LATEST FINDINGS ON PM HEALTH EFFECTS

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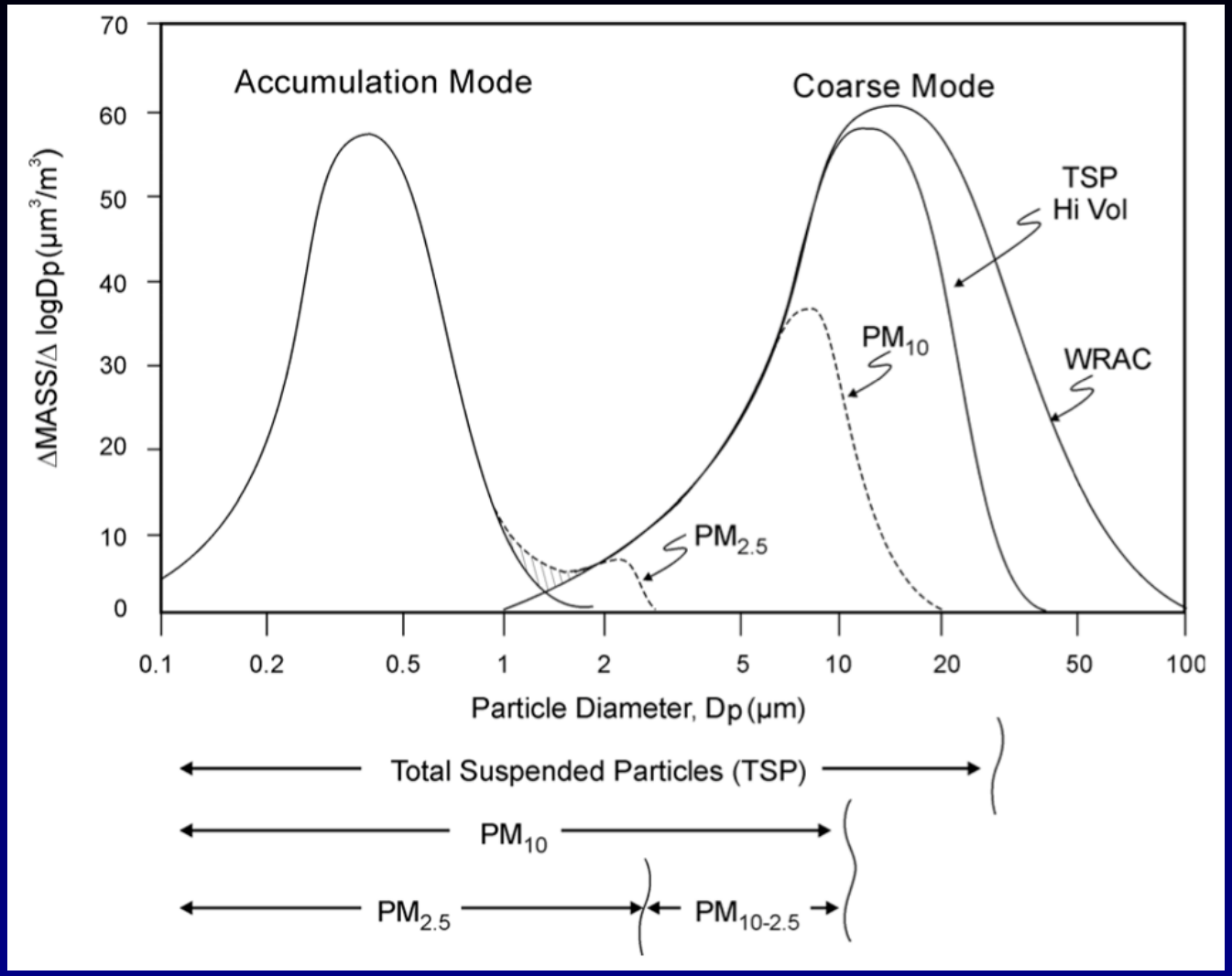


Figure 9-6. An idealized distribution of ambient particulate matter showing the accumulation mode and the coarse mode and the size fractions collected by size-selective samplers. (WRAC is the Wide Range Aerosol Classifier which collects the entire coarse model [Lundgren and Burton, 1995]).

Source: 4th Draft PM Criteria Document, June 2003.

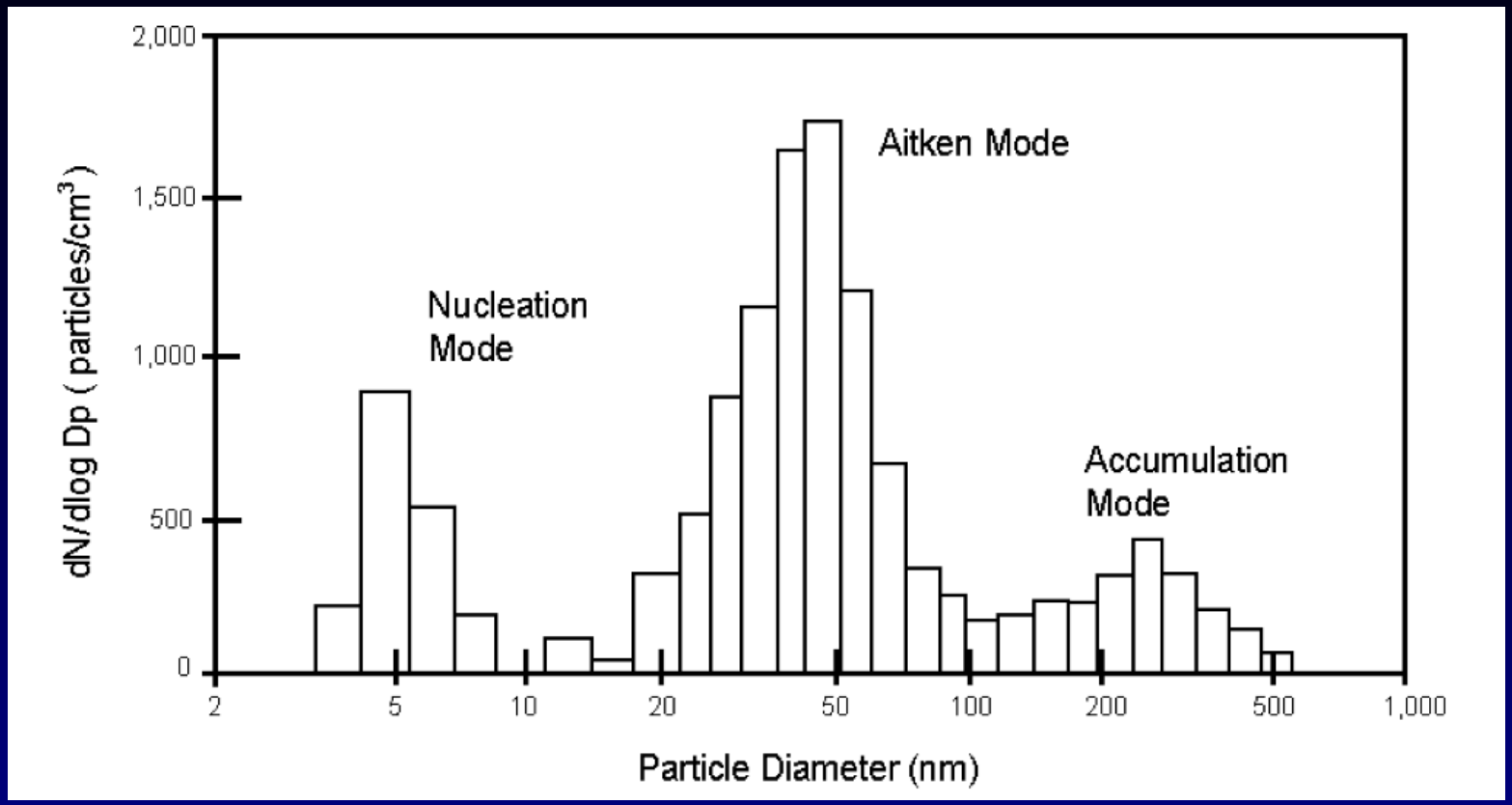


Figure 9-4. Submicron number size distributions observed in a boreal forest in Finland showing the tri-modal structure of fine particles. The total particle number concentration was 1011 particles/cm^3 (10 minute average).

Source: 4th Draft PM Criteria Document, June 2003.

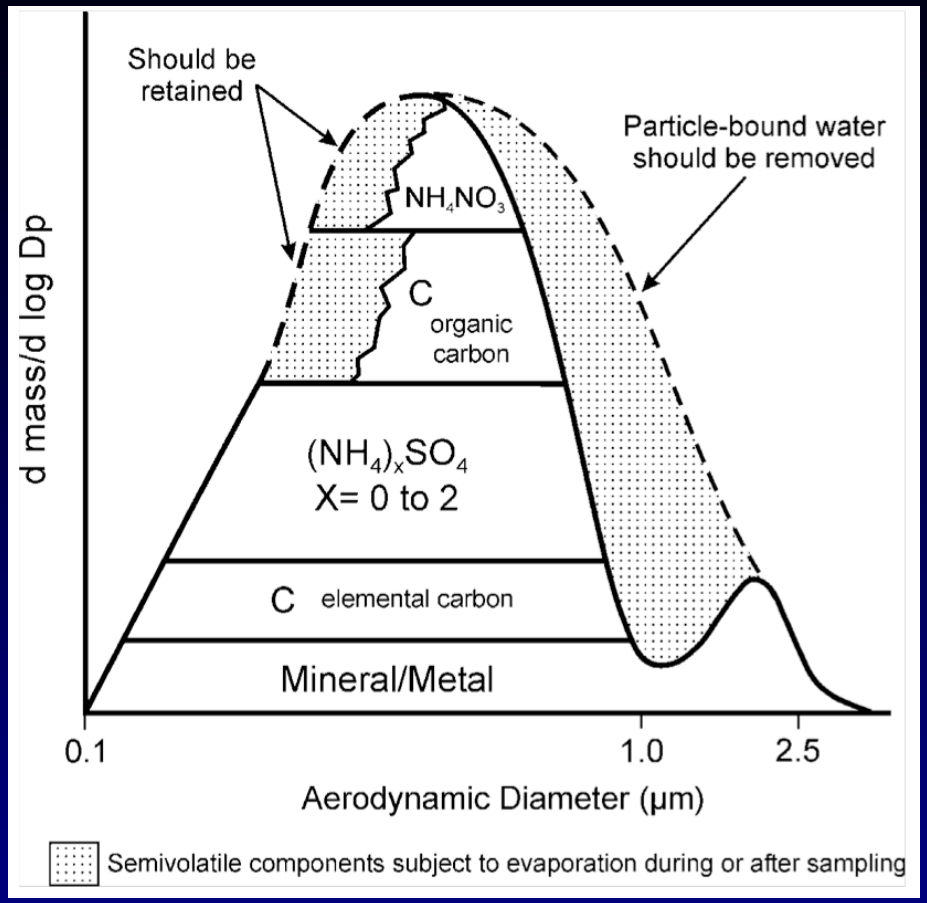


Figure 9-7. Schematic showing major nonvolatile and semivolatile components of $PM_{2.5}$. Semivolatile components are subject to partial to complete loss during equilibration or heating. The optimal technique would be to remove all particle-bound water but no ammonium nitrate or semivolatile organic PM.

Source: 4th Draft PM Criteria Document, June 2003.

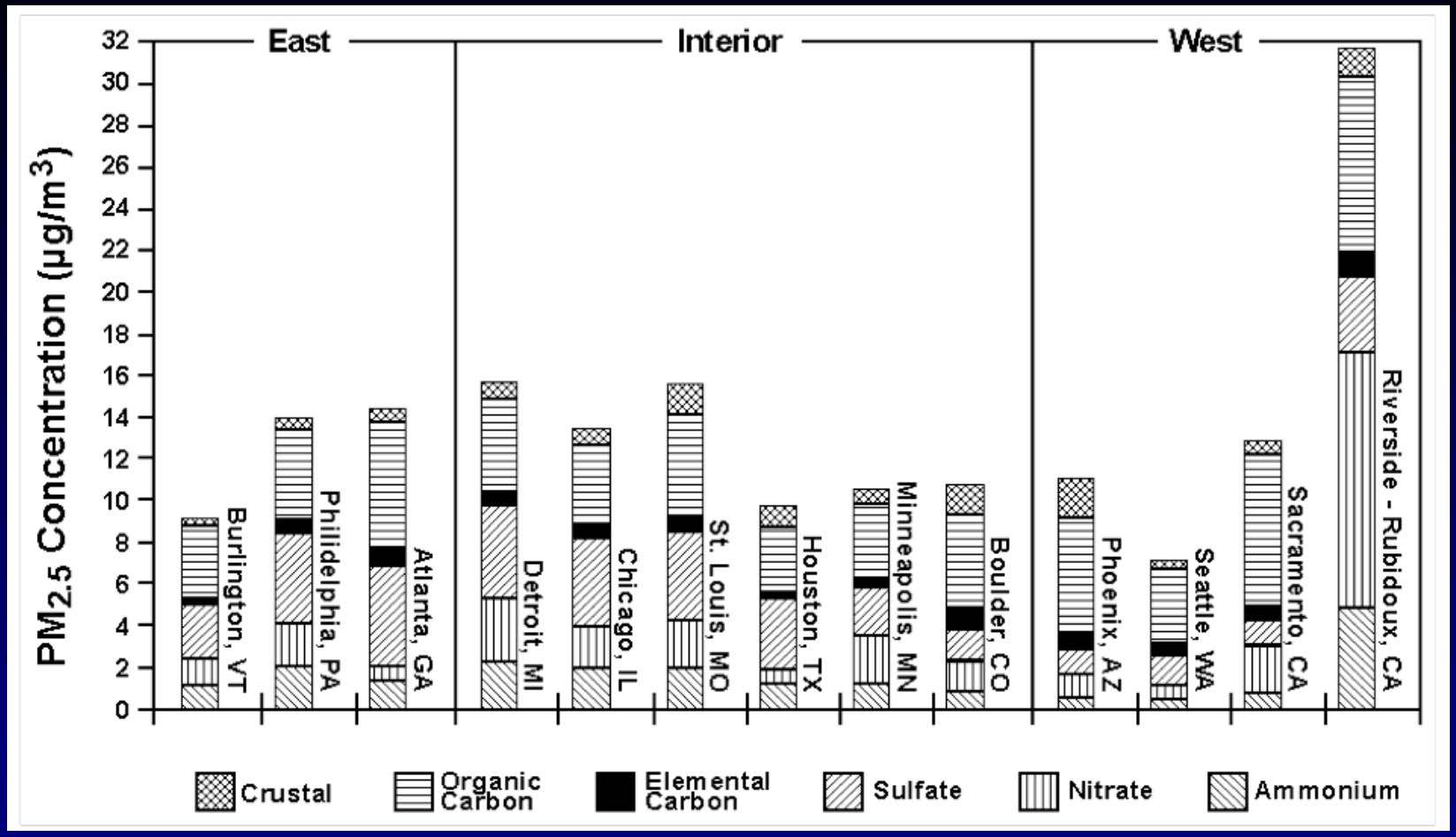
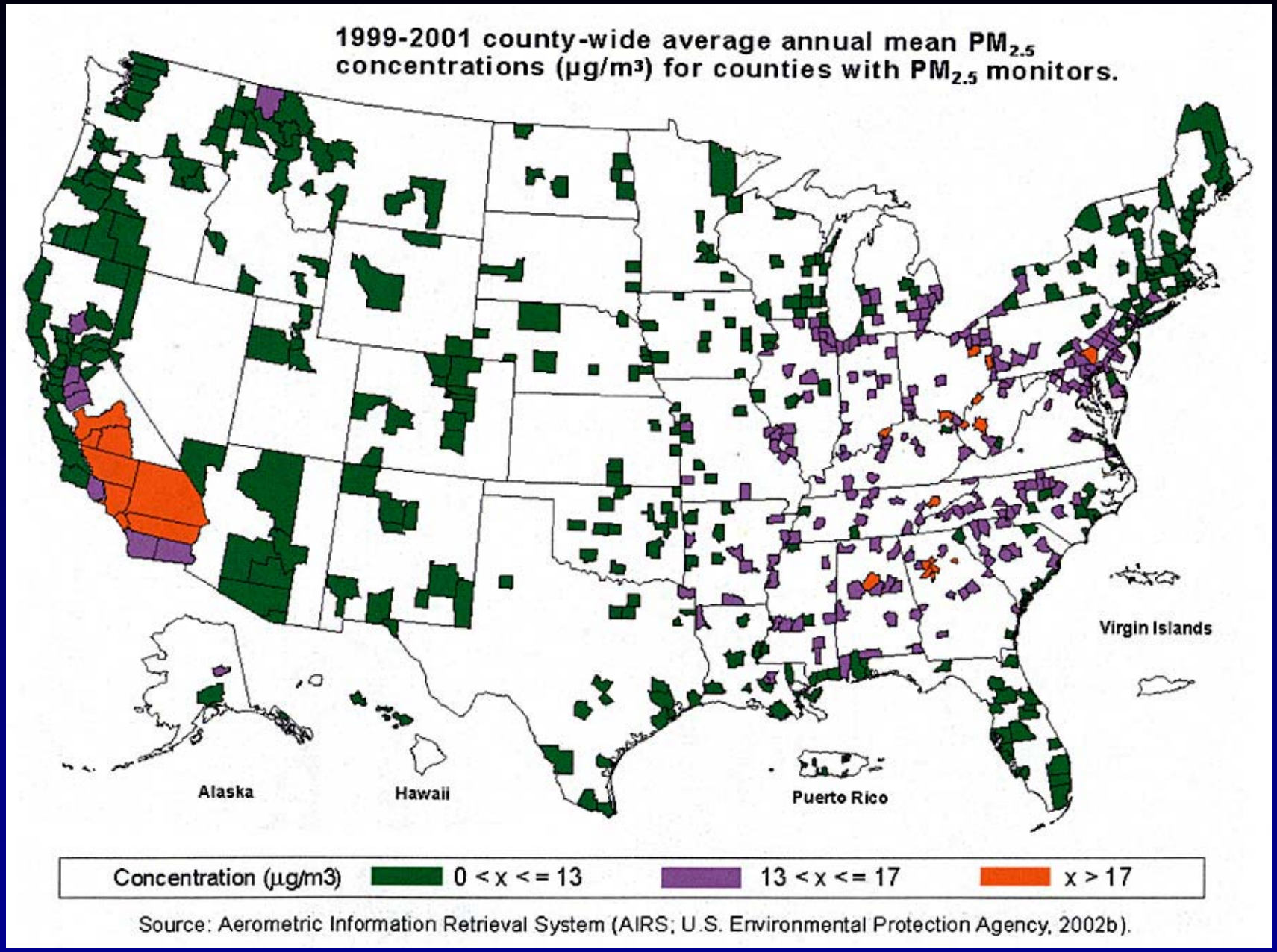
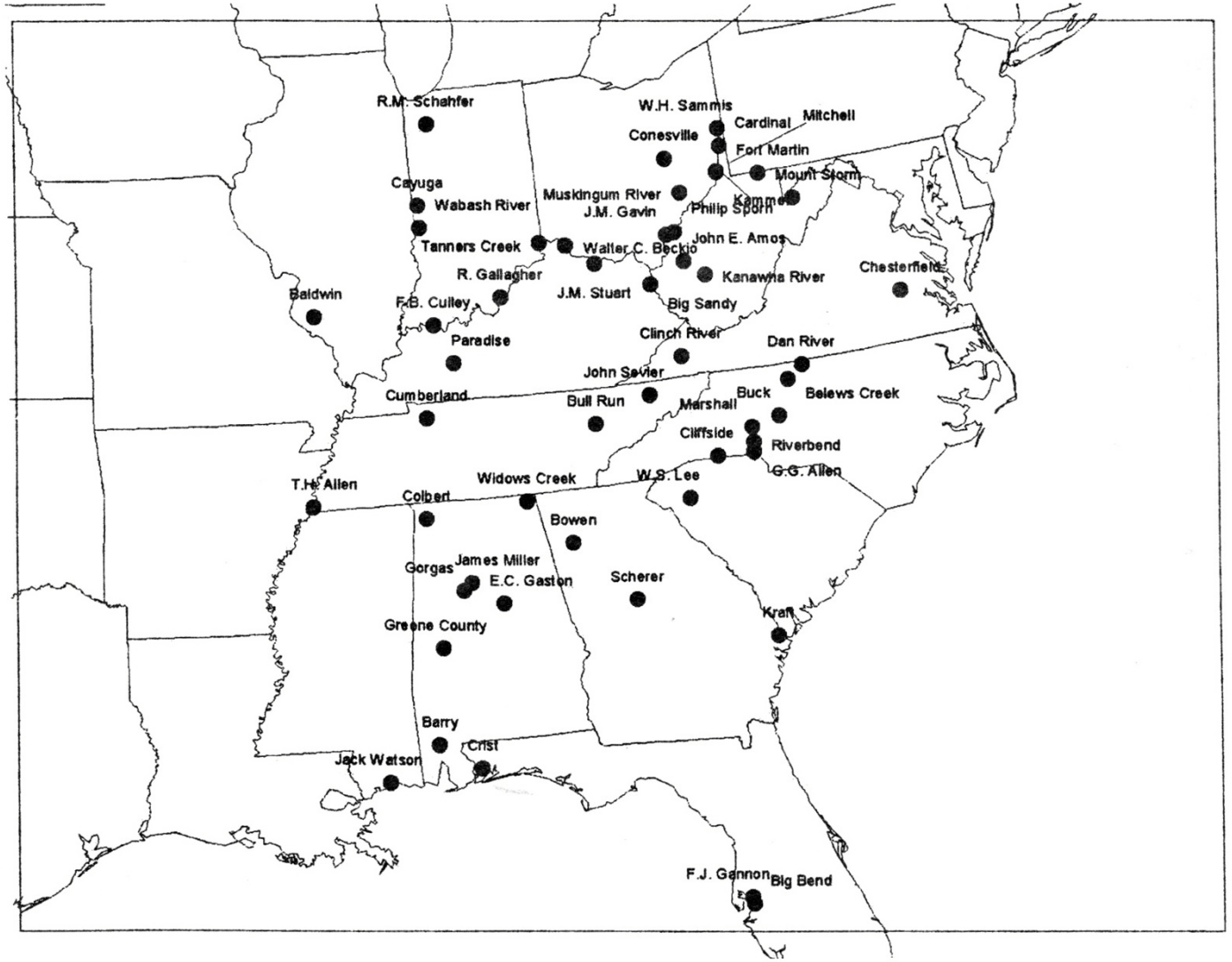


Figure 9 8. Major chemical components of $PM_{2.5}$ as determined in the U.S. Environmental Protection Agency's national speciation network from October 2001 to September 2002.

Source: 4th Draft PM Criteria Document, June 2003.



Source: Aerometric Information Retrieval System (AIRS; U.S. Environmental Protection Agency, 2002b).



Power plants charged with violations of the Clean Air Act's New Source Review provisions.

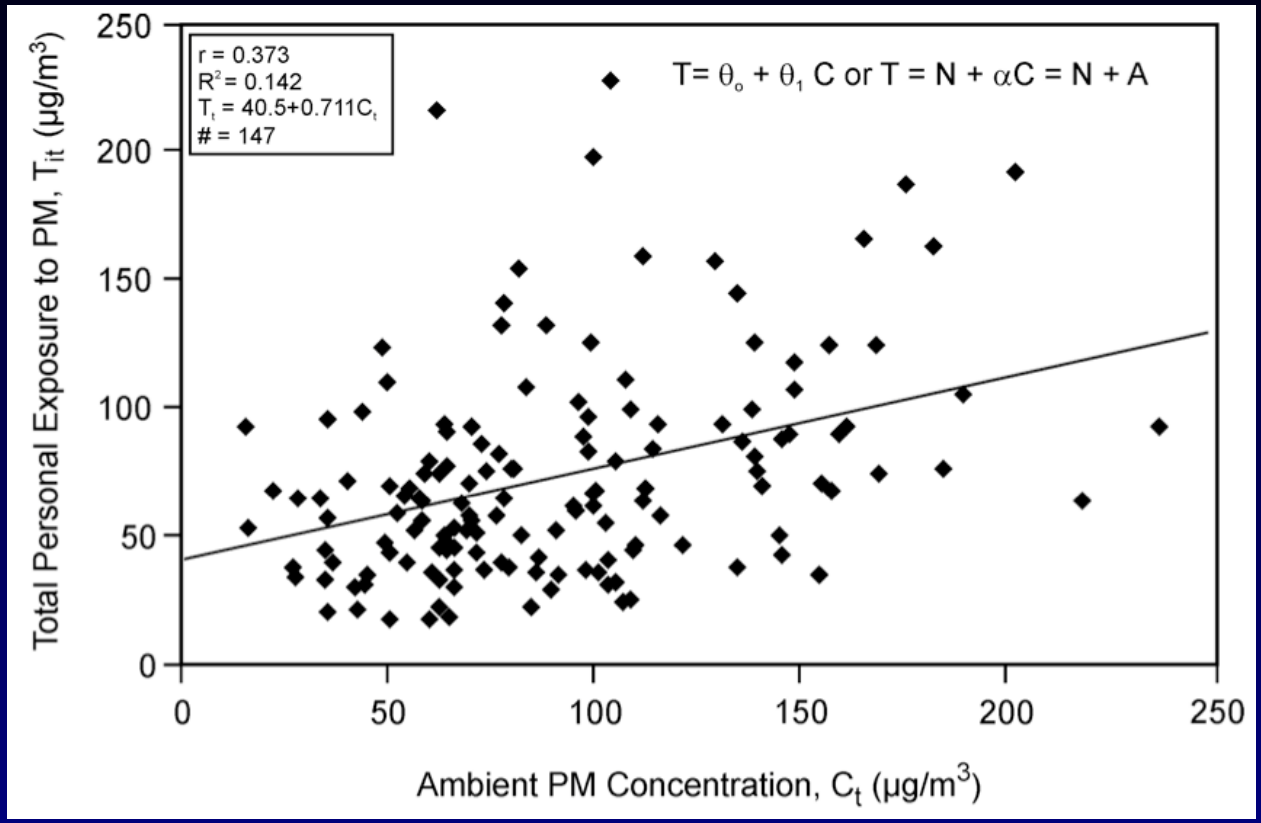


Figure 9-10. Regression analysis of daytime total personal exposures to PM_{10} versus ambient PM_{10} concentrations using data from the PTEAM study. The slope of the regression line is interpreted by exposure analysts as the average α, where $\alpha C = A$.

Source: 4th Draft PM Criteria Document, June 2003.

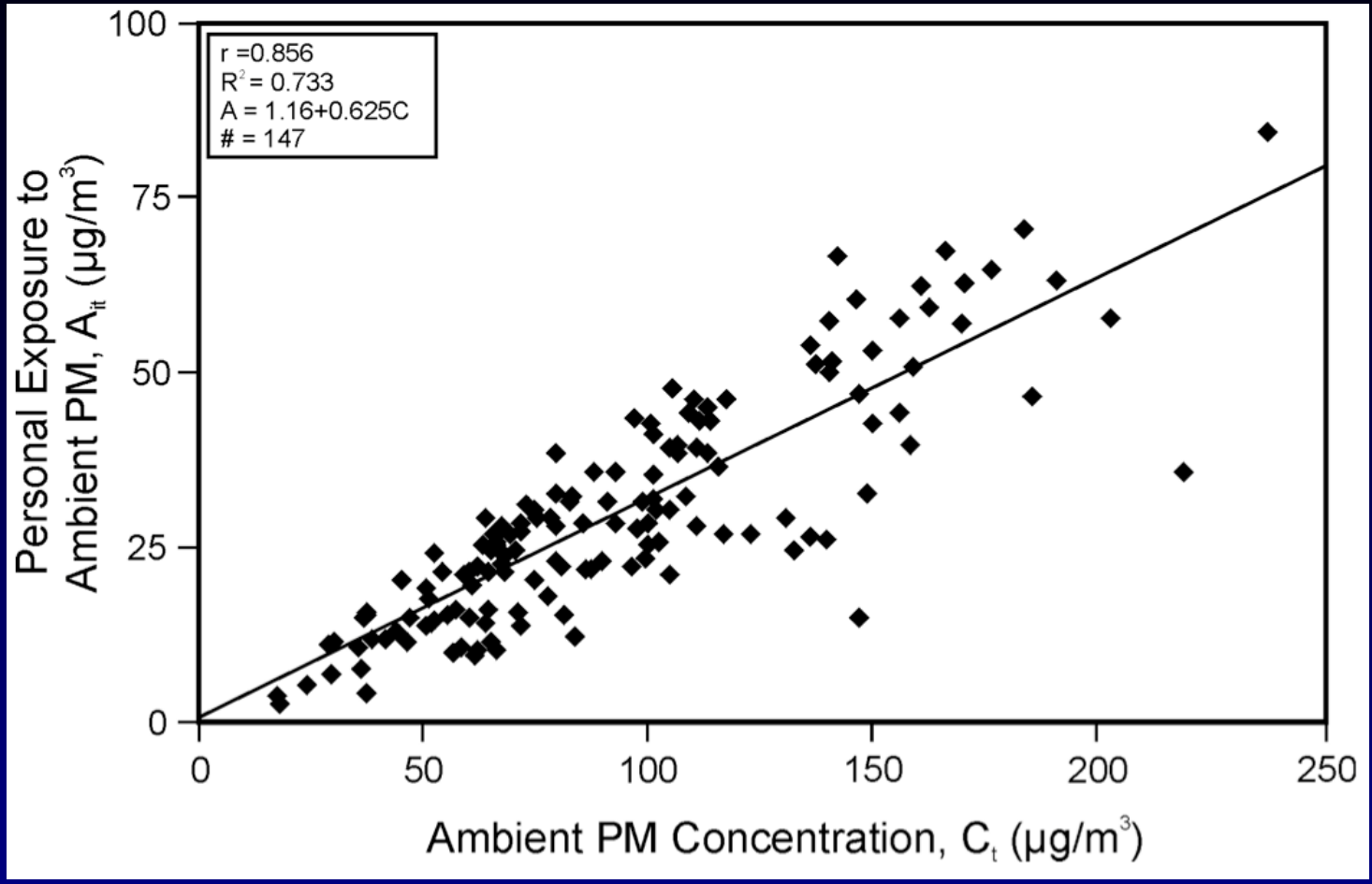


Figure 9 11. Regression analysis daytime exposures to the ambient component of personal exposure to PM_{10} (ambient exposure) versus ambient PM_{10} concentrations.

Source: 4th Draft PM Criteria Document, June 2003.

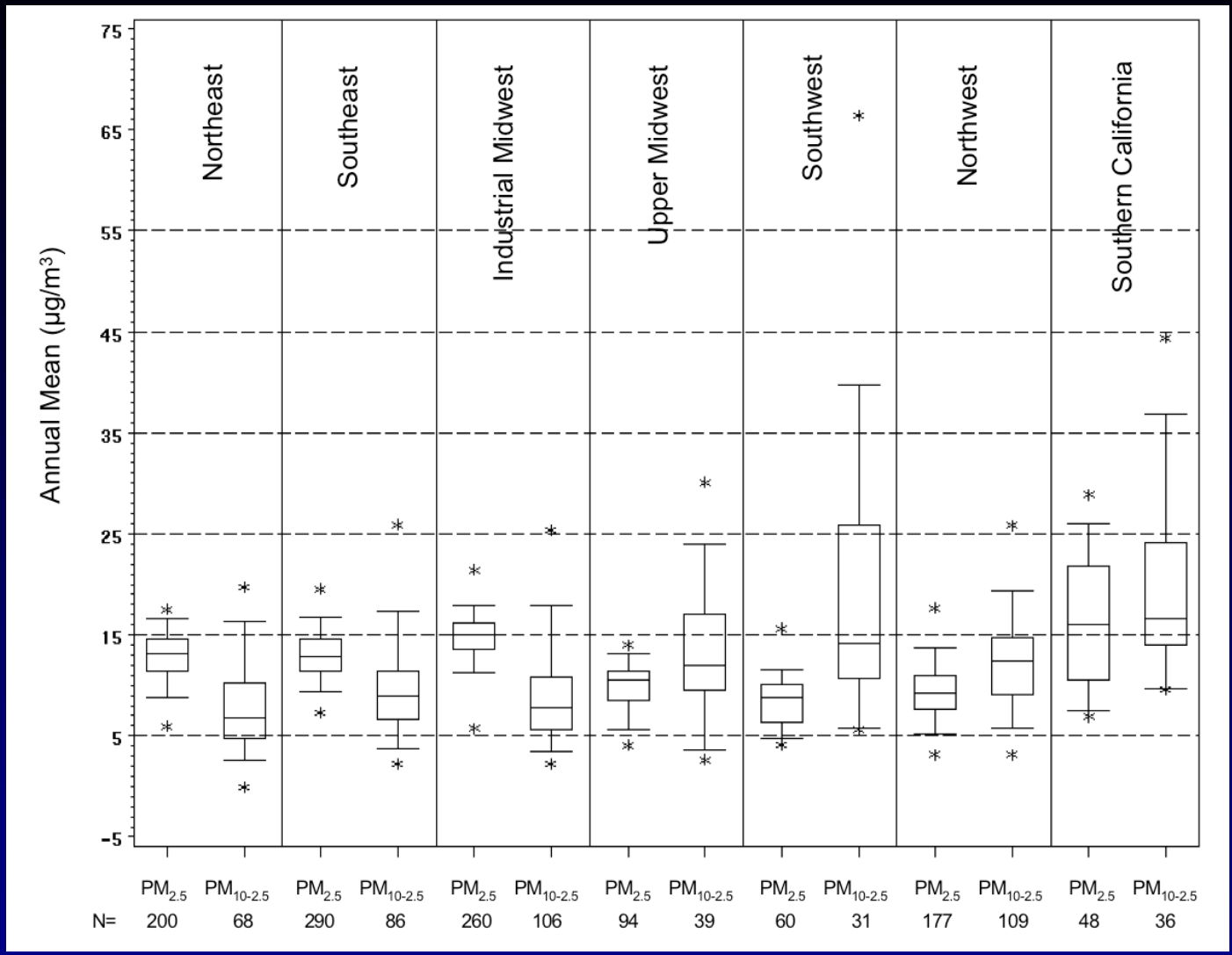


Figure 2-4. Distribution of annual mean $PM_{2.5}$ and estimated annual mean $PM_{10\ 2.5}$ concentrations by region, 2000-2002. Box depicts interquartile range and median; whiskers depict 5th and 95th percentiles; asterisks depict minimum and maximum. Number below indicates the number of sites in each region.

Source: 1st Draft PM Staff Paper, August 2003.

Fig 6-4 ICRP-1994 model for structure, function, epithelial cell types, and nomenclature of the human respiratory tract.

Functions	Cylology (Epithelium)	Histology (Walls)	Generation Number	Anatomy	Regions used in Model: New	Regions used in Model: Old*	Zones (Air)	Location	Airway Surface	Number of Airways
Air Conditioning; Temperature and Humidily, and Cleaning; Fast Particle Clearance; Air Conduction	Respiratory Epithelium with Goblet Cells: Cell Types: - Ciliated Cells - Nonciliated Cells: • Goblet Cells • Mucous (Secretory) Cells • Serous Cells • Brush Cells • Endocrine Cells • Basal Cells • Intermediate Cells	Mucous Membrane, Respiratory Epithelium (Pseudostratified, Ciliated, Mucous), Glands		Anterior Nasal Passages	ET_1				$2 \times 10^{-3} m^2$	—
		Mucous Membrane, Respiratory or Stratified Epithelium, Glands		Nose, Mouth, Pharynx, Posterior, Larynx, Esophagus	ET_2	LN_{ET} (N-P)	Conditioning	Extrathoracic	$4.5 \times 10^{-2} m^2$	—
		Mucous Membrane, Respiratory Epithelium, Cartilage Rings, Glands	0	Trachea	BB	(T-B)	Conditioning	Extrapulmonary	$3 \times 10^{-2} m^2$	511
			1	Main Bronchi						
		Mucous Membrane, Respiratory Epithelium, Cartilage plates, Smooth Muscle Layer, Glands	2 - 8	Bronchi						
	Respiratory Epithelium with Clara Cells (No Goblet Cells) Cell Types: - Ciliated Cells - Nonciliated Cells • Clara (Secretory) Cells	Mucous Membrane, Respiratory Epithelium, No Cartilage, No Glands, Smooth Muscle Layer	9 - 14	Bronchioles	bb		Conduction	Thoracic	$2.6 \times 10^{-1} m^2$	6.5×10^4
		Mucous Membrane, Single-Layer Respiratory Epithelium, Less Ciliated, Smooth Muscle Layer	15	Terminal Bronchioles		LN_{TH} †	$0.175 \times 10^{-3} m^3$ (Anatomical Dead Space)	Pulmonary		
Air Conduction; Gas Exchange; Slow Particle Clearance	Respiratory Epithelium Consisting Mainly of Clara Cells (Secretory) and Few Ciliated Cells	Mucous Membrane, Single-Layer Respiratory Epithelium of Cuboidal Cells, Smooth Muscle Layers	16 - 18	Respiratory Bronchioles			Gas-Exchange Transitory		$7.5 m^2$	4.6×10^5
Gas Exchange; Very Slow Particle Clearance	Squamous Alveolar Epithelium Cells (Type I), Covering 93% of Alveolar Surface Areas	Wall Consists of Alveolar Entrance Rings, Squamous Epithelial Layer, Surfactant	**	Alveolar Ducts	AI	P	$0.2 \times 10^{-3} m^3$			
	Cuboidal Alveolar Epithelial Cells (Type II. Surfactant-Producing), Covering 7% of Alveolar Surface Area	Interalveolar Septa Covered by Squamous Epithelium, Containing Capillaries, Surfactant	**	Alveolar Sacs			$4.5 \times 10^{-3} m^3$		$140 m^2$	4.5×10^7
	Alveolar Macrophages			Lymphatics		L				

* Previous ICRP Model
** Unnumbered because of imprecise information
† Lymph nodes are located only in BB region but drain the bronchial and alveolar interstitial regions as well as the bronchial region.

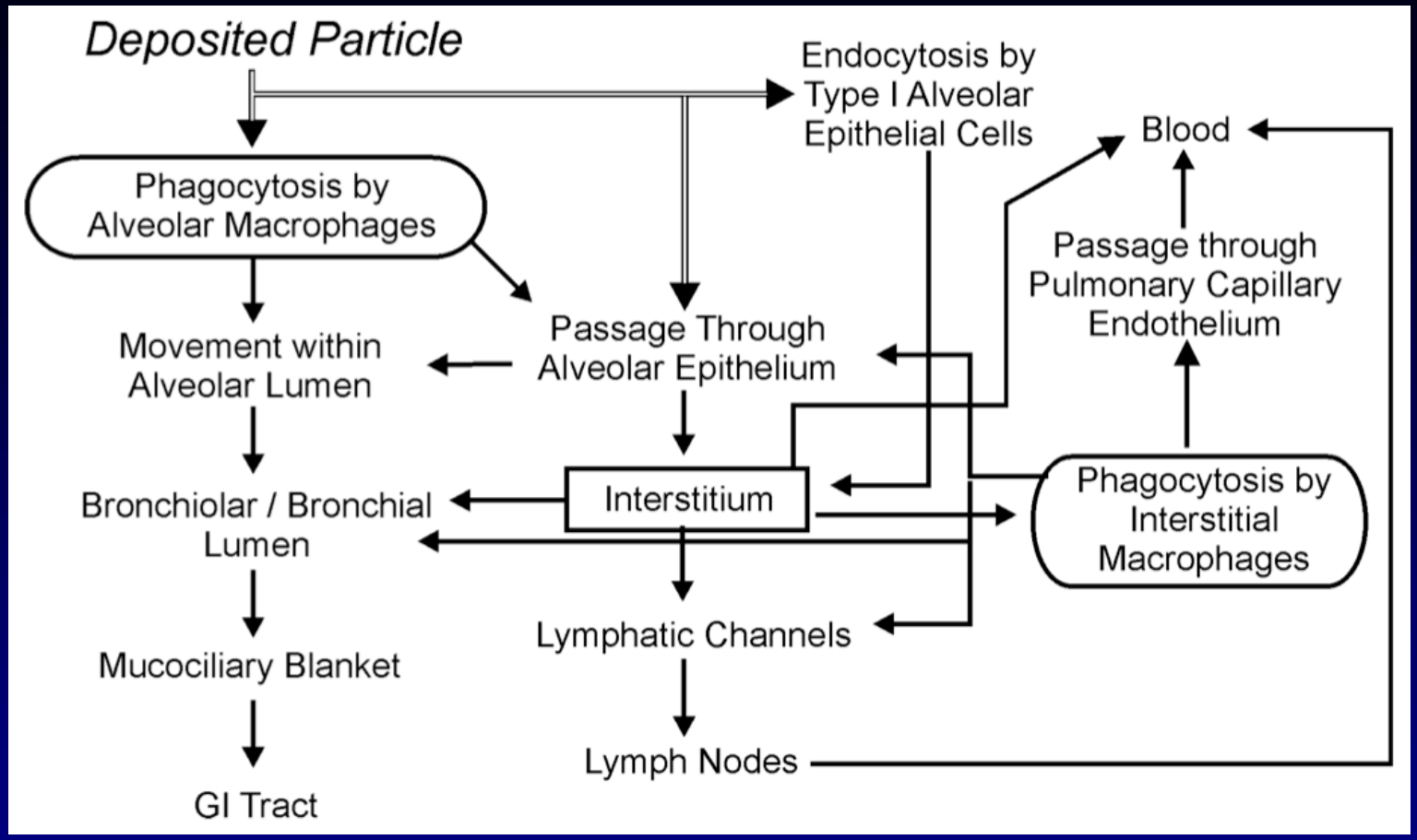


Figure 6-12. Diagram of known and suspected clearance pathways for poorly soluble particles depositing in the alveolar region. (The magnitude of various pathways may depend upon size of deposited particle.)

Source: 4th Draft PM Criteria Document, June 2003.

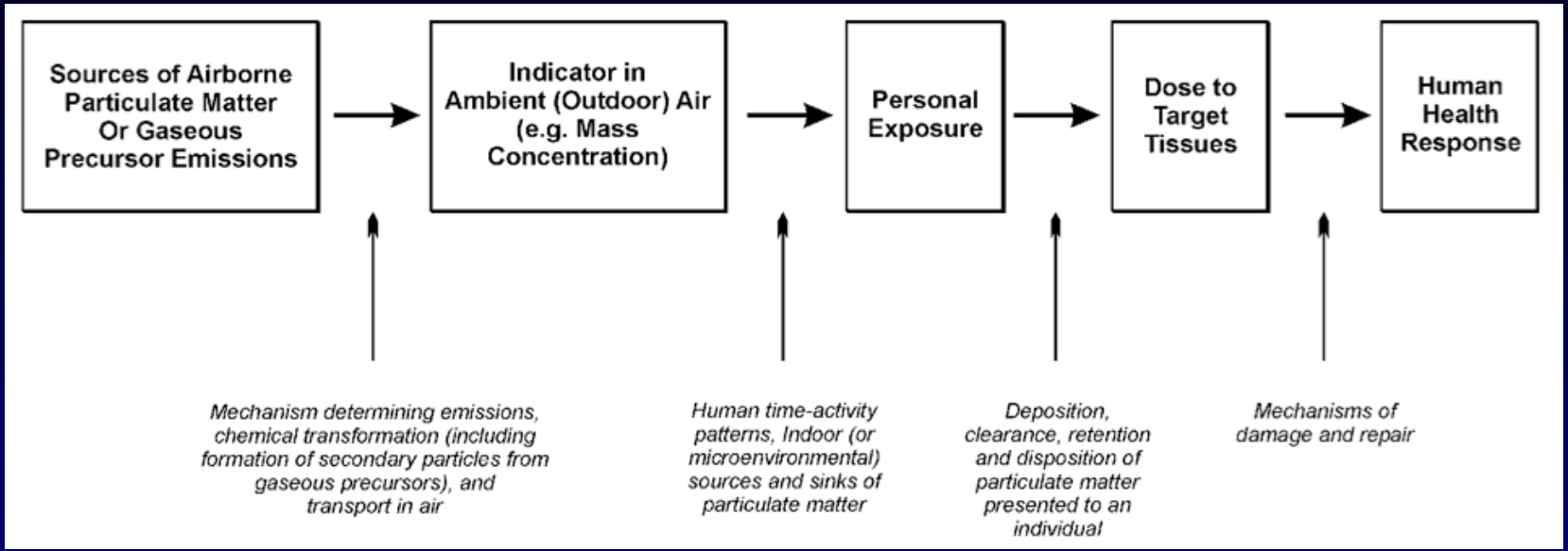


Figure 9 1. A general framework for integrating particulate-matter research. Note that this figure is not intended to represent a framework for research management. Such a framework would include multiple pathways for the flow of information.

Source: 4th Draft PM Criteria Document, June 2003.

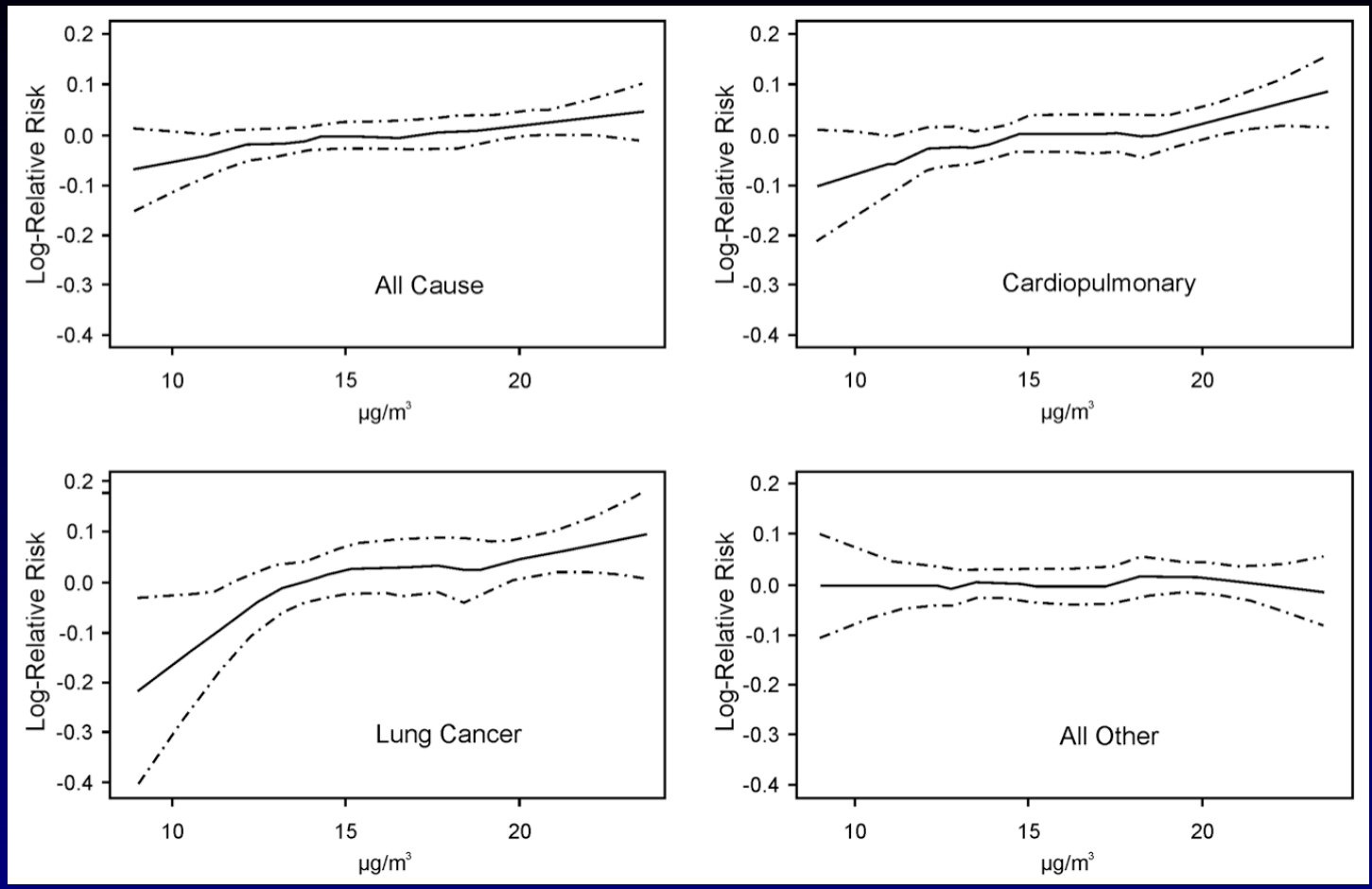


Figure 8-9. Natural logarithm of relative risk for total and cause-specific mortality per 10 µg/m^3 $PM_{2.5}$ (approximately the excess relative risk as a fraction), with smoothed concentration-response functions. Based on Pope et al. (2002) mean curve (solid line) with pointwise 95% confidence intervals (dashed lines).

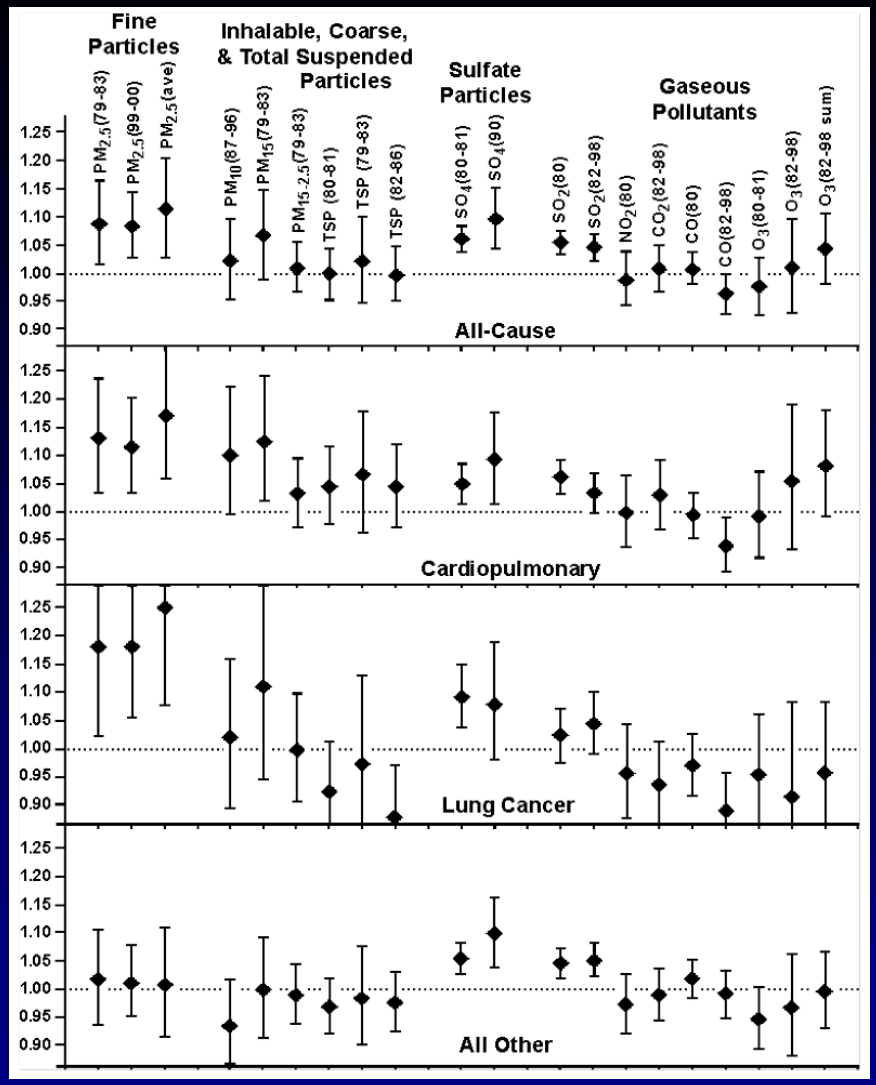
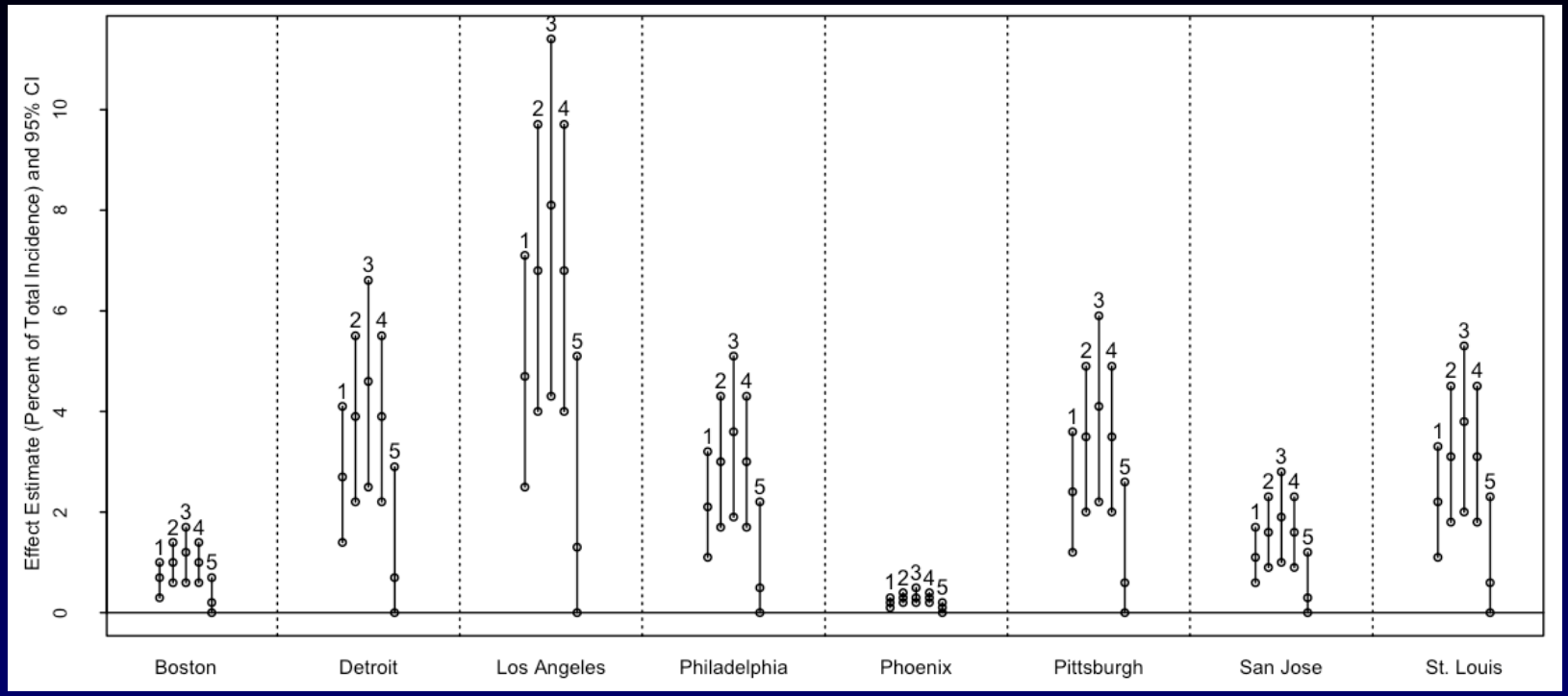


Figure 8-11. Relative risk of total and cause specific mortality for particle metrics and gaseous pollutants over different averaging periods (years 1979-2000 in parentheses).

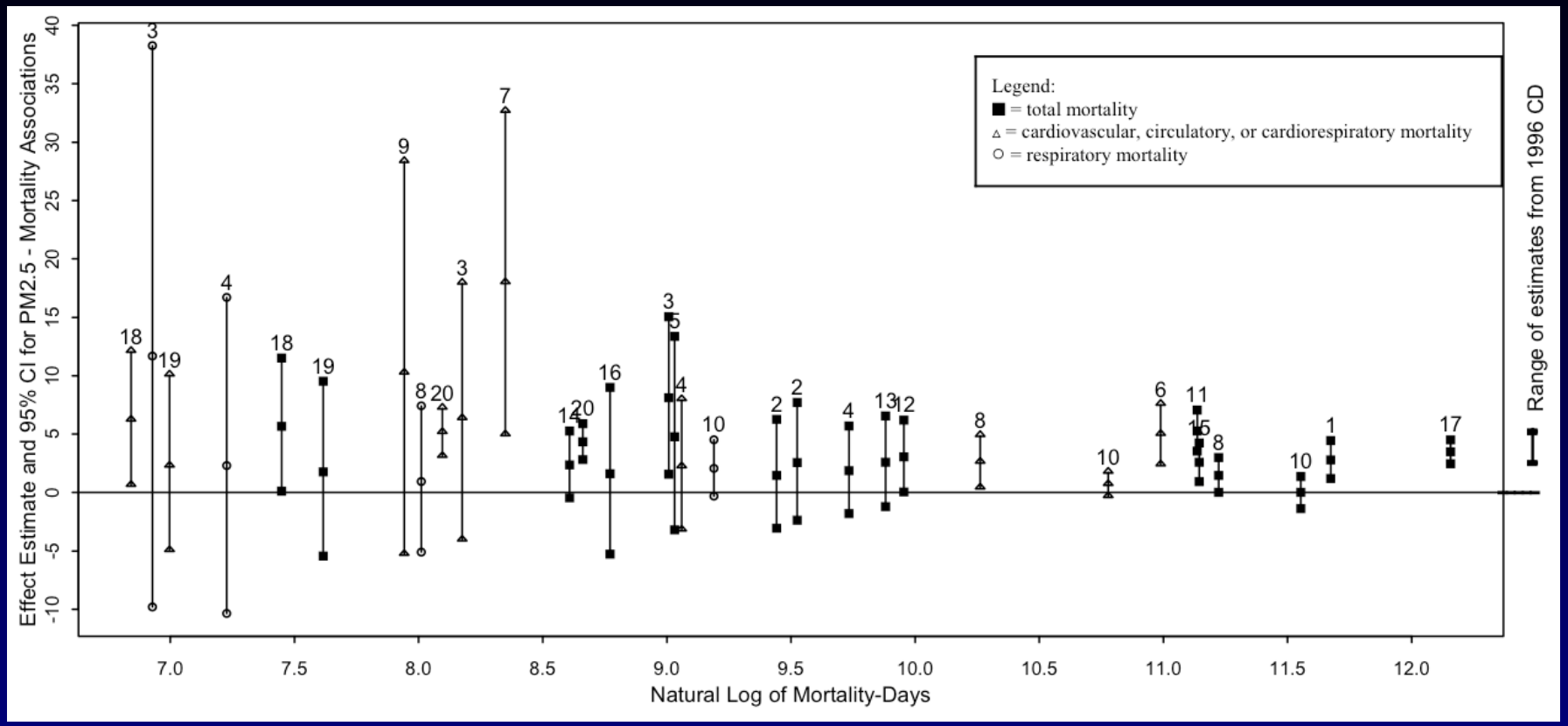
Source: 4th Draft PM Criteria Document, June 2003.



1. Krewski et al. (2000) – ACS
2. Krewski et al. (2000) – ACS – with CO
3. Krewski et al. (2000) – ACS – with NO2
4. Krewski et al. (2000) – ACS – with O3
5. Krewski et al. (2000) – ACS – with SO2

Figure 4-8. Estimated annual percent of mortality associated with long-term exposure to $PM_{2.5}$ (and 95% confidence interval): Single-pollutant and multi-pollutant models. (Single-pollutant models are always on the left, followed by the corresponding multi-pollutant models.)

Source: 1st Draft PM Staff Paper, August 2003.



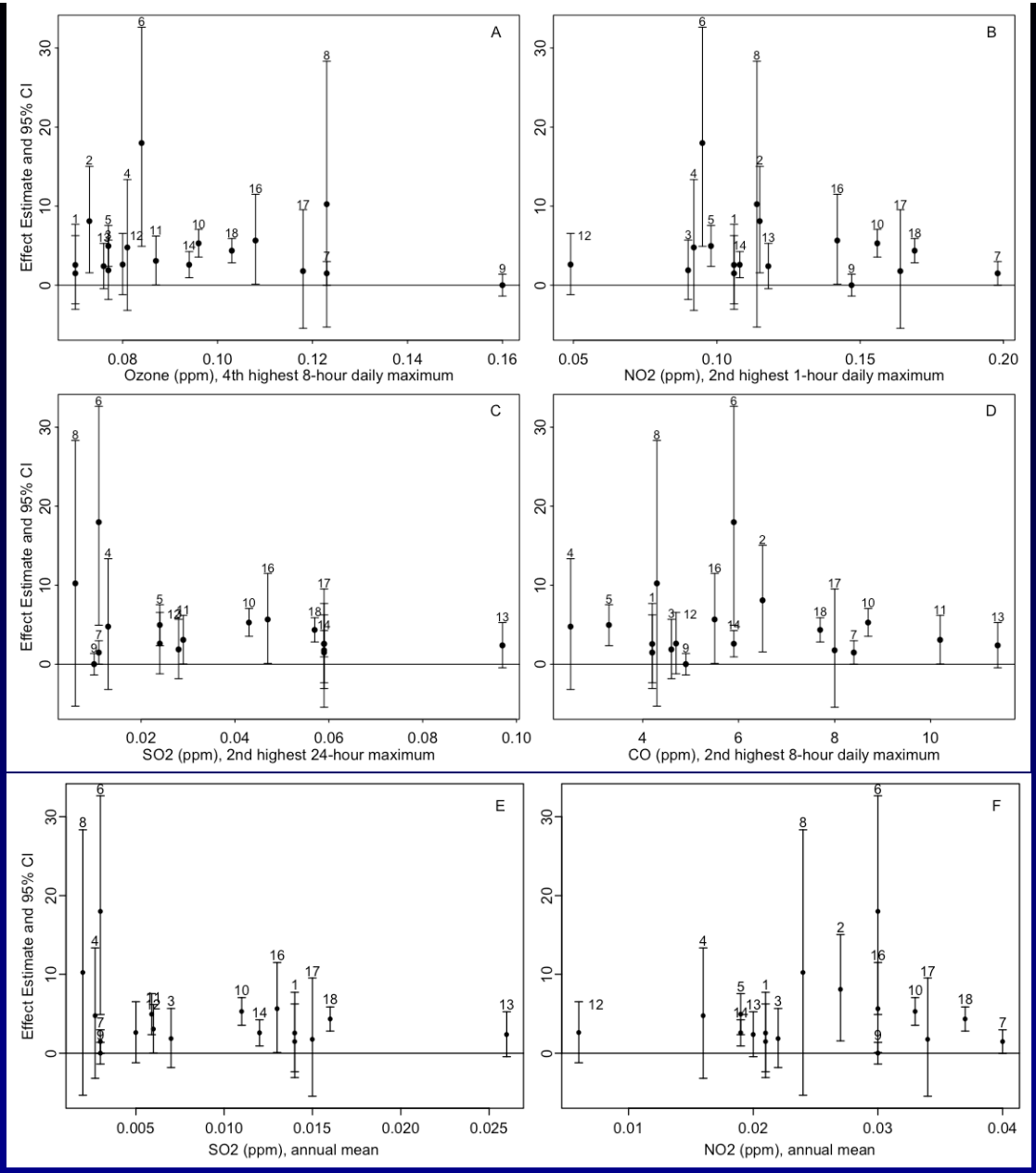
1. ★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Chock et al., 2000, Pittsburgh •
3. Fairley, 2003,, Santa Clara
4. Ito, 2003, Detroit
5. Klemm and Mason, 2000, Atlanta •
6. Lipfert et al., 2000a, Philadelphia •
7. Mar et al., 2003, Phoenix
8. Moolgavkar., 2003, LA
9. Ostro et al., 2003, Coachella Valley
10. Ostro et al., 1995, So. California •
11. Schwartz, 2003a, Boston
12. Schwartz, 2003a, Kingston/ Harriman
13. Schwartz, 2003a, Portage
14. Schwartz, 2003a, Steubenville
15. Schwartz, 2003a, St. Louis
16. Schwartz, 2003a, Topeka
17. ★ Schwartz, 2003a, Six Cities overall
18. Tsai et al., 2000, Camden NJ •
19. Tsai et al., 2000, Elizabeth NJ •
20. Tsai et al., 2000, Newark NJ •

Figure 3-5. Effect estimates for $PM_{2.5}$ and mortality from total, respiratory and cardiovascular causes from U.S. and Canadian cities in relation to the mortality-days product (the product of study days and the number of deaths per day - an indicator of study precision). Study locations are identified below; multi-city studies denoted by a star. Results of GAM stringent reanalyses; studies not originally using GAM denoted by •.

Source: 1st Draft PM Staff Paper, August 2003.

Figure 3-12. Associations between $PM_{2.5}$ and total mortality from U.S. studies, plotted against gaseous pollutant concentrations from the same locations. Air quality data obtained from the Aerometric Information Retrieval System (AIRS) for each study time period: (A) mean of 4th highest 8-hour ozone concentration; (B) mean of 2nd highest 1-hour NO_2 concentration; (C) mean of 2nd highest 24-hour SO_2 concentration; (D) mean of 2nd highest 8-hour CO concentration; (E) annual mean SO_2 concentration; (F) annual mean NO_2 concentration. Study locations are identified below.

Source: 1st Draft PM Staff Paper, August 2003.



1. Chock et al., 2000, Pittsburgh
2. Fairley, 2003, Santa Clara
3. Ito, 2003, Detroit
4. Klemm and Mason, 2000, Atlanta
5. Lipfert et al., 2000a
6. Mar et al., 2003, Phoenix
7. Moolgavkar, 2003
8. Ostro et al., 2003, Coachella Valley
9. Ostro et al., 1995, So. California
10. Schwartz, 2003a, Boston
11. Schwartz, 2003a, Knoxville
12. Schwartz, 2003a, Portage
13. Schwartz, 2003a, St. Louis
14. Schwartz, 2003a, Steubenville
15. Schwartz, 2003a, Topeka
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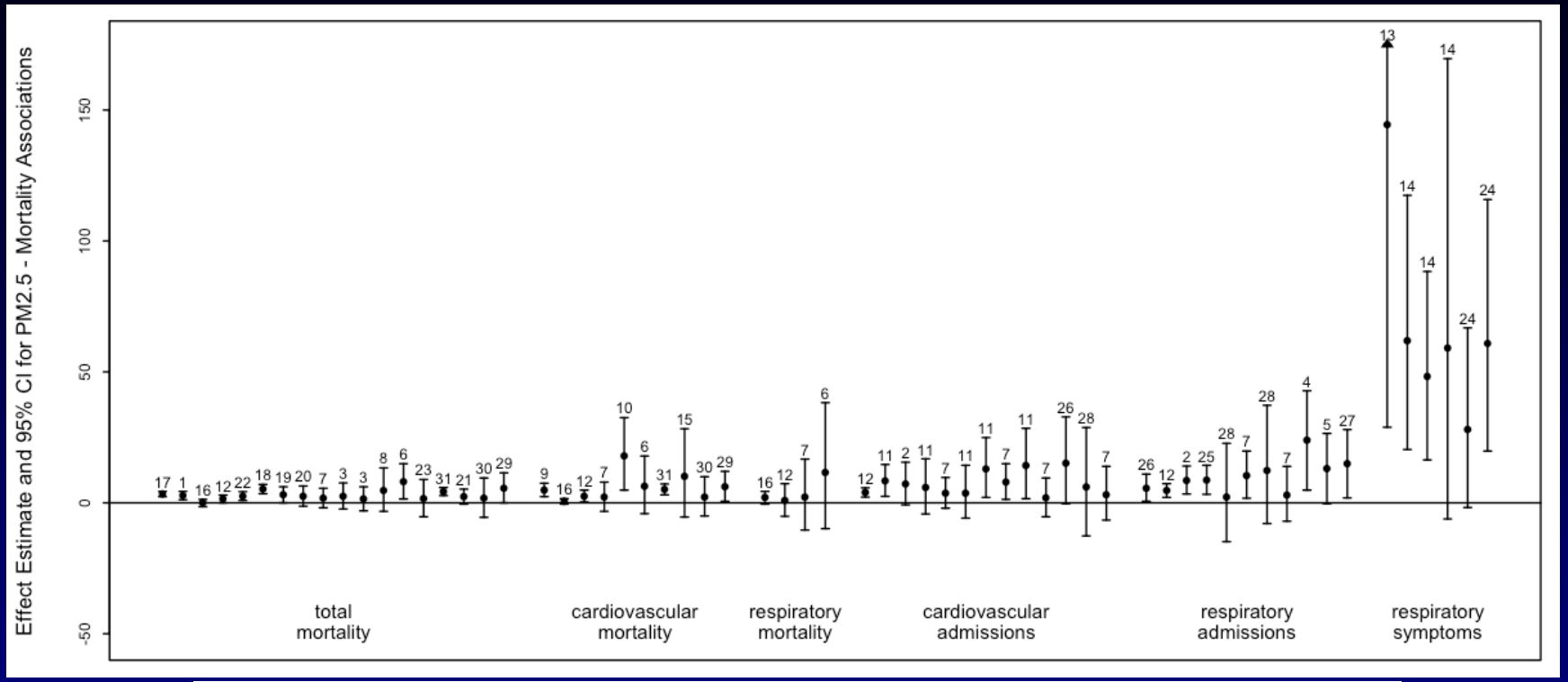
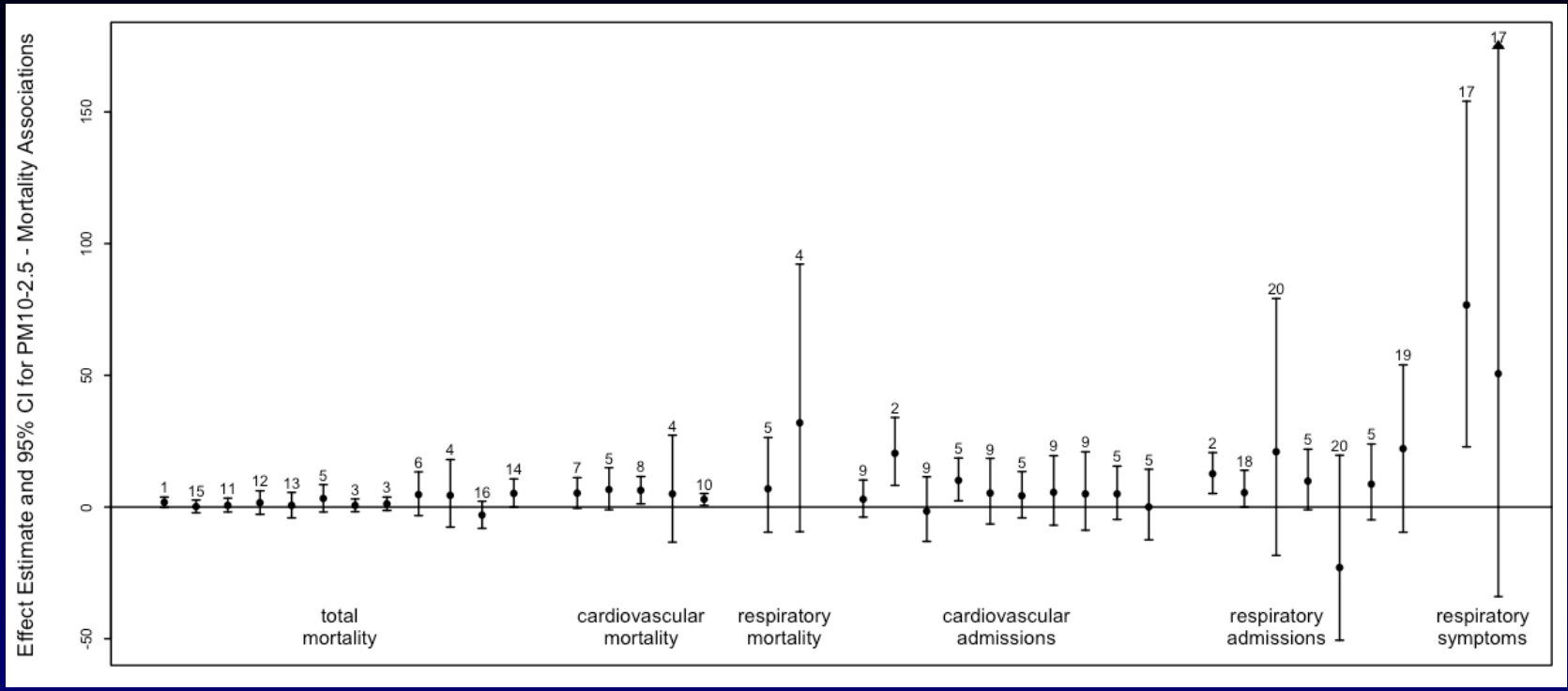
17. Tsai et al., 2000, Elizabeth NJ
18. Tsai et al., 2000, Newark NJ



1.★ Burnett and Goldberg, 2003, 8 Canadian cities
2. Burnett et al., 1997, Toronto •
3. Chock et al., 2000, Pittsburgh •
4. Delfino et al., 1997, Montreal •
5. Delfino et al., 1998, Montreal •
6. Fairley, 2003,, Santa Clara
7. Ito, 2003, Detroit
8. Klemm and Mason, 2000, Atlanta
7. Lipfert et al., 2000a, Philadelphia •
10. Mar et al., 2003, Phoenix
11. Metzger et al., 2003 •
12. Moolgavkar, 2003, Los Angeles Co.
13. Neas et al., 1995, Uniontown •
14. Neas et al., 1996, State College •
15. Ostro et al., 2003, Coachella Valley
16. Ostro et al., 1995, So. California •
17. ★ Schwartz 2003a, 6 cities overall
18. Schwartz 2003a, Boston
19. Schwartz 2003a, Kingston/Harriman
20. Schwartz 2003a, Portage
21. Schwartz 2003a, Steubenville
22. Schwartz 2003a, St. Louis
23. Schwartz 2003a, Topeka
24. ★ Schwartz and Neas, 2000, 6 cities •
25. Sheppard, 2003, Seattle
26. Stieb et al., 2000, St. John •
27. Thurston et al., 1994, Toronto •
28. Tolbert et al., 2000a, Atlanta •
29. Tsai et al., 2000, Newark •
30. Tsai et al., 2000, Elizabeth •
31. Tsai et al., 2000, Camden •

Figure 3-11a. Estimated excess mortality and morbidity risks per 25 μg/m^3 $PM_{2.5}$ from U.S. and Canadian studies (above). Results of GAM stringent reanalyses; studies not originally using GAM denoted by •. Multi-city studies denoted by a star.

Source: 1st Draft PM Staff Paper, August 2003.



1.★ Burnett and Goldberg, 2003, 8 Canadian cities
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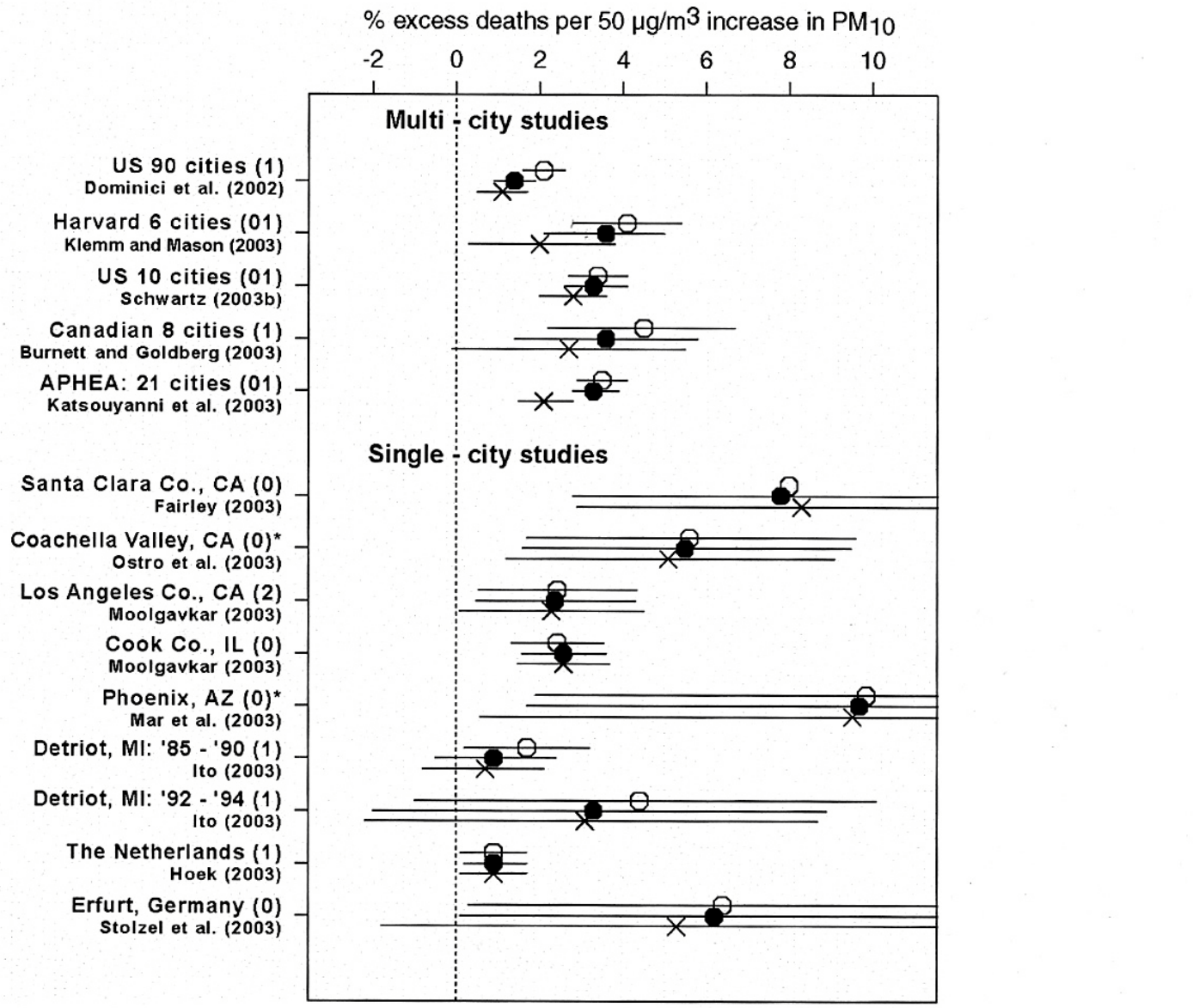
8. Mar et al., 2003, Phoenix
9. Metzger et al., 2003 •
10. Ostro et al., 2003, Coachella Valley
11. Schwartz 2003a, Boston
12. Schwartz 2003a, Kingston/Harriman
13. Schwartz 2003a, Portage
14. Schwartz 2003a, Steubenville
15. Schwartz 2003a, St. Louis
16. Schwartz 2003a, Topeka
17. ★ Schwartz and Neas, 2000, 6 cities •
18. Sheppard, 2003, Seattle
19. Thurston et al., 1994, Toronto •
20. Tolbert et al., 2000a, Atlanta •

Figure 3-11b. Estimated excess mortality and morbidity risks per 25 μg/m^3 $PM_{10-2.5}$ from U.S. and Canadian studies (above). Results of GAM stringent reanalyses; studies not originally using GAM denoted by •. Multi-city studies denoted by a star.

Source: 1st Draft PM Staff Paper, August 2003.



Reanalysis results for PM_{10} excess risk estimates for total non-accidental mortality for numerous locations (cardiovascular mortality for Phoenix & Coachella Valley), using: GAM with default convergence criteria (O); GAM stringent criteria (●); & GLM/natrual splines (X) ala original GAM model.

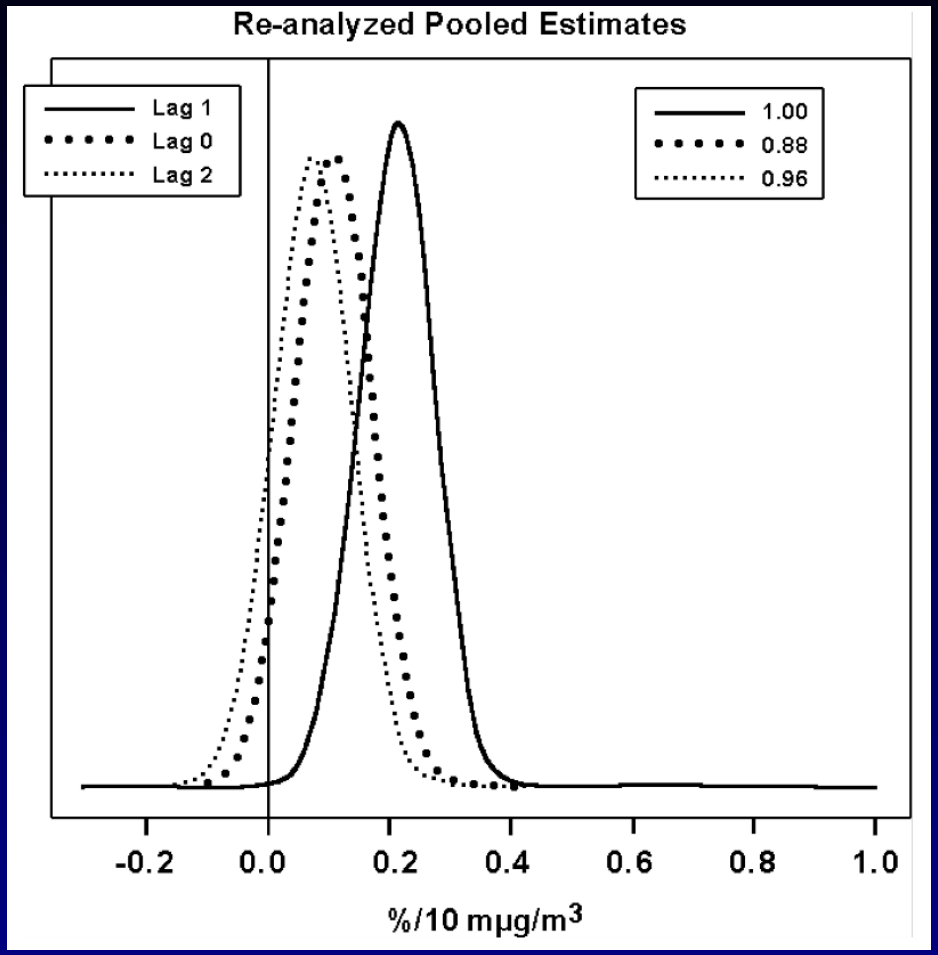


Figure 8-19. Marginal posterior distribution for effects of PM_{10} on all cause mortality at lag 0, 1, and 2 for the 90 cities. From Dominici et al. (2002a). The numbers in the upper right legend are posterior probabilities that overall effects are greater than 0.

Source: 4th Draft PM Criteria Document, June 2003.

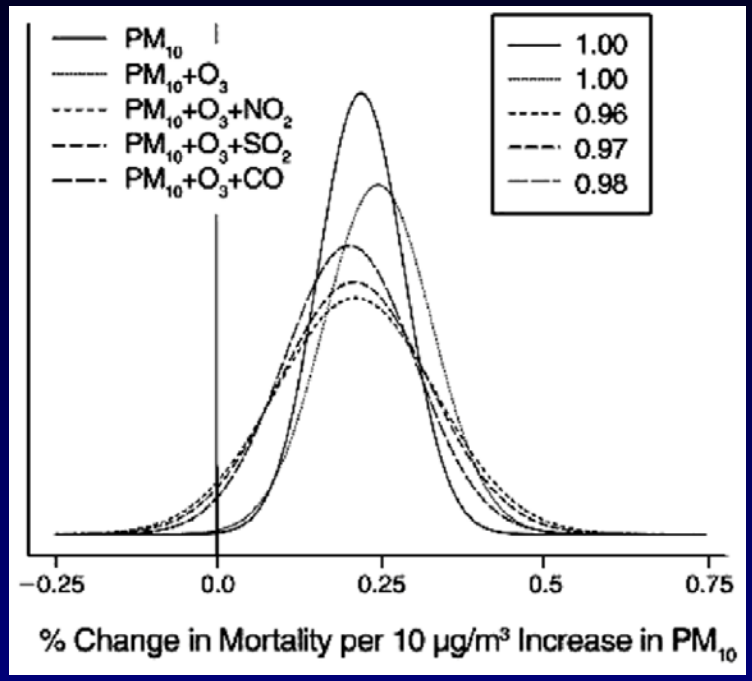
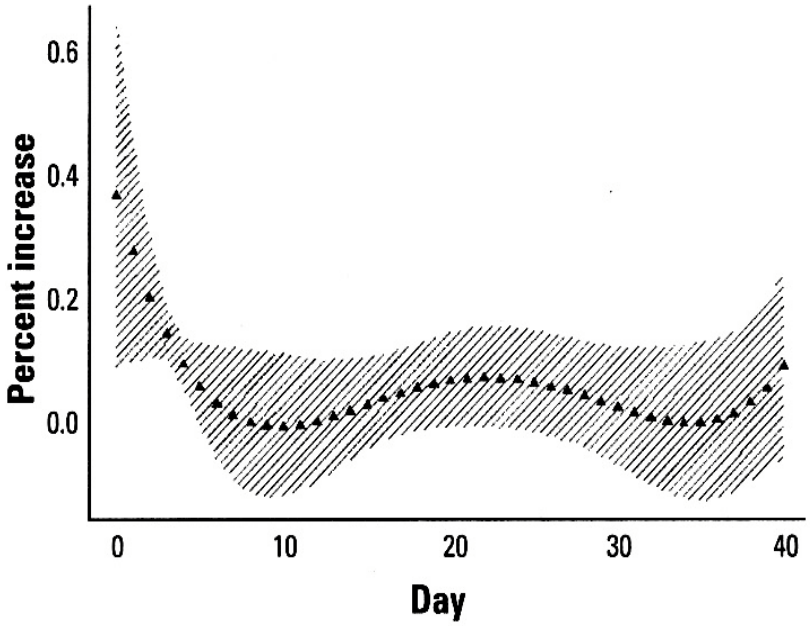
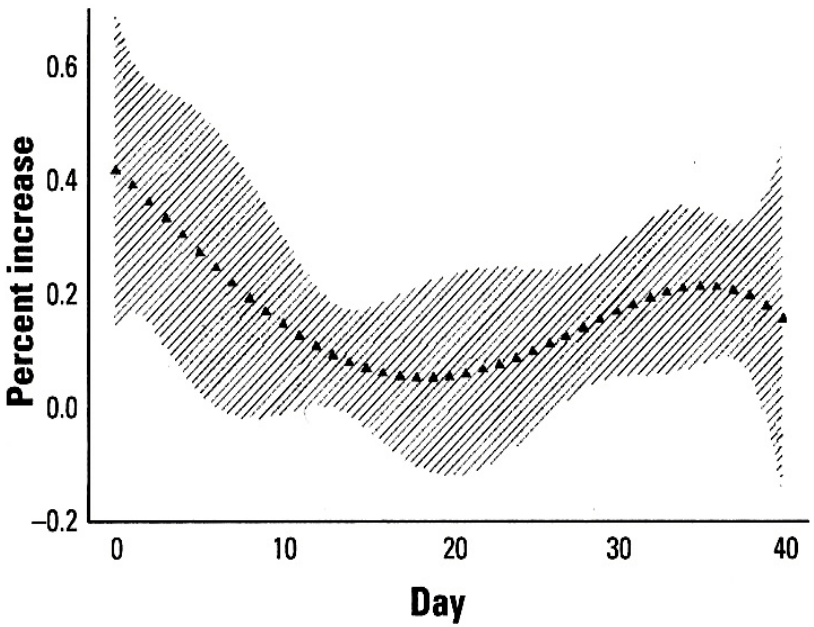


Figure 8-6. Marginal posterior distributions for effect of PM_{10} on total mortality at lag 1 with and without control for other pollutants, for the 90 cities. The numbers in the upper right legend are the posterior probabilities that the overall effects are greater than 0.

Source: 4th Draft PM Criteria Document, June 2003.



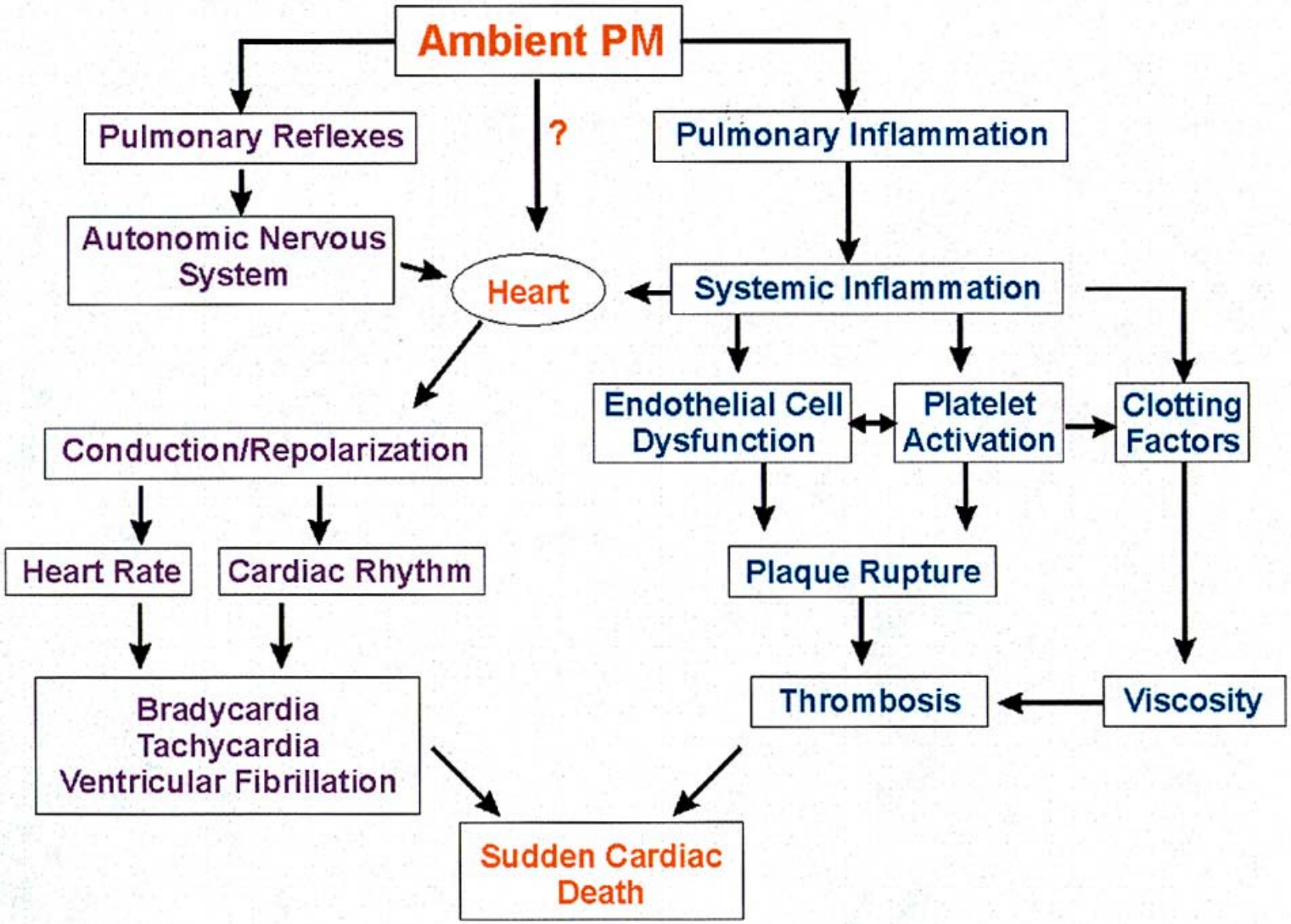
The estimated shape of the association of PM_{10} for each lag with daily deaths for CVD with a fourth-degree distributed lag model with random effect in 10 cities (percentage increase in deaths for a 10 $\mu g/m^3$ increase in PM_{10}). The shaded area represents the 95% CIs.



The estimated shape of the association of PM_{10} for each lag with daily deaths for respiratory disease with a fourth-degree distributed lag model with random effect in 10 cities (percentage increase in deaths for a 10 $\mu g/m^3$ increase in PM_{10}). The shaded area represents the 95% CIs.

From: Zanobetti, et al., Environ. Health Perspect. 111:1188-1193 (2003).

Mechanisms/Pathways for PM Toxicity



Schematic representation of potential pathophysiological pathways and mechanisms by which ambient PM may increase risk of cardiovasular morbidity and/or mortality.

Table 3-1. Summary of Potential Mechanisms Based on Emerging Toxicological Evidence

Effect	Potential Mechanisms
Direct Pulmonary Effects	Lung injury and inflammation
	Increased susceptibility to respiratory infections
	Increased airway reactivity and exacerbation of asthma
Systemic Effects Secondary to Lung Injury	Lung injury leading to impairment of heart function by lowering blood oxygen levels and increasing the work of breathing
	Lung inflamation and cytokine production leading to adverse systemic hemodynamic effects (e.g., arrhythmia)
	Lung inflammation leading to increased risk of heart attacks and strokes due to increased blood coagulability
	PM/lung interactions potentially affecting hematopoiesis (e.g., blood cell formation)
Direct Effects on the Heart	Uptake of particles and/or distribution of soluble components from the lungs into the systemic circulation
	Effects on the autonomic control of the heart and cardiovascular system

Source: 1st Draft PM Staff Paper, August 2003.

Chapter 7. Toxicology of Particulate Matter

Toxicological Evidence of Health Effects Related to Ambient PM

1. Utah Valley PM collected before, during, and after steel mill closure provided natural experiment; demonstrated coherence of PM effects in in-vitro, in animals, and in human clinical studies with epidemiologic results.
2. Health effects associated with PM constituents/sources (e.g., metals, sulfates, traffic, coal combustion).
3. Exposure of humans and animals to concentrated air particles (CAPs) result in physiological changes and dose-related impacts on cardiovascular functions.

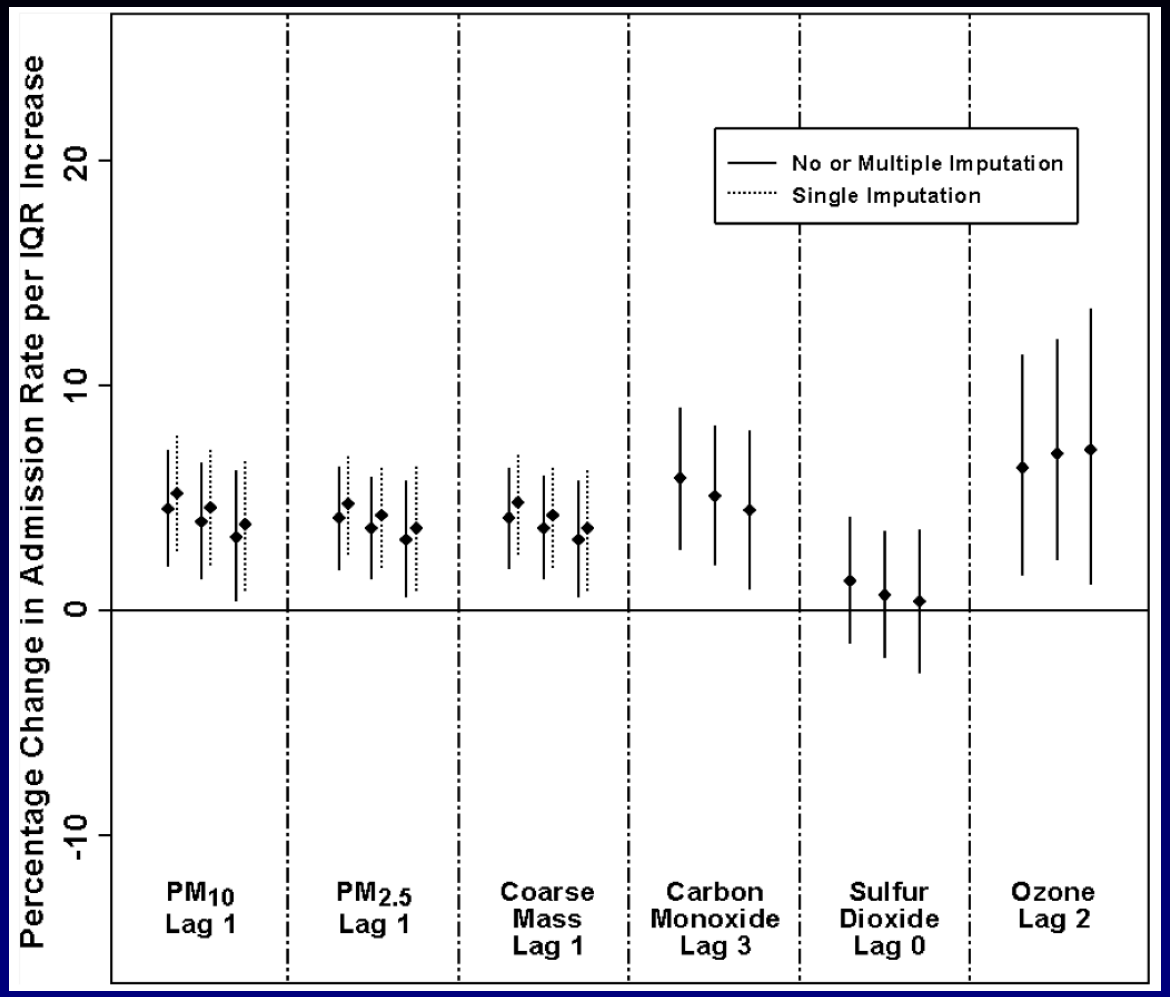


Figure 8-13. Percent change in hospital admission rates and 95% CIs for an IQR increase in pollutants from single-pollutant models for asthma. Poisson regression models are adjusted for time trends (64-df spline), day-of-week, and temperature (4-df spline). The IQR for each pollutant equals: 19 $\mu g/m^3$ for PM_{10}, 11.8 $\mu g/m^3$ for $PM_{2.5}$, 9.3 $\mu g/m^3$ for coarse PM, 20 ppb for O_3, 4.9 ppb for SO_2, and 924 ppb for CO. Triplets of estimates for each pollutant are for the original GAM analysis using smoothing splines, the revised GAM analysis with stricter convergence criteria, and the GLM analysis with natural splines. For pollutants that required imputation (i.e., estimation of missing value) estimates ignoring (single imputation) or adjusting for (multiple imputation) the imputation are shown.

Source: 4th Draft PM Criteria Document, June 2003.

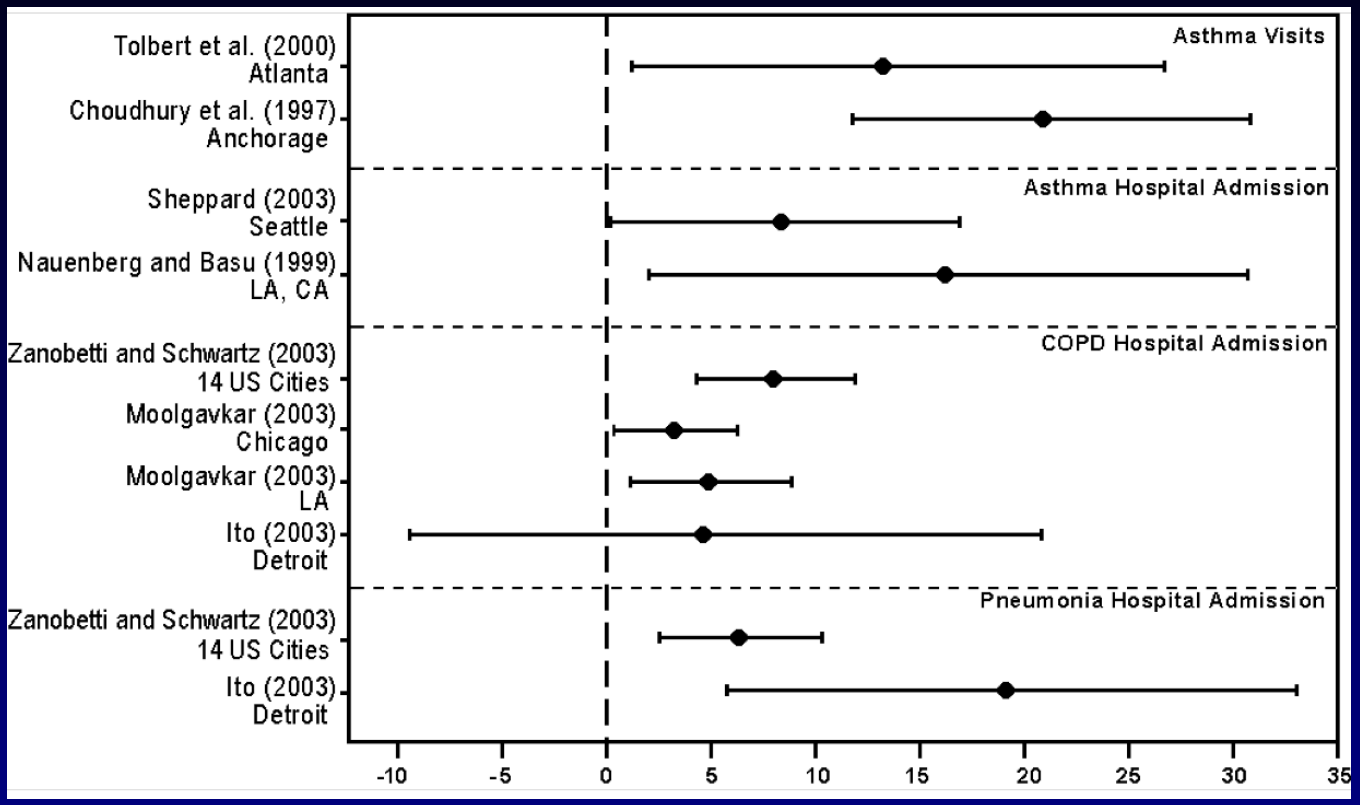


Figure 8-14. Maximum excess risk of respiratory-related hospital admissions and visits per 50 $\mu g/m^3$ PM_{10} increment in selected studies of U.S. cities based on single pollutant models.

Source: 4th Draft PM Criteria Document, June 2003.

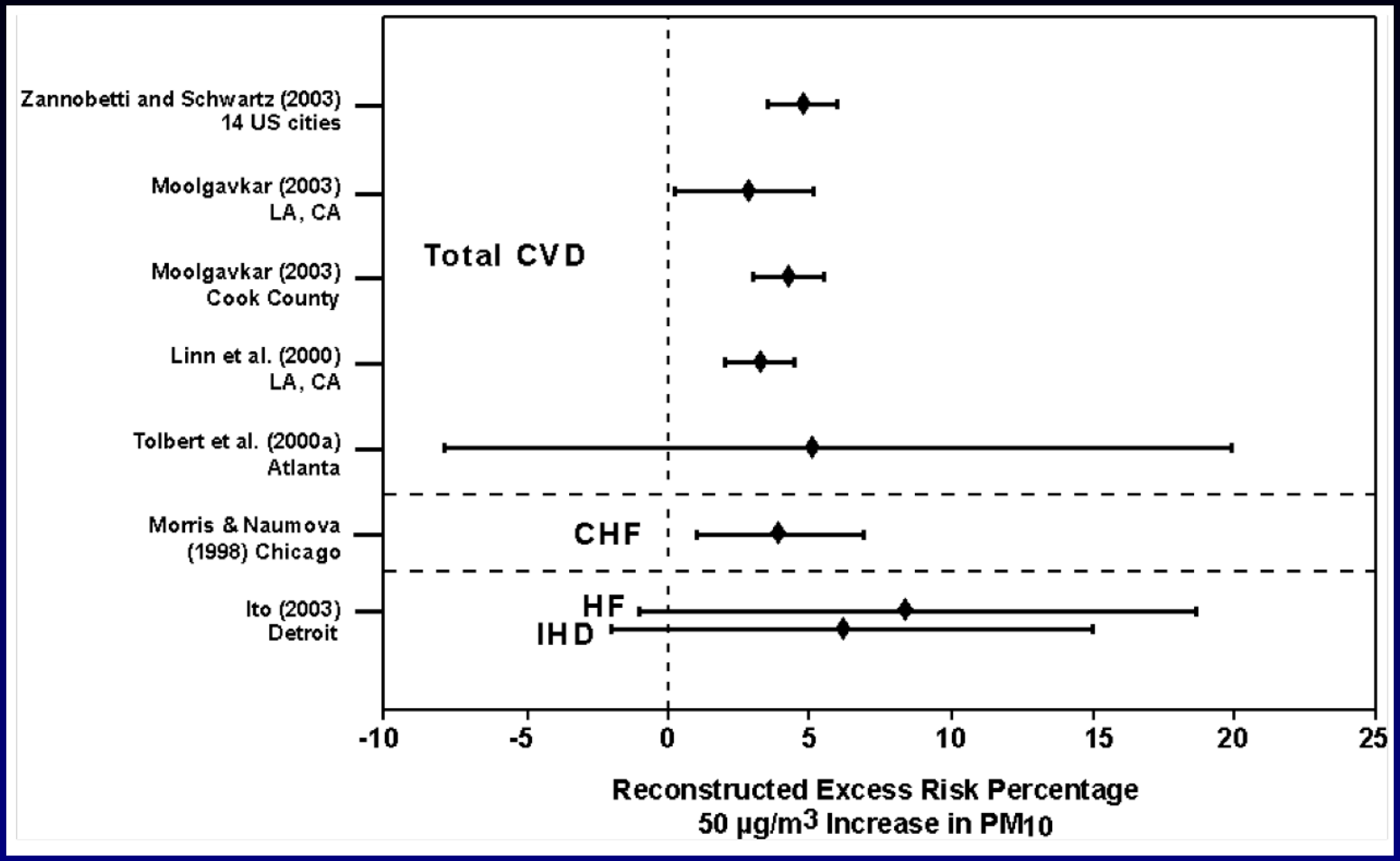


Figure 9-18. Acute cardiovascular hospitalizations and PM exposure excess risk estimates derived from selected U.S. PM_{10} studies. CVD = cardiovascular disease and CHF = congestive heart failure. IHD = ischemic heart disease.

Source: 4th Draft PM Criteria Document, June 2003.

Associations Between Pollutants and Respiratory Health Outcomes from the Children's Health Study

Respiratory Health Outcome	Associated Pollutants*	Reference
Slowed Lung Growth	NO_2, PM_{10}, $PM_{2.5}$, HNO_3	Gauderman et al., 2000; 2002 Avol et al., 2001
Asthma Causation	O_3	McConnell et al., 2002
Asthma Exacerbation	NO_2, PM_{10}	McConnell et al., 1999
Acute Respiratory Illness	O_3	Gilliland et al., 2001

*These were the main pollutants provided in the cited analyses. Pollutants were usually highly correlated, thus, effects may be due to mixtures.

Unresolved Problems in Characterizing Health Effects of Ambient Air Pollution

- lack of demonstrated biological mechanisms for PM-related effects,
- potential influence of measurement error and exposure error,
- potential confounding by copollutants,
- evaluation of the effects of components, surface coatings or other characteristics of PM,
- the shape of concentration-response relationships,
- methodological uncertainties in epidemiological analyses,
- the extent of life span shortening,
- characterization of annual and daily background concentrations,
- understanding of the effects of coarse fraction PM, and
- effects, if any, of air toxics.