

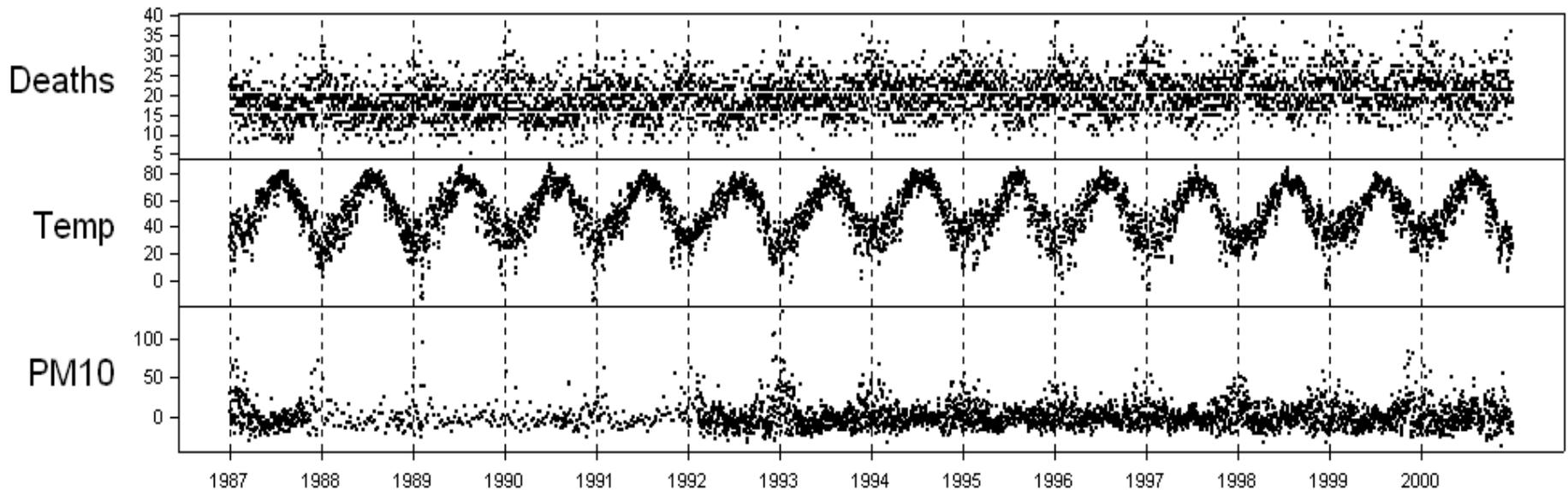
# *Particulate Matter (PM): Adverse Health Effects*

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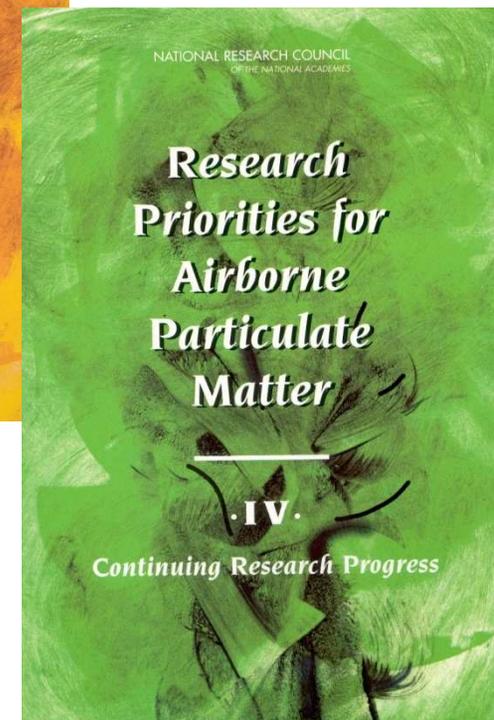
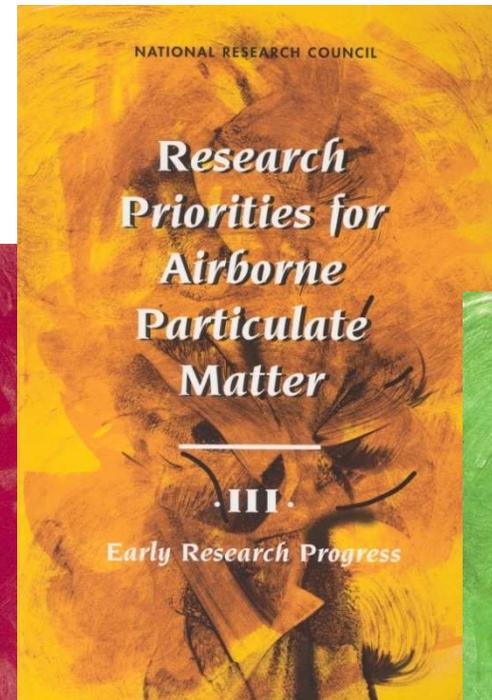
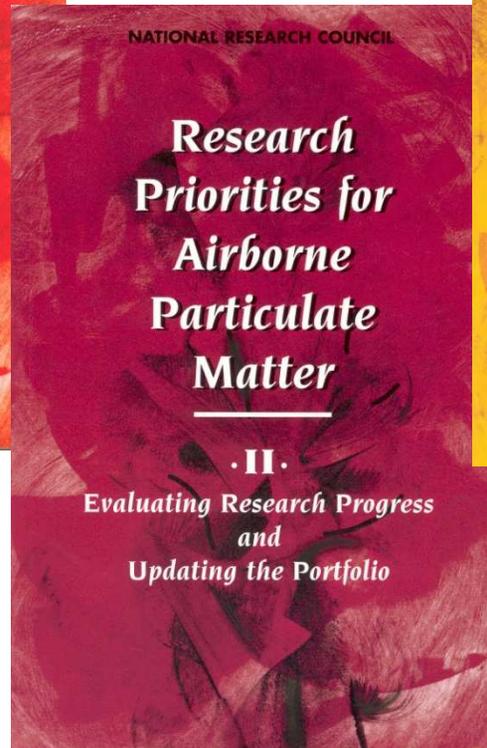
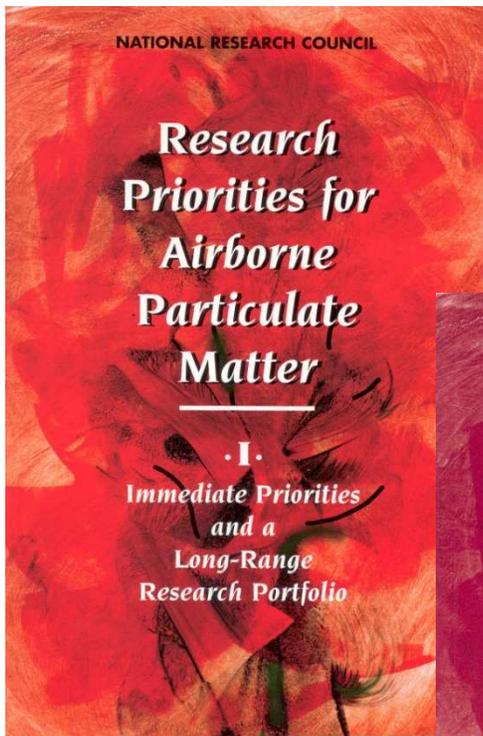
**California Air Resources Board**  
**Thursday, November 19, 2009**



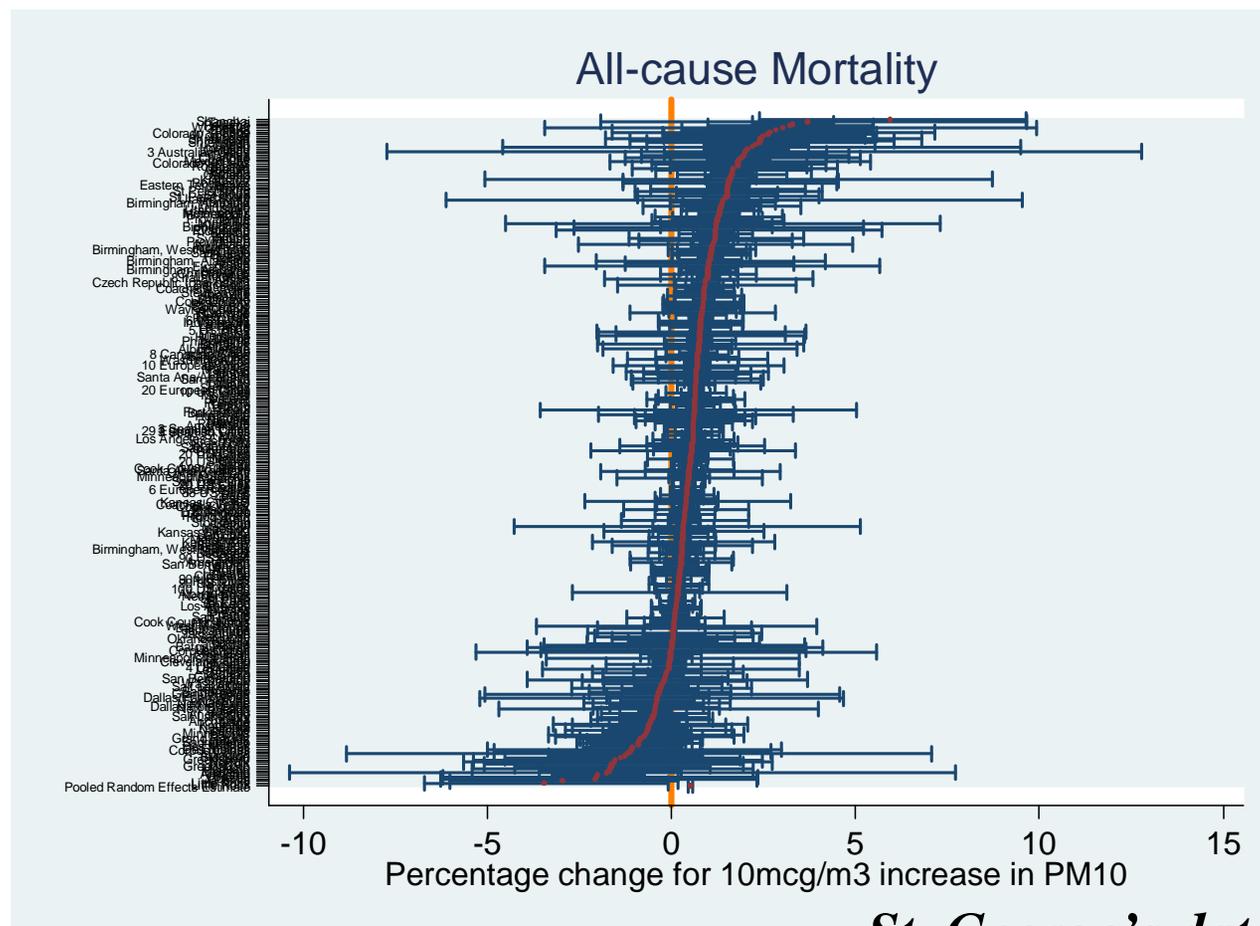
# National Morbidity Mortality Air Pollution Study



# National Research Council's PM Committee



# Time-series estimates to 2006: Daily all-cause mortality and $PM_{10}$ (n=314)



# **Integrated Science Assessment for Particulate Matter**

## **Second External Review Draft**

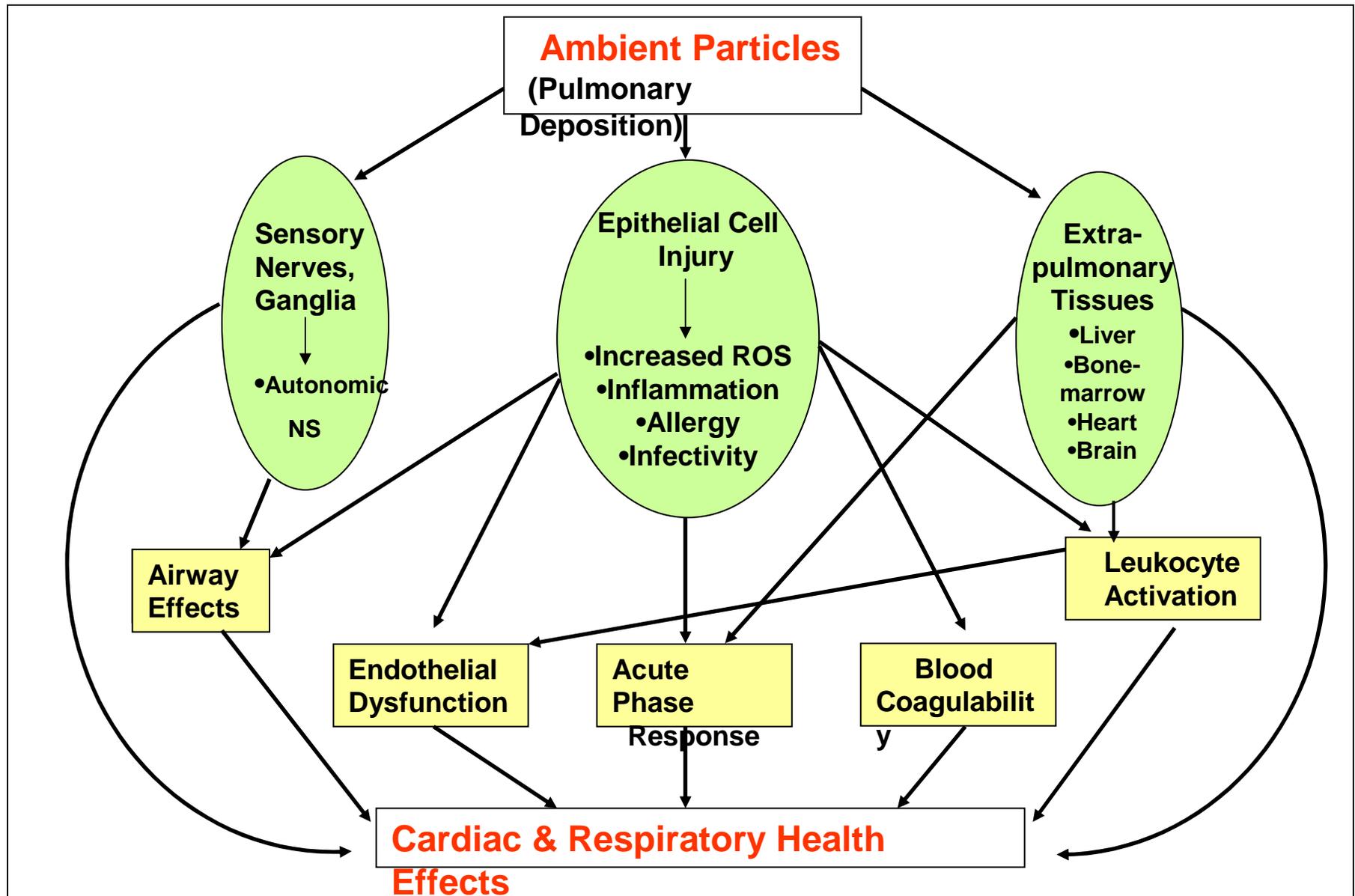
ISA: EPA/600/R-08/139B  
ANNEXES: EPA/600/R-08/139BA

National Center for Environmental Assessment-RTP Division  
Office of Research and Development  
U.S. Environmental Protection Agency  
Research Triangle Park, NC

# EPA's Evidence Classification

**Table 1-3. Weight of evidence for causal determination.**

Determination	Health Effects	Ecological and Welfare Effects
<b>Causal relationship</b>	Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures. That is, the pollutant has been shown to result in health effects in studies in which chance, bias, and confounding could be ruled out with reasonable confidence. For example: a) controlled human exposure studies that demonstrate consistent effects; or b) observational studies that cannot be explained by plausible alternatives or are supported by other lines of evidence (e.g., animal studies or mode of action information). Evidence includes replicated and consistent high-quality studies by multiple investigators.	Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures. That is, the pollutant has been shown to result in effects in studies in which chance, bias, and confounding could be ruled out with reasonable confidence. Controlled exposure studies (laboratory or small- to medium-scale field studies) provide the strongest evidence for causality, but the scope of inference may be limited. Generally, determination is based on multiple studies conducted by multiple research groups, and evidence that is considered sufficient to infer a causal relationship is usually obtained from the joint consideration of many lines of evidence that reinforce each other.
<b>Likely to be a causal relationship</b>	Evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain. That is, the pollutant has been shown to result in health effects in studies in which chance and bias can be ruled out with reasonable confidence but potential issues remain. For example: a) observational studies show an association, but copollutant exposures are difficult to address and/or other lines of evidence (controlled human exposure, animal, or mode of action information) are limited or inconsistent; or b) animal toxicological evidence from multiple studies from different laboratories that demonstrate effects, but limited or no human data are available. Evidence generally includes replicated and high-quality studies by multiple investigators.	Evidence is sufficient to conclude that there is a likely causal association with relevant pollutant exposures. That is, an association has been observed between the pollutant and the outcome in studies in which chance, bias and confounding are minimized, but uncertainties remain. For example, field studies show a relationship, but suspected interacting factors cannot be controlled, and other lines of evidence are limited or inconsistent. Generally, determination is based on multiple studies in multiple research groups.
<b>Suggestive of a causal relationship</b>	Evidence is suggestive of a causal relationship with relevant pollutant exposures, but is limited because chance, bias and confounding cannot be ruled out. For example, at least one high-quality epidemiologic study shows an association with a given health outcome but the results of other studies are inconsistent.	Evidence is suggestive of a causal relationship with relevant pollutant exposures, but chance, bias and confounding cannot be ruled out. For example, at least one high-quality study shows an effect, but the results of other studies are inconsistent.
<b>Inadequate to infer a causal relationship</b>	Evidence is inadequate to determine that a causal relationship exists with relevant pollutant exposures. The available studies are of insufficient quantity, quality, consistency or statistical power to permit a conclusion regarding the presence or absence of an effect .	The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of an effect.
<b>Not likely to be a causal relationship</b>	Evidence is suggestive of no causal relationship with relevant pollutant exposures. Several adequate studies, covering the full range of levels of exposure that human beings are known to encounter and considering susceptible or vulnerable subpopulations, are mutually consistent in not showing an effect at any level of exposure.	Several adequate studies, examining relationships with relevant exposures, are consistent in failing to show an effect at any level of exposure.



Source: Utell, Univ. Rochester, 2003

# Short-term Exposure to PM<sub>2.5</sub> and Mortality

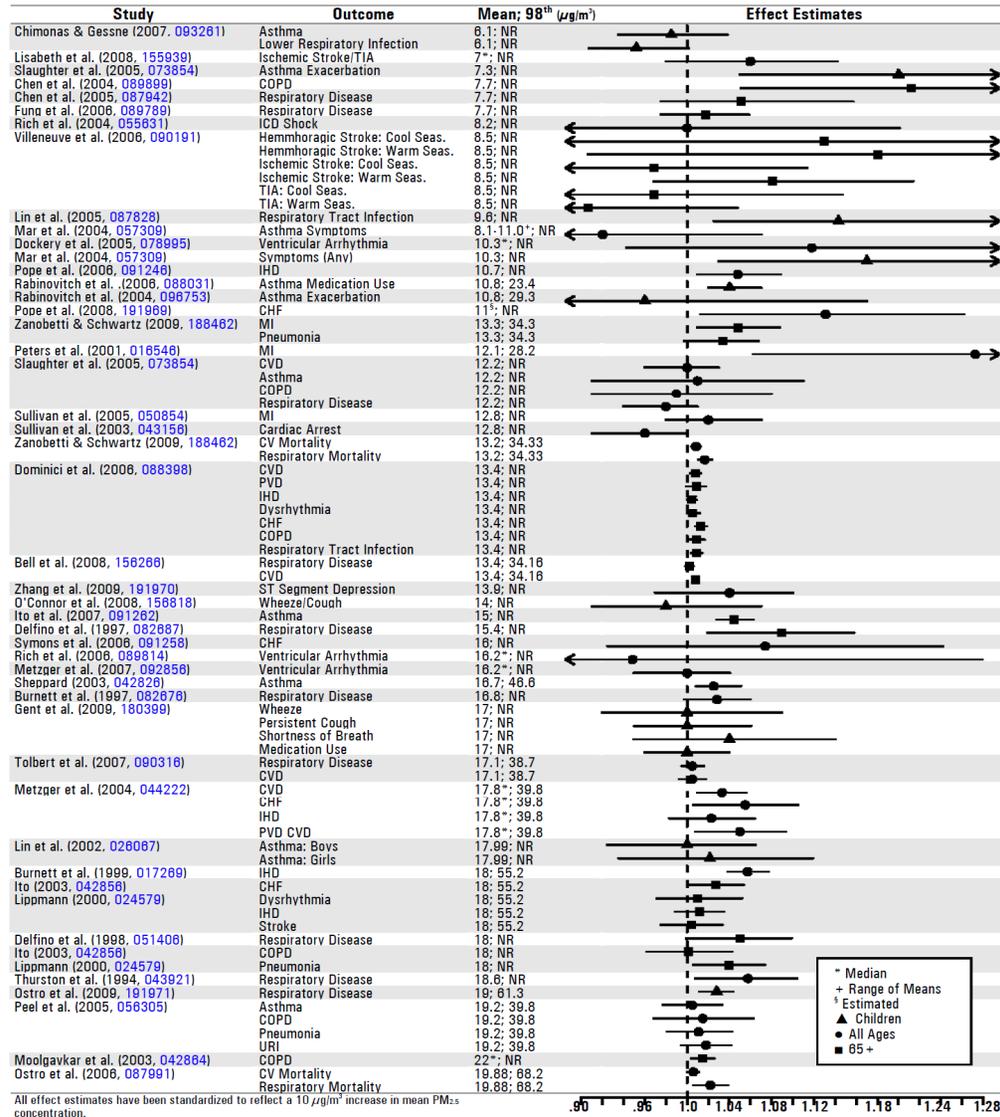
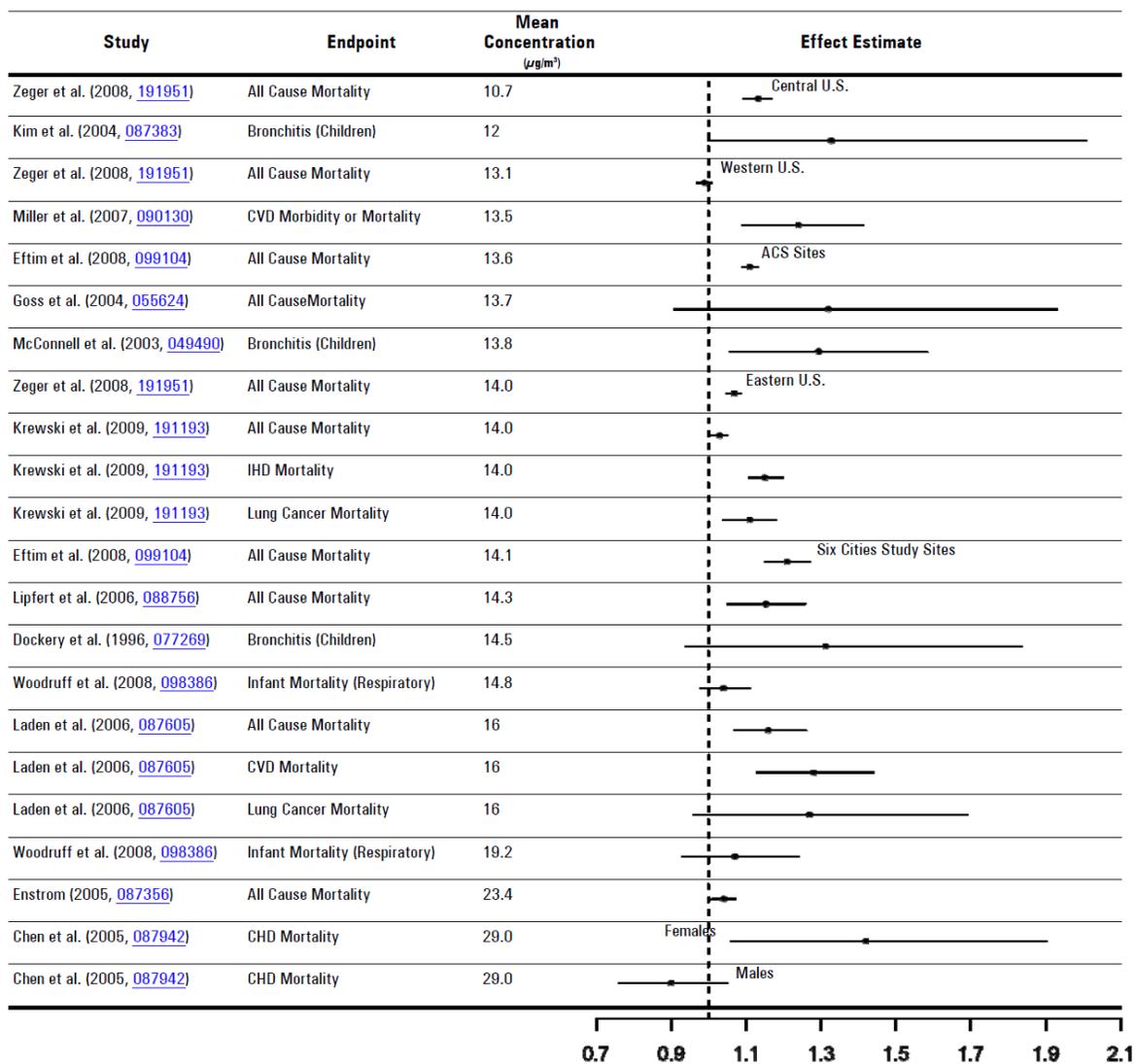


Figure 2-1. Excess risk estimates from epidemiologic studies of PM<sub>2.5</sub> ordered by mean 24-h avg concentration as reported by the investigator.

**Table 2-1 Summary of causal determinations for short-term exposure to PM<sub>2.5</sub>.**

Size Fraction	Outcome	Causality Determination
PM <sub>2.5</sub>	Cardiovascular Effects	Causal
	Respiratory Effects	Likely to be causal
	Mortality	Likely to be causal

# Long-term Exposure to PM<sub>2.5</sub> and Mortality



**Figure 2-2. Summary of U.S. studies examining the association between long-term exposure to PM<sub>2.5</sub> and CVD morbidity/mortality, respiratory morbidity/mortality, and all-cause mortality conducted in locations where the mean annual PM<sub>2.5</sub> concentration ranged from 10.7-29 μg/m<sup>3</sup>. All effect estimates have been standardized to reflect a 10 μg/m<sup>3</sup> increase in mean annual PM<sub>2.5</sub> concentration.**

**Table 2-2. Summary of causal determinations for long-term exposure to PM<sub>2.5</sub>.**

Size Fraction	Outcome	Causality Determination
PM <sub>2.5</sub>	Cardiovascular Effects	Causal
	Respiratory Effects	Likely to be causal
	Mortality	Likely to be causal
	Reproductive and Developmental	Suggestive
	Cancer, Mutagenicity, and Genotoxicity	Suggestive

**Table 2-3. Summary of causal determinations for short-term exposure to PM<sub>10-2.5</sub>.**

Size Fraction	Outcome	Causality Determination
PM <sub>10-2.5</sub>	Cardiovascular Effects	Suggestive
	Respiratory Effects	Suggestive
	Mortality	Suggestive

**Table 2-4. Summary of causal determinations for short-term exposure to UFPs.**

<b>Size Fraction</b>	<b>Outcome</b>	<b>Causality Determination</b>
Ultrafine Particles	Cardiovascular Effects	Suggestive
	Respiratory Effects	Suggestive

