EPA United States Environmental Protection Agency

Overview of Main Conclusions of the Integrated Science Assessment for Particulate Matter (External Review Draft)

Bay Area Air Quality Management District Advisory Council

Jason Sacks
National Center for Environmental Assessment
Office of Research and Development
U.S. Environmental Protection Agency
March 11, 2019

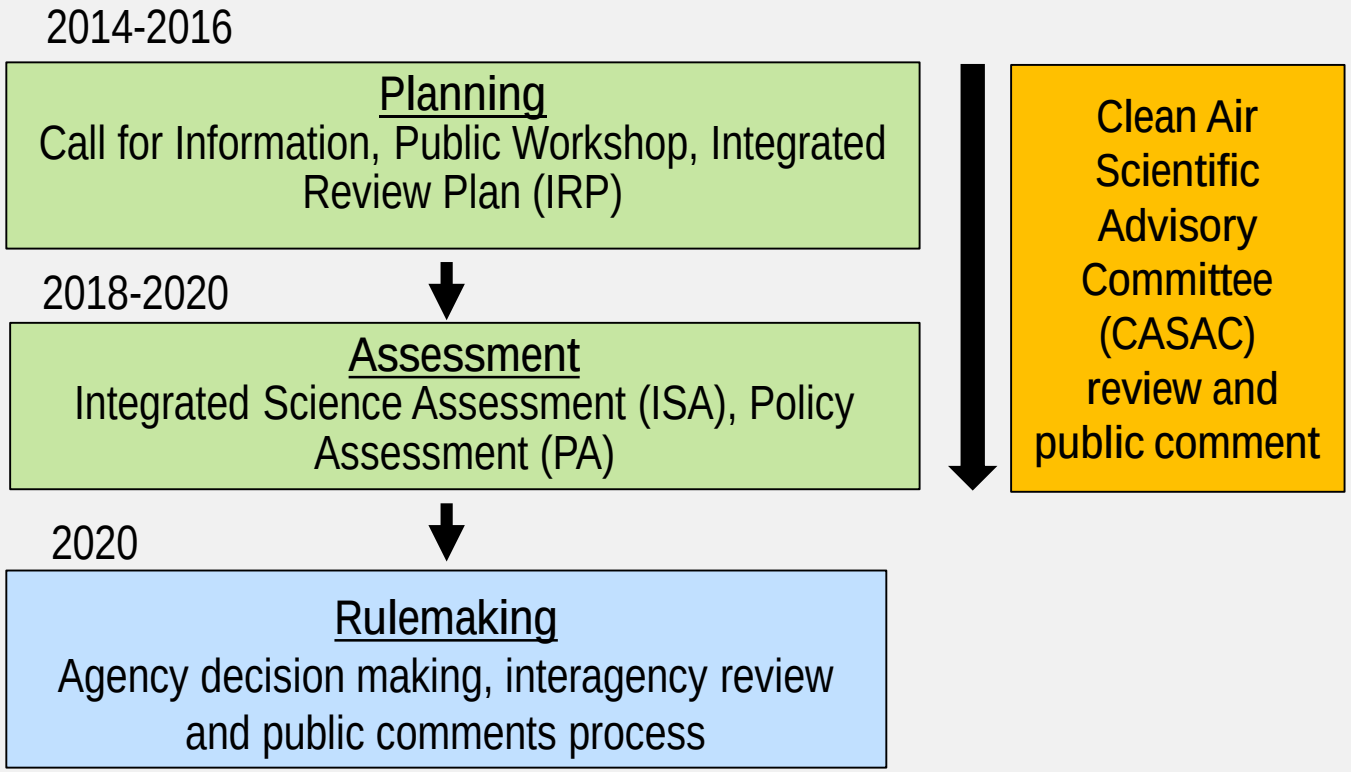
EPA United States Environmental Protection Agency

Disclaimer

This presentation is based on information provided in the external review draft Integrated Science Assessment for Particulate Matter. This information is distributed solely for the purpose of pre-dissemination peer review under applicable information quality guidelines. It has not been formally disseminated by EPA. It does not represent and should not be construed to represent any Agency determination or policy. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Overview of the Process for Reviewing the PM NAAQS

- IRP: Planned approach, schedule
- ISA: Assesses the available scientific information on public health and welfare effects; provides the science foundation for the review
- PA: Transparent analysis of the adequacy of the current standards and, as appropriate, potential alternatives



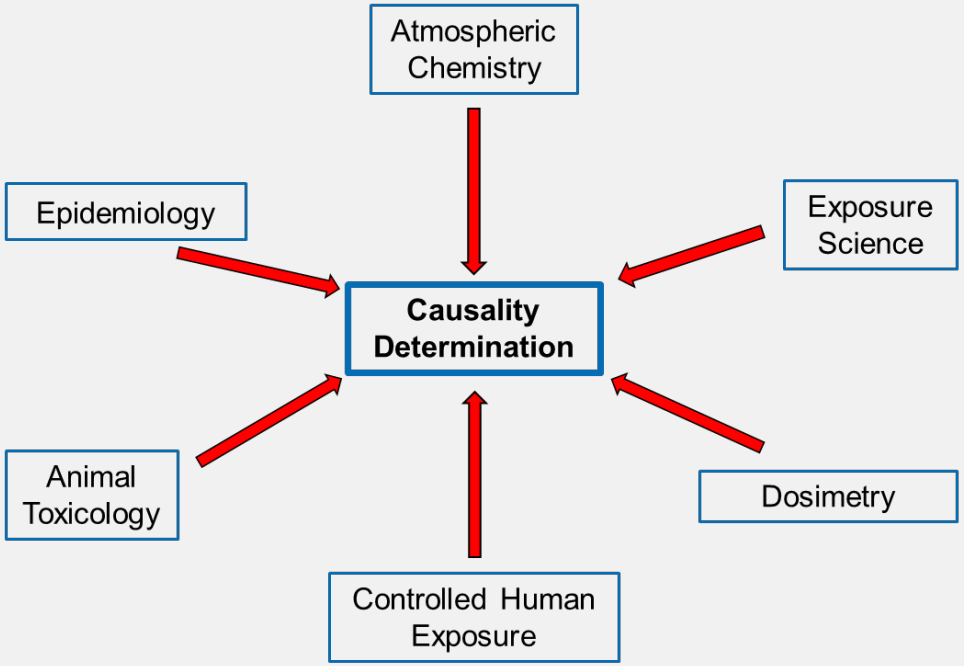
Note: This NAAQS Review Process was originally outlined in Administrator Pruitt's May 9, 2018 "Back to Basics" Memo.

Weight-of-Evidence Approach for Causality Determinations for Health and Welfare Effects

- Provides transparency through structured framework
- Developed and applied in ISAs for all criteria pollutants
- Emphasizes synthesis of evidence across scientific disciplines (e.g., controlled human exposure, epidemiologic, and toxicological studies)
- Five categories based on overall weight-of-evidence:
  - Causal relationship
  - Likely to be a causal relationship
  - Suggestive of, but not sufficient to infer, a causal relationship
  - Inadequate to infer the presence or absence of a causal relationship
  - Not likely to be a causal relationship
- ISA Preamble describes this framework
  - Preamble is now stand-alone document (http://www.epa.gov/isa)
- CASAC extensively reviewed the Agency's causal framework in the process of reviewing ISAs from 2008 – 2015; its use was supported in all ISAs

Evaluation of the Scientific Evidence

- Organize relevant literature for broad health outcome categories
- Evaluate studies, characterize results, extract relevant data
- Integrate evidence across disciplines for health outcome categories
- Develop causality determinations using established framework
- Evaluate evidence for populations potentially at increased risk
- Consideration of evidence spans many scientific disciplines from source to effect:
  - Atmospheric chemistry
  - Exposure
  - Controlled human exposure studies
  - Epidemiologic studies
  - Animal toxicologic studies
  - At-risk populations/lifestages



****Informs Hazard Identification step of Risk Assessment Process****

Framework for Causality Determinations in the ISA

	Health Effects	Ecological and Other Welfare Effects
Causal relationship	Evidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures (e.g., doses or exposures generally within one to two orders of magnitude of recent concentrations). That is, the pollutant has been shown to result in health effects in studies in which chance, confounding, and other biases could be ruled out with reasonable confidence. For example: (1) controlled human exposure studies that demonstrate consistent effects, or (2) observational studies that cannot be explained by plausible alternatives or that are supported by other lines of evidence (e.g., animal studies or mode of action information). Generally, the determination is based on multiple high-quality studies conducted by multiple research groups.	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, confounding, and other biases could be ruled out with reasonable confidence. Controlled exposure studies (laboratory studies, clinical studies, and field studies) provide the strongest evidence for causality, but the scope of inference may be limited. Generally, the 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.
Likely to be a causal relationship	Evidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures. That is, the pollutant has been shown to result in health effects in studies where results are not explained by chance, confounding, and other biases, but uncertainties remain. For example: (1) observational studies show an association, but copollutant exposures are difficult to address and/or other lines of evidence (e.g., human exposure, animal, or mode of action information) are limited or inconsistent, or (2) animal toxicological evidence from multiple studies from different laboratories demonstrate effects, but limited or no human data are available. Generally, the determination is based on multiple high-quality studies.	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, confounding, and other biases are minimized but uncertainties remain. For example, field studies show a relationship, but suspected interacting factors and/or other lines of evidence are limited or inconsistent. Generally, the determination is based on multiple studies by multiple research groups.
Suggestive of, but not sufficient to infer, a causal relationship	Evidence is suggestive of a causal relationship with relevant pollutant exposures but is limited, and chance, confounding, and other biases cannot be ruled out. For example: (1) when the body of evidence is relatively small, at least one high-quality epidemiologic study shows an association with a given health outcome and/or at least one high-quality animal toxicological study shows effects relevant to humans in animals of a single species, or (2) when the body of evidence is relatively large, evidence from studies of varying quality is generally supportive but not entirely consistent, and there may be coherence across lines of evidence (e.g., animal studies or mode of action information) to support the determination.	Evidence is suggestive of a causal relationship with relevant pollutant exposures, but chance, confounding, and other biases cannot be ruled out. For example, at least one high-quality study shows an effect, but the results of other studies are inconsistent.
Inadequate to infer a causal relationship	Evidence is inadequate to determine that a causal relationship exists with relevant pollutant exposures. The available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of an effect.	Evidence is inadequate to determine that a causal relationship exists with relevant pollutant exposures. The available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence or absence of an effect.
Not likely to be a causal relationship	Evidence indicates there is 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 at-risk populations and lifestages, are mutually consistent in failing to show an effect at any level of exposure.	Evidence indicates there is no causal relationship with relevant pollutant exposures. Several adequate studies examining relationships with relevant exposures are consistent in failing to show an effect at any level of exposure.

Multiple, high-quality studies
Rule out chance, confounding, and other biases with reasonable confidence

Multiple, high-quality studies
Important uncertainties remain

Evidence is suggestive but limited

Evidence is of insufficient quantity, quality, consistency, or statistical power

Multiple studies show no effect across exposure concentrations

Contents of the Draft PM ISA

Preface: Legislative Requirements of the PM NAAQS, Purpose and Overview of the ISA, Process for Developing ISA

Executive Summary

Chapter 1. Integrated Synthesis

Chapter 2. Sources, Atmospheric Chemistry, and Ambient Concentrations

Chapter 3. Exposure to Ambient PM

Chapter 4. Dosimetry of PM

Chapters 5 - 11. Respiratory Effects, Cardiovascular Effects, Metabolic Effects, Nervous System Effects, Reproductive and Developmental Effects, Cancer, and Mortality

Chapter 12. Lifestages and Populations Potentially at Increased Risk of a PM-related Health Effect

Chapter 13. Welfare Effects

Scope

- **Scope:** The ISA is tasked with answering the question "Is there an independent effect of PM on health and welfare at relevant ambient concentrations?"
  - Health Effects
    - Studies will be considered if they include a composite measure of PM (e.g., $PM_{2.5}$ mass, $PM_{10-2.5}$ mass, ultrafine particle (UFP) number)
      - Studies of source-based exposures that contain PM (e.g., diesel exhaust, wood smoke, etc.) if they have a composite measure of PM and examine effects with and without particle trap to assess the particle effect
      - Studies of components of PM if they include a composite measure of PM to relate toxicity of component(s) to current indicator
    - Studies will be considered if PM exposures are relevant to ambient concentrations (< 2 mg/m^3; 1 to 2 orders of magnitude above ambient concentrations)

Scope (cont.)

- Welfare Effects
  - Focus is on non-ecological welfare effects
    - Visibility Impairment
    - Climate Effects
    - Materials Effects
  - Ecological effects resulting from the deposition of PM and PM components are being considered as part of the review of the secondary (welfare-based) NAAQS for oxides of nitrogen, oxides of sulfur and PM

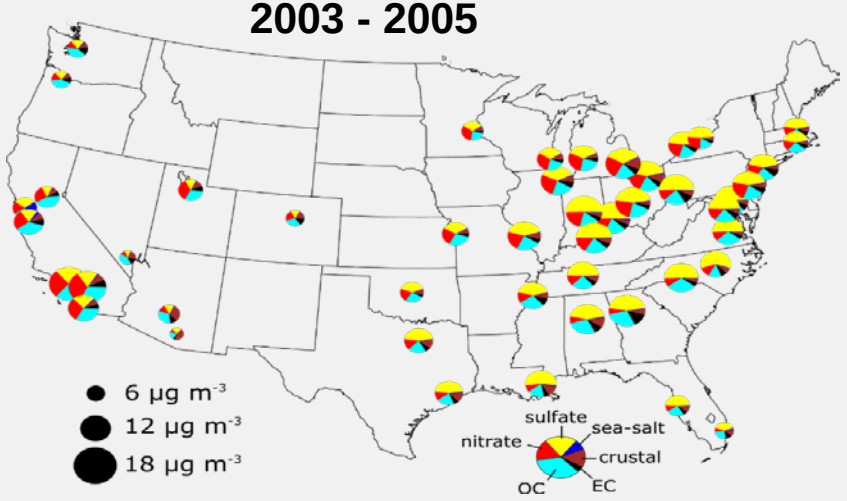
Executive Summary and Chapter 1

- Executive Summary
  - High-level overview of main conclusions of the entire ISA
  - Briefly captures strengths, limitations, and remaining uncertainties in the evidence base
- Integrated Synthesis (Chapter 1)
  - More detailed synthesis of the scientific evidence compared to the Executive Summary
    - Focus is on those health and welfare effects where it was concluded that a *causal* or *likely to be causal* relationship exists
    - Broad characterization of uncertainties and limitations in the evidence for $PM_{10-2.5}$ and UFPs that contributed to a *suggestive of, but not sufficient to infer* and *inadequate* causality determination
  - Integrated discussion of policy-relevant issues (e.g., copollutant confounding, concentration-response relationship, sources and components, etc.) spanning the health effects evidence
  - More detailed characterization of the strengths, limitations, and remaining uncertainties in the evidence base

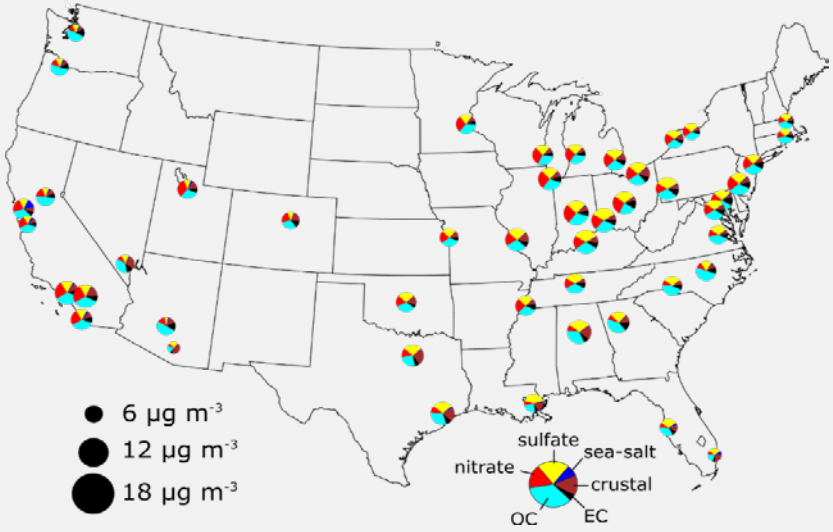
PM Concentrations and Trends (Chapter 2)

- $PM_{2.5}$
  - Steady declining trend 2000 to 2015, with most of the U.S. with annual average < 12 µg/m^3
  - Annual average decreased from 12 µg/m^3 to 8.6 µg/m^3 from 2006 to 2014
- $PM_{10\text{-}2.5}$
  - Federal Reference Method (FRM) in 2011
  - Recent data indicates that the contribution of $PM_{10\text{-}2.5}$ to PM_{10} is higher than previously reported
- UFPs
  - Highly variable concentration in space and over time due to physical and chemical processing in the atmosphere
  - UFP measured using multiple methods, varying in the size ranges examined
  - No U.S. monitoring network
- $PM_{2.5}$ Components
  - Organic carbon has replaced sulfate as the most abundant component of $PM_{2.5}$ in many locations, specifically in the eastern U.S.

2003 - 2005



2013 - 2015



Exposure to PM (Chapter 3)

- **Potential Errors and Uncertainty**
  - Vary depending on the exposure assessment method used
  - Evaluations more often occur for methods used in long-term exposure studies
- **Exposure Error**
  - **Short-term exposure studies:** exposure error produces underestimation of health effects
  - **Long-term exposure studies:** exposure error produces underestimation or overestimation of health effects
    - Overestimation of health effects occurs if the exposure model has low spatial resolution and underestimates exposures
- **Overall**
  - Necessary to examine individual study details to evaluate potential errors and uncertainty as well as quality of the exposure assessment method used

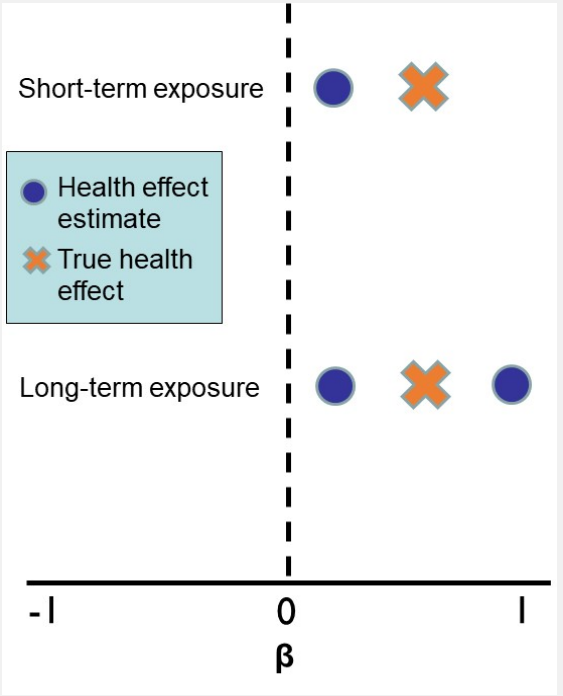
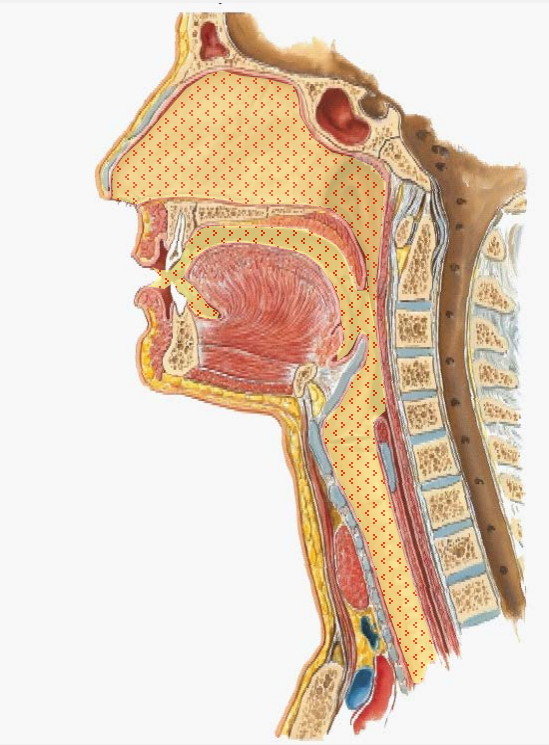


Figure. Influence of exposure error on health effects associations.

Dosimetry of PM (Chapter 4)

- New information in this review:
  - Demonstrates that children inhale less through the nose and have lower nasal deposition efficiency than adults resulting in increased exposure of the lungs to inhaled PM
  - Shows the translocation of a small fraction of particles (≤ 0.2 μm) out of the respiratory tract from the:
    - Olfactory mucosa to the brain
    - Alveolar region of the lung into blood
  - Indicates that PM_{10} overestimates the size of particles likely to enter the human lung



Oronasal breathing

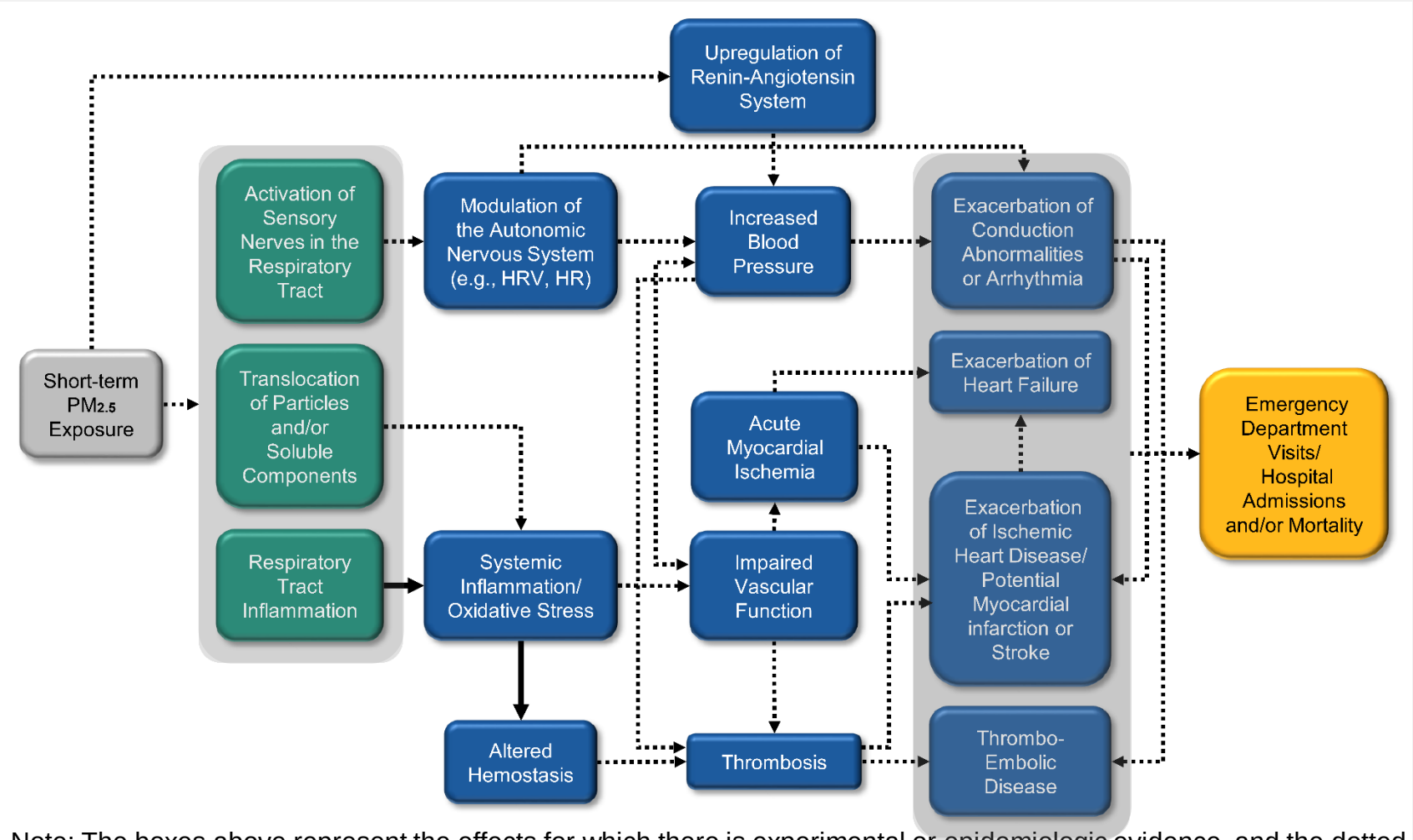
Draft PM ISA
Health Effects: Causality Determinations

HUMAN HEALTH EFFECTS					
ISA			Current PM Draft ISA		
Indicator			$PM_{2.5}$	$PM_{10-2.5}$	UFP
Health Outcome	Respiratory	Short-term exposure			
		Long-term exposure			
	Cardiovascular	Short-term exposure			
		Long-term exposure		*	
	Metabolic	Short-term exposure	*	*	*
		Long-term exposure	*	*	*
	Nervous System	Short-term exposure	*		*
		Long-term exposure	*	*	*
	Reproductive: Male/Female Reproduction and Fertility	Long-term exposure			
	Reproductive: Pregnancy and Birth Outcomes				
	Cancer	Long-term exposure	*	*	
	Mortality	Short-term exposure			
		Long-term exposure		*	

Causal **Likely causal** **Suggestive** **Inadequate**

* = new determination or change in causality determination from 2009 PM ISA

Example: Potential Biological Pathways Figure



Note: The boxes above represent the effects for which there is experimental or epidemiologic evidence, and the dotted arrows indicate a proposed relationship between those effects. Solid arrows denote direct evidence of the relationship as provided, for example, by an inhibitor of the pathway or a genetic knock-out model used in an experimental study. Shading around multiple boxes denotes relationships between groups of upstream and downstream effects. Progression of effects is depicted from left to right and color-coded (gray, exposure; green, initial event; blue, intermediate event; orange, apical event). Here, apical events generally reflect results of epidemiologic studies, which often observe effects at the population level. Epidemiologic evidence may also contribute to upstream boxes. When there are gaps in the evidence, there are complementary gaps in the figure.

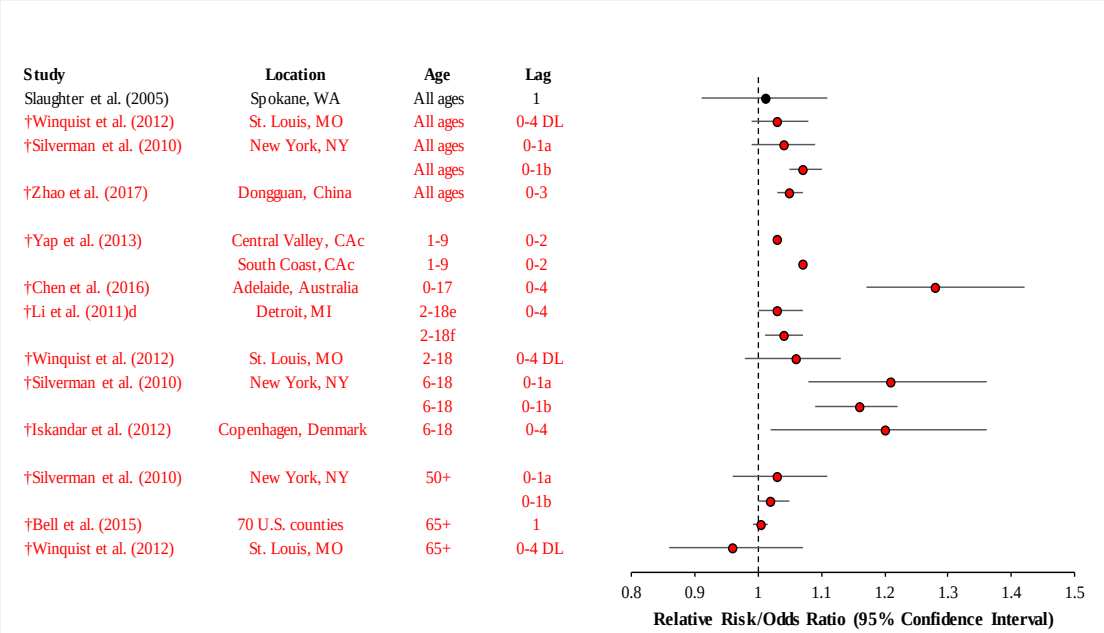
Respiratory Effects (Chapter 5)

Recent evidence supports the conclusions of the 2009 PM ISA, and continues to support a likely to be causal relationship between short- and long-term $PM_{2.5}$ exposure and respiratory effects

- Short-term $PM_{2.5}$ Exposure **(Likely to be Causal)**
  - Epidemiologic evidence: consistent evidence for asthma exacerbation in children and COPD exacerbation in adults, as well as respiratory mortality.
  - Experimental evidence: worsening of allergic airways disease and/or subclinical effects related to COPD, provide biological plausibility for asthma and COPD exacerbations
- Long-term $PM_{2.5}$ Exposure **(Likely to be Causal)**
  - Epidemiologic evidence: consistent changes in lung function and lung function growth rate, increased asthma incidence, asthma prevalence and wheeze in children; acceleration of lung function decline in adults; and respiratory mortality
  - Experimental evidence: impaired lung development and development of allergic airways disease, biological plausibility for decrements in lung function growth in children and asthma development

Respiratory Effects (Chapter 5)

Example: Short-term $PM_{2.5}$ Exposure and Asthma

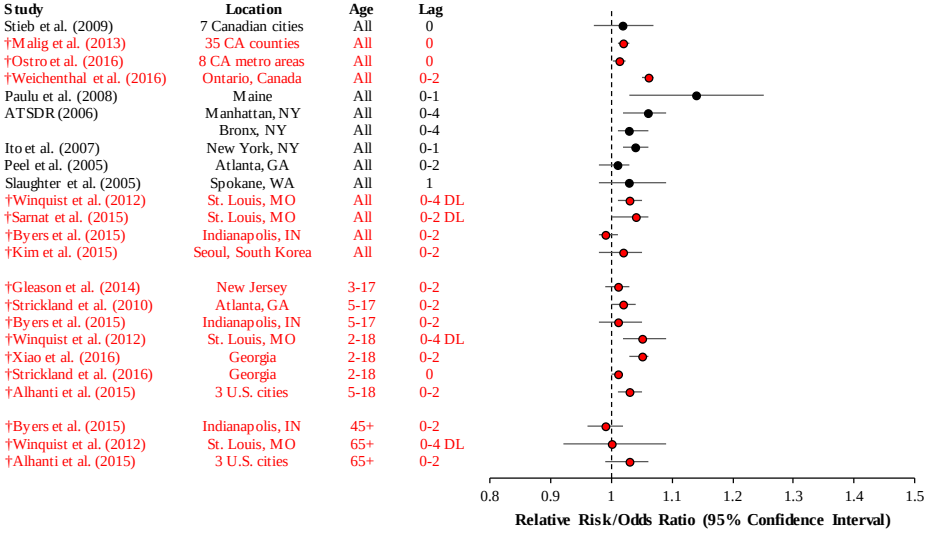


Hospital Admissions

Red = recent studies;
Black = U.S. study evaluated in the 2009 PM ISA

Emergency Department Visits

Red = recent studies;
Black = U.S. and Canadian studies evaluated in the 2009 PM ISA



Cardiovascular Effects (Chapter 6)

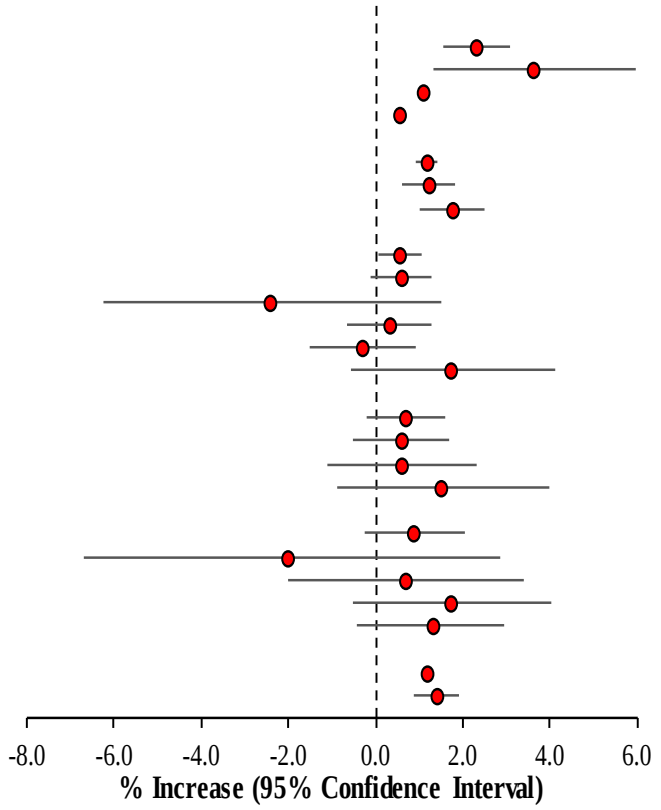
A large body of recent evidence supports and extends the conclusions of the 2009 PM ISA that there is a causal relationship between short- and long-term $PM_{2.5}$ exposure and cardiovascular effects

- Short-term $PM_{2.5}$ Exposure **(Causal)**
  - Epidemiologic evidence: generally consistent positive associations for hospital admissions and ED visits, particularly for ischemic heart disease (IHD) and heart failure (HF), as well as cardiovascular mortality
  - Experimental evidence: endothelial dysfunction, effects indicating impaired cardiac function, arrhythmia, changes in heart rate variability (HRV), increases in blood pressure (BP), and indicators of systemic inflammation, oxidative stress, and coagulation
- Long-term $PM_{2.5}$ Exposure **(Causal)**
  - Epidemiologic evidence: consistent positive associations for cardiovascular mortality; evidence for coronary heart disease (CHD) and stroke particularly in populations with pre-existing disease; evidence for coronary artery calcification (CAC)
  - Experimental evidence: impaired heart function, increased blood pressure, endothelial dysfunction, and atherosclerotic plaque progression

Cardiovascular Effects (Chapter 6)

Example: Short-term $PM_{2.5}$ Exposure and Cardiovascular-related Mortality

Study	Location	Lag	Outcome
†Lee et al. (2015)a	3 Southeast states, U.S.	0-1	Cardiovascular CHF MI Stroke
†Dai et al. (2014)	75 U.S. cities	0-1	Cardiovascular MI Stroke
†Samoli et al. (2013) †Samoli et al. (2014)	10 European Med cities 10 European Med cities	0-1	Cardiovascular Cardiac CHF Cerebrovascular Acute Coronary Events Arrhythmias
†Pascal et al. (2014)	9 French cities	0-1	Cardiovascular Cardiac IHD Cerebrovascular
†Milojevic et al. (2014)	England and Wales	0-1	Cardiovascular CHF MI Stroke IHD
†Shah et al. (2015) †Wang et al. (2014)	Meta-analysis Meta-analysis	--- ---	Stroke Stroke



Red = recent studies

Figure 6-7. Percent increase in cause-specific cardiovascular mortality outcomes for a 10 µg/m³ increase in 24-hour average $PM_{2.5}$ concentrations observed in multicity studies and meta-analyses.

Nervous System Effects (Chapter 8)

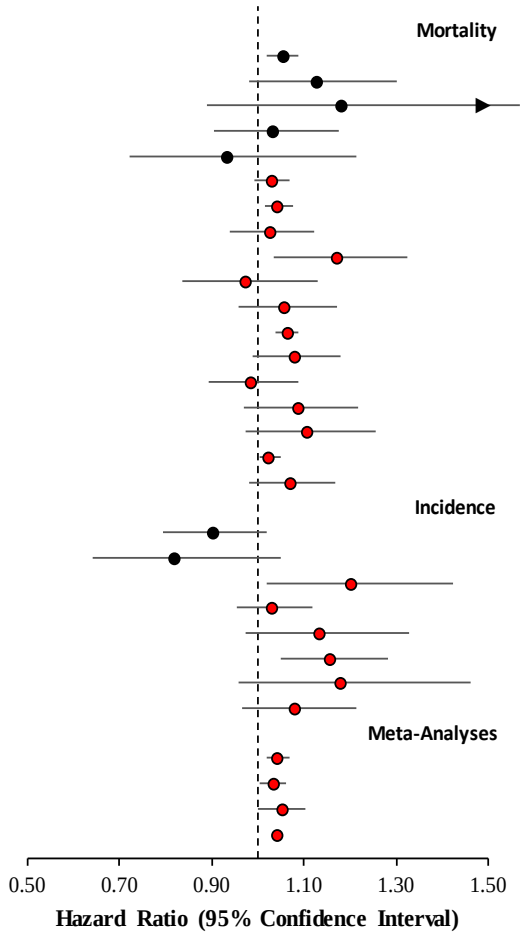
- Long-term $PM_{2.5}$ Exposure **(Likely to be Causal – NEW conclusion)**
  - Epidemiologic evidence
    - Consistent evidence for cognitive decline/impairment and decreased brain volume; more limited evidence for Alzheimer's disease and dementia
  - Experimental evidence
    - Consistent evidence for inflammation, oxidative stress, morphologic changes, and neurodegeneration in multiple brain regions of adult animals
    - Limited evidence for early indicators of Alzheimer's disease, impaired learning/memory, altered behavior in adult animals, and morphologic changes during development
- Long-term UFP Exposure **(Likely to be Causal – NEW conclusion)**
  - Epidemiologic evidence
    - Limited evidence for effects on cognitive development in children
  - Experimental evidence
    - Consistent evidence for inflammation, oxidative stress, and neurodegeneration in adult animals
    - Limited evidence of Alzheimer's disease pathology in a susceptible animal model
    - Strong evidence of developmental effects, mainly from one laboratory, for inflammation, morphologic changes including persistent ventriculomegaly, and behavioral effects following pre/postnatal exposure

Cancer (Chapter 10)

- Long-term $PM_{2.5}$ Exposure **(Likely to be Causal – NEW conclusion)**
  - Recent epidemiologic studies greatly expand upon the limited number of studies in the 2009 PM ISA that examined lung cancer incidence and mortality
    - Primarily positive associations, supported by analyses focusing on never smokers
  - Experimental and epidemiologic studies provide evidence for a relationship between $PM_{2.5}$ exposure and genotoxicity, epigenetic effects, and carcinogenic potential.
  - $PM_{2.5}$ exhibits several characteristics of carcinogens providing biological plausibility for $PM_{2.5}$ exposure contributing to cancer development

Cancer (Chapter 10)

Study	Cohort	Location	Follow-up Years	Qualifier
Krewski et al. (2009)	ACS (Re-analysis)	U.S.	1982-2000	
Laden et al. (2006)	HSC	6 U.S. cities	1974-1998	
McDonnell et al. (2000)	AHSMOG	California	1973-1977	Men
Brunekreef et al. (2009)a	NLCS - Air	Netherlands	1987-1996	Full Cohort
Brunekreef et al. (2009)a	NLCS - Air	Netherlands	1987-1996	Case Cohort
†Thurston et al. (2013)	ACS-CPS II	U.S.	1982-2004	
†Turner et al. (2016)	ACS-CPS II	U.S.	1982-2004	
†Hart et al. (2011)	TrIPS	U.S.	1985-2000	Men
†Lepeule et al. (2012)	HSC	6 U.S. cities	1974-2009	
†Lipsett et al. (2011)	CTS	California	2000-2005	Women
†Jerrett et al. (2013)	ACS-CPS II	California	1982-2000	
†Crouse et al. (2015)	CanCHEC	Canada	1991-2006	
†Pinault et al. (2016)	CCHS	Canada	2000-2011	
†Villeneuve et al. (2015)	CNBSS	Canada	1980-2005	Women
†Weichenthal et al. (2016)	CanCHEC	Ontario	1991-2009	
†Carey et al. (2013)	National English	United Kingdom	2003-2007	
†Cesaroni et al. (2013)	RoLS	Rome, Italy	2001-2010	
†Wong et al. (2016)	---	Hong Kong	1998-2011	
Brunekreef et al. (2009)b	NLCS - Air	Netherlands	1987-1996	Full Cohort
Brunekreef et al. (2009)b	NLCS - Air	Netherlands	1987-1996	Case Cohort
†Gharibvand et al. (2016)	AHSMOG-2	U.S.	2002-2011	
†Puett et al. (2014)	NHS	U.S.	1994-2010	Women
†Hystad et al. (2013)	NECSS	Canada	1994-1997	
†Tomczak et al. (2016)	CNBSS	Canada	1980-2004	Women
†Raaschou-Nielsen et al. (2013)	ESCAPE	Europe	1990s	
†Hart et al. (2015)	NCLS	Netherlands	1986-2003	
†Hamra et al. (2014)c	---	---	---	14 studies
†Yang et al. (2015)c	---	---	---	10 studies
†Chen et al. (2015)c	---	---	---	6 studies
†Cui et al. (2015)d	---	---	---	12 studies

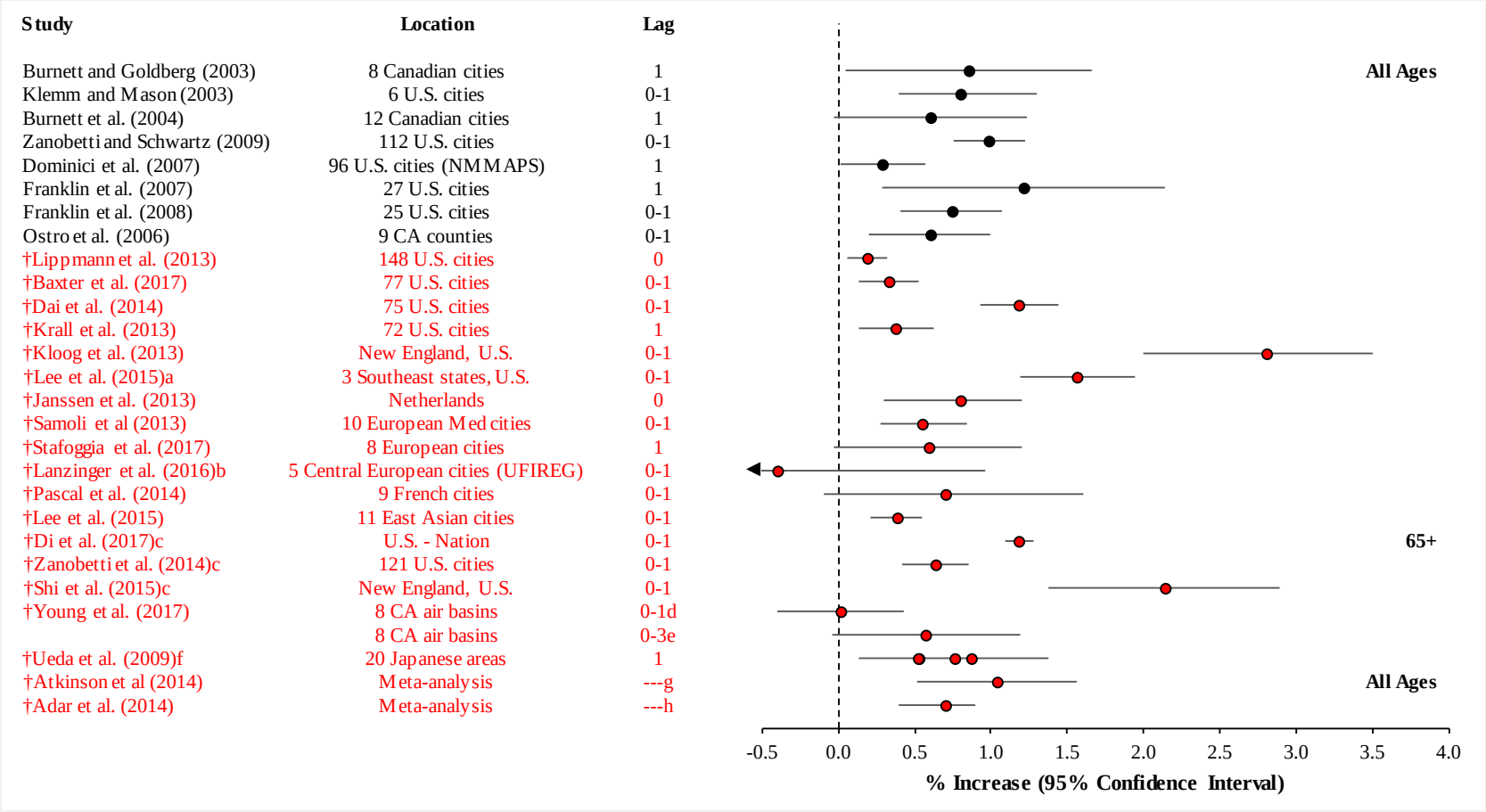


Note: Red = recent studies; Black = studies evaluated in the 2009 PM ISA

Figure 10-3. Summary of associations reported in previous and recent cohort studies that examined long-term $PM_{2.5}$ exposure and lung cancer mortality and incidence.

Mortality – Short-term $PM_{2.5}$ Exposure (Chapter 11) (Causal)

Recent evidence <u>supports and extends</u> the conclusions of the 2009 PM ISA that there is a <u>causal relationship</u> between short-term $PM_{2.5}$ exposure and mortality



Note: Red = recent multi-city studies; Black = multi-city studies evaluated in the 2009 PM ISA

Figure 11-1. Summary of associations between short-term $PM_{2.5}$ exposure and total (nonaccidental) mortality in multicity studies for a 10 μg/m³ increase in 24-hour average concentrations.

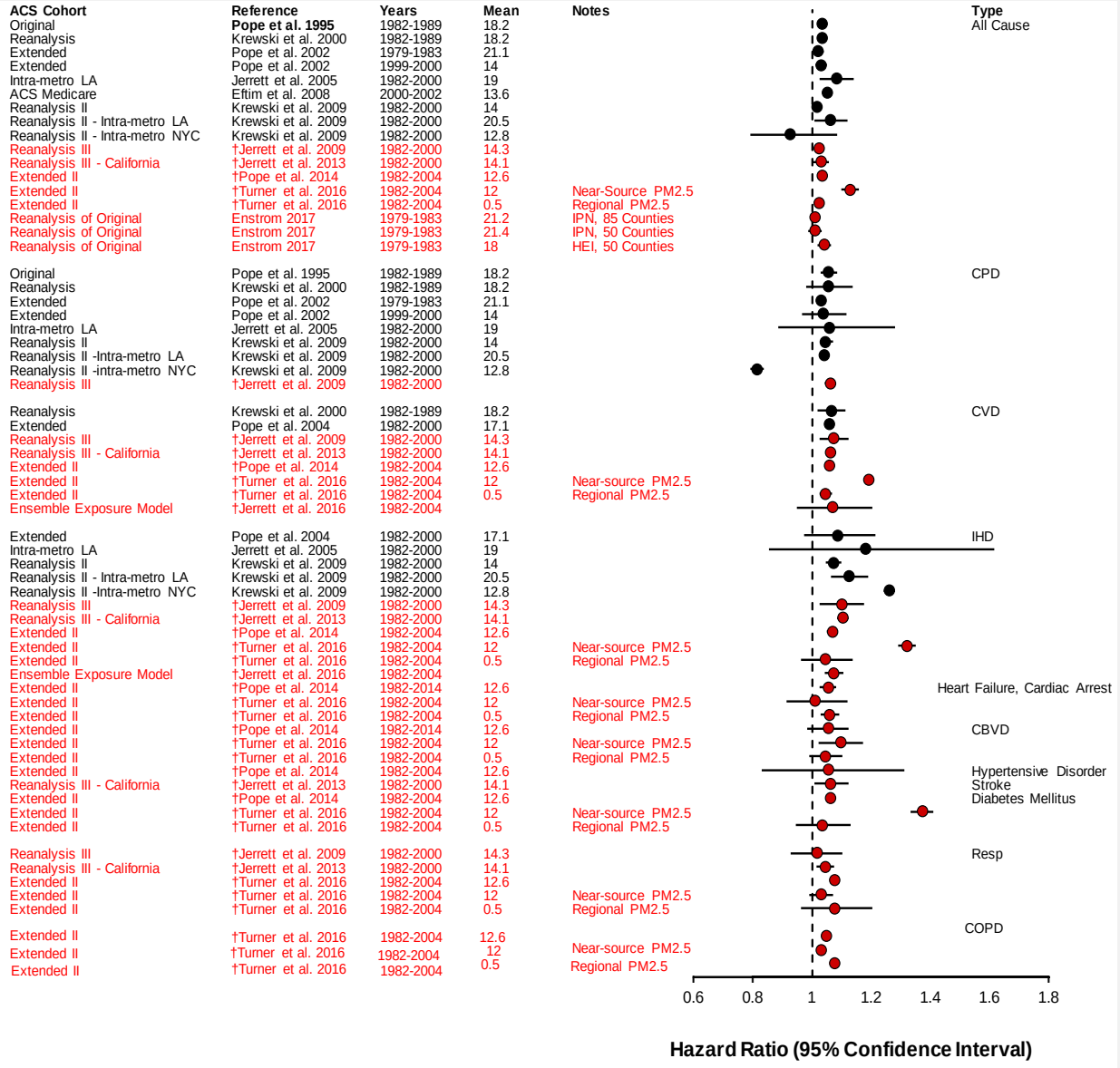
Mortality – Long-term $PM_{2.5}$ Exposure (Chapter 11) (Causal)

Recent evidence supports and extends the conclusions of the 2009 PM ISA that there is a causal relationship between long-term $PM_{2.5}$ exposure and mortality

Figure 11-17. Associations between long-term exposure to $PM_{2.5}$ and total (nonaccidental) mortality in the American Cancer Society (ACS) cohort.

Note: Associations are presented per 5 µg/m³ increase in pollutant concentration.

Red = recent studies;
Black = studies evaluated in the 2009 PM ISA

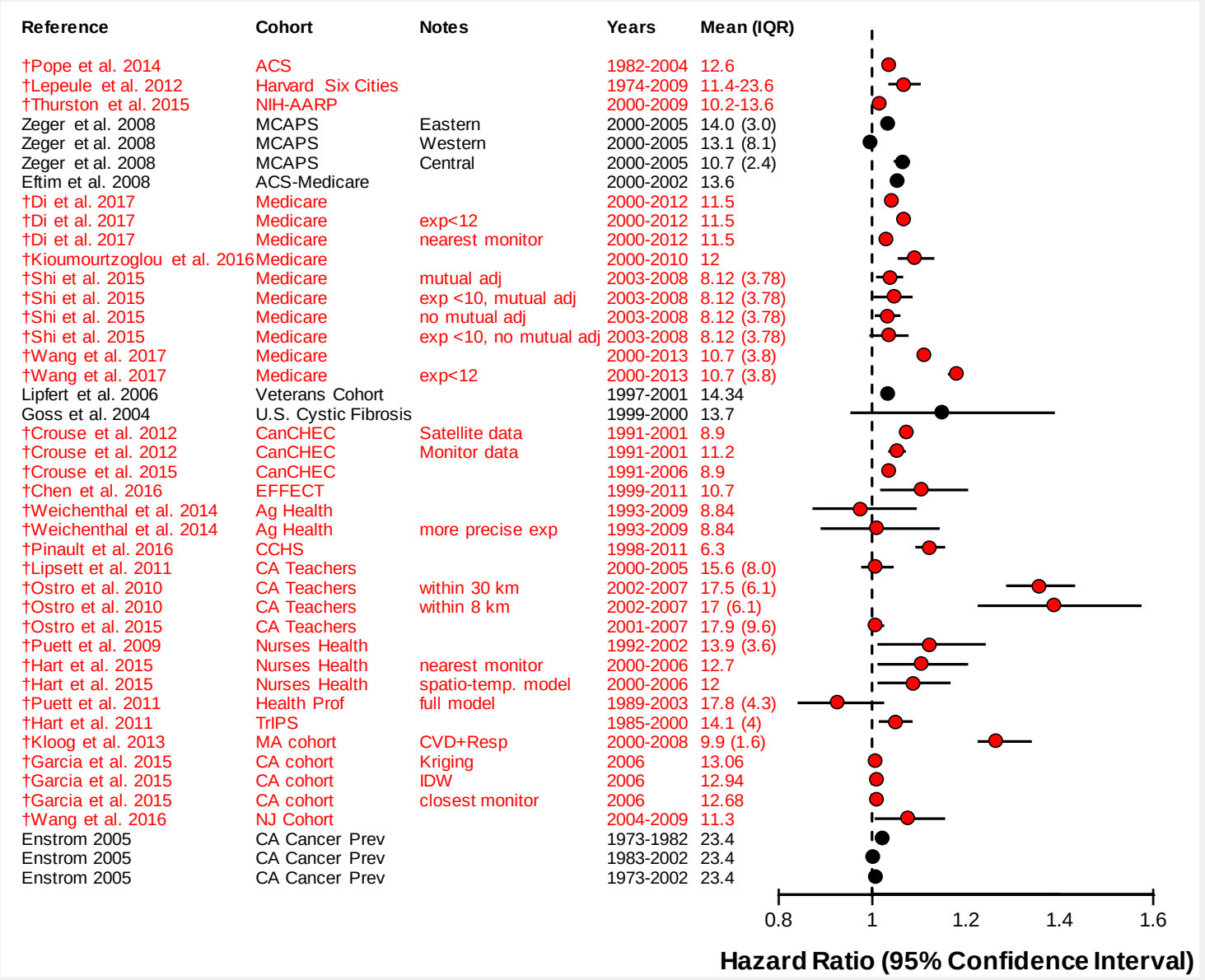


Mortality – Long-term $PM_{2.5}$ Exposure (Chapter 11) (Causal)

Figure 11-18. Associations between long-term $PM_{2.5}$ and total (nonaccidental) mortality in recent North American cohorts.

Note: Associations are presented per 5 µg/m^3 increase in pollutant concentration.

Red = recent studies;
Black = studies evaluated in the 2009 PM ISA



Other Causality Determinations (Chapters 5 – 10)

- Limitations and uncertainties in the evidence, along with few or no epidemiologic and experimental studies resulted in conclusions of:
  - Suggestive of, but not sufficient to infer, a causal relationship, for:
    - $PM_{2.5}$: repro/dev, nervous system (ST)
    - $PM_{10\text{-}2.5}$: mortality (ST), respiratory (ST), cardiovascular (ST/LT), metabolic (LT), cancer, nervous system (LT)
    - UFP: respiratory (ST), cardiovascular, (ST), nervous system (ST).
  - Inadequate to determine the presence or absence of a causal relationship, for:
    - $PM_{10\text{-}2.5}$: respiratory (LT), metabolic (ST), repro/dev, nervous system (ST)
    - UFP: mortality (ST/LT), respiratory (LT), cardiovascular (LT), metabolic (ST/LT), repro/dev, cancer

Note: ST = short-term exposure; LT = long-term exposure

Policy-Relevant Considerations (Chapter 1)

- **Copollutant Confounding**: Across recent studies examining various health effects and both short- and long-term $PM_{2.5}$ exposures, associations remain relatively unchanged in copollutant models

- **Concentration-Response (C-R) Relationship**: Across studies evidence continues to support a linear, no-threshold C-R relationship

- **PM Components and Sources**: Many $PM_{2.5}$ components and sources are associated with many health effects, and the evidence does not indicate that any one source or component is more strongly related with health effects than $PM_{2.5}$ mass

Populations Potentially at Increased Risk of a PM-related Health Effect (Chapter 12)

- The NAAQS are intended to protect both the population as a whole and those potentially at increased risk for health effects in response to exposure to criteria air pollutants
  - *Are there specific populations and lifestages at increased risk of a PM-related health effect, compared to a reference population?*
- The ISA identified and evaluated evidence for factors that may increase the risk of $PM_{2.5}$-related health effects in a population or lifestage, classifying the evidence into four categories:
  - Adequate evidence; suggestive evidence; inadequate evidence; evidence of no effect
- Conclusions:
  - Adequate: children and nonwhite populations
  - Suggestive: pre-existing cardiovascular and respiratory disease, overweight/obese, genetic variants glutathione pathways, low SES
  - Inadequate: pre-existing diabetes, older adults, residential location, sex, diet, and physical activity

Draft PM ISA
Welfare Effects: Causality Determinations

NONECOLOGICAL WELFARE EFFECTS		
	ISA	Current PM Draft ISA
		PM
Welfare Effect	Visibility	
	Climate	
	Materials	

Causal Likely causal Suggestive Inadequate

* = new determination or change in causality determination from 2009 PM ISA

Welfare Effects (Chapter 13)

Recent evidence supports and extends the conclusions of the 2009 PM ISA that there is a causal relationship between PM and welfare effects

- Visibility Impairment **(Causal)**
  - Long-term visibility improvements throughout the U.S as PM concentrations have decreased
  - Regional and seasonal patterns in atmospheric visibility parallel PM concentration patterns
  - More evidence supporting the relationship between visibility and PM composition
- Climate Effects **(Causal)**
  - New evidence provides greater specificity about radiative forcing
  - Increased understanding of additional climate impacts driven by PM radiative effects
  - Improved characterization of key sources of uncertainty particularly with response to PM-cloud interactions
- Materials Effects **(Causal)**
  - New information for glass and metals including modeling of glass soiling
  - Progress in the development of quantitative dose-response relationships and damage functions for materials in addition to stone, including glass and metals
  - Quantitative research on PM impacts on energy yield from photovoltaic systems

PM ISA Team

United States Environmental Protection Agency

NCEA Team

Jason Sacks (Assessment Lead)
Barbara Buckley (Deputy Lead)
Michelle Angrish
Renee Beardslee**†
Adam Benson*†
James Brown
Evan Coffman
Elizabeth Chan*+
Allen Davis
Steve Dutton
Brooke Hemming
Erin Hines
Ellen Kirrane
Dennis Kotchmar
Meredith Lassiter
Vijay Limaye##†
Tom Long
Tom Luben
April Maxwell*†
Joseph McDonald***
Steve McDow
Ihab Mikati*†
Jennifer Nichols
Molini Patel†
Rob Pinder+
Joseph Pinto++
Kristen Rappazzo
Jennifer Richmond-Bryant
Lindsay Stanek#
Michael Stewart
Chris Weaver

* ORISE
** Postdoctoral Fellow
*** NRMRL/OTAQ
# NERL
Region 5
+ OAQPS
++ Retired
† Separated

NCEA Management (Current)

John Vandenberg, NCEA-RTP Director
Steve Dutton, Deputy Director
Tara Greaver, Branch Chief (Acting)

NCEA Management (Retired/Previously Acting)

Debra Walsh, Deputy Director (Retired)
Reeder Sams, Deputy Directory (Acting)
Andrew Hotchkiss, Branch Chief (Acting)
Alan Vette, Branch Chief (Acting)
Jennifer Richmond-Bryant, Branch Chief (Acting)

Technical Support

Marieka Boyd
Ryan Jones
Connie Meacham++
Shane Thacker

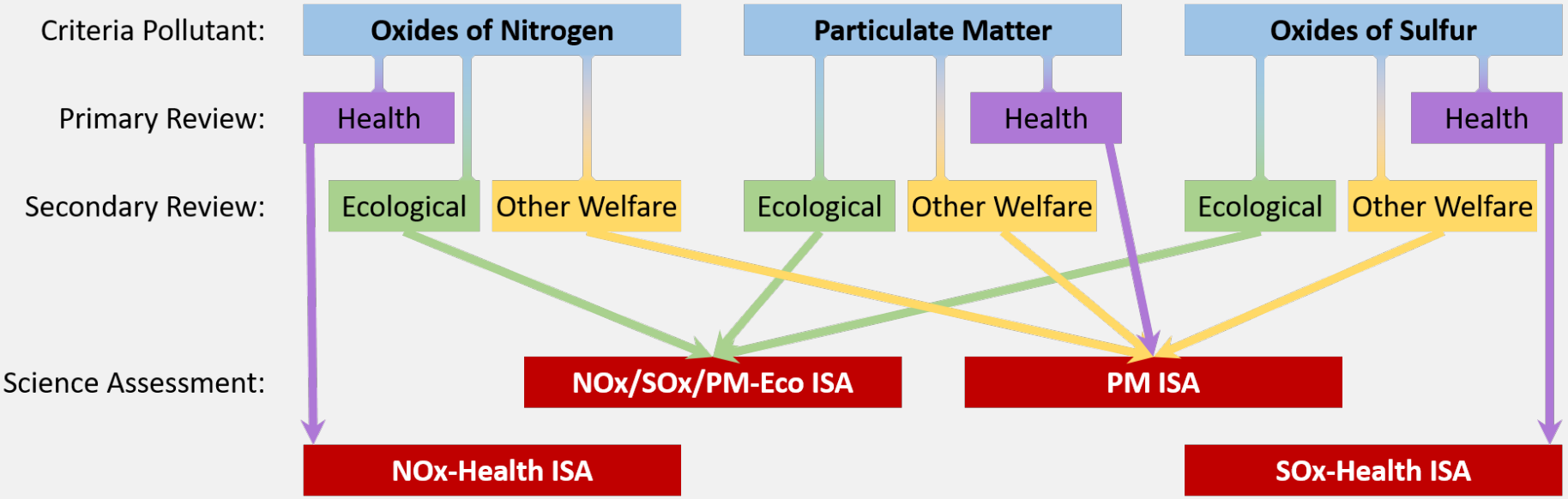
External Authors

Neil Alexis
Matt Campen
Sorina Eftim
Allison Elder
Jay Gandy
Katie Holliday
Veli Matti Kerminen
Igor Koturbash
Markku Kulmala
Petter Ljungman
William Malm
Loretta Mickley
Marianthi-Anna Kioumourtzoglou
James Mulholland
Maria Rosa
Armistead Russell
Brett Schichtel
Michelle Turner
Laura Van Winkle
James Wagner
Greg Wellenius
Eric Whitsel
Catherine Yeckel
Antonella Zanobetti
Max Zhang

Supplemental Materials

EPA United States Environmental Protection Agency

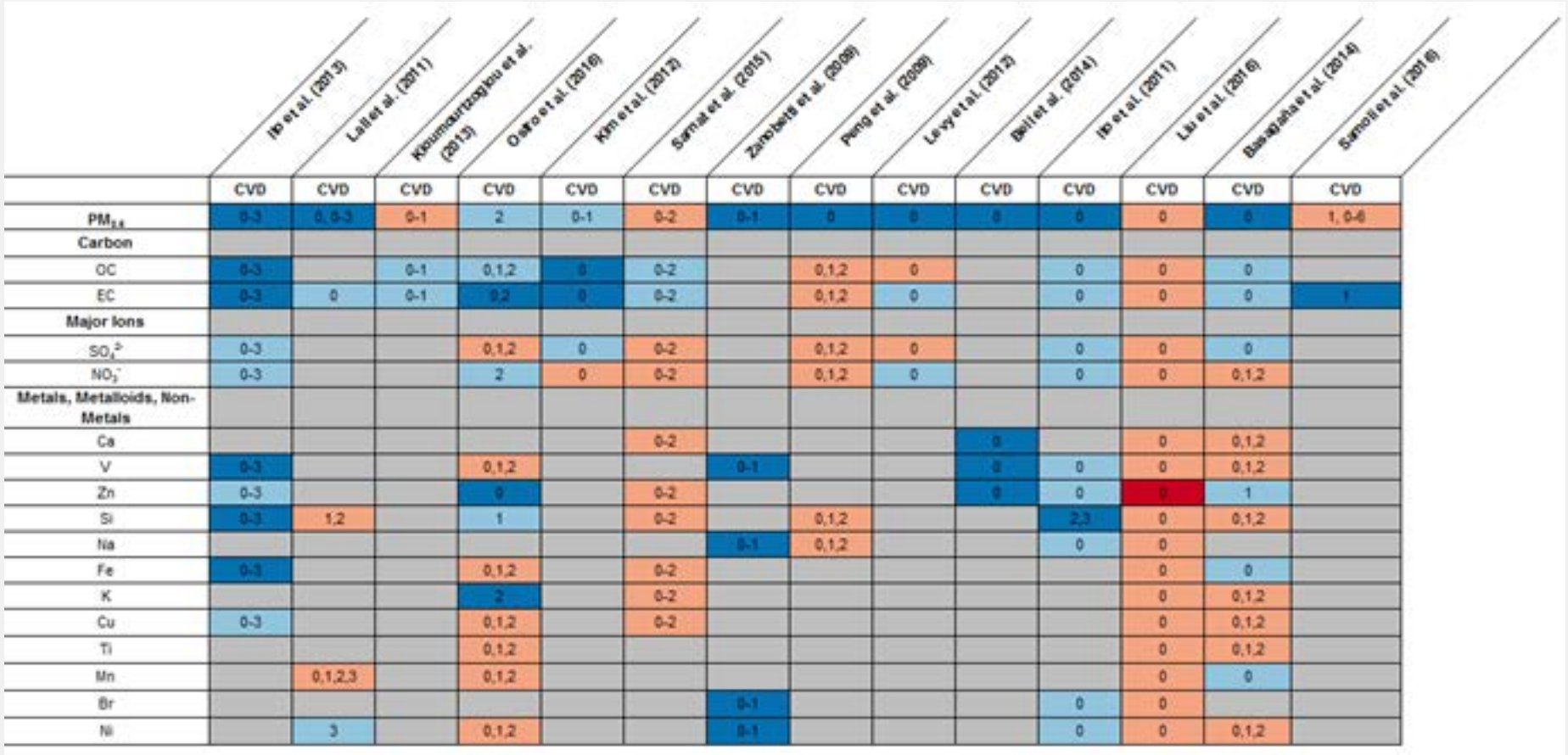
Relationship among Integrated Science Assessments



Notes: Primary (health-based) review of effects on public health = Health
Secondary (welfare-based) review of effects on pubic welfare = Ecological + Other Welfare
Ecological = effects on soil, water, crops, vegetation, animals, wildlife
Other Welfare = effects on manmade materials, weather, visibility, climate

Example: Evaluation of PM Components Studies

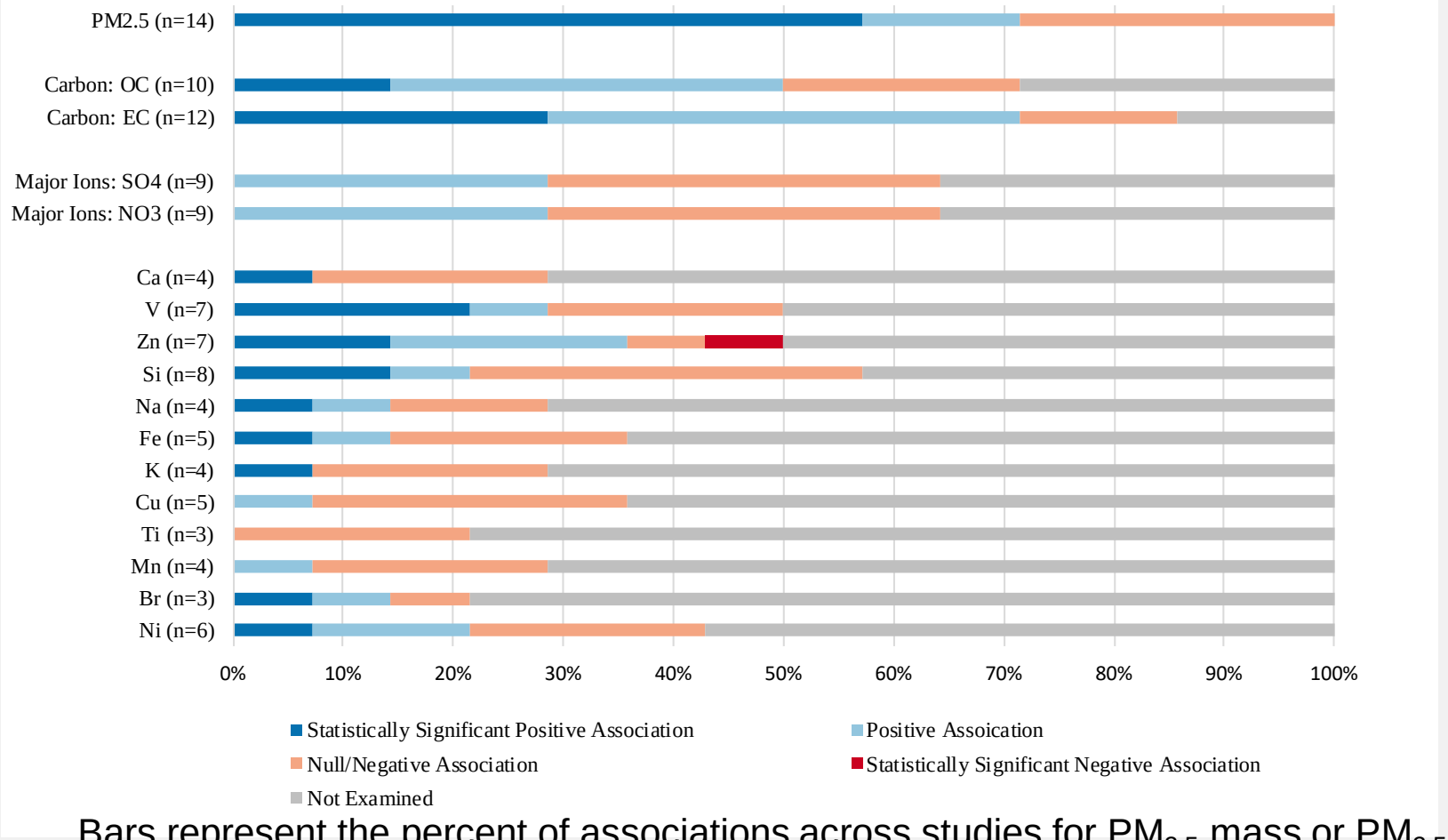
Short-term $PM_{2.5}$ and $PM_{2.5}$ Components Exposure and Cardiovascular Effects: Hospital Admissions and Emergency Department (ED) visits – Heat Map



- Numbers represent lags for which associations observed.
- $PM_{2.5}$ mass or $PM_{2.5}$ components associations categorized by results that are statistically significant positive (dark blue), positive/null (light blue), null/negative (light orange), statistically significant negative (red), or not examined (gray).

Example: Evaluation of PM Components Studies

Short-term $PM_{2.5}$ and $PM_{2.5}$ Components Exposure and Cardiovascular Effects: Hospital Admissions and ED visits – Distribution of Risk Estimates



Bars represent the percent of associations across studies for $PM_{2.5}$ mass or $PM_{2.5}$ components that are statistically significant positive (dark blue), positive (light blue), null/negative (light orange), statistically significant negative (red), or not examined (gray). n = number of studies that provided an estimate for $PM_{2.5}$ mass and individual $PM_{2.5}$ components.

At-Risk Framework Description

Classification	Health Effects
Adequate evidence	There is substantial, consistent evidence within a discipline to conclude that a factor results in a population or lifestage being at increased risk of air pollutant-related health effect(s) relative to some reference population or lifestage. Where applicable, this evidence includes coherence across disciplines. Evidence includes multiple high-quality studies.
Suggestive evidence	The collective evidence suggests that a factor results in a population or lifestage being at increased risk of air pollutant-related health effect(s) relative to some reference population or lifestage, but the evidence is limited due to some inconsistency within a discipline or, where applicable, a lack of coherence across disciplines.
Inadequate evidence	The collective evidence is inadequate to determine whether a factor results in a population or lifestage being at increased risk of air pollutant-related health effect(s) relative to some reference population or lifestage. The available studies are of insufficient quantity, quality, consistency, and/or statistical power to permit a conclusion to be drawn.
Evidence of no effect	There is substantial, consistent evidence within a discipline to conclude that a factor does not result in a population or lifestage being at increased risk of air pollutant-related health effect(s) relative to some reference population or lifestage. Where applicable, the evidence includes coherence across disciplines. Evidence includes multiple high-quality studies.

Excerpt from Preamble to ISAs

AGENDA: 6

Particulate Matter Symposium

Advisory Council Meeting
March 11, 2019

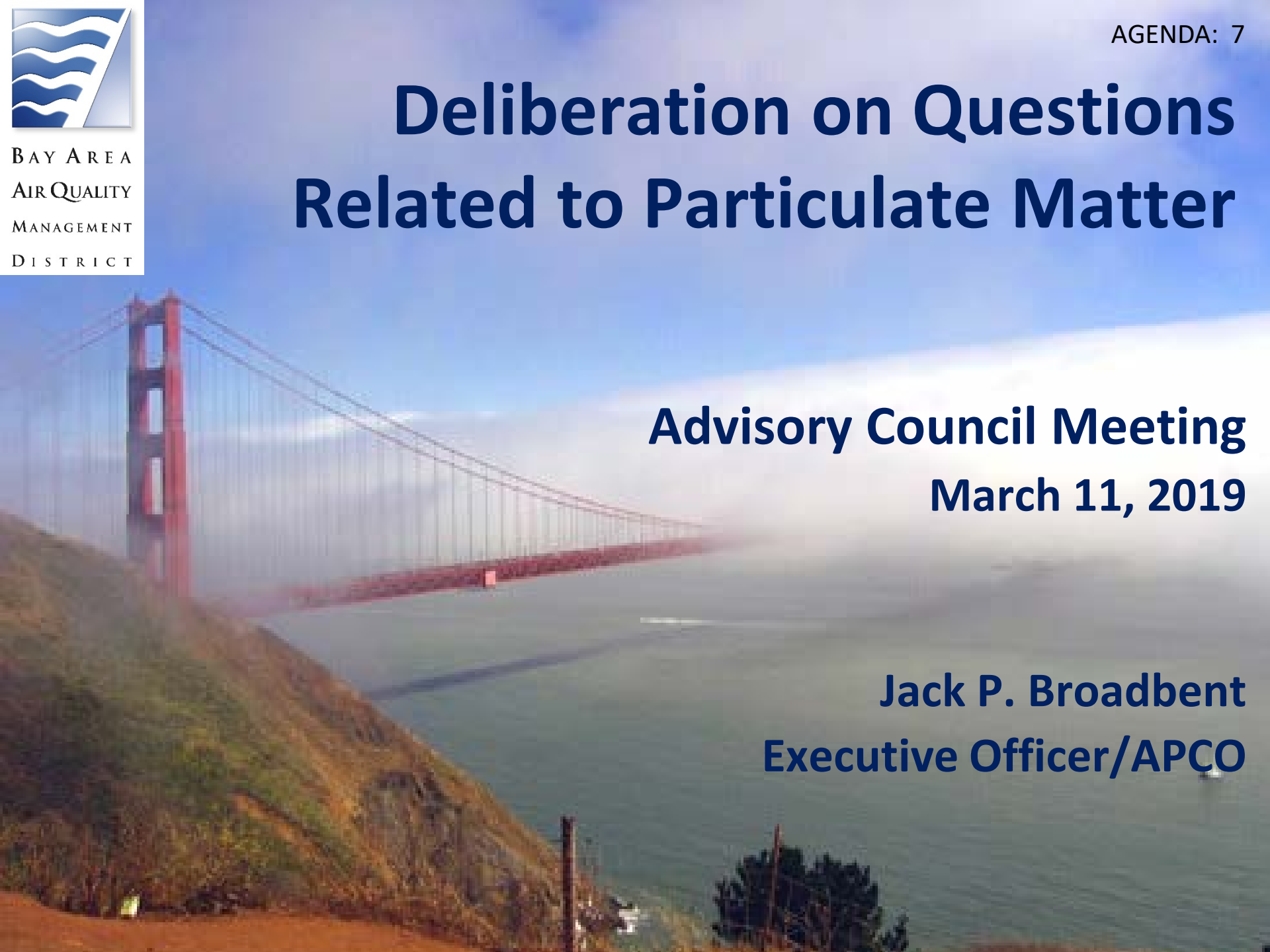
Jack P. Broadbent
Executive Officer/APCO

Proposed Symposium on Particulate Matter

- Convened by the Advisory Council as its October 2019 Meeting
- Primary Goal of enabling identification of health focused attainment guidelines beyond those already in effect
- Location to host up to 300 people
- Engage experts who were previously engaged at the Federal level
- Include local health officials and community groups

AGENDA: 7

Deliberation on Questions Related to Particulate Matter



Advisory Council Meeting
March 11, 2019

Jack P. Broadbent
Executive Officer/APCO

Deliberation on Questions Related to Particulate Matter (PM)

- The Council is invited to deliberate on topics such as:
  - Are current standards sufficiently health protective?
  - What health inequities exist relative to PM?
  - What metrics can be used to address these questions?
    - Concentration?
    - Exposure?
    - Risk (complexities of PM characterization)?
    - Health Outcomes?
  - What are the most important actions that can be taken now? And in the future?