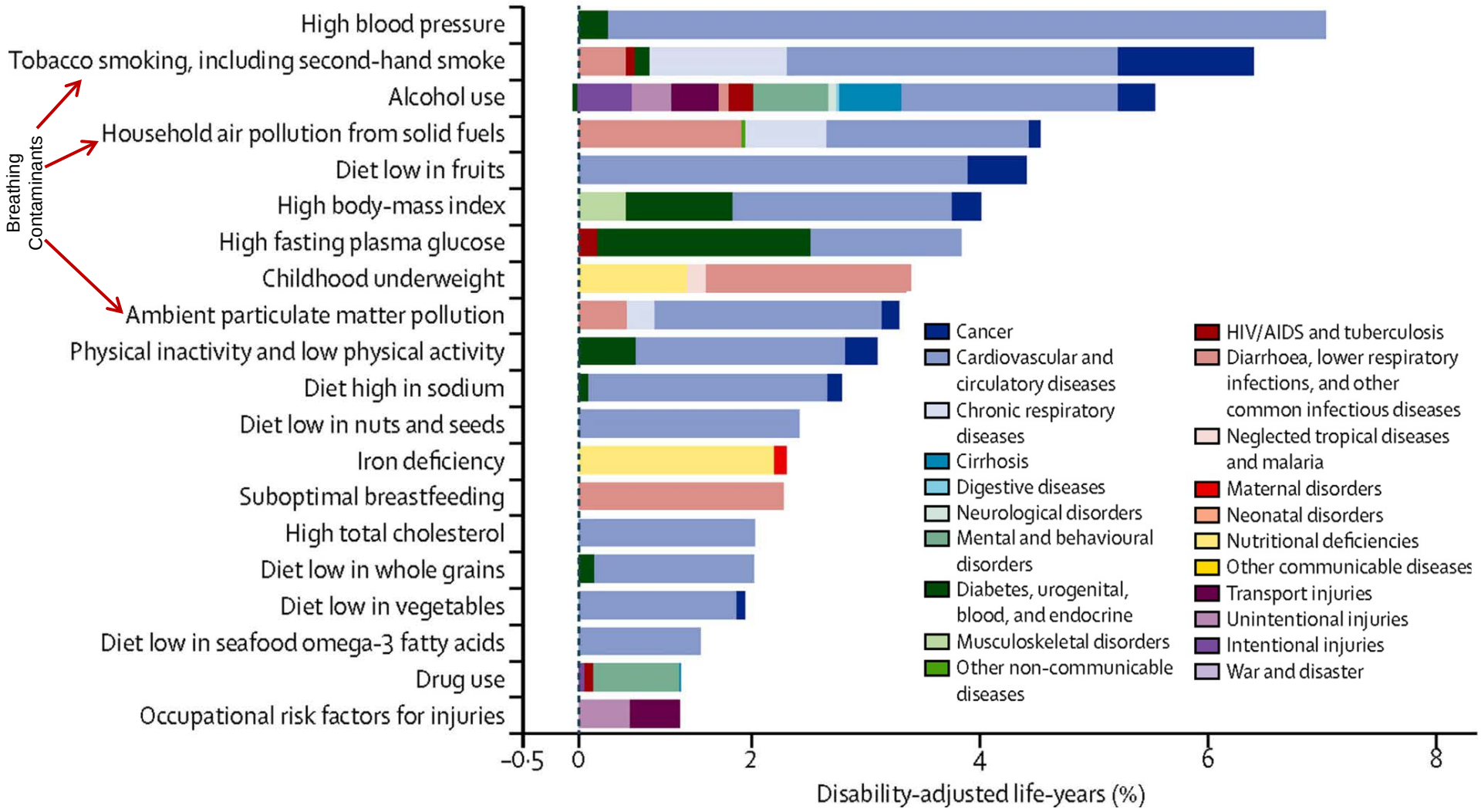
The Global Burden of Disease 2010

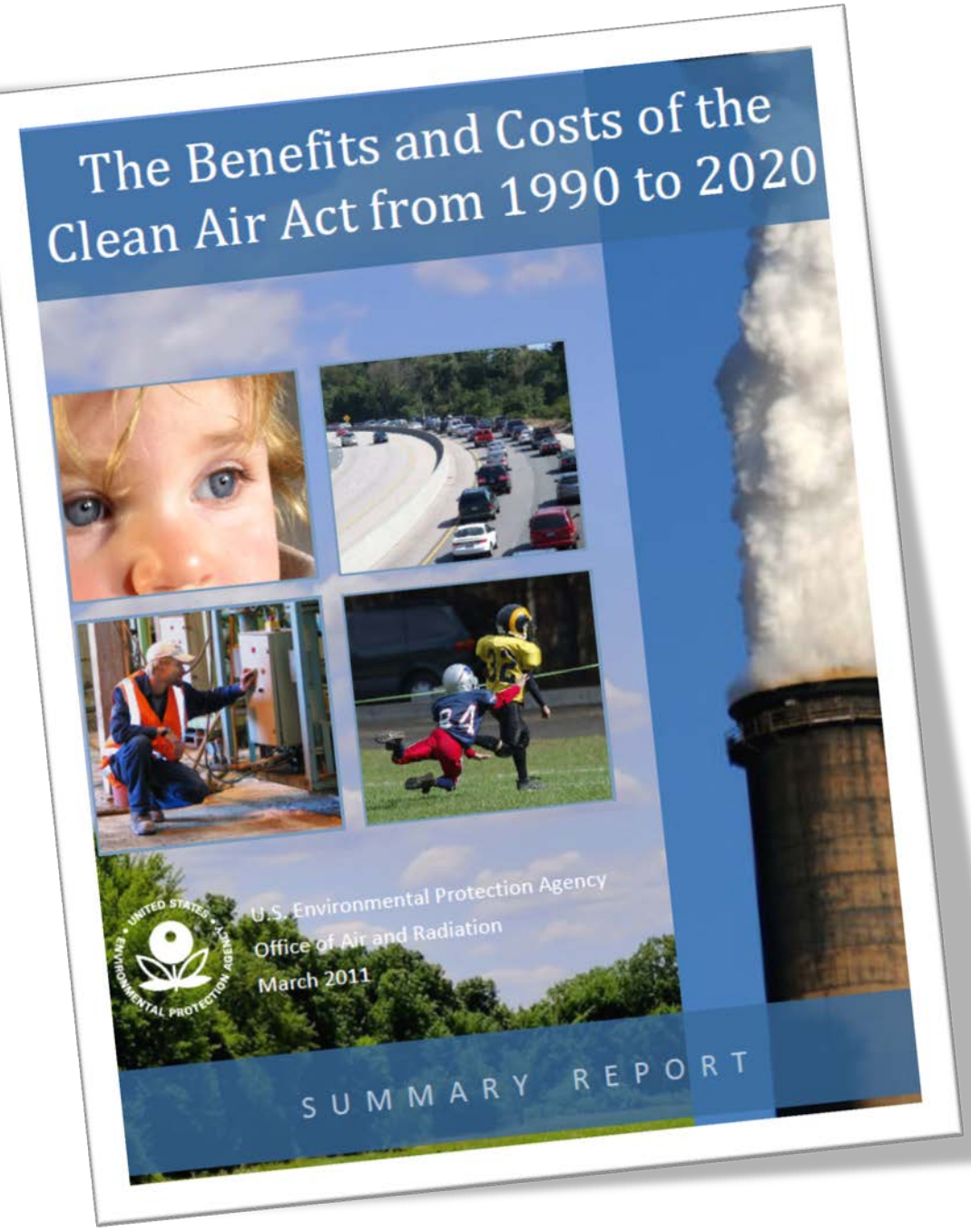


THE LANCET
Volume 376 · Number 9734 · Pages 1-68 · July 3-9, 2010 www.thelancet.com

The Global Burden of Disease 2010

Breathing contaminates contributes to global burden of disease (GBD)

	Number of attributable deaths	Disability adjusted life-years (DALYs)
Tobacco Smoking	5.7 mil.	5.7%
Second Hand Smoke	0.6 mil.	0.6%
Household air pollution from solid fuels	3.5 mil.	4.5%
Ambient PM air pollution	3.2 mil.	3.1%
Ambient Ozone	0.2 mil.	0.1%



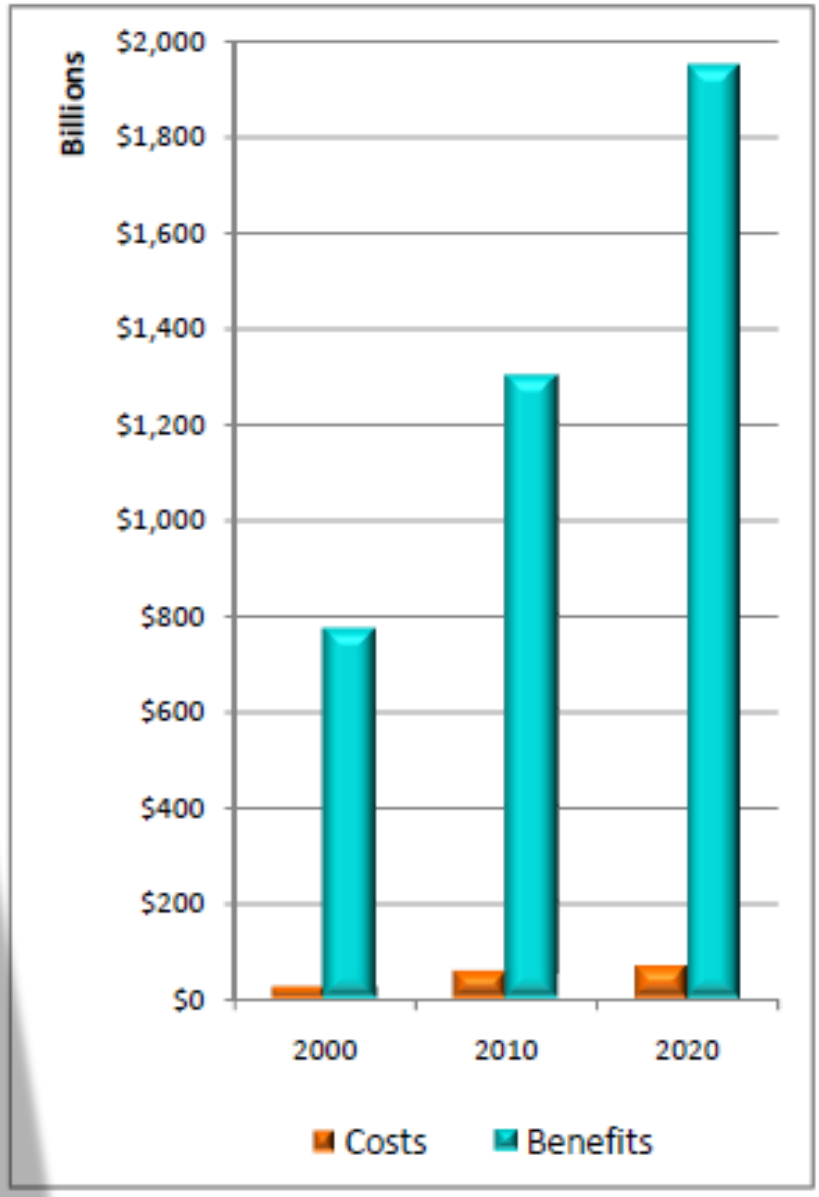
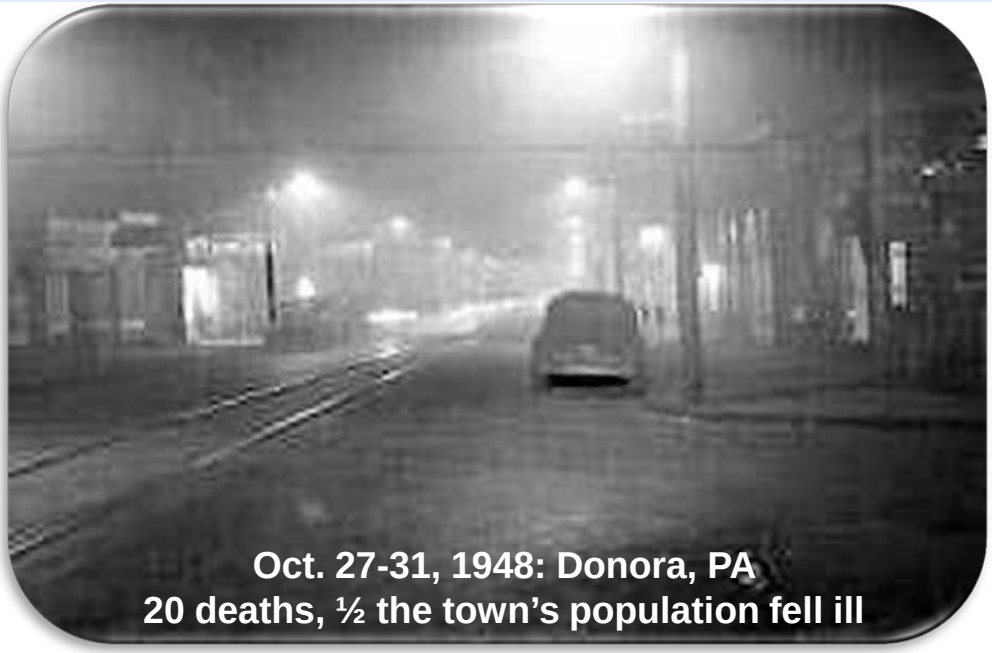


Exhibit 1. Primary Central Estimates of direct benefits and direct costs for the 2000, 2010, and 2020 study target years. (In billions of 2006 dollars). The graph shows the extent to which benefits exceed costs throughout the study period.

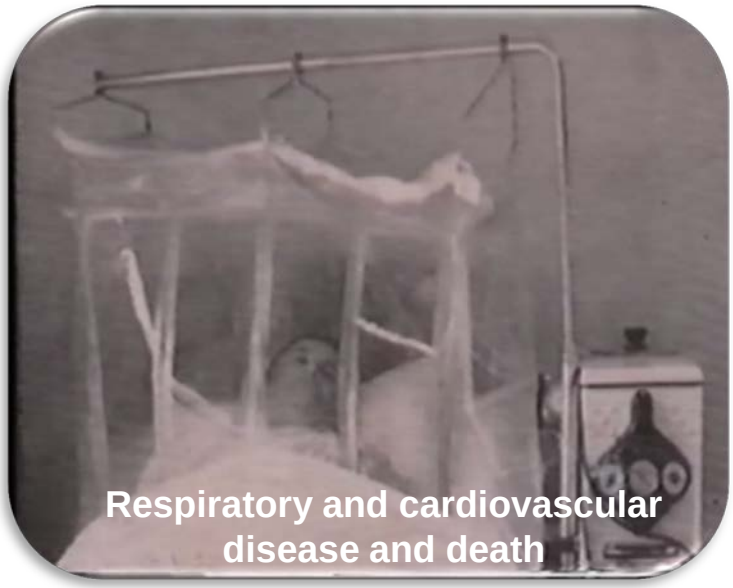
Early **“Killer smog”** episodes demonstrated that air pollution at extreme levels can contribute to respiratory and cardiovascular disease and death

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

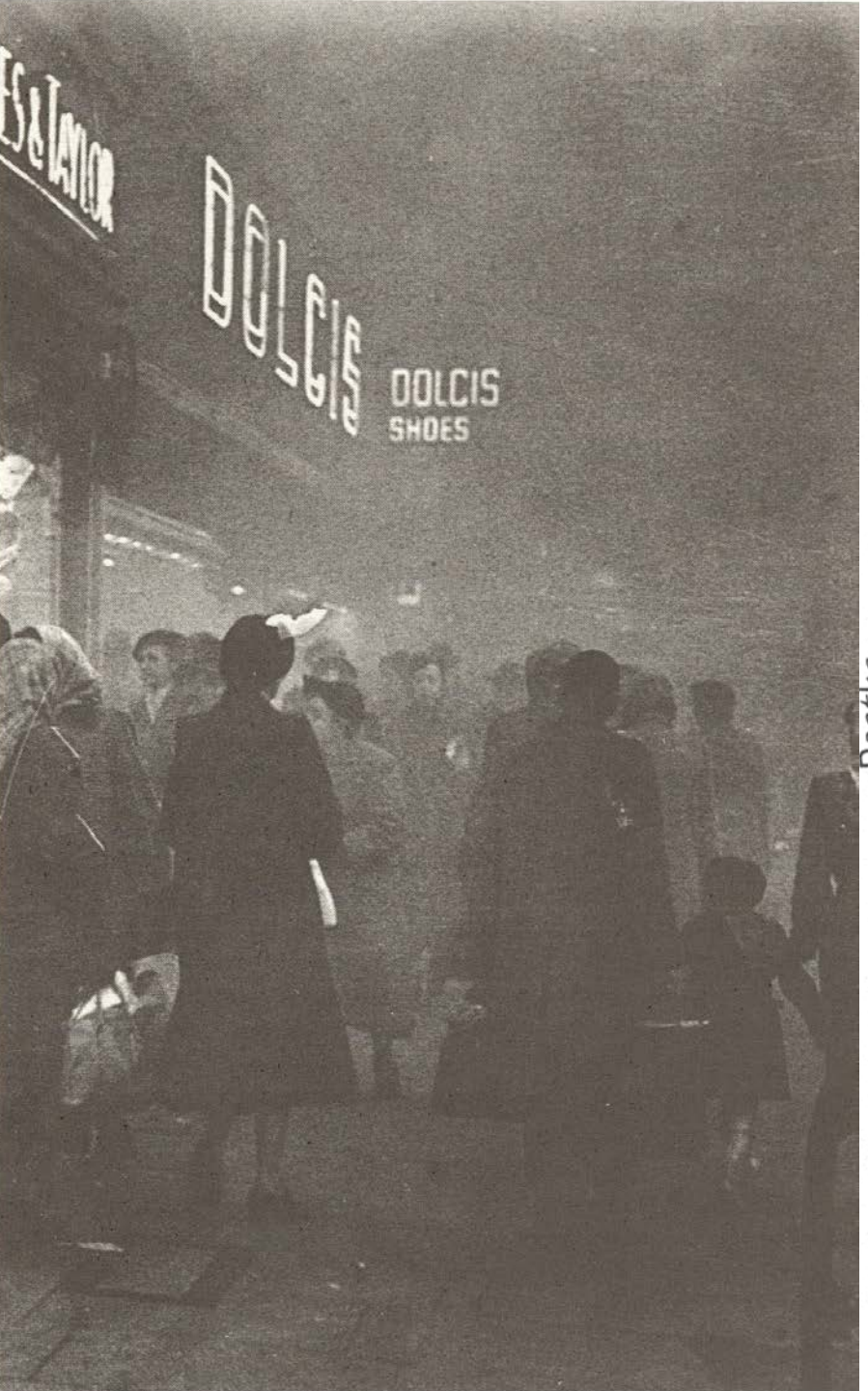


Oct. 27-31, 1948: Donora, PA
20 deaths, ½ the town’s population fell ill

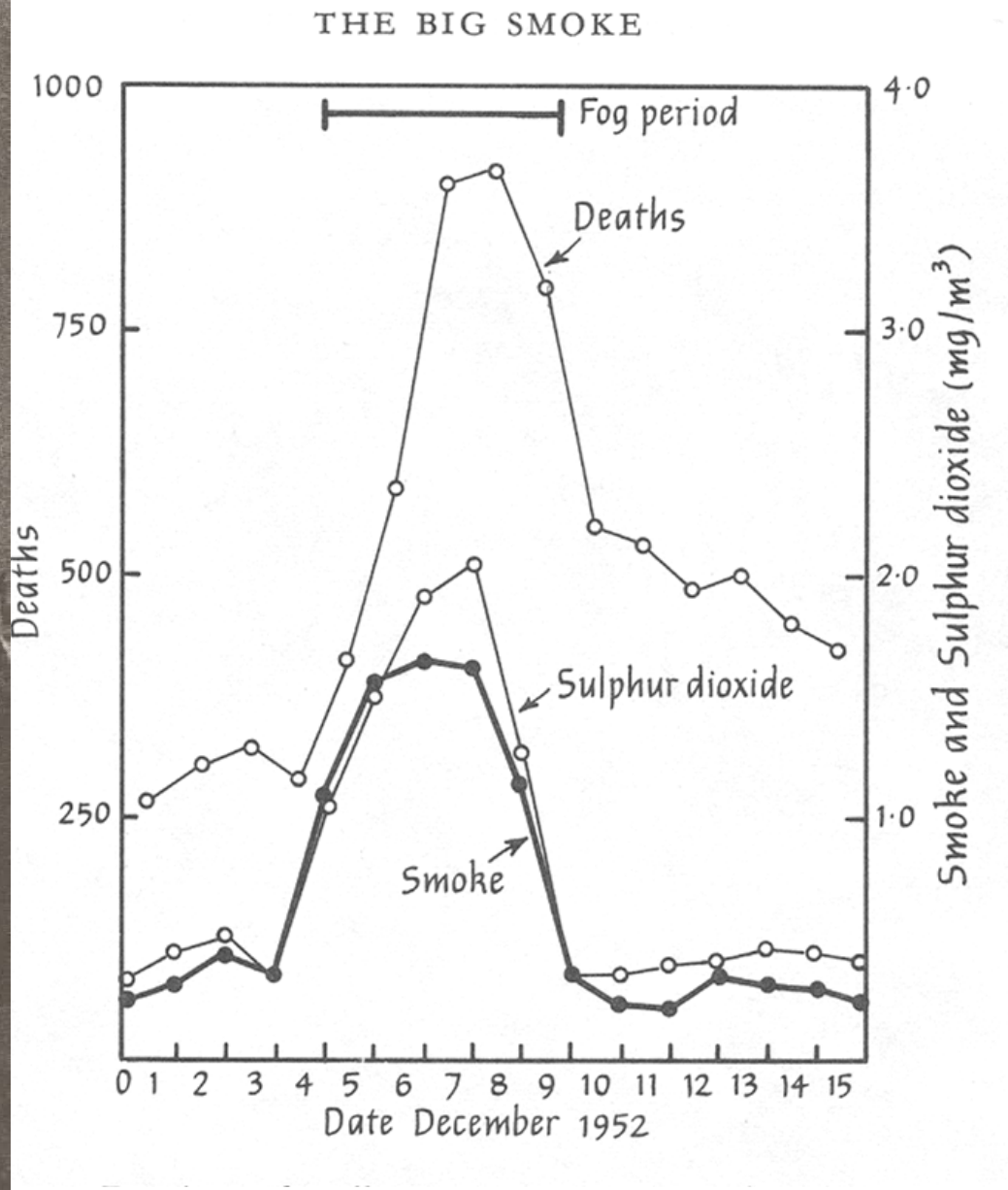
Dec. 5-9, 1952: London--1000’s of excess deaths



Respiratory and cardiovascular disease and death



London Fog Episode, Dec. 1952

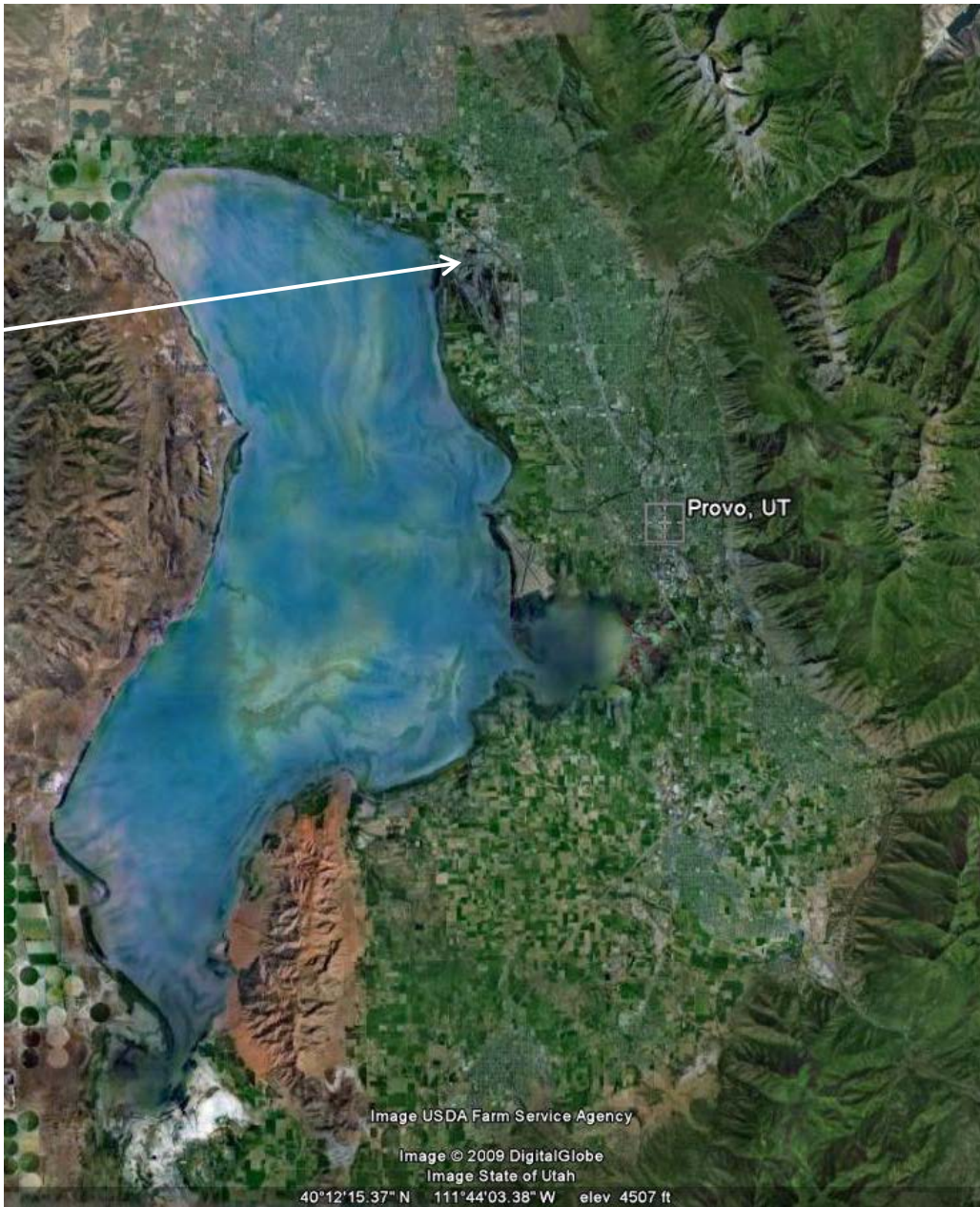


From: Brimblecombe P. The Big Smoke, Methu

Utah Valley, 1980s

- **Winter inversions trap local pollution**
- **Natural test chamber**

- **Local Steel mill contributed ~50% $PM_{2.5}$**
- **Shut down July 1986-August 1987**
- **Natural Experiment**

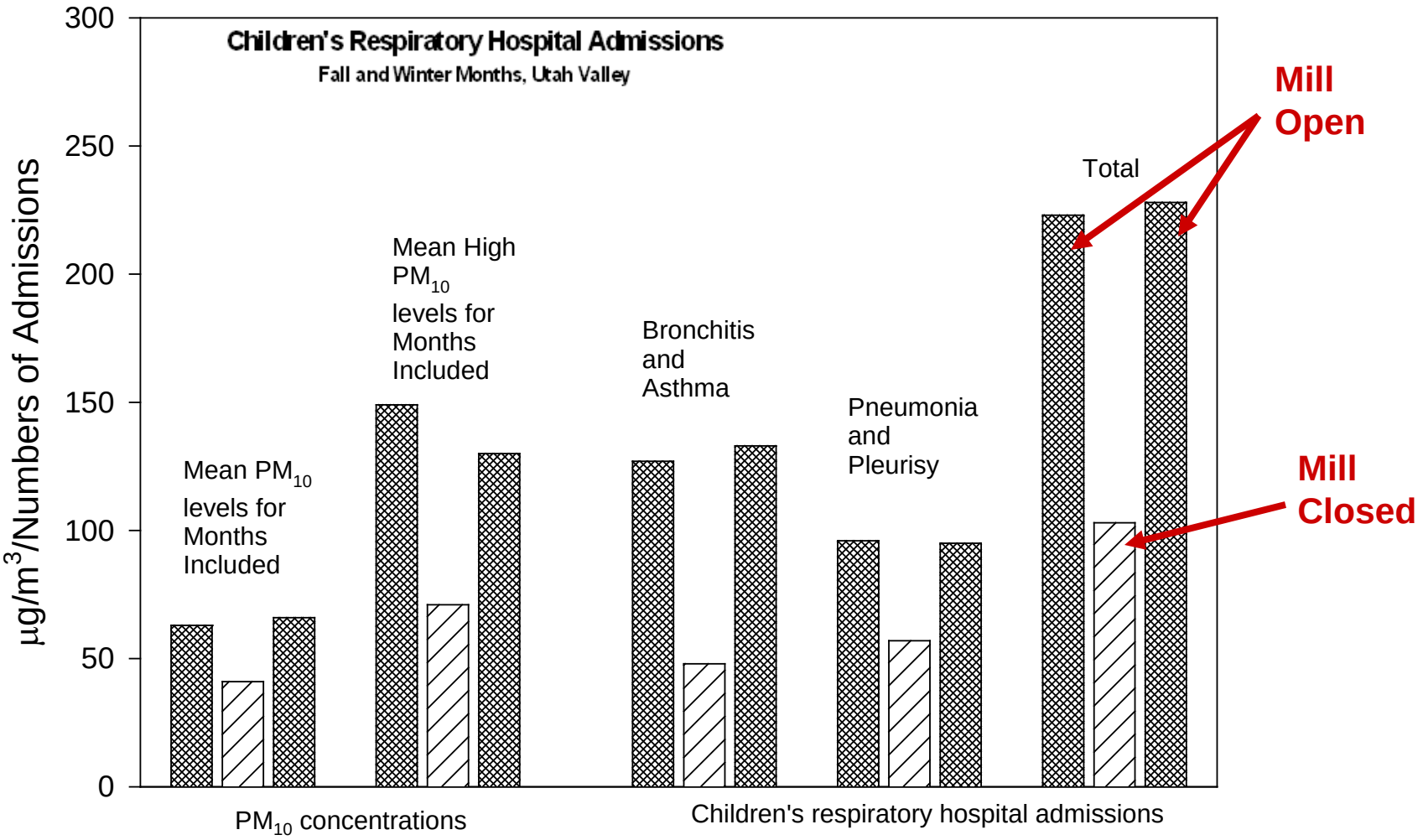


Large difference in air quality when inversions trap air pollution in valley

Utah Valley: Clean day

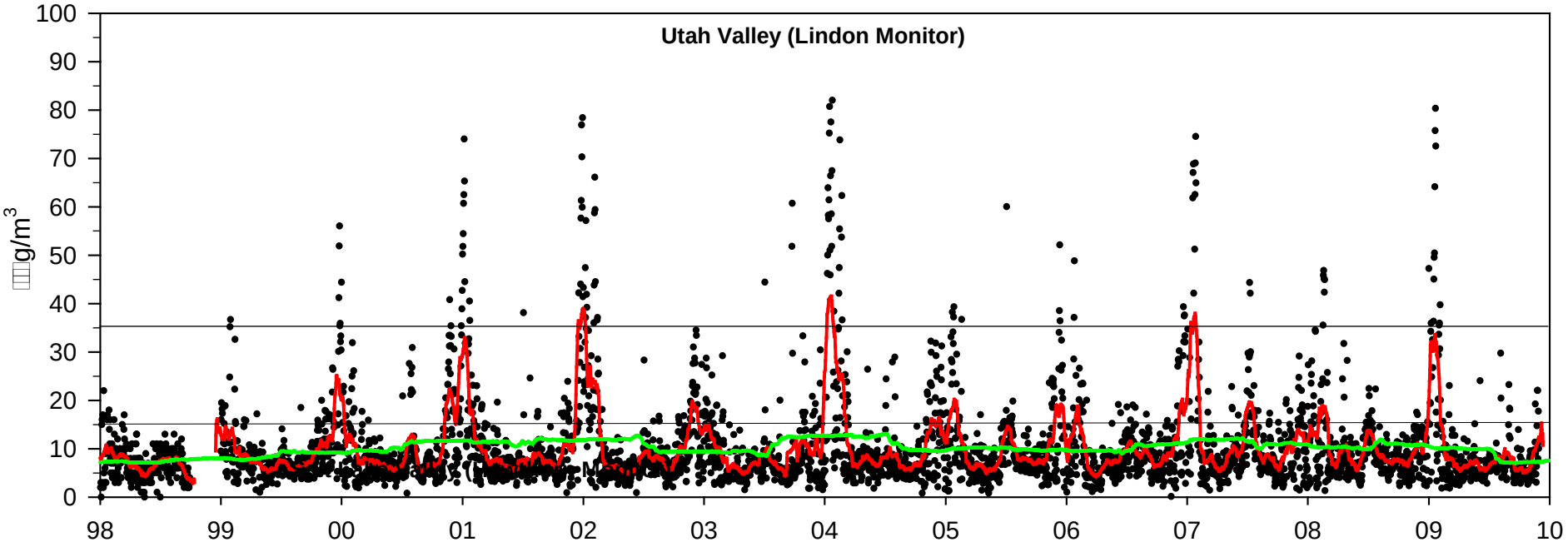
Utah Valley: Dirty day
(PM_{10} = 220 μg/m^3)

When the steel mill was open, total children's hospital admissions for respiratory conditions **approx. doubled.**



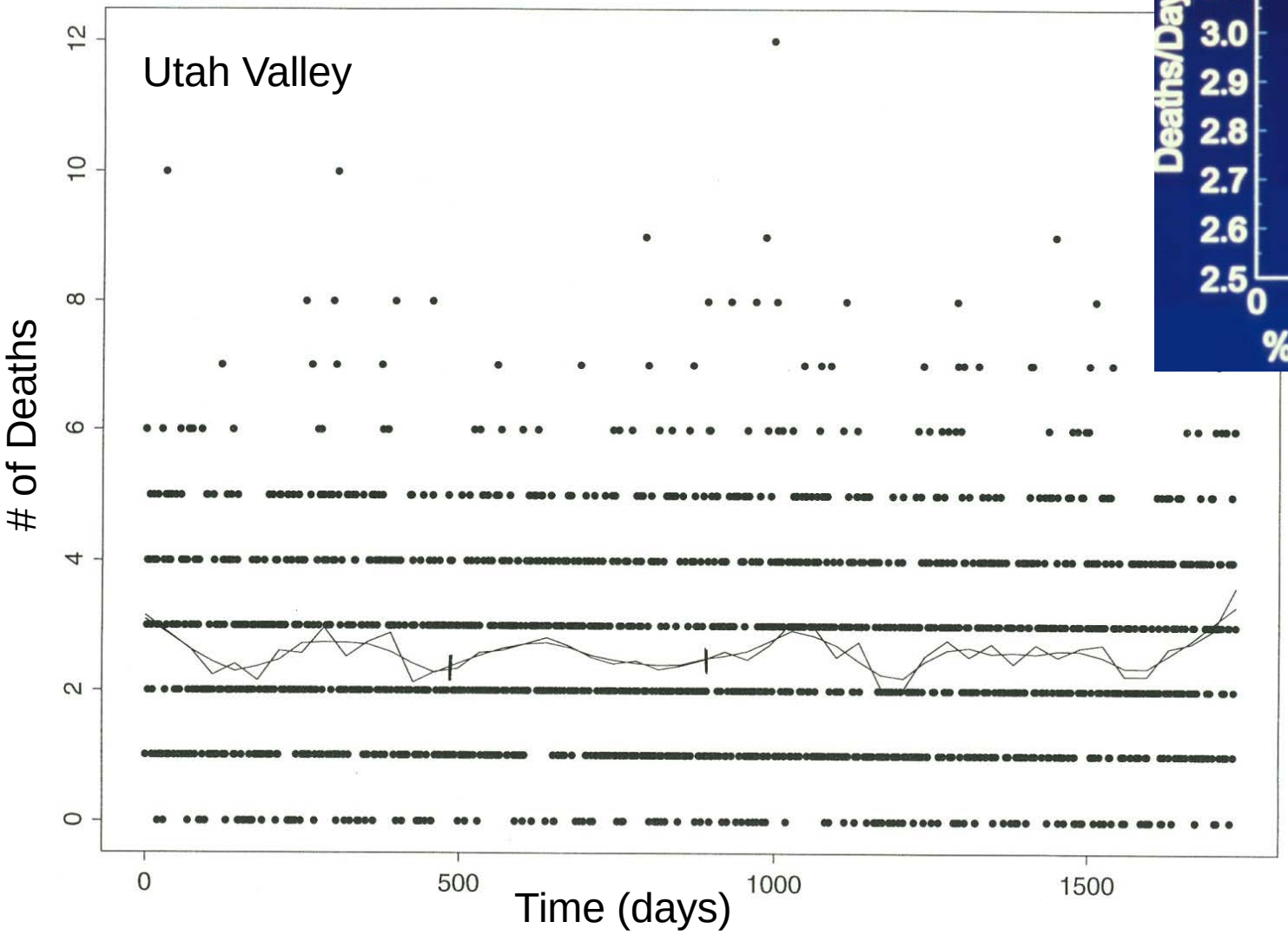
Sources: Pope. Am J Pub Health.1989; Pope. Arch Environ Health. 1991

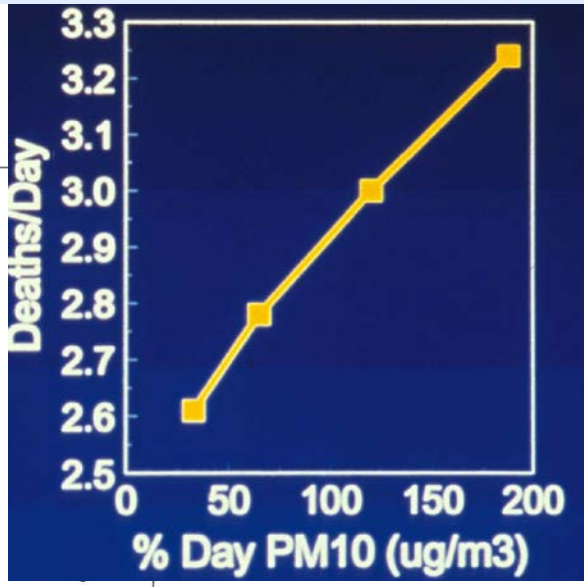
Health studies take advantage of **highly variable** air pollution levels that result from inversions.



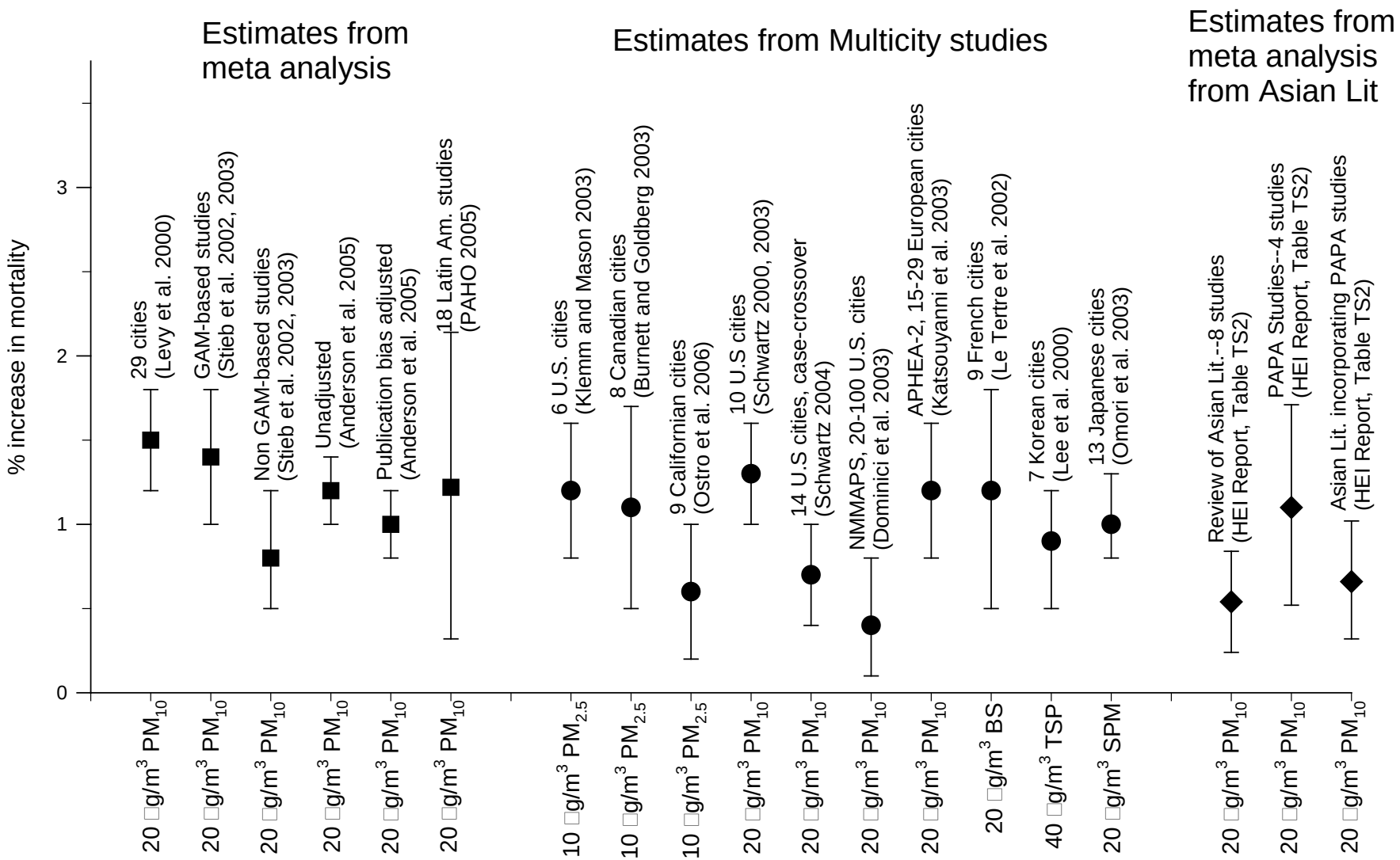
$PM_{2.5}$ concentrations January 1 1998-December 12 2009. Black dots, 24-hr $PM_{2.5}$; Red line, 30-day moving average $PM_{2.5}$; Green line, 1-yr moving average $PM_{2.5}$.

Daily changes in air pollution ➡ daily death counts





Daily time-series studies ***of over 200 cities***



10 μg/m³ $PM_{2.5}$ or 20 μg/m³ PM_{10} → 0.4% to 1.5% increase in relative risk of mortality—Small but remarkably consistent across meta-analyses and multi-city studies.

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION 2006;114:2443-48

Ischemic Heart Disease Events Triggered by Short-Term Exposure to Fine Particulate Air Pollution

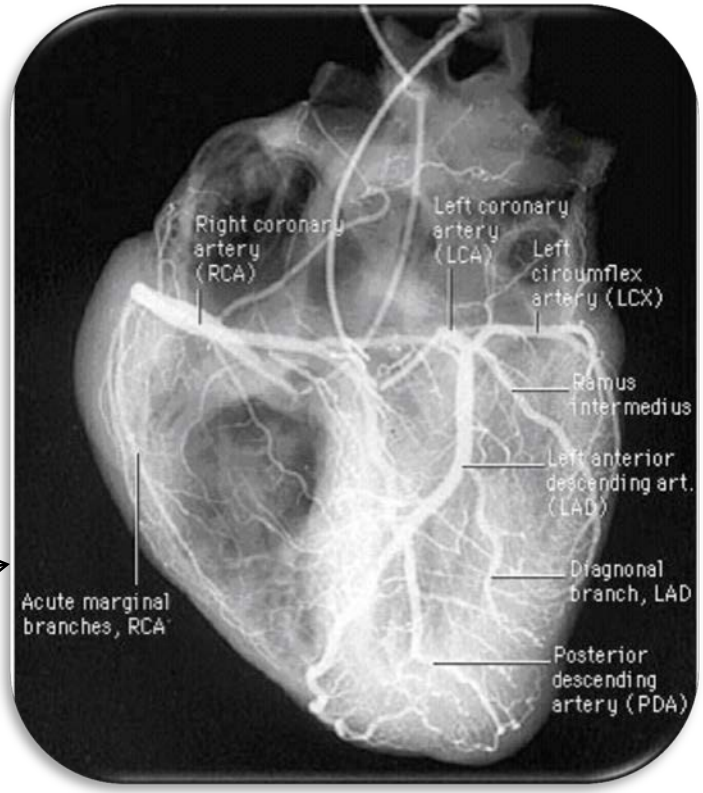
C. Arden Pope III, PhD; Joseph B. Muhlestein, MD; Heidi T. May, MSPH; Dale G. Renlund, MD; Jeffrey L. Anderson, MD; Benjamin D. Horne, PhD, MPH

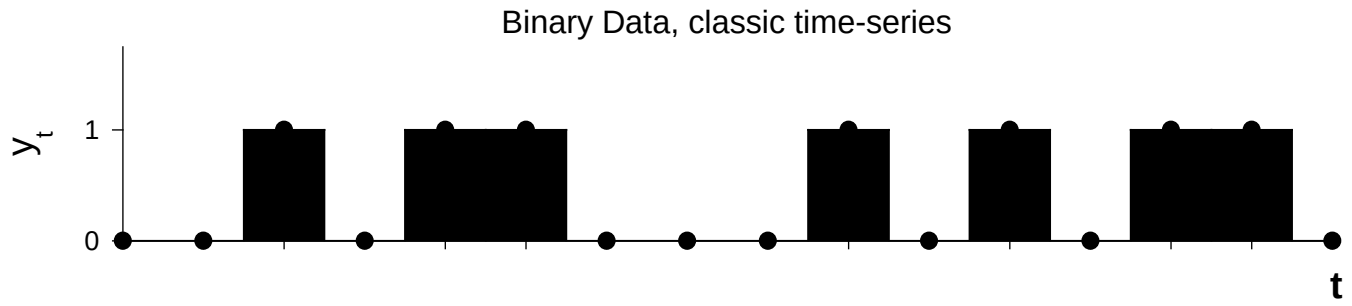
Jeffrey Anderson

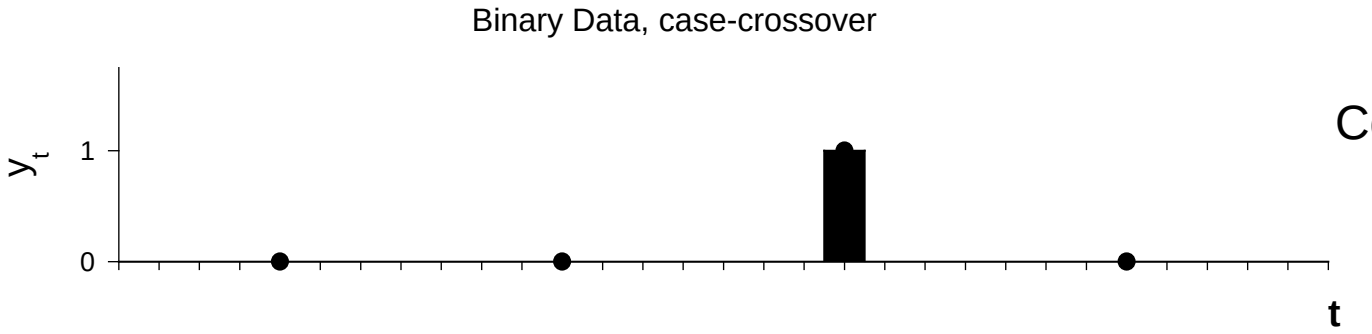
Methods:

Case-crossover study of acute ischemic coronary events (heart attacks and unstable angina) in 12,865 well-defined and followed up cardiac patients who lived on Utah's Wasatch Front

…and who underwent coronary angiography







Conditional Logistic Reg.

Each subject serves as his/her own control.

Control for subject-specific effects, day of week, season, time-trends, etc.—by matching

Conditional logistic regression:

$$\ln\left(\frac{\text{Prob}(Y_t = 1)}{1 - \text{Prob}(Y_t = 1)}\right) =$$

$$\alpha_1 + \alpha_2 + \alpha_3 + \ldots + \alpha_{12,865} + \beta(w_0 P_t + w_1 P_{t-1} + w_2 P_{t-2} + \ldots)$$

Control by matching for:

All cross-subject differences
(in this case, 12,865 subject-level fixed effects),
Season and/or month of year,
Time trends,
Day of week

Modeling controversies: How to select control or referent periods. Time stratified referent selection approach (avoids bias that can occur due to time trends in exposure) (**Holly Janes, Lianne Sheppard, Thomas Lumley** Statistics in Medicine and Epidemiology 2005)

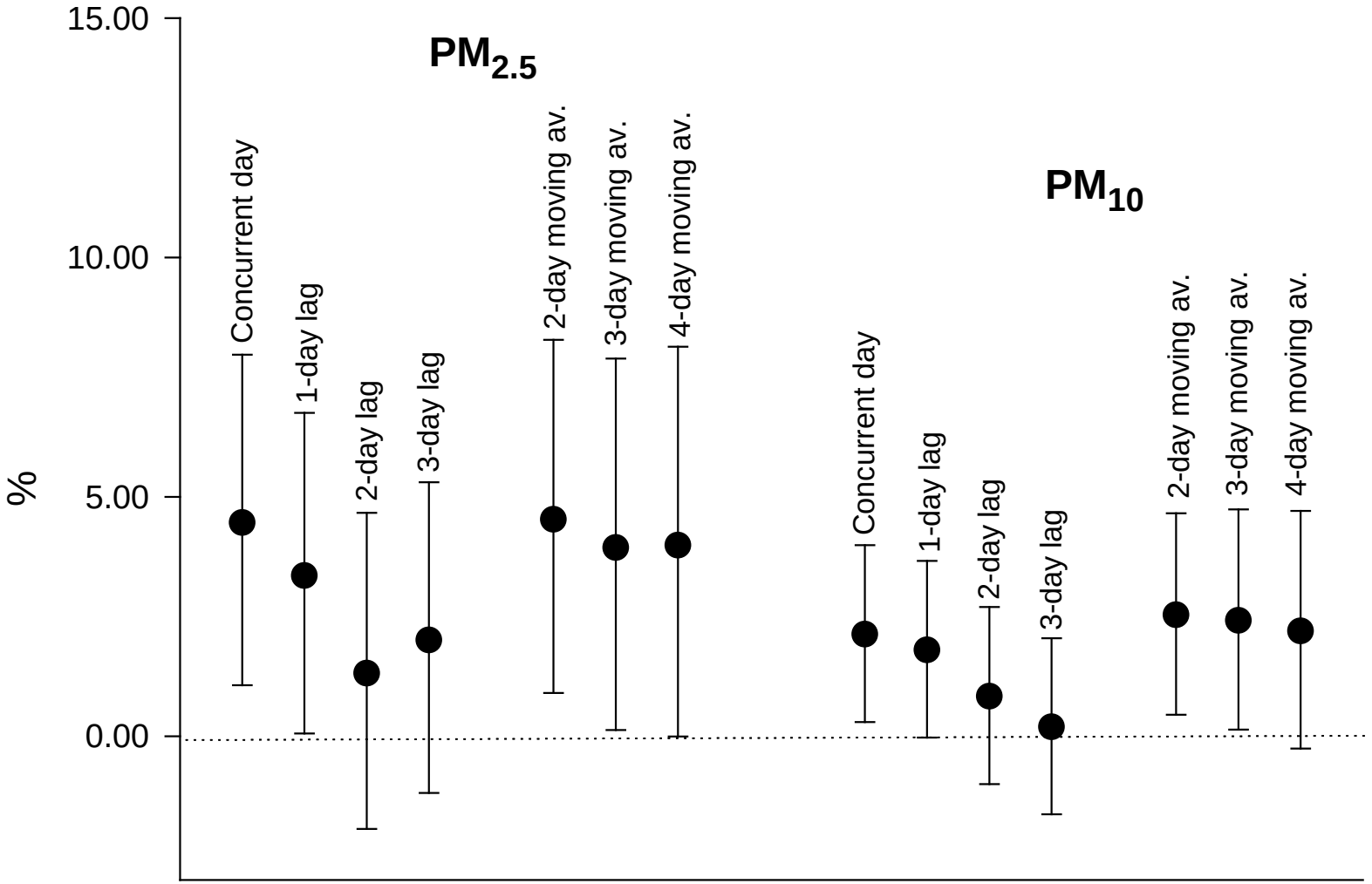


Figure 1. Percent increase in risk (and 95% CI) of acute coronary events associated with 10 □g/m^3 of $PM_{2.5}$, or PM_{10} for different lag structures.

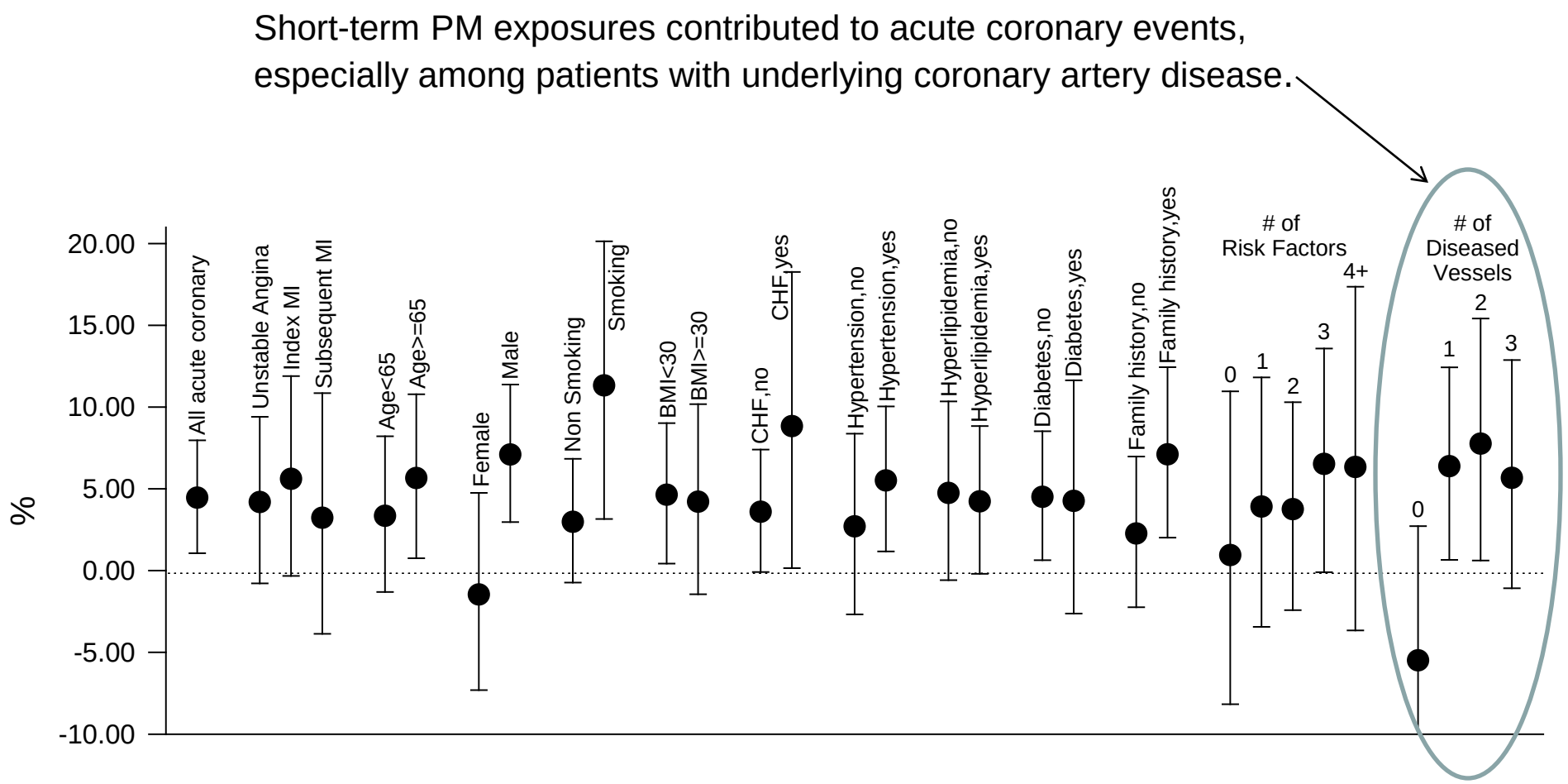


Figure 2. Percent increase in risk (and 95% CI) of acute coronary events associated with 10 μg/m^3 of $PM_{2.5}$, stratified by various characteristics.

Relation of Heart Failure Hospitalization to Exposure to Fine Particulate Air Pollution

C. Arden Pope III, PhD[a,*], Dale G. Renlund, MD[b,c], Abdallah G. Kfoury, MD[b,c], Heidi T. May, MSPH[b], and Benjamin D. Horne, PhD, MPH[b,c]

Am J Cardiol 2008;102:1230–1234

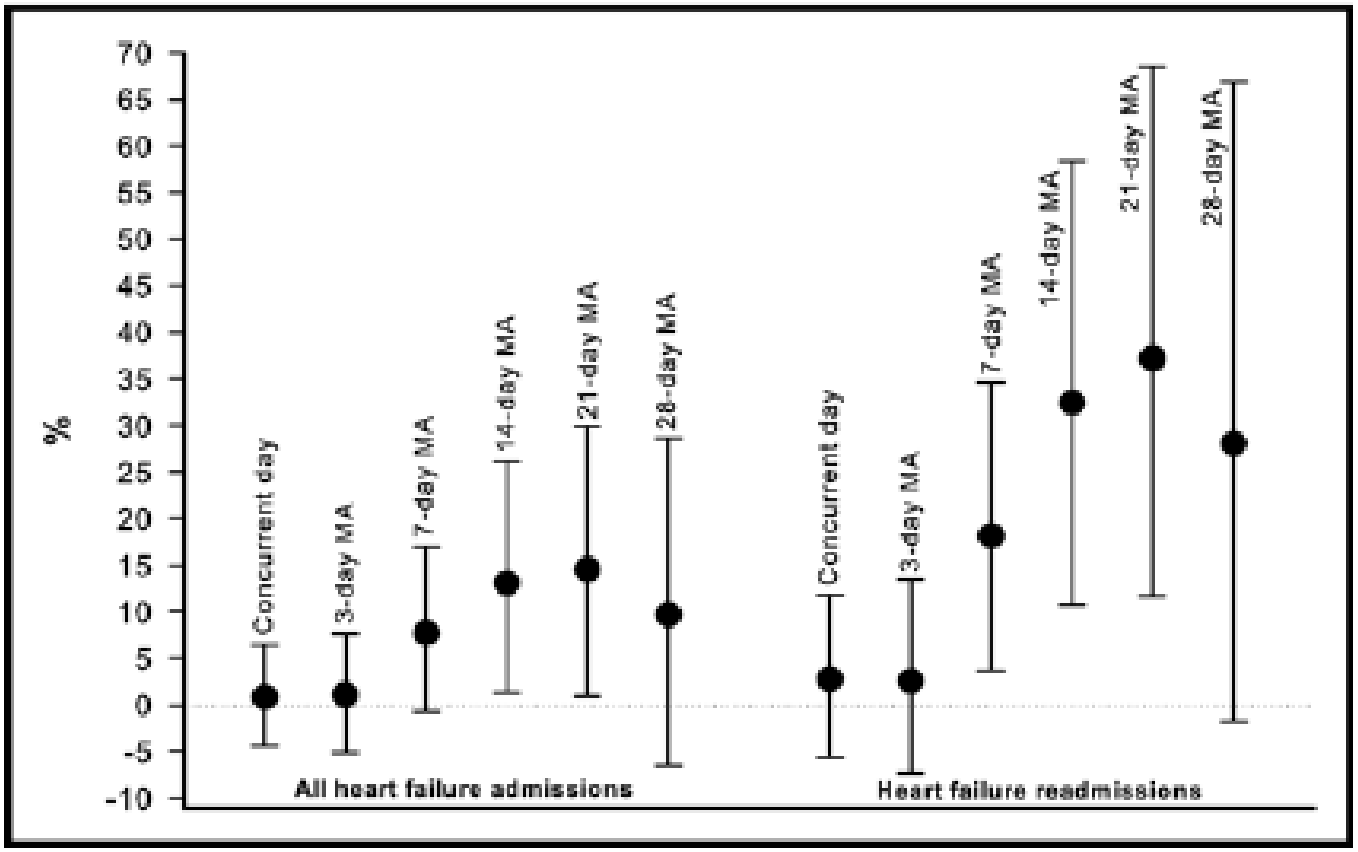


Figure 1. Percent increase in risk and 95% CIs of HF admissions and readmissions, associated with a 10 $\mu g/m^3$ of $PM_{2.5}$ for selected lagged moving average (MA) exposures 0 to 28 days.

Dale Renlund

Abdallah Kfoury

Benjamin Horne

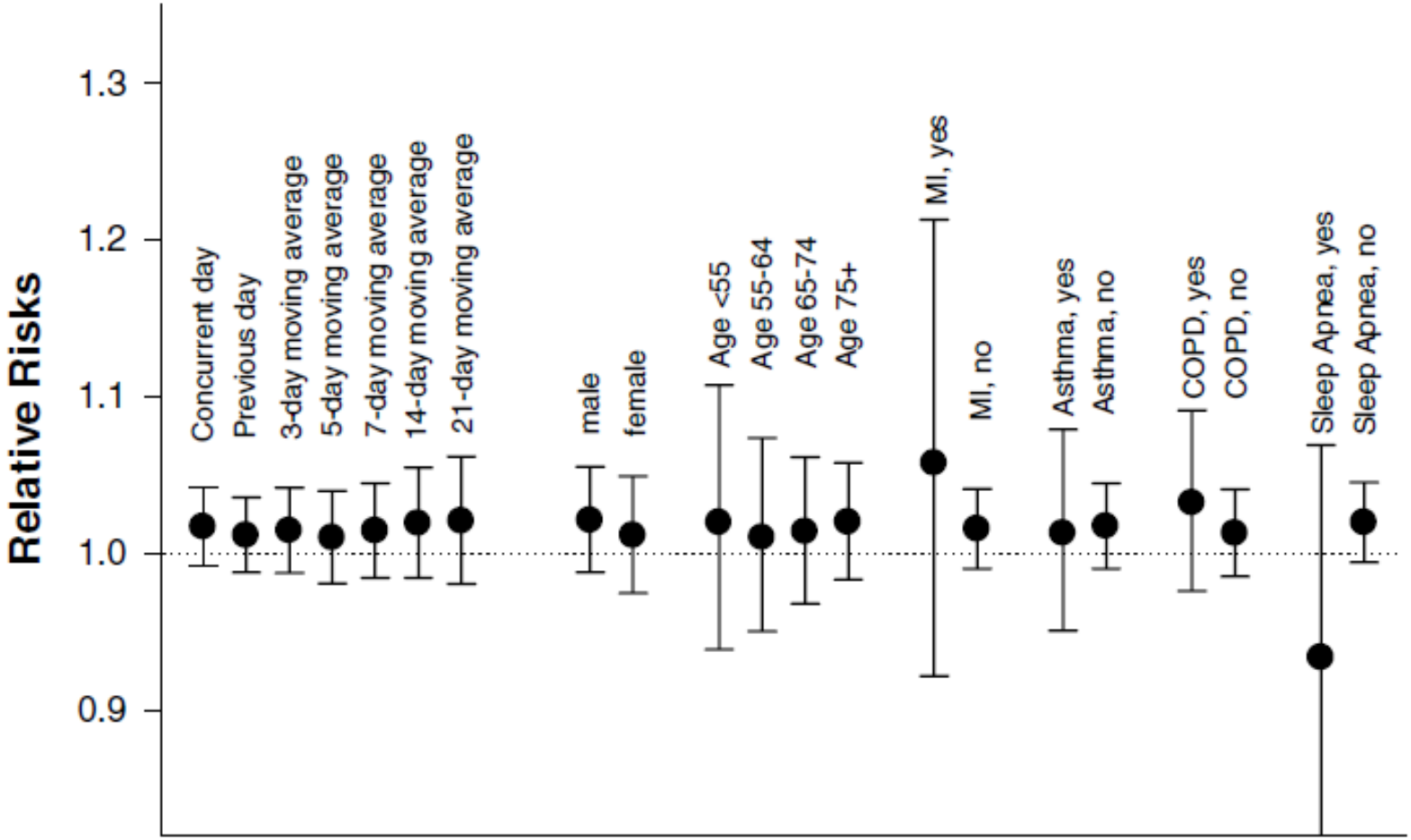
Atrial Fibrillation Hospitalization Is Not Increased with Short-Term Elevations in Exposure to Fine Particulate Air Pollution

T. JARED BUNCH, M.D.,* BENJAMIN D. HORNE, PH.D., M.P.H.,† SAMUEL J. ASIRVATHAM, M.D.,‡ JOHN D. DAY, M.D.,* BRIAN G. CRANDALL, M.D.,* J. PETER WEISS, M.D.,* JEFFREY S. OSBORN, M.D.,* JEFFREY L. ANDERSON, M.D.,† JOSEPH B. MUHLESTEIN, M.D.,† DONALD L. LAPPE, M.D.,† and C. ARDEN POPE III, PH.D.§

Jared Bunch

PACE
Pacing And Clinical Electrophysiology

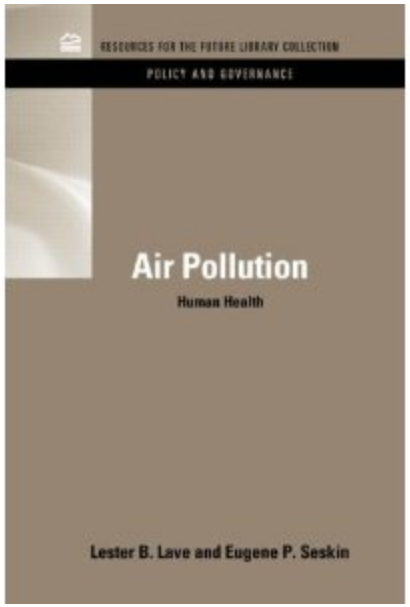
2011

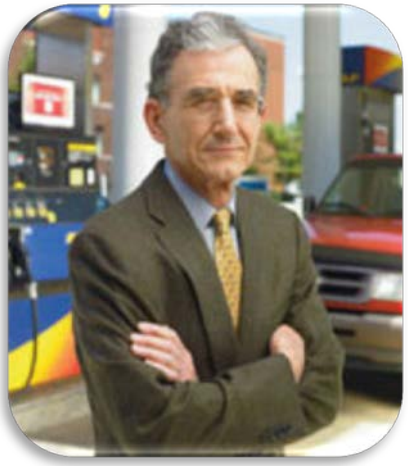


Short-term changes in air pollution exposure are associated with:

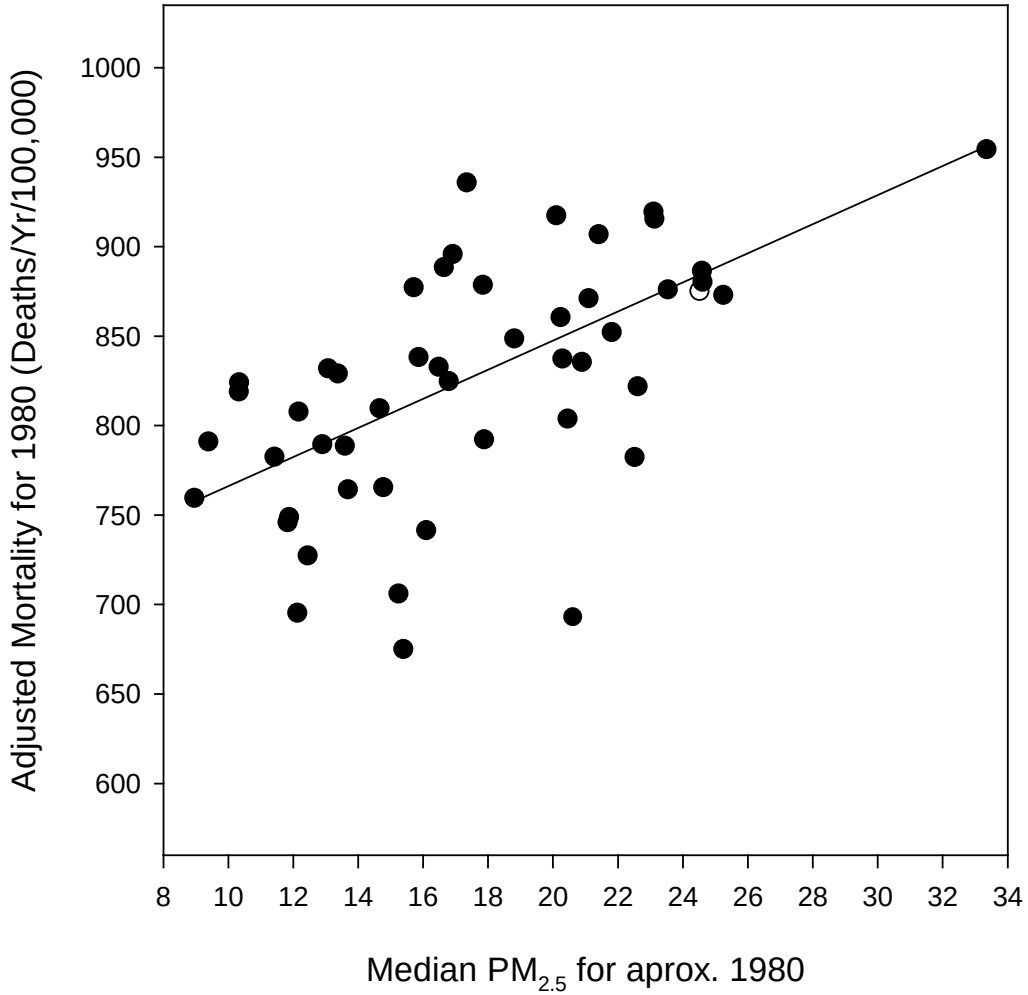
- Daily death counts (respiratory and cardiovascular)
- Hospitalizations
- Lung function
- Symptoms of respiratory illness
- School absences
- Ischemic heart disease
- Etc.

Longer-term air pollution exposure has been linked to even substantially larger health effects.





Age-, sex-, and race- adjusted population-based mortality rates in U.S. cities for 1980 plotted over various indices of particulate air pollution (From Pope 2000).



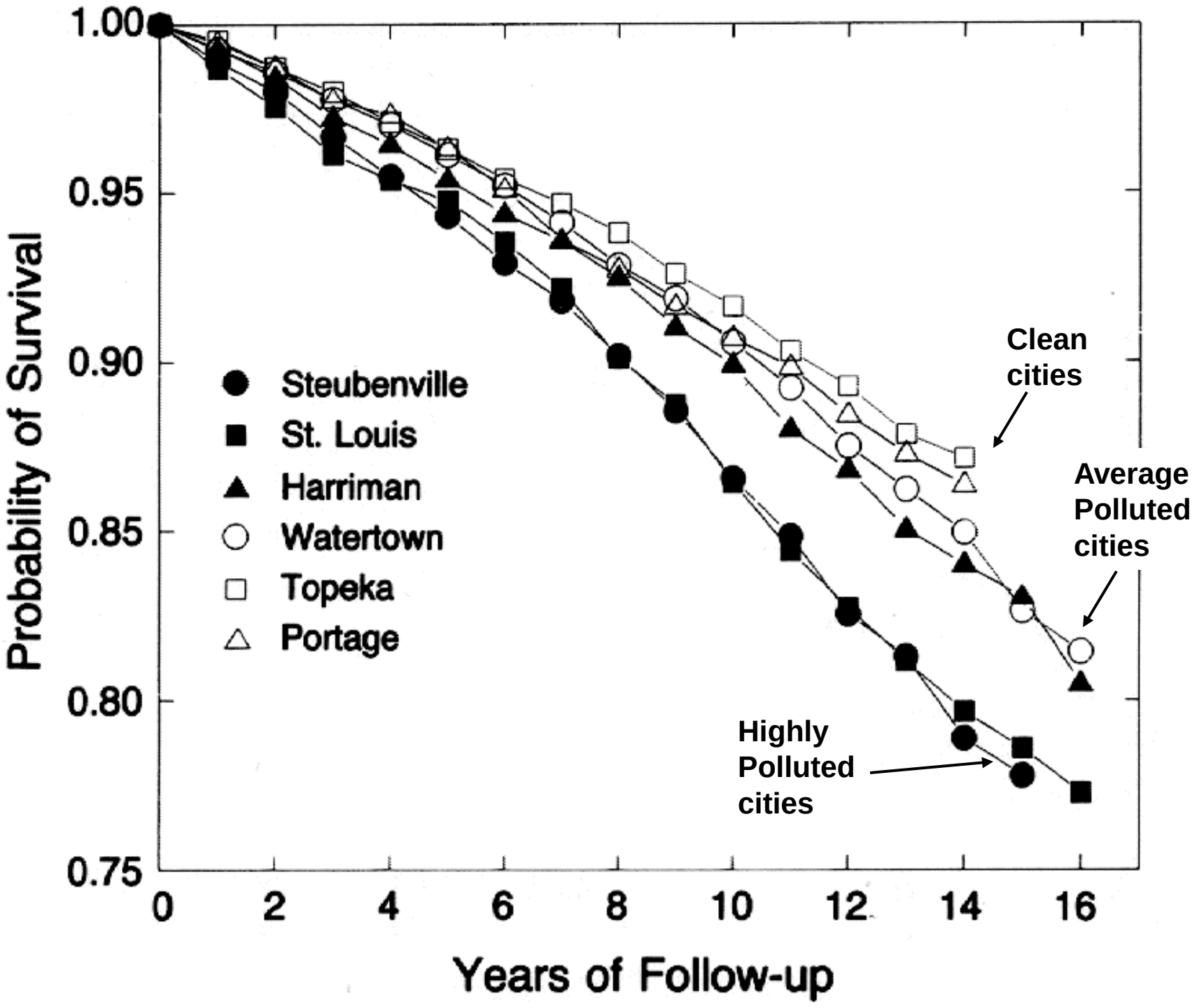
An Association Between Air Pollution and Mortality in Six U.S. Cities

The NEW ENGLAND JOURNAL of MEDICINE 1993

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

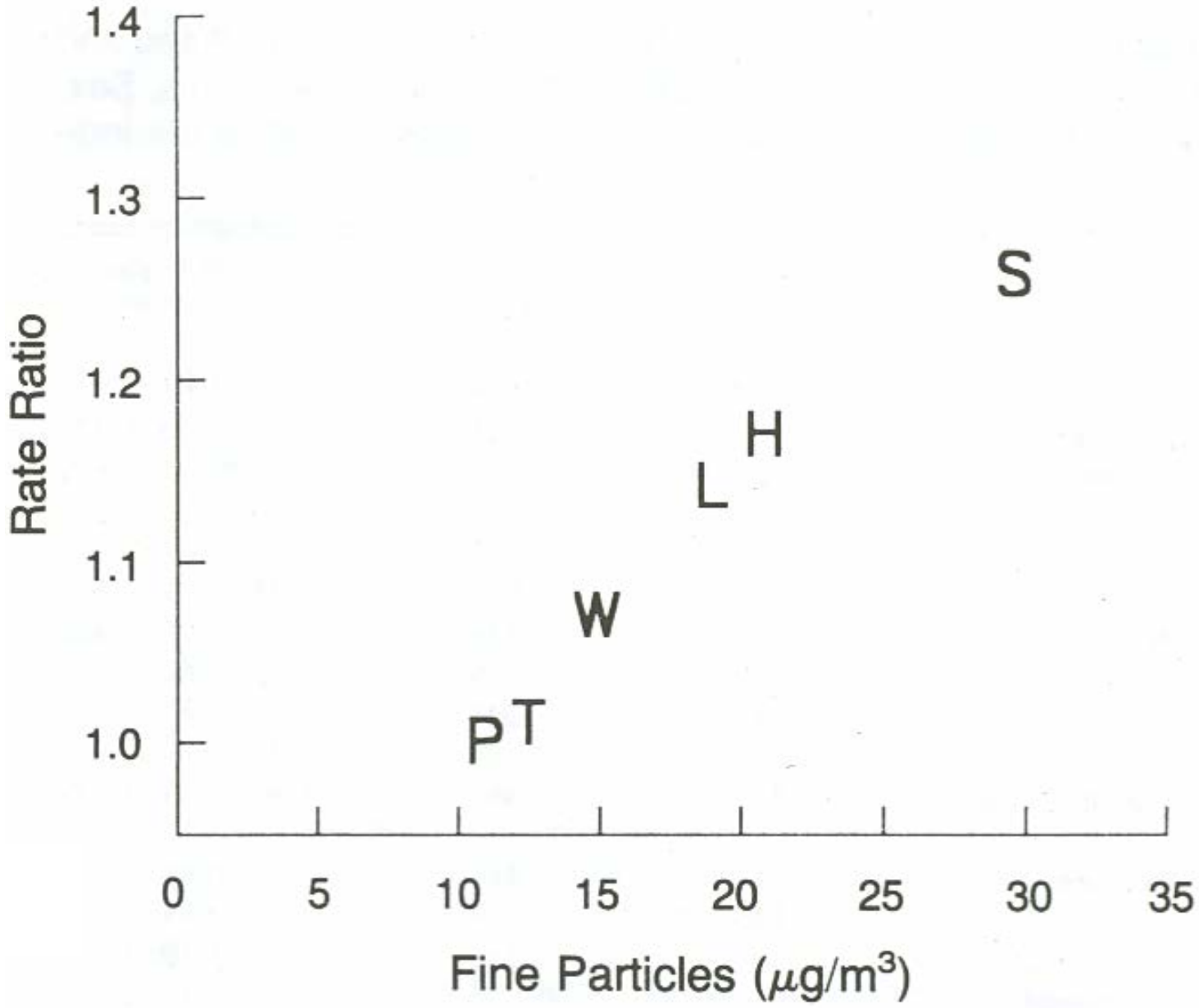
Methods:

- 14-16 yr prospective follow-up of 8,111adults living in six U.S. cities.
- Monitoring of TSP PM_{10}, $PM_{2.5}$, SO_4, H^+, 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.


1.00
0.95
0.90
0.85
0.80
0.75
Probability of Survival
0
2
4
6
8
10
12
14
16
Years of Follow-up
Steubenville
St. Louis
Harriman
Watertown
Topeka
Portage
Clean cities
Average Polluted cities
Highly Polluted cities

Adjusted risk ratios (and 95% CIs) for cigarette smoking and $PM_{2.5}$

Cause of Death	Current Smoker, 25 Pack years	Most vs. Least Polluted City
All	2.00 (1.51-2.65)	**1.26** (1.08-1.47)
Lung Cancer	8.00 (2.97-21.6)	**1.37** (0.81-2.31)
Cardio-pulmonary	2.30 (1.56-3.41)	**1.37** (1.11-1.68)
All other	1.46 (0.89-2.39)	**1.01** (0.79-1.30)


1.4
1.3
1.2
1.1
1.0
Rate Ratio
S
H
L
W
P
T
0
5
10
15
20
25
30
35
Fine Particles (μg/m3)

JAMA®
The Journal of the American Medical Association
March 6, 2002—Vol 287, No. 9

Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution

C. Arden Pope III, PhD
Richard T. Burnett, PhD
Michael J. Thun, MD
Eugenia E. Calle, PhD
Daniel Krewski, PhD
Kazuhiko Ito, PhD
George D. Thurston, ScD

Context Associations have been found between day-to-day particulate ai and increased risk of various adverse health outcomes, including cardiopulmc tality. However, studies of health effects of long-term particulate air pollu been less conclusive.

Objective To assess the relationship between long-term exposure to fir late air pollution and all-cause, lung cancer, and cardiopulmonary mortalit

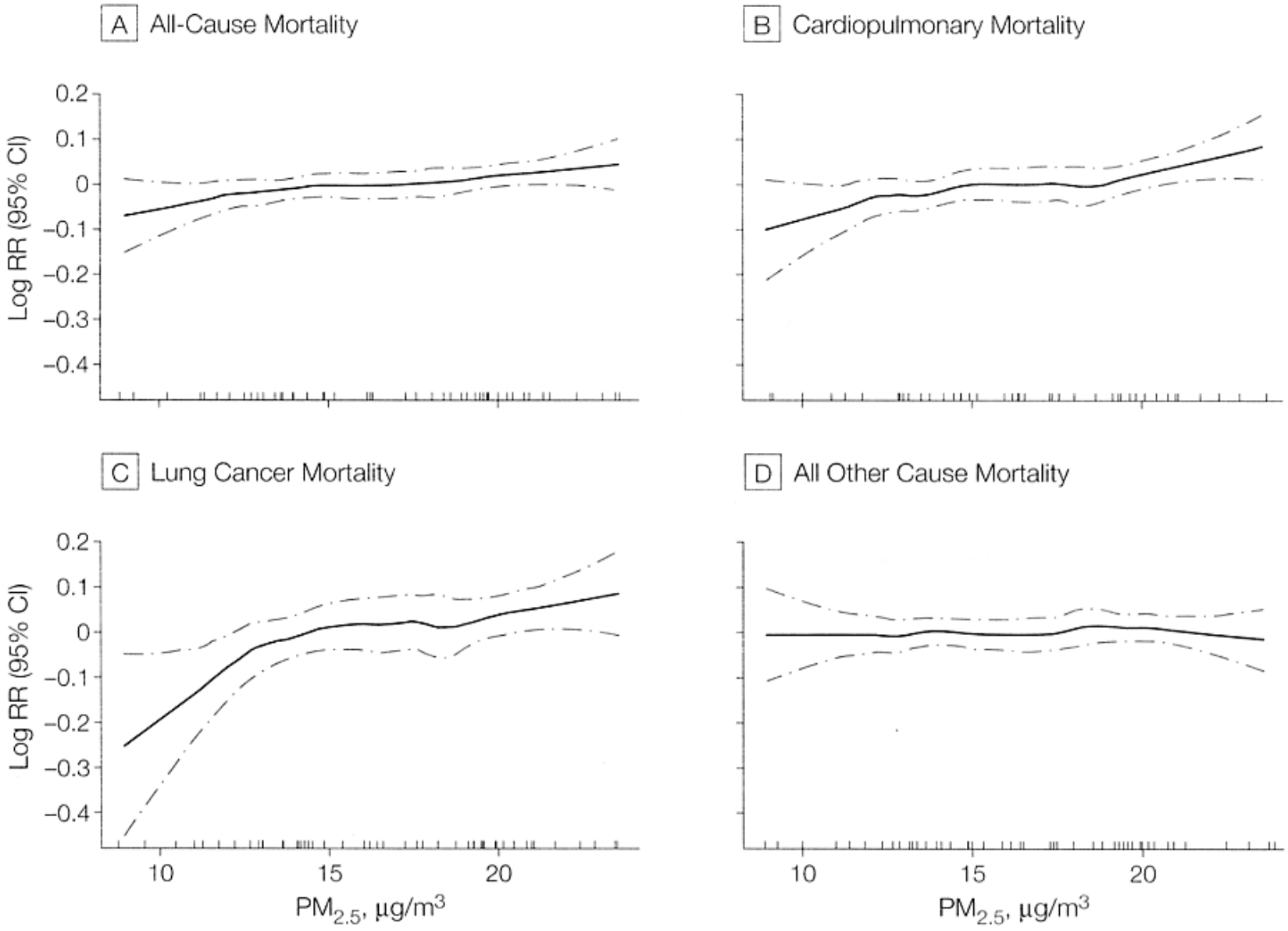
Design, Setting, and Participants Vital status and cause of death data lected by the American Cancer Society as part of the Cancer Prevention II stu going prospective mortality study, which enrolled approximately 1.2 million adu







Figure 2. Nonparametric Smoothed Exposure Response Relationship



Science
25 July 1997 AAAS

Showdown Over Clean Air Science

Jocelyn Kaiser

Industry and environmental researchers are squaring off over studies linking air pollution and illness in what some are calling the biggest environmental fight of the decade



Dan Krewski
Rick Burnett
Mark Goldberg
and 28 others

HEI

SPECIAL REPORT

HEALTH EFFECTS INSTITUTE

July 2000

Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality

A Special Report of the Institute's Particle Epidemiology Reanalysis Project

SUPREME COURT OF THE UNITED STATES

WHITMAN, ADMINISTRATOR OF ENVIRONMENTAL PROTECTION AGENCY, ET AL. *v.* AMERICAN TRUCKING ASSOCIATIONS, INC., ET AL.

No. 99–1257. Argued November 7, 2000—Decided February 27, 2001*

Legal uncertainty largely resolved with 2001 unanimous ruling by the U.S. Supreme Court.

U.S. Medicare Cohort Studies

U.S. Medicare Cohort studies:

- Eftim et al. Epidemiology 2008
- Zegar et al. EHP 2008



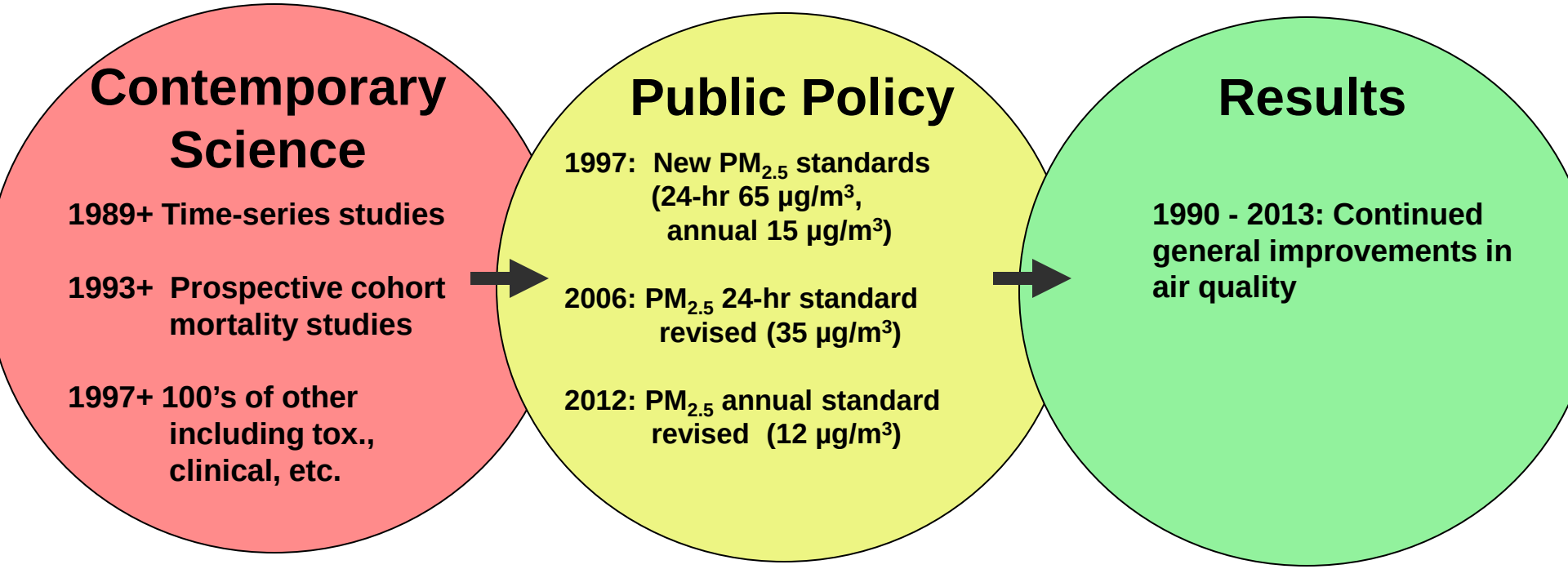

Cohorts of Medicare participants cities of the 6-cities and ACS study, plus all U.S.

All Cause | CPD | CVD | IHD

Percent increase in mortalily risk (95% CI)

190
180
120
110
100
90
80
70
60
50
40
30
20
10
0
-10

Modern air pollution science has resulted in new and tighter standards in the U.S. for air pollution—especially $PM_{2.5}$



So, an obvious question—

Has reducing air pollution resulted in substantial and measurable **improvements in human health?**

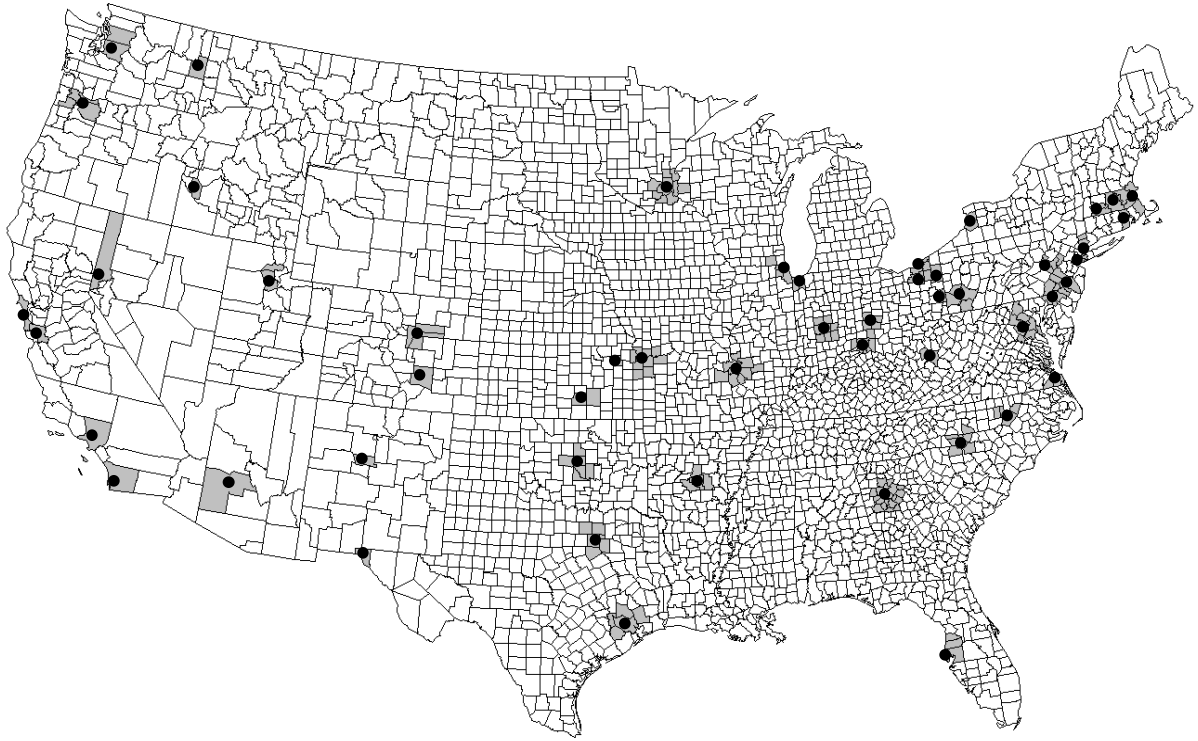
Do cities with bigger improvements in air quality have bigger improvements in health, measured by life expectancy?

The NEW ENGLAND JOURNAL of MEDICINE

January 22, 2009

Fine-Particulate Air Pollution and Life Expectancy in the United States

C. Arden Pope, III, Ph.D., Majid Ezzati, Ph.D., and Douglas W. Dockery, Sc.D.

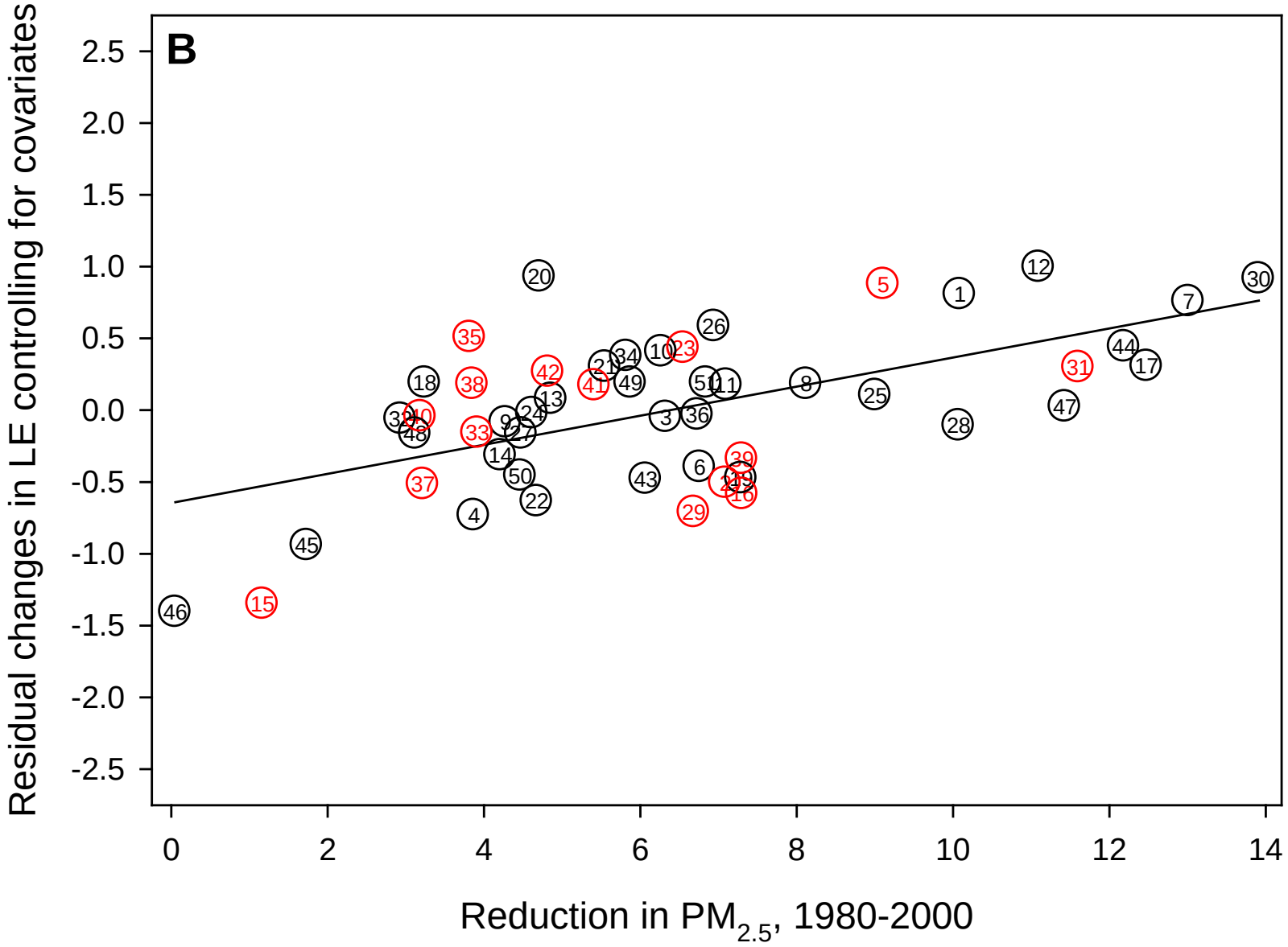


- Matching $PM_{2.5}$ data for 1979-1983 and 1999-2000 in 51 Metro Areas

- Life Expectancy data for 1978-1982 and 1997-2001 in 211 counties in 51 Metro areas

- Evaluate changes in Life Expectancy with changes in $PM_{2.5}$ for the 2-decade period of approximately 1980-2000.

YES. On average, the greater the reduction in air pollution, the greater the increase in life expectancy.



Effect of Air Pollution Control on Life Expectancy in the United States

An Analysis of 545 U.S. Counties for the Period from 2000 to 2007

Andrew W. Correia,[a] C. Arden Pope III,[b] Douglas W. Dockery,[c] Yun Wang,[a] Majid Ezzati,[d] and Francesca Dominici[a]

Epidemiology 2013;

Francesca Dominici

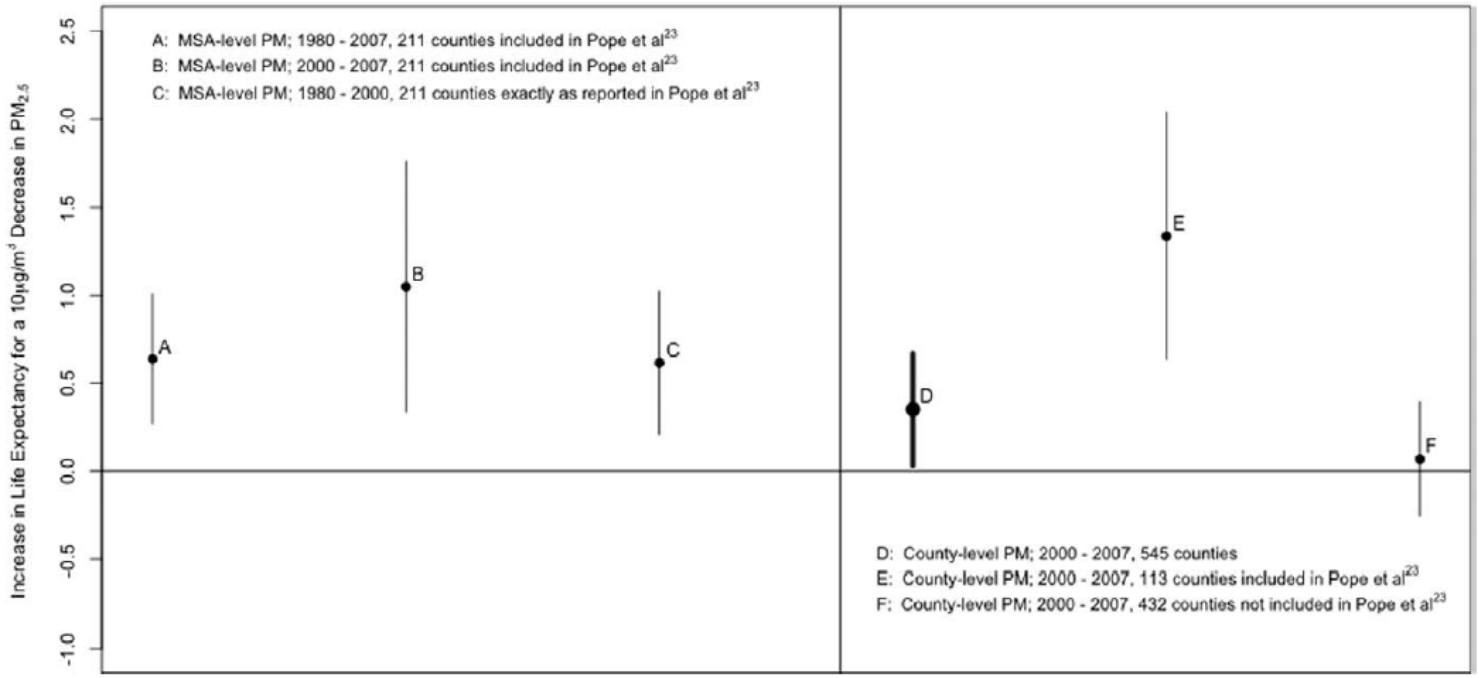
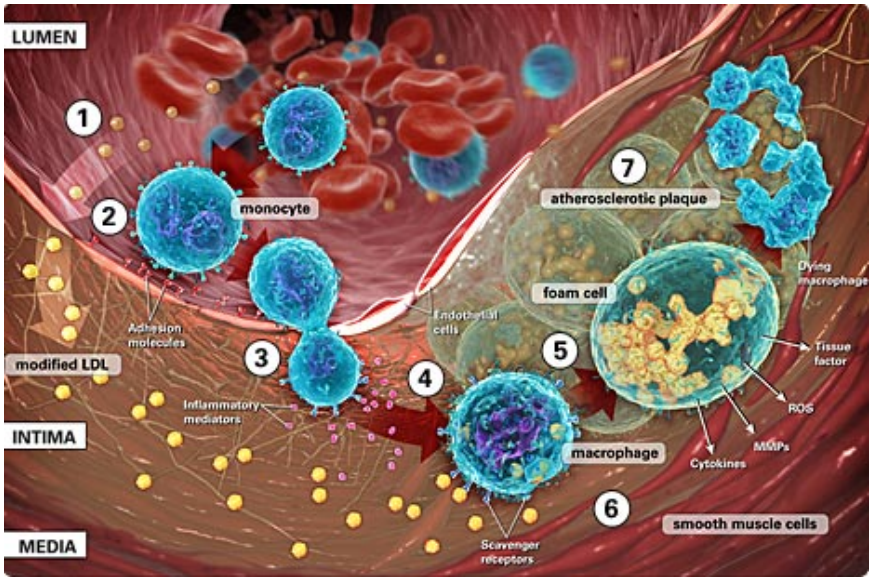


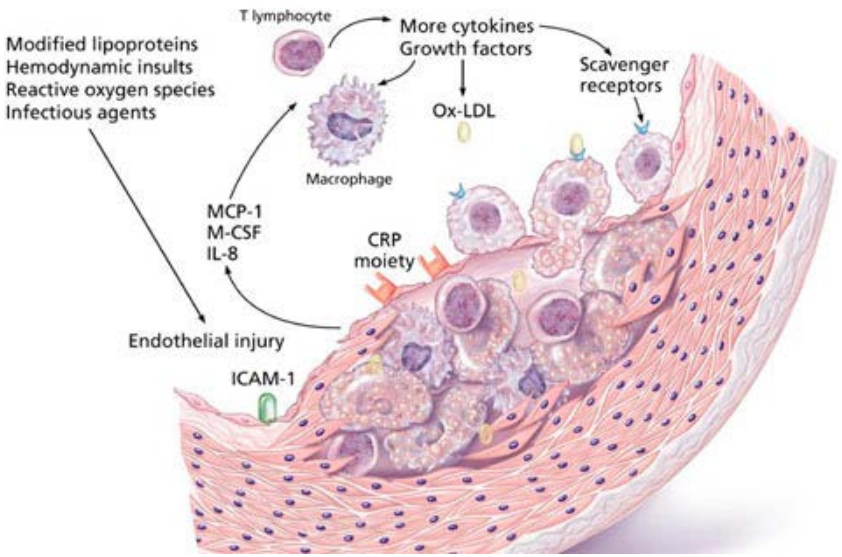
FIGURE 3. Point estimates (circles) and 95% confidence intervals (vertical lines) for the effect of a 10 $\mu g/m^3$ decrease in $PM_{2.5}$ on life expectancy. Estimates A and B were obtained from data set 3; estimate C was obtained from data set 2. Estimates A, B, and C were adjusted for changes in income, population, proportion of the population that is black, lung cancer death rate, and COPD death rate (model 4, eTable 2a, b, http://links.lww.com/EDE/A630). Estimates D, E, and F were obtained from data set 1, adjusted for changes in income, population, proportion of high-school graduates, proportion of the population that is black, proportion of the population that is Hispanic, lung cancer death rate, and COPD death rate (model 3, Table 2).

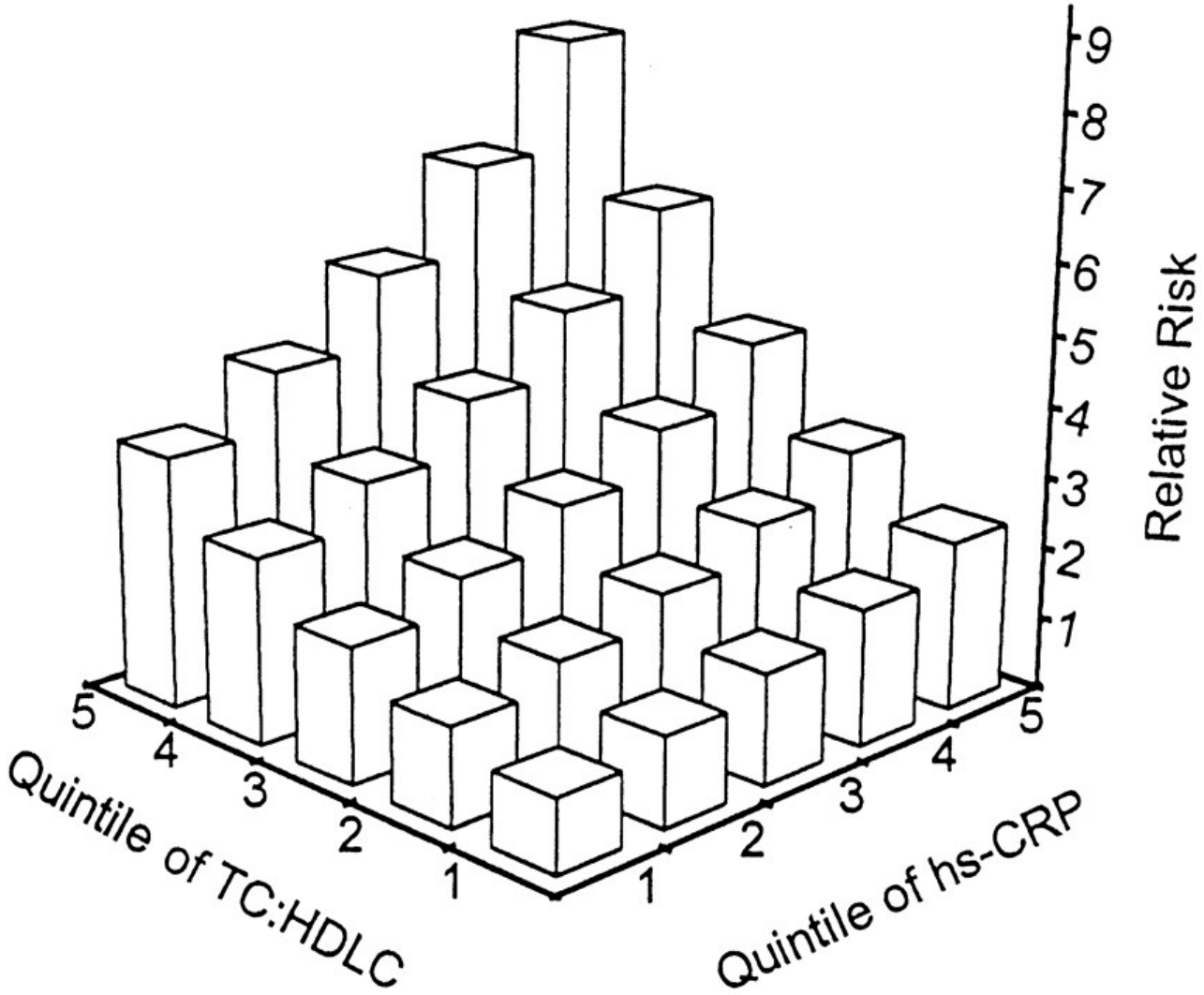
Cardiovascular disease as part of chronic and acute inflammatory processes.

By the early 2000s, there was increasingly compelling evidence that inflammation is a major accomplice with LDL cholesterol in the initiation and progression of atherosclerosis.

Furthermore, inflammation contributes to acute thrombotic complications of atherosclerosis, increasing the risk of making atherosclerotic plaques more vulnerable to rupture, clotting, and precipitating acute cardiovascular or cerebrovascular events (MI or ischemic stroke).







Paul Ridker

Interactive effects of hs-CRP (marker of inflammation) and blood lipids.

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

Ridker PM. 2001;103:1813-1818.

Fine Particulate exposure

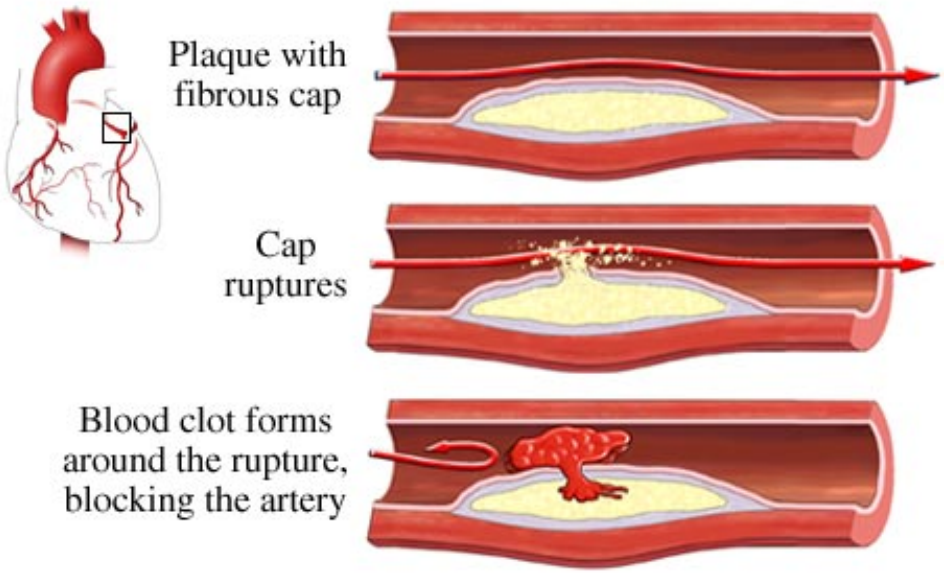
↓

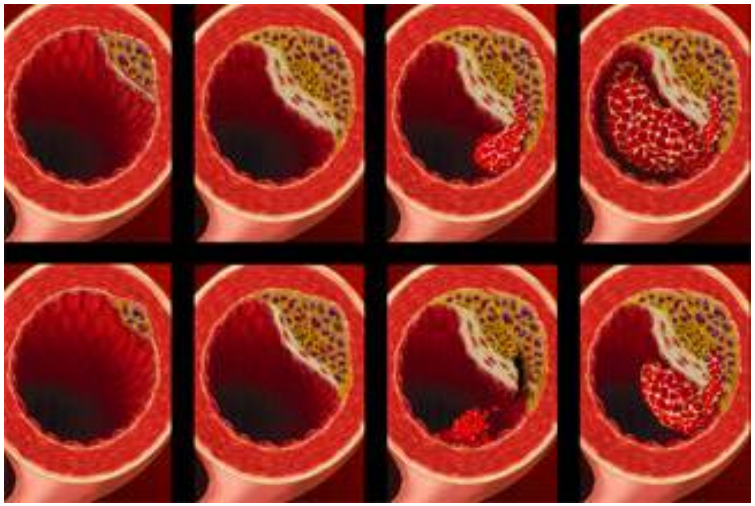
Pulmonary and systemic inflammation and oxidative stress

(along with blood lipids)

↓

Progression and destabilization of atherosclerotic plaques





Experimental evidence of biological effects of PM extracted from filters
(Ghio, Costa, Devlin, Kennedy, Frampton, Dye, et al. 1998-2004)

- Acute airway injury and inflammation in rats and humans
- *In vitro* oxidative stress and release of proinflammatory mediators by cultured respiratory epithelial cells
- Differential toxicities of PM when the mill was operating versus when it was not (metals content and mixtures?)

A series of studies by van Eeden, Hogg, Suwa et al. (1997-2002) suggest:

PM exposure

↓

Pulmonary inflammation

↓

Systemic inflammatory responses (including release of inflammatory mediators, bone marrow stimulation and release of leukocytes and platelets)

↓

Progression and destabilization of atherosclerotic plaques

Stephan van Eeden

In rabbits naturally prone to develop atherosclerosis they found that:

PM exposure

↓↓

Accelerated progression of atherosclerotic plaques with greater vulnerability to plaque rupture

James Hogg


Sun QH


Lippmann M

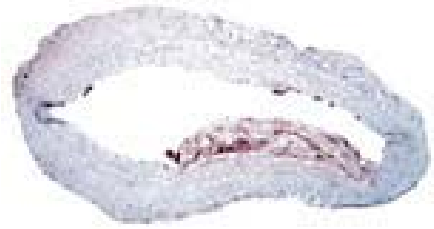
Sun et al. (*JAMA* 2005)

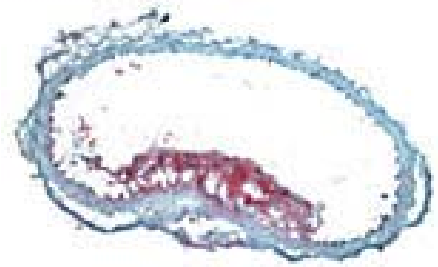
apoE$^{-/-}$ mouse

Representative Photomicrographs of Aortic Arch Sections

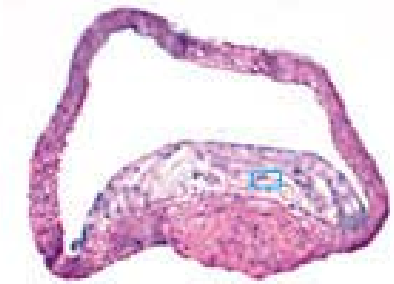
Normal Chow

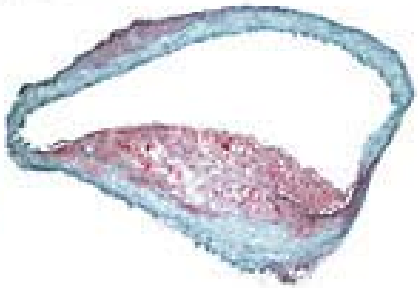
Clean Filtered Air





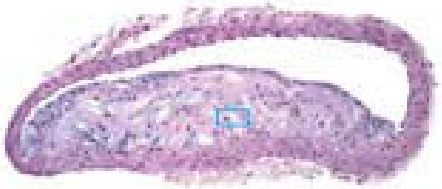
PM Polluted Air

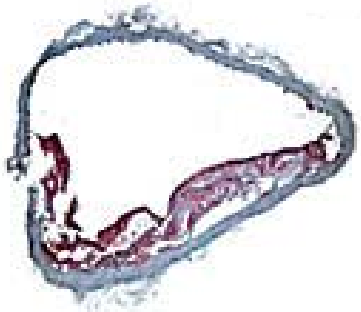




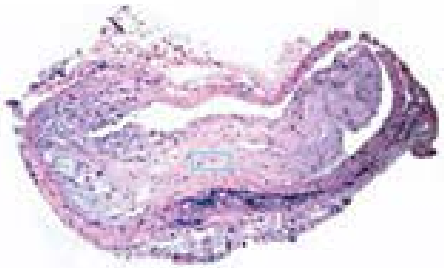
High-Fat Chow

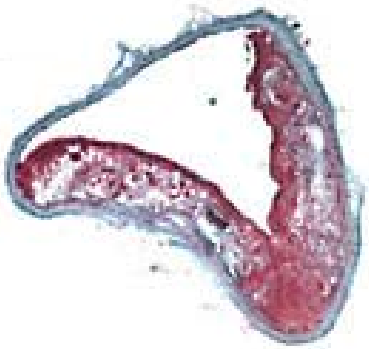
Clean Filtered Air



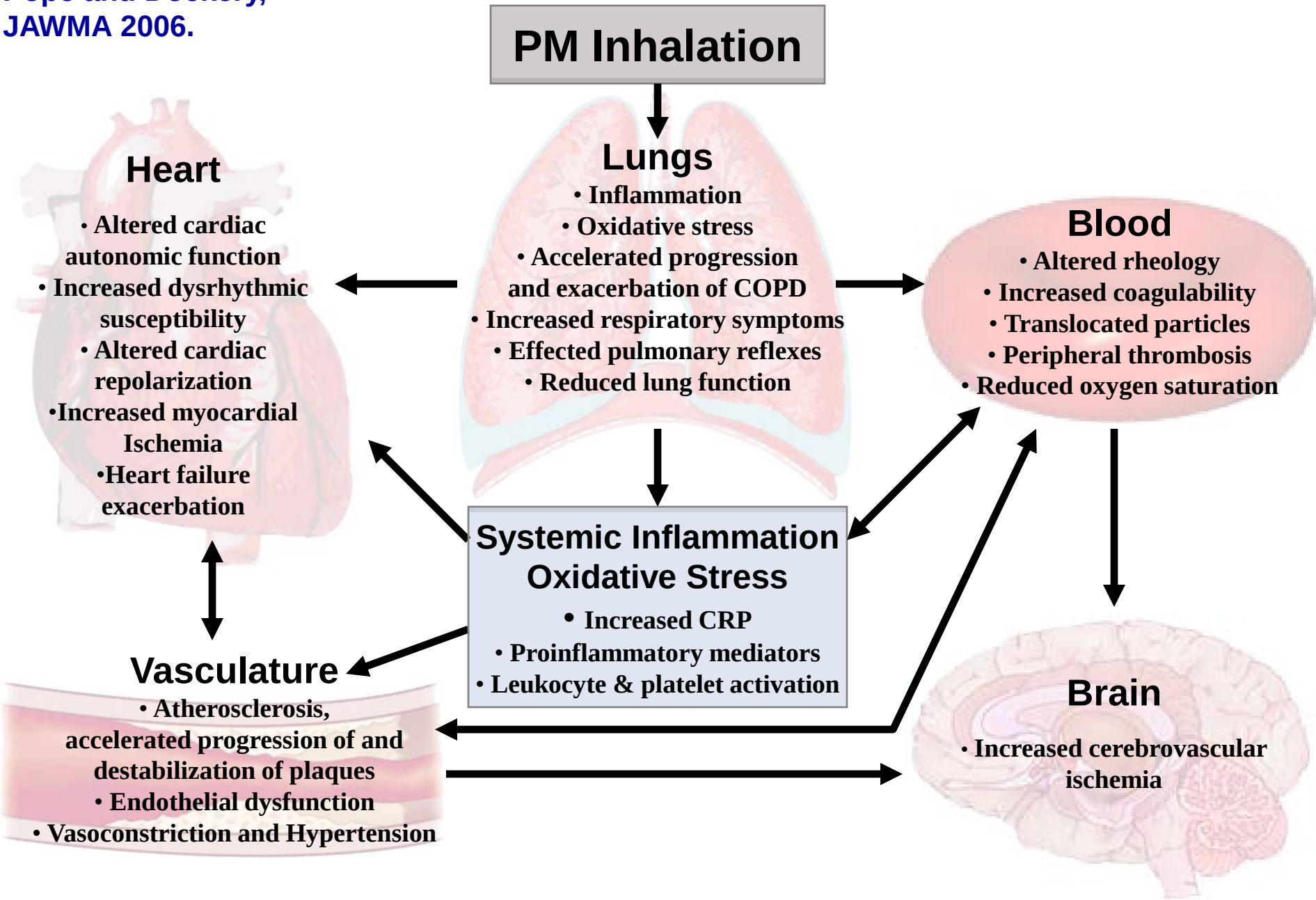


PM Polluted Air





Pope and Dockery, JAWMA 2006.



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Brook, Rajagopalan, Pope, et al. 2011
AHA Scientific Statement, PM and CVD

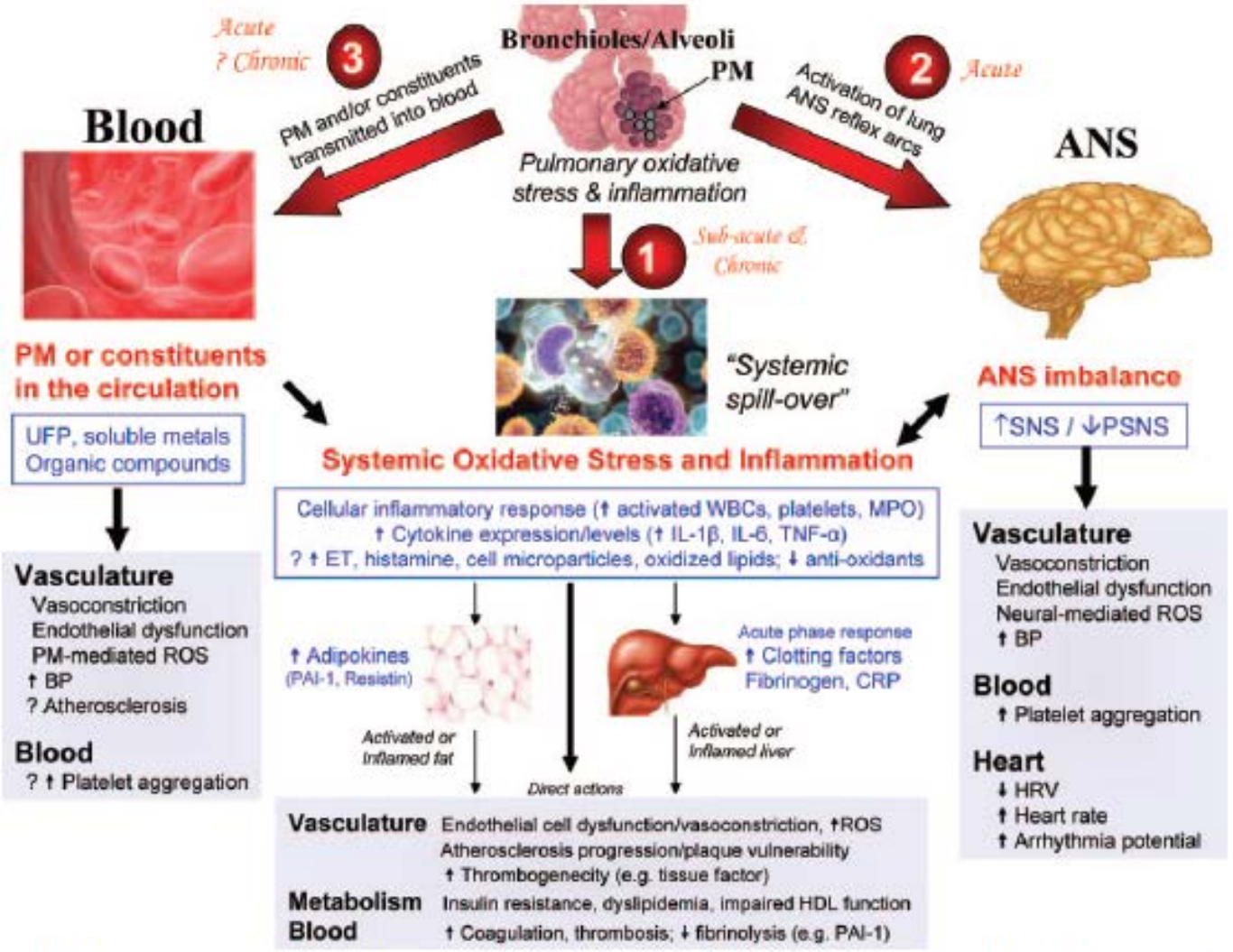
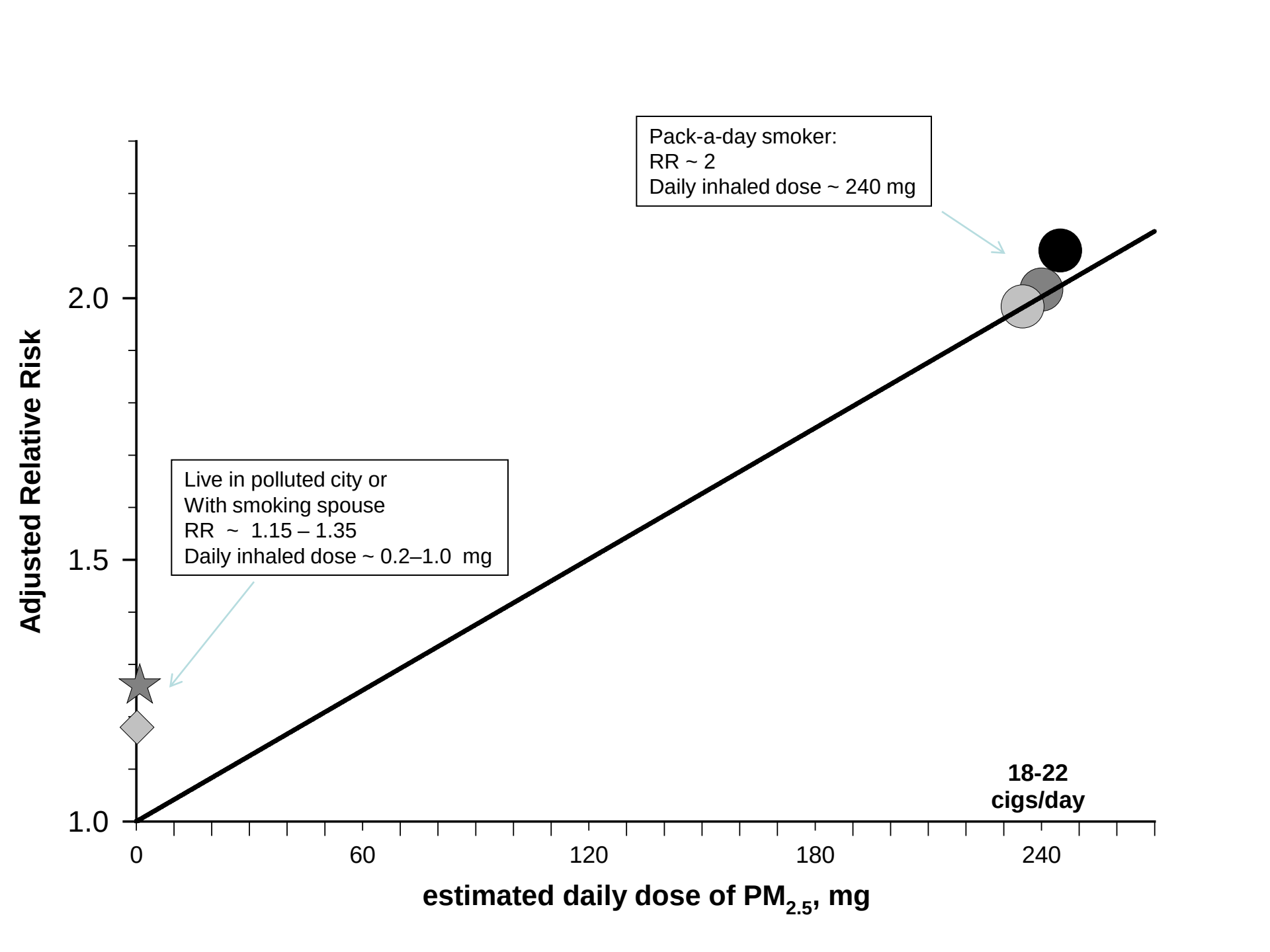


Figure 3. Biological pathways linking PM exposure with CVDs. The 3 generalized intermediary pathways and the subsequent specific biological responses that could be capable of instigating cardiovascular events are shown. MPO indicates myeloperoxidase; PAI, plasminogen activator inhibitor; PSNS, parasympathetic nervous system; SNS, sympathetic nervous system; and WBCs, white blood cells. A question mark (?) indicates a pathway/mechanism with weak or mixed evidence or a mechanism of likely yet primarily theoretical existence based on the literature.

Biggest criticisms regarding the overall results:

1. The effects aren't big enough to be compelling (need RR > 2.0)

2. The effects are too large to be biologically plausible based on an extrapolation of smoking literature.


Pack-a-day smoker:
RR ~ 2
Daily inhaled dose ~ 240 mg
Live in polluted city or
With smoking spouse
RR ~ 1.15 – 1.35
Daily inhaled dose ~ 0.2–1.0 mg
18-22
cigs/day
Adjusted Relative Risk
2.0
1.5
1.0
0
60
120
180
240
estimated daily dose of PM2.5, mg

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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

Table 2. Adjusted Relative Cardiovascular and Cardiopulmonary Risk Estimates for Various Increments of Exposure From Cigarette Smoking, Secondhand Cigarette Smoke, and Ambient Air Pollution From the Present Analysis and Selected Comparison Studies

Source of Risk Estimate	Increments of Exposure	Adjusted Relative Risk (95% CI)			Estimated Daily Dose of $PM_{2.5}$, mg	
		Ischemic Heart Disease	Cardiovascular Disease	Cardiopulmonary Disease	Baseline*	Alternative†
Cigarette smoking estimates based on ACS CPS-II cohort						
ACS full cohort, present analysis	≤3 (1.5) cigarettes/day	1.63 (1.36–1.96)	1.64 (1.42–1.89)	1.72 (1.50–1.96)	18.0	10.5
ACS full cohort, present analysis	4–7 (5.5) cigarettes/day	1.54 (1.34–1.77)	1.61 (1.45–1.78)	1.65 (1.50–1.82)	66.0	38.5
ACS full cohort, present analysis	8–12 (10) cigarettes/day	1.85 (1.69–2.02)	1.79 (1.67–1.93)	1.87 (1.75–2.00)	120.0	70.0
ACS full cohort, present analysis	13–17 (15) cigarettes/day	1.79 (1.59–2.02)	1.67 (1.52–1.85)	1.75 (1.60–1.92)	180.0	105.0
ACS full cohort, present analysis	18–22 (20) cigarettes/day	1.98 (1.87–2.10)	2.02 (1.93–2.11)	2.09 (2.01–2.18)	240.0	140.0
ACS full cohort, present analysis	≥23 (27) cigarettes/day	1.97 (1.86–2.10)	2.03 (1.93–2.13)	2.17 (2.08–2.27)	324.0	189.0
Ambient air pollution estimates based on ACS CPS-II cohort						
ACS $PM_{2.5}$ subcohort, original[2]	24.5 $\mu g/m^3$ ambient $PM_{2.5}$	…	…	1.31 (1.17–1.46)	0.44	0.56
ACS $PM_{2.5}$ subcohort, extended[4,5]	10 $\mu g/m^3$ ambient $PM_{2.5}$	1.18 (1.14–1.23)	1.12 (1.08–1.15)	1.09 (1.03–1.16)	0.18	0.23
Comparison ambient air pollution estimates based on alternative cohorts						
Harvard Six Cities original[1]	18.6 $\mu g/m^3$ ambient $PM_{2.5}$	…	…	1.37 (1.11–1.68)	0.33	0.42
Harvard Six Cities extended[7]	10 $\mu g/m^3$ ambient $PM_{2.5}$	…	1.28 (1.13–1.44)	…	0.18	0.23
Women's Health Initiative[8]	10 $\mu g/m^3$ ambient $PM_{2.5}$	…	1.24 (1.09–1.41)‡	…	0.18	0.23
Comparison SHS estimates						
Surgeon General's report[32]	Low-moderate SHS exposure	…	1.16 (1.03–1.32)	…	0.36	0.46
Surgeon General's report[32]	Moderate-high SHS exposure	…	1.26 (1.12–1.42)	…	0.90	1.15
INTERHEART study[33]	1–7 h/wk SHS exposure	1.24 (1.17–1.32)§	…	…	0.36	0.46
INTERHEART study[33]	Live with smoking spouse	1.28 (1.12–1.47)§	…	…	0.72	0.92

*The baseline estimated daily dose assumes an inhalation rate of 18 m^3/d and a dose of 12 mg per cigarette.
†The alternative estimated daily dose assumes an inhalation rate of 23 m^3/d and a dose of 7 mg per cigarette.
‡First cardiovascular disease event.
§Myocardial infarction.

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Pope, Burnett, Krewski, et al. 2009.

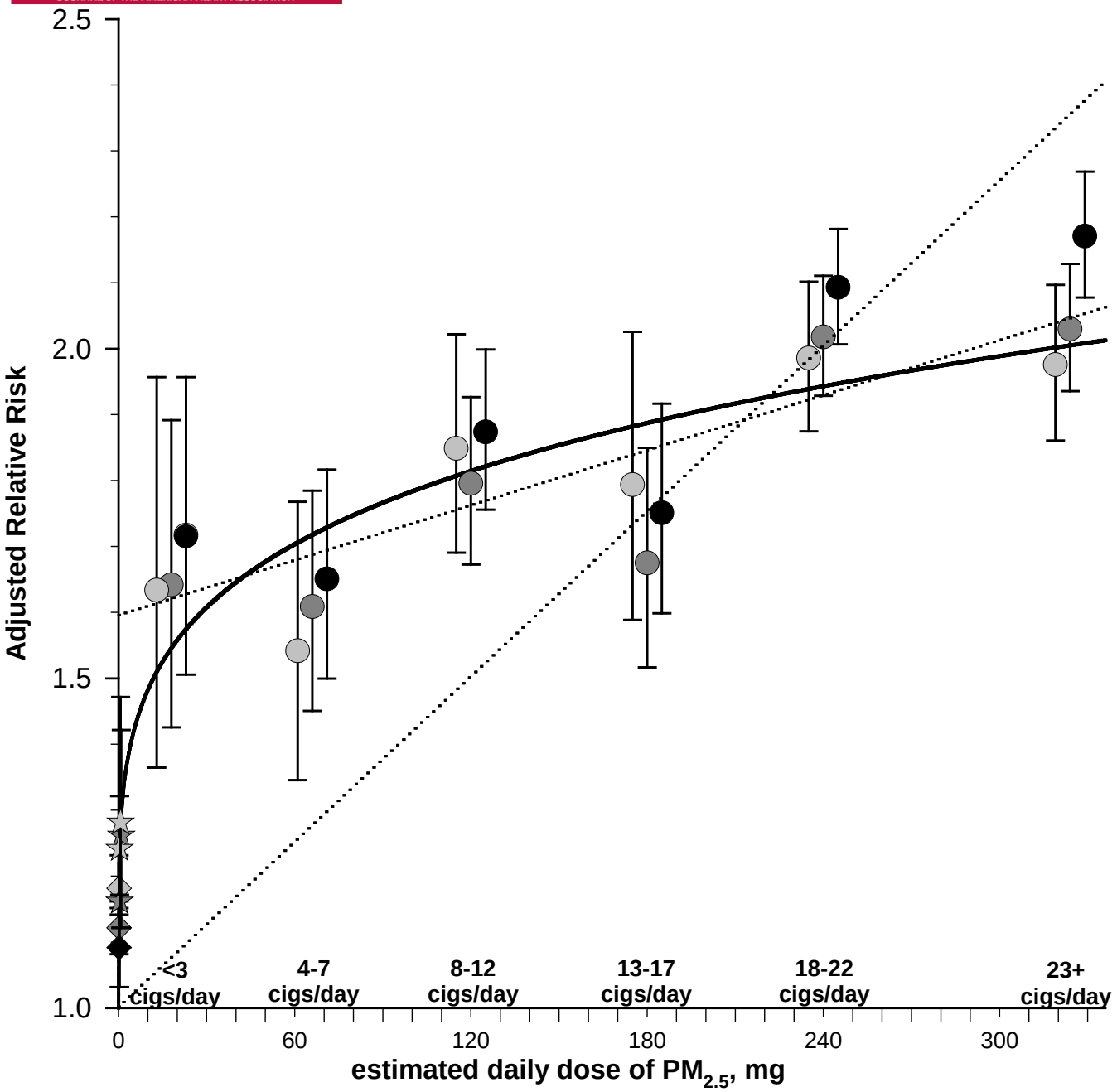


Figure 1. Adjusted relative risks (and 95% CIs) of IHD (light gray), CVD (dark gray), and CPD (black) mortality plotted over estimated daily dose of $PM_{2.5}$ from different increments of current cigarette smoking. Diamonds represent comparable mortality risk estimates for $PM_{2.5}$ from air pollution.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

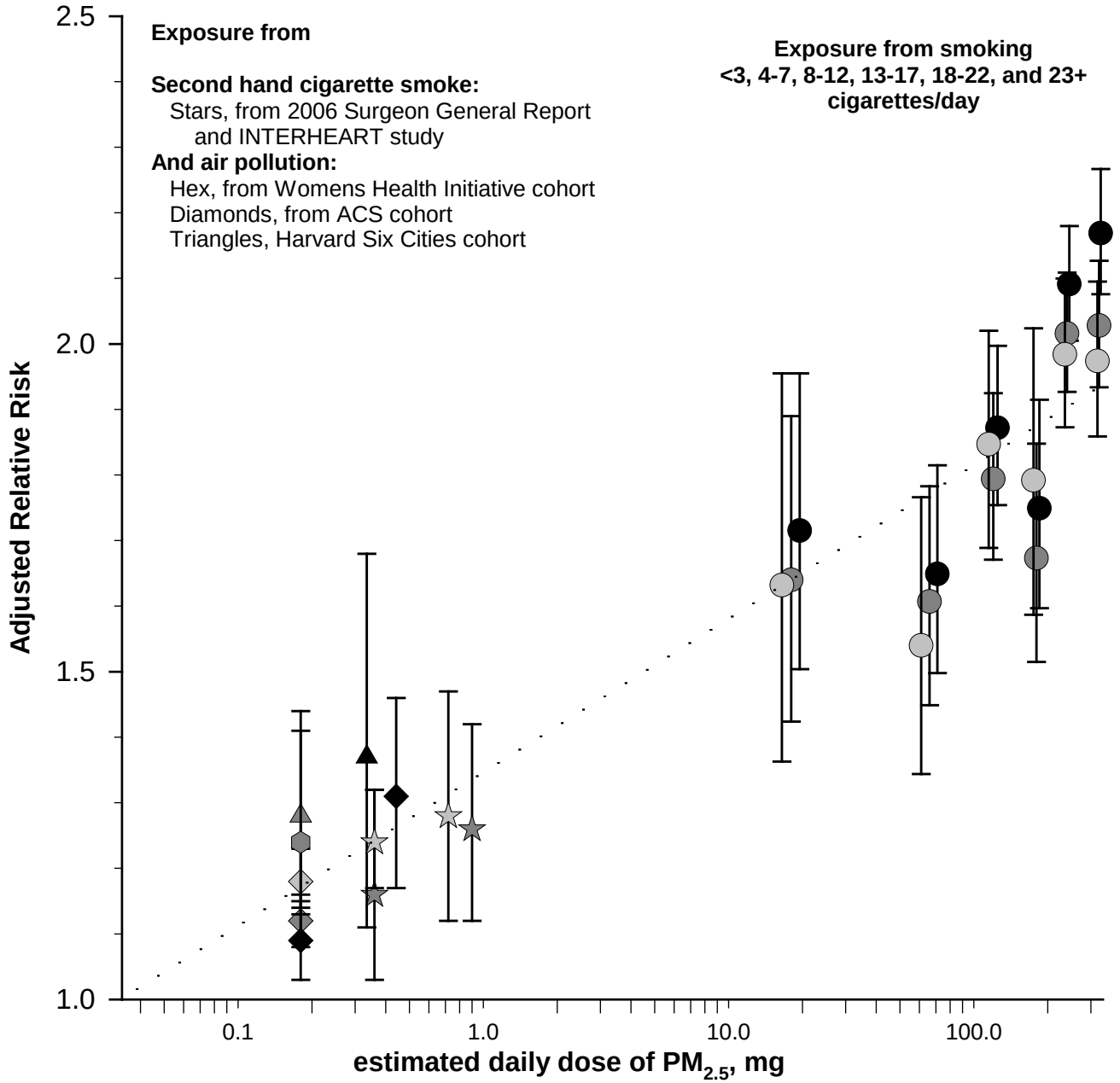
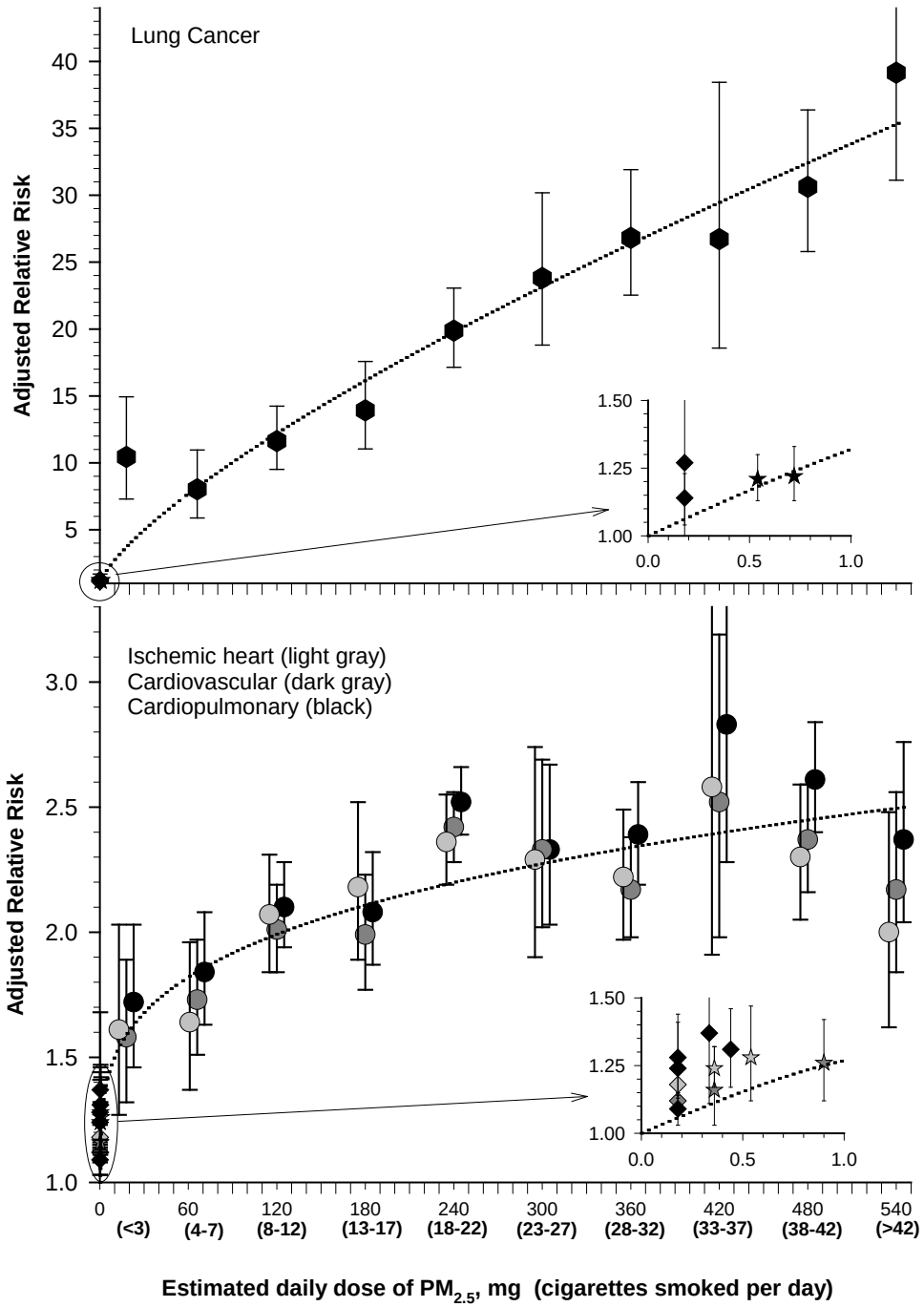


Figure 2. Adjusted relative risks (and 95% CIs) of ischemic heart disease (light gray), cardiovascular (dark gray), and cardiopulmonary (black) mortality plotted over baseline estimated daily dose (using a log scale) of $PM_{2.5}$ from current cigarette smoking (relative to never smokers), SHS, and air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


Lung Cancer
Adjusted Relative Risk
40
35
30
25
20
15
10
5
1.50
1.25
1.00
0.0
0.5
1.0
Ischemic heart (light gray)
Cardiovascular (dark gray)
Cardiopulmonary (black)
Adjusted Relative Risk
3.0
2.5
2.0
1.5
1.0
0
60
120
180
240
300
360
420
480
540
(<3)
(4-7)
(8-12)
(13-17)
(18-22)
(23-27)
(28-32)
(33-37)
(38-42)
(>42)
Estimated daily dose of PM2.5, mg (cigarettes smoked per day)

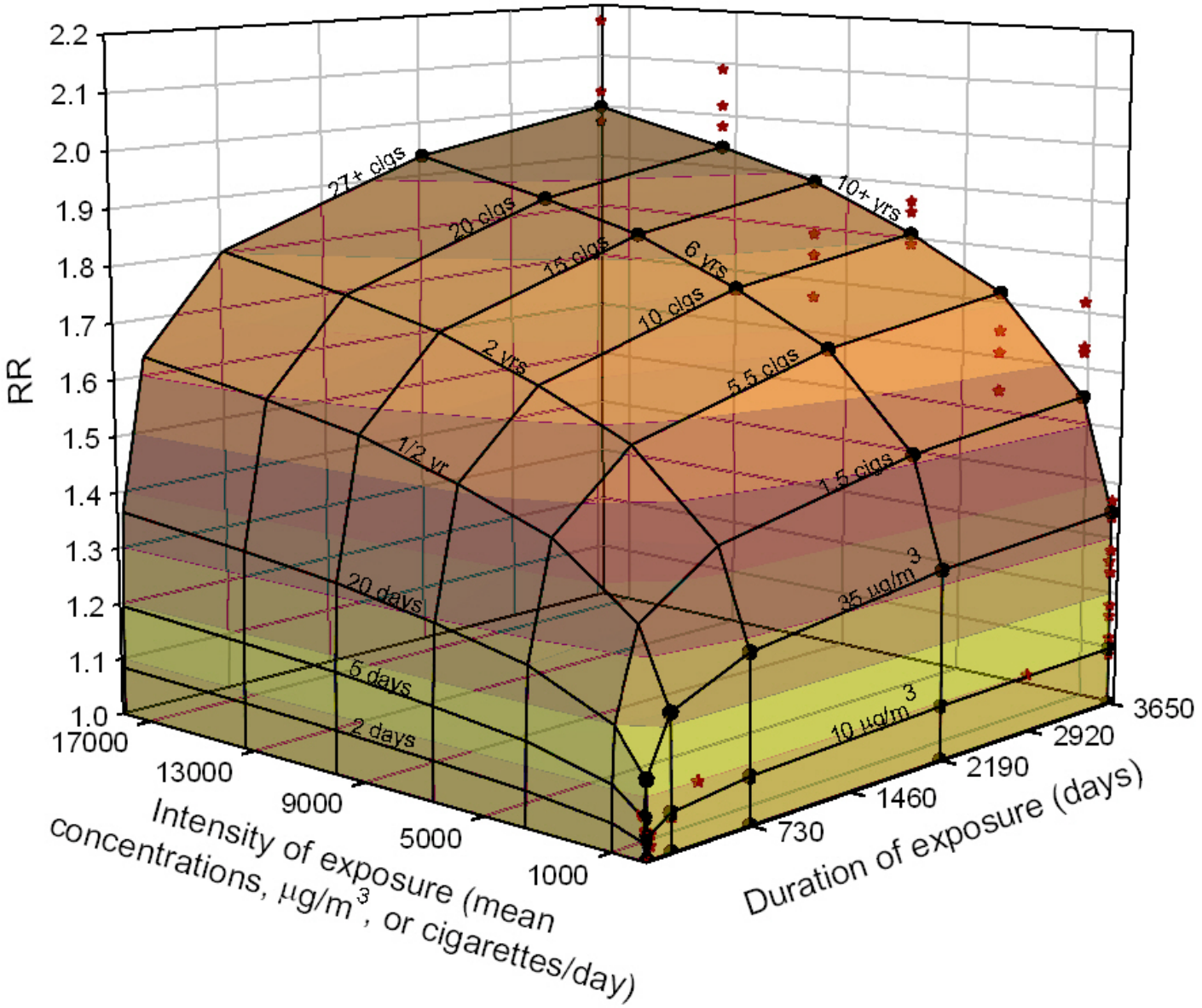
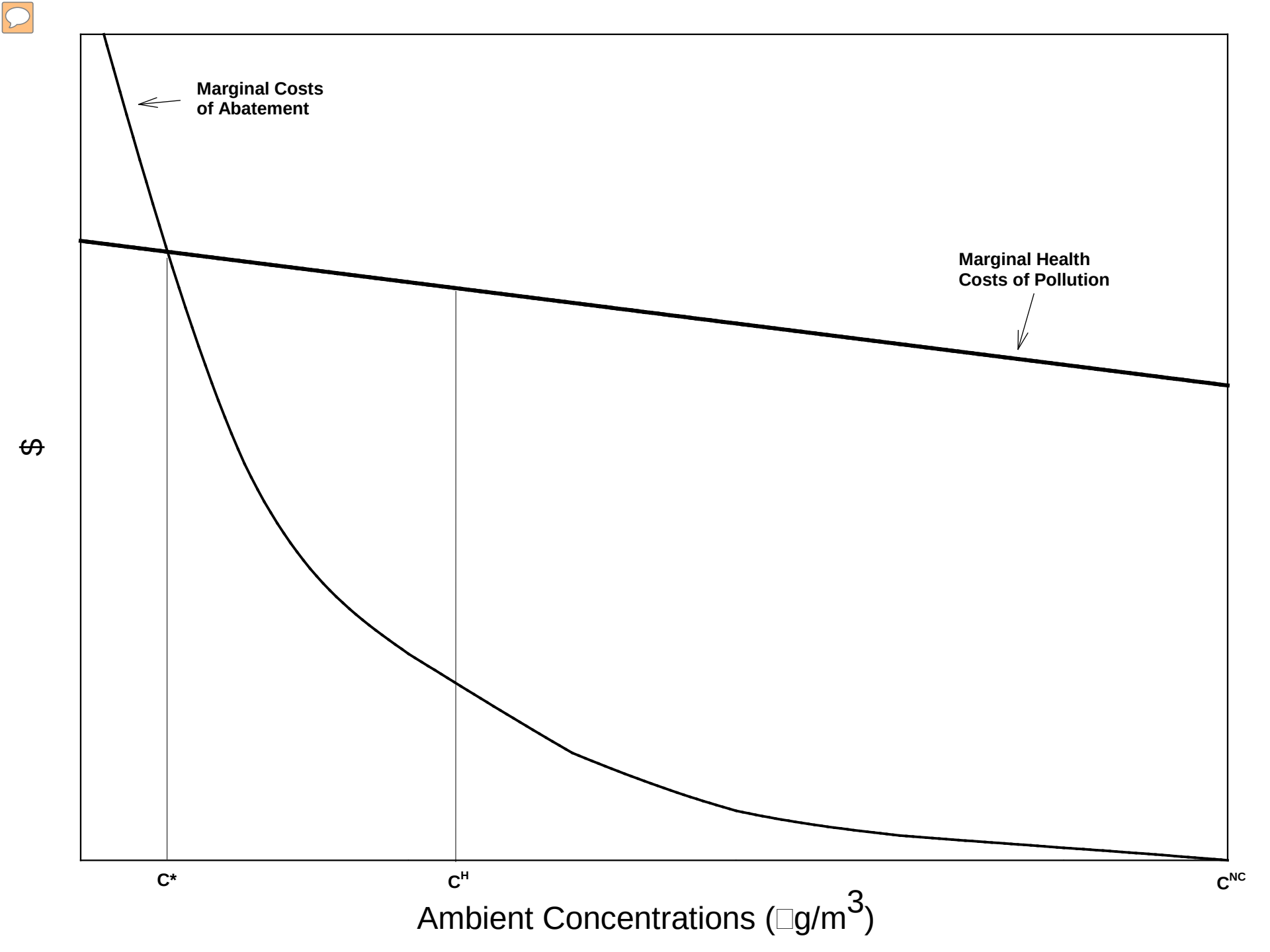
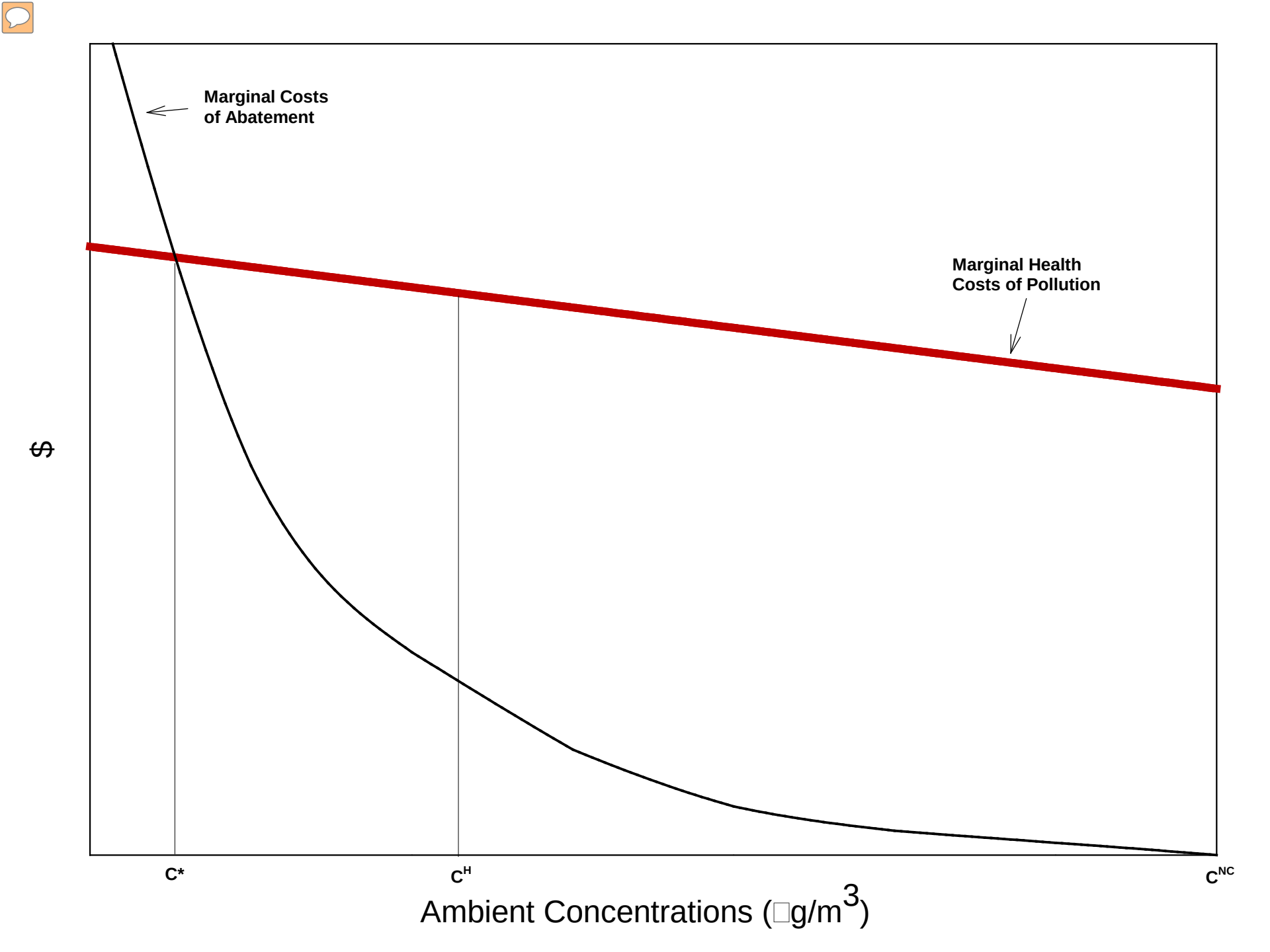
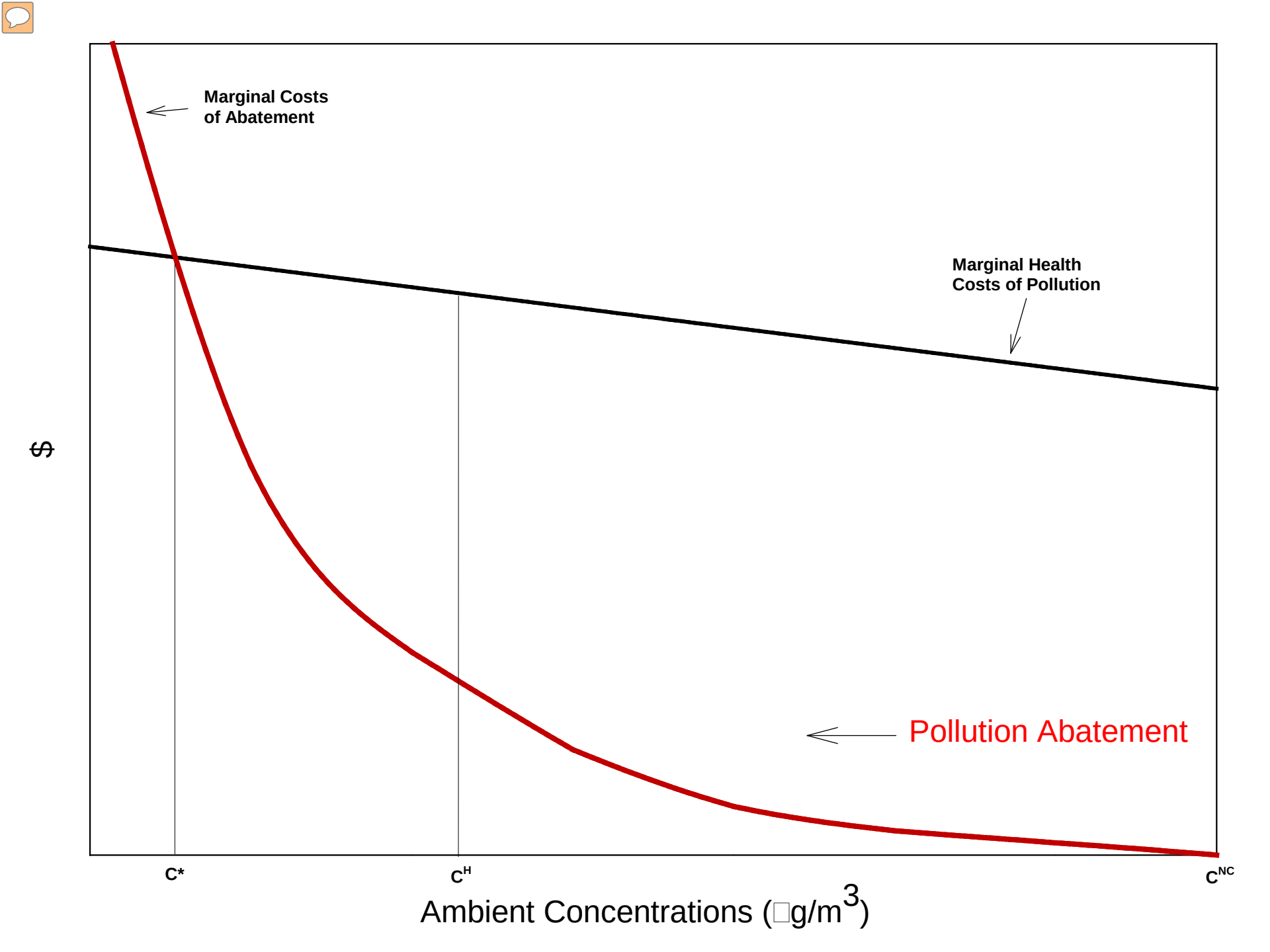
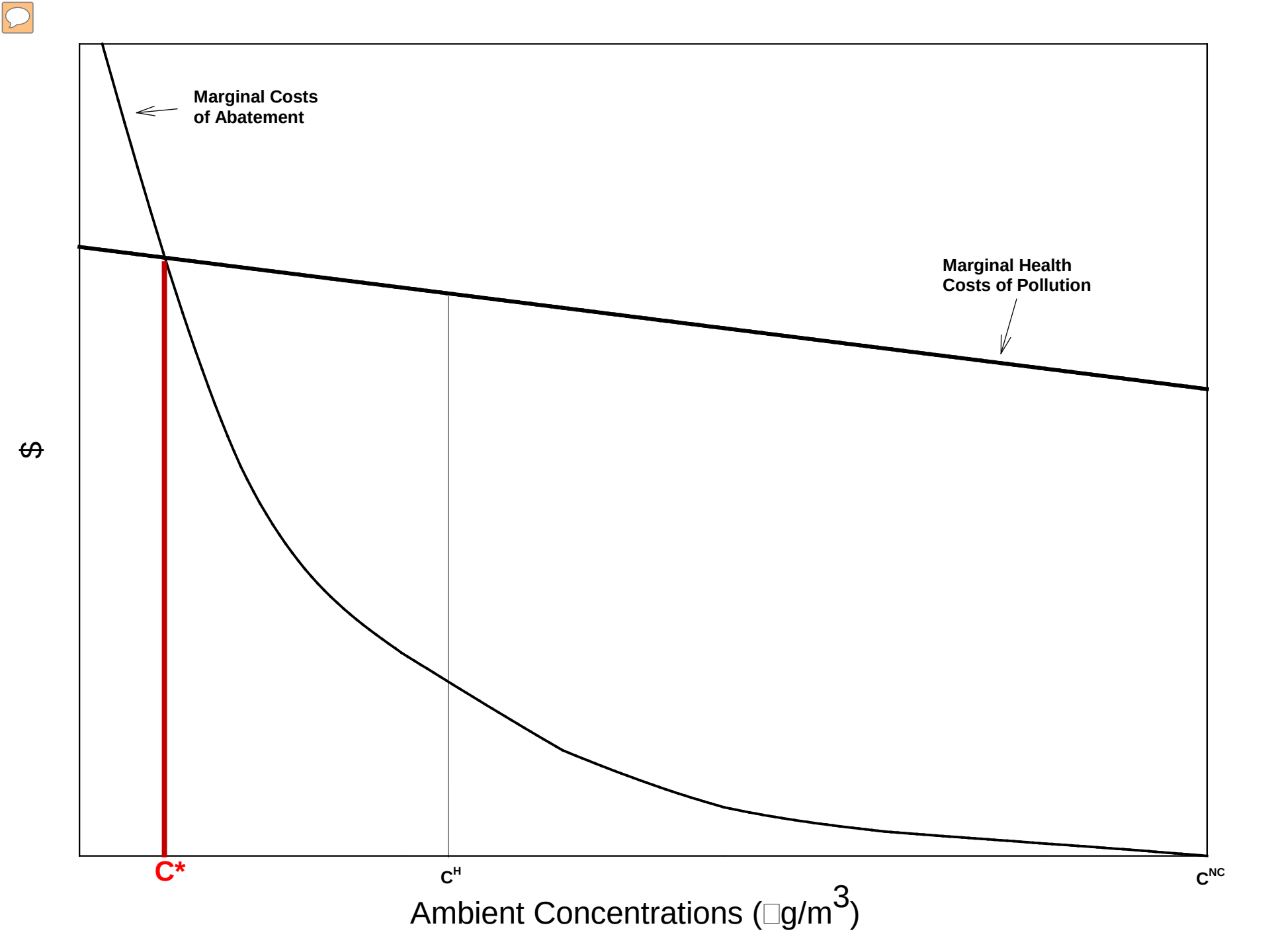


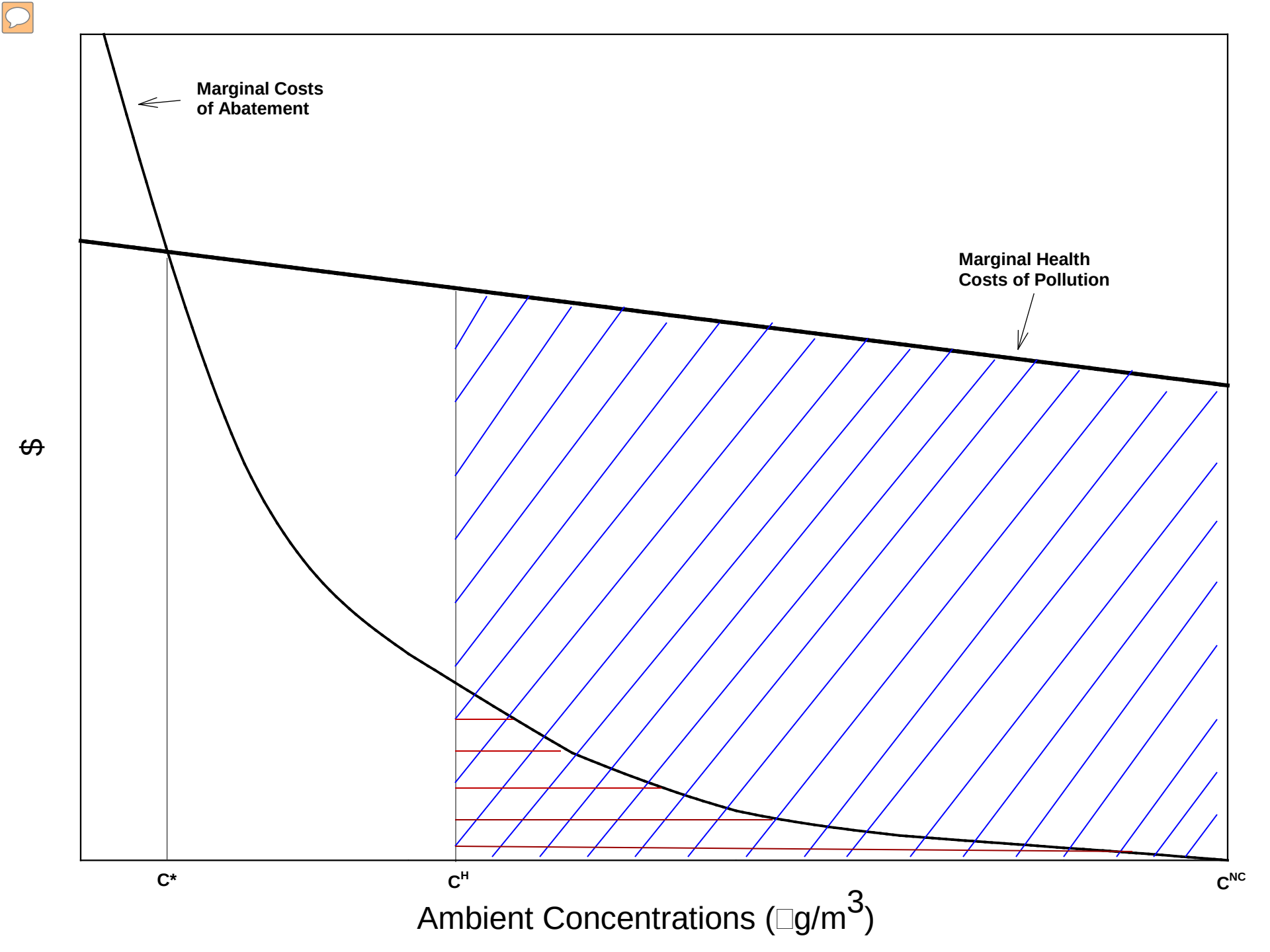
Figure 1. Stylized representation of the risk-response relationship between cardiopulmonary mortality and two primary dimensions of cumulative exposure to $PM_{2.5}$ (intensity and duration).

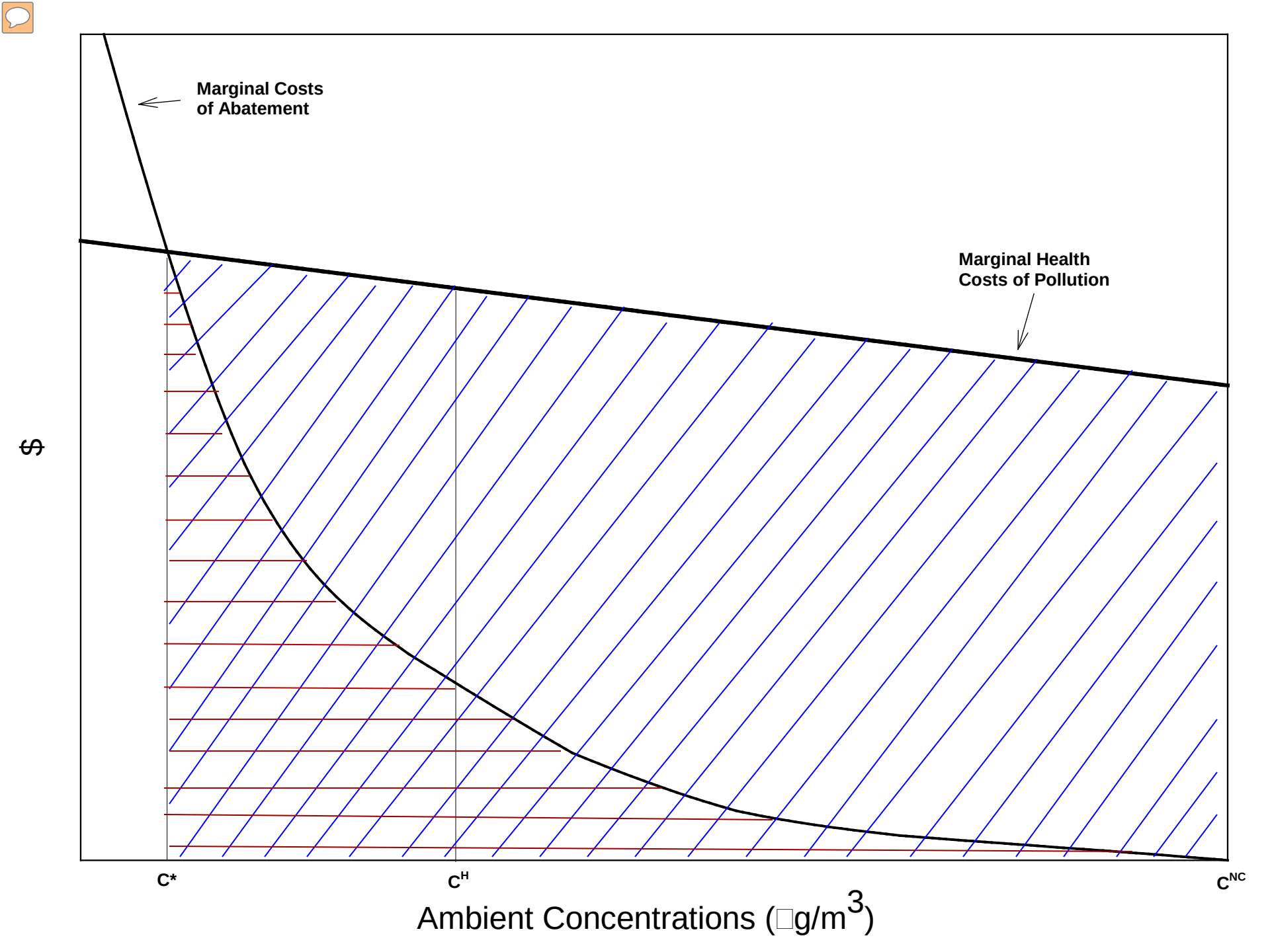

Marginal Costs
of Abatement
Marginal Health
Costs of Pollution
$
C*
C^H
C^NC
Ambient Concentrations (□g/m3)


Marginal Costs
of Abatement
Marginal Health
Costs of Pollution
$
C*
C^H
C^NC
Ambient Concentrations (□g/m3)


Marginal Costs
of Abatement
Marginal Health
Costs of Pollution
$
Pollution Abatement
C*
C^H
C^NC
Ambient Concentrations (□g/m3)


Marginal Costs
of Abatement
Marginal Health
Costs of Pollution
$
C*
C^H
C^NC
Ambient Concentrations (□g/m3)


Marginal Costs
of Abatement
Marginal Health
Costs of Pollution
$
C*
C^H
C^NC
Ambient Concentrations (□g/m3)


Marginal Costs
of Abatement
Marginal Health
Costs of Pollution
$
C*
C^H
C^NC
Ambient Concentrations (□g/m3)