Particulate Matter:
Spotlight on Health Protection

Call to Order
Pledge of Allegiance
Public Comment
Approval of Minutes

Stan Hayes

Particulate Matter:
Spotlight on Health Protection

Welcome Remarks

Jack Broadbent

Particulate Matter:
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Introduction

Jeff McKay

Particulate Matter:
Spotlight on Health Protection

PM Symposium Series




Health Effects

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- Assessment lead for the Particulate Matter Integrated Science Assessment
- Key leadership roles in synthesizing the health effects evidence of air pollution for various National Ambient Air Quality Standards reviews
- International training on U.S. EPA's Environmental Benefits Mapping and Analysis Program – Community Edition
- M.P.H. from Johns Hopkins University in 2003

Current State of Particulate Matter Science: Particulate Matter Integrated Science Assessment (PM ISA) (Working Draft Conclusions)

Particulate Matter: Spotlight on Health Protection

Bay Area Air Quality Management District

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Center for Public Health and Environmental Assessment
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October 28, 2019

Disclaimer

This presentation is based on information provided in the external review draft Integrated Science Assessment for Particulate Matter (PM ISA) as well as ongoing revisions to the PM ISA based on comments provided by the public and Clean Air Scientific Advisory Committee (CASAC). 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.

Outline

- PM NAAQS Milestones
- PM ISA
  - Weight-of-Evidence Evaluation
  - Scope
  - Ultrafine Particles (UFPs)
  - Causality Determinations: Health Effects
    - Likely to be Causal
    - $PM_{2.5}$ Sources and Components
    - Populations/Lifestages at Increased Risk
  - Next Steps

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

2014-2016

Planning
Call for Information and Public Workshop: Feb. 2015
Integrated Review Plan (IRP): Dec. 2016

2018-2020

Assessment
Integrated Science Assessment (ISA):
- External Review Draft: Oct. 2018
- Final ISA: Dec. 2019

Policy Assessment (PA): Sep. 2019

2020

Rulemaking
Agency decision making, interagency review and public comments process

Clean Air Scientific Advisory Committee (CASAC) review and public comment:
ISA: Dec. 2018
PA: Oct. 2019

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 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

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)

Ultrafine Particles (UFPs)

- Ultrafine particles are generally considered to be PM with a diameter less than or equal to 0.1 µm (100 nm)
- Uncertainties:
  - Highly variable concentration in space and over time due to physical and chemical processing in the atmosphere
    - UFP concentrations are highest in urban areas and during rush hour, and are highly episodic during winter
  - Lack of U.S. monitoring network and limited data on spatial and temporal UFP concentrations
  - UFP measured using multiple methods, varying in the size ranges examined - some capturing multiple size ranges below 100 nm, while others can include sizes above 100 nm
    - Contributed to difficulty in evaluating evidence within and across epidemiologic and experimental studies

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Draft PM ISA Health Effects: Causality Determinations

Table 1-5. Summary of causality determinations for health effect categories for the draft PM ISA.

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	Long-term exposure			
	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

Respiratory Effects

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

- Epidemiologic evidence:
  - Consistent evidence for asthma exacerbation in children and COPD exacerbation in adults; respiratory mortality.
- Experimental evidence:
  - Animal models of asthma and COPD demonstrate worsening of allergic airway disease and/or subclinical effects
- Remaining Uncertainties:
  - Lack of coherence between epidemiologic and animal toxicological evidence because most effects demonstrated in healthy animals
  - Minimal evidence from controlled human exposure studies for respiratory effects
  - Limited assessment of potential copollutant confounding

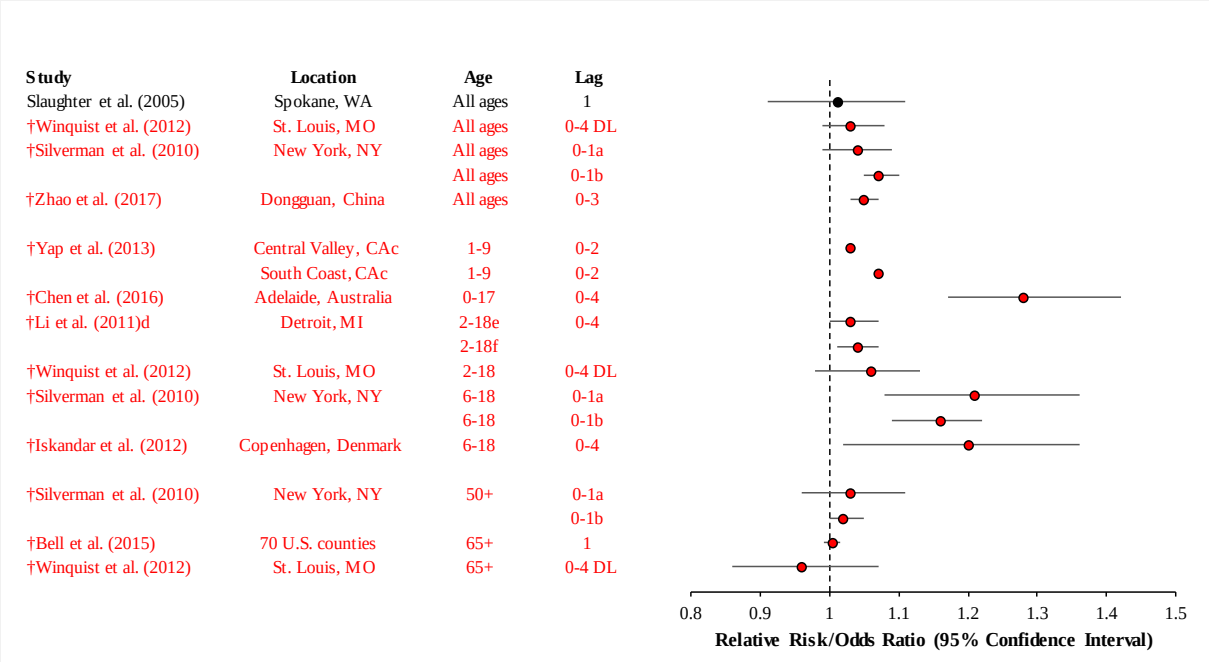


Figure 5-2. Summary of associations between short-term $PM_{2.5}$ exposures and asthma hospital admissions for a 10 µg/m³ increase in 24-hour average $PM_{2.5}$ concentrations.

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

Respiratory Effects (cont.)

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

- Epidemiologic evidence:
  - Consistent changes in lung function and lung function growth
  - Increased asthma incidence, asthma prevalence and wheeze in children
  - Acceleration of lung function decline in adults
  - Improvements in lung function growth with declining $PM_{2.5}$ concentrations
  - Consistent evidence for increased risk of respiratory mortality
- Experimental evidence:
  - Impaired lung development and development of allergic airway disease
  - Biological plausibility for decrements in lung function growth in children and asthma development
- Remaining Uncertainties:
  - Limited evidence from animal toxicological studies
  - Limited assessment of potential copollutant confounding

Nervous System Effects

- Long-term $PM_{2.5}$ Exposure **(Likely to be Causal – NEW conclusion)**
  - Epidemiologic evidence:
    - Consistent evidence for cognitive decline/impairment and decreased brain volume
    - Limited evidence for neurodegeneration (e.g., 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
  - Remaining Uncertainties:
    - Challenge conducting epidemiologic studies of neurodegeneration because often a genetic component
    - Epidemiologic studies of neurodevelopmental effects limited due to the small number of studies, and uncertainty regarding critical exposure windows
    - Limited assessment of potential copollutant confounding

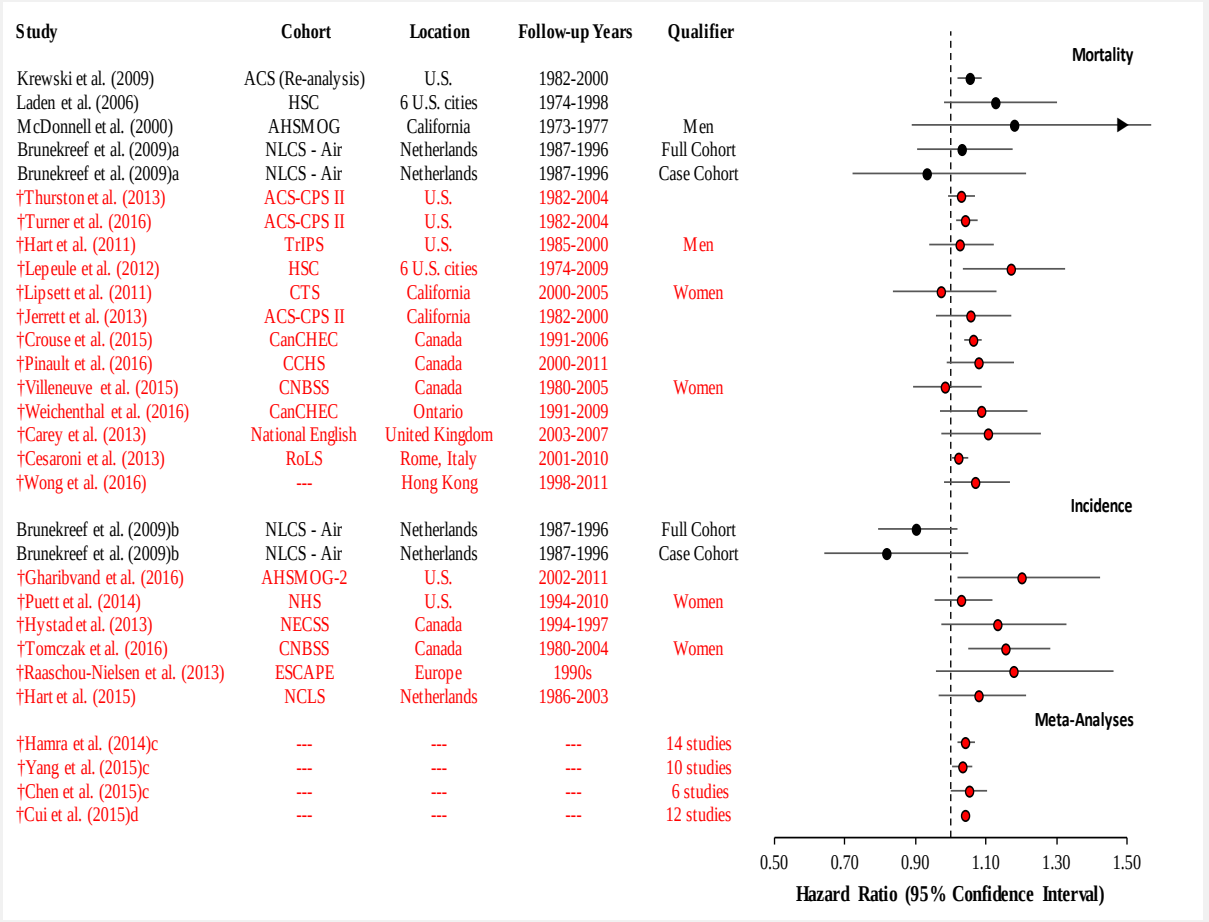
Nervous System Effects

- 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
  - Remaining Uncertainties:
    - Relative lack of epidemiologic studies
    - Inconsistency in size range of UFPs examined across disciplines
    - Spatial and temporal variability in UFP concentrations
    - Relative lack of UFP monitoring data
    - Long-term exposure to UFPs

Cancer

Long-term $PM_{2.5}$ Exposure (Likely to be Causal – NEW conclusion)

- Decades of research on whole PM exposures:
  - Genotoxicity
  - Epigenetic effects
  - Carcinogenic potential
  - Characteristics of carcinogens
- Experimental and epidemiologic studies examining $PM_{2.5}$ support:
  - Genotoxicity
  - Epigenetic effects
  - Carcinogenic potential
  - Characteristics of carcinogens
- Epidemiologic evidence:
  - Lung cancer incidence and mortality
- Remaining Uncertainties:
  - Inconsistency in specific cancer-related biomarkers across disciplines
  - Limited assessment of copollutant confounding



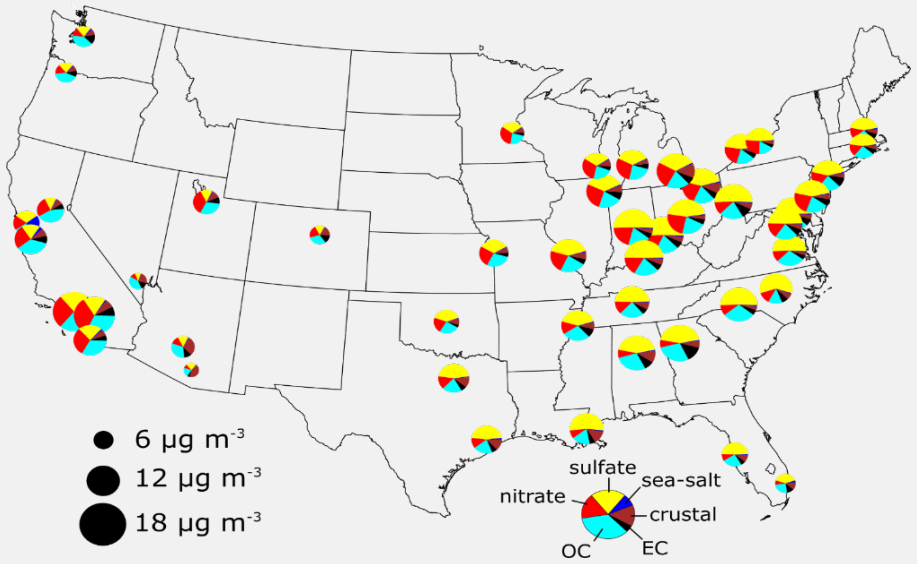
Note: Red = recent studies; Black = studies evaluated in the 2009 PM ISA

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.

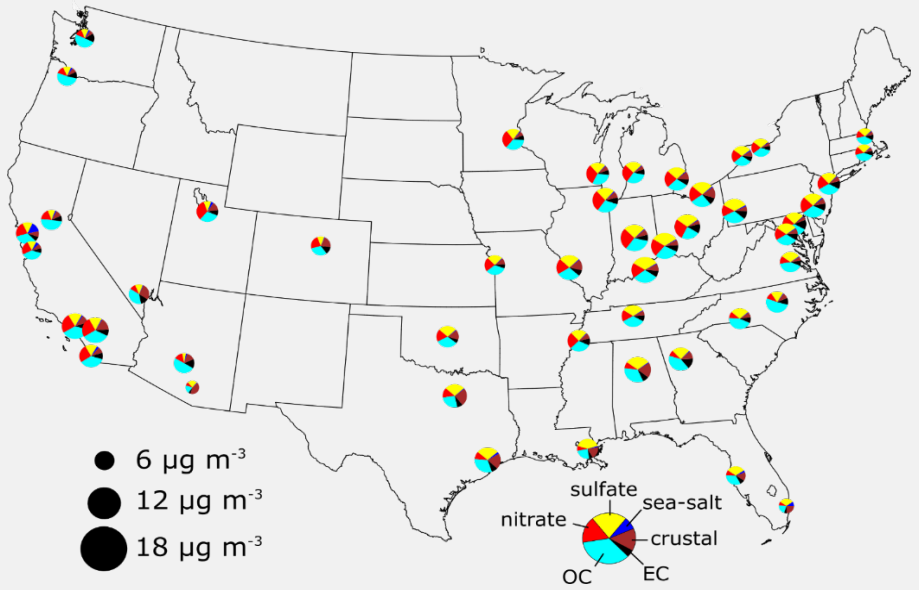
PM Components and Sources

- Conclusion:
  - 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
    - Evaluation of individual components, based largely on evidence from epidemiologic studies
    - Evaluation of sources limited to a smaller subset of studies
      - Across studies, consistent evidence for effects with various combustion-related sources (e.g., industrial activities, traffic, wildfires, biomass burning, etc.)

National Trend in $PM_{2.5}$ Component Concentrations



2003 - 2005



2013 - 2015

- 2003 - 2005: As % of total mass, sulfate higher in East; OC in West
- 2013 – 2015: Reduction in sulfate contribution in East; contributions similar to 2003 – 2005 in West
- Overall: Organic carbon has replaced sulfate as the most abundant component of $PM_{2.5}$ in many locations, specifically in the eastern U.S.

Example: $PM_{2.5}$ Components and Cardiovascular Effects

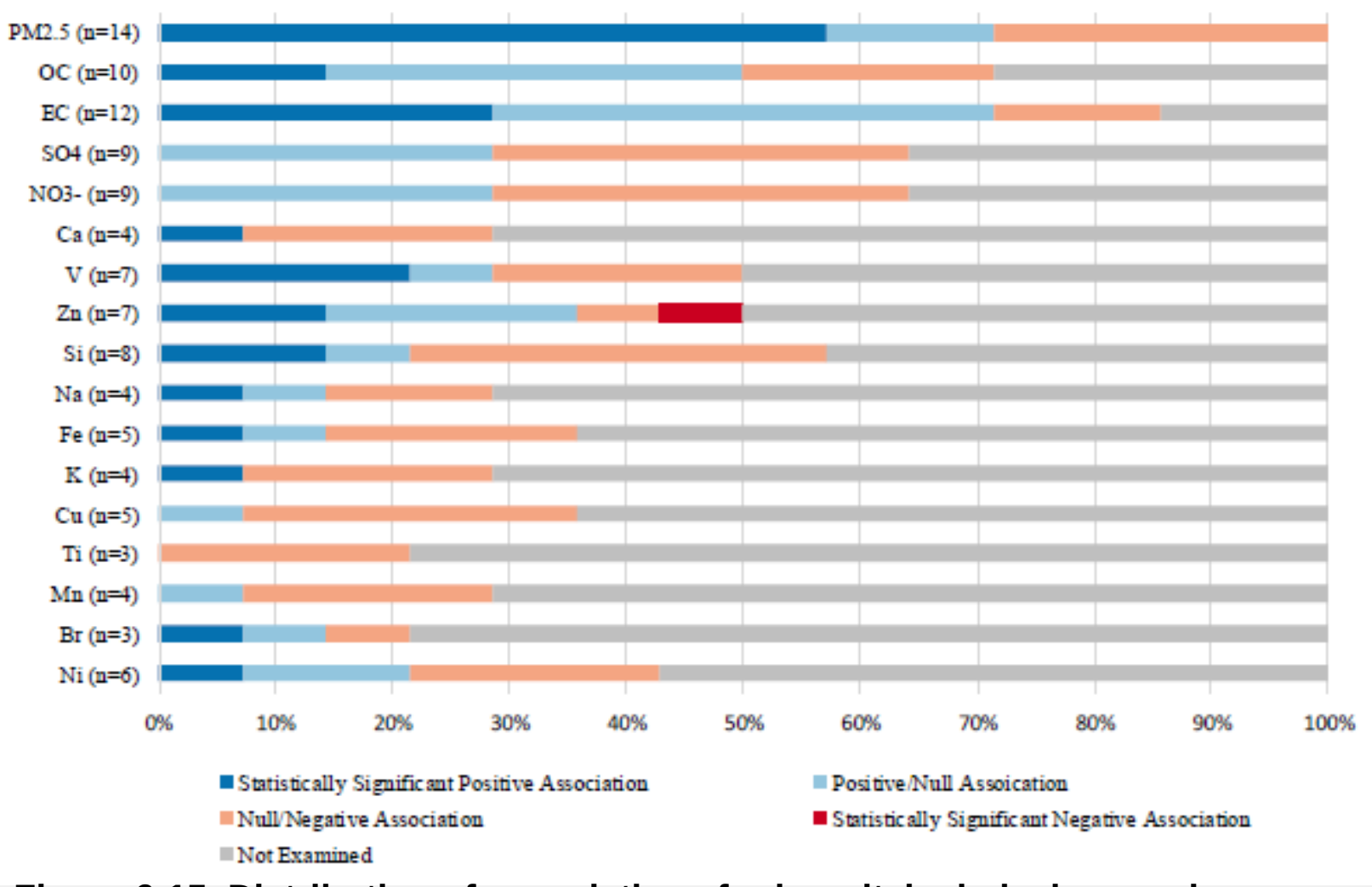


Figure 6-15. Distribution of associations for hospital admissions and emergency department visits for cardiovascular-related effects and short-term $PM_{2.5}$ and $PM_{2.5}$ components exposure.

Populations Potentially at Increased Risk of a PM-related Health Effect

- 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 transferase pathways, low SES
  - Inadequate: pre-existing diabetes, older adults, residential location, sex, diet, and physical activity

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Supplemental Materials

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) 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 are not adequately addressed and 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 study shows effects relevant to humans in animal 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

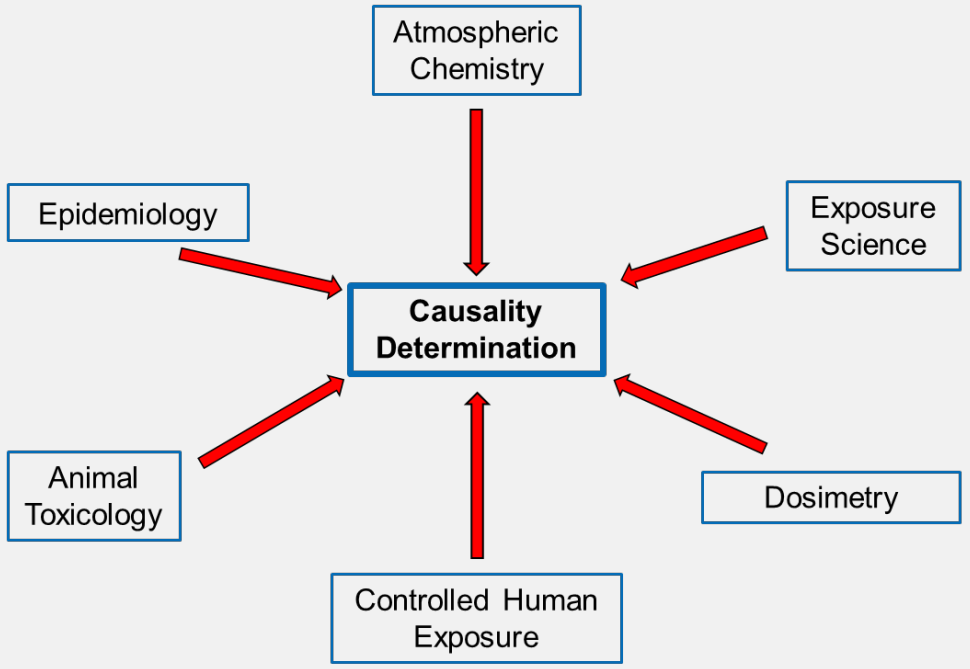
Evidence is suggestive but limited

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

Multiple studies show no effect across exposure concentrations

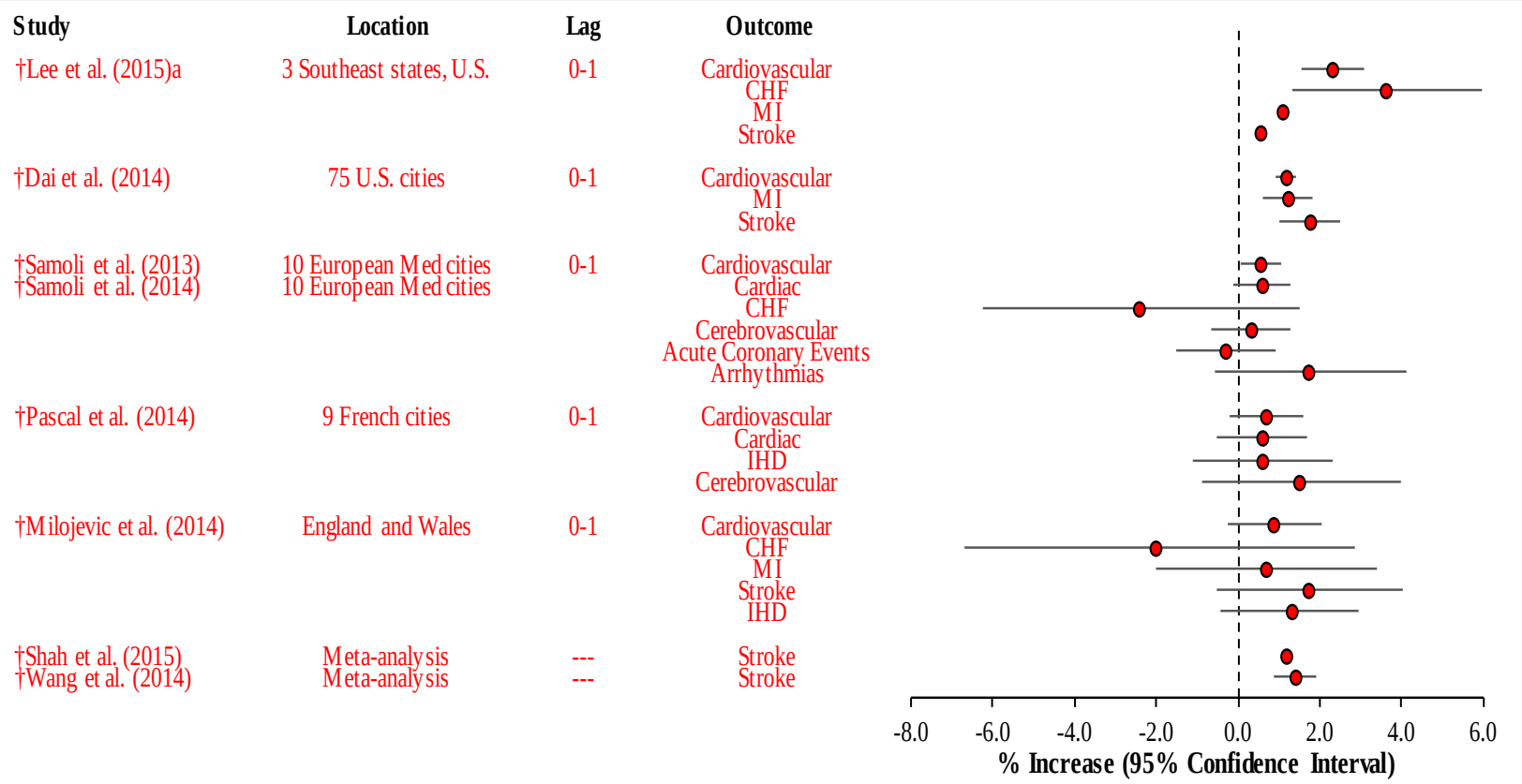
Evaluation of the Scientific Evidence

- Organize relevant literature for broad outcome categories
- Evaluate studies, characterize results, extract relevant data
- Integrate evidence across disciplines for 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



Cardiovascular Effects

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

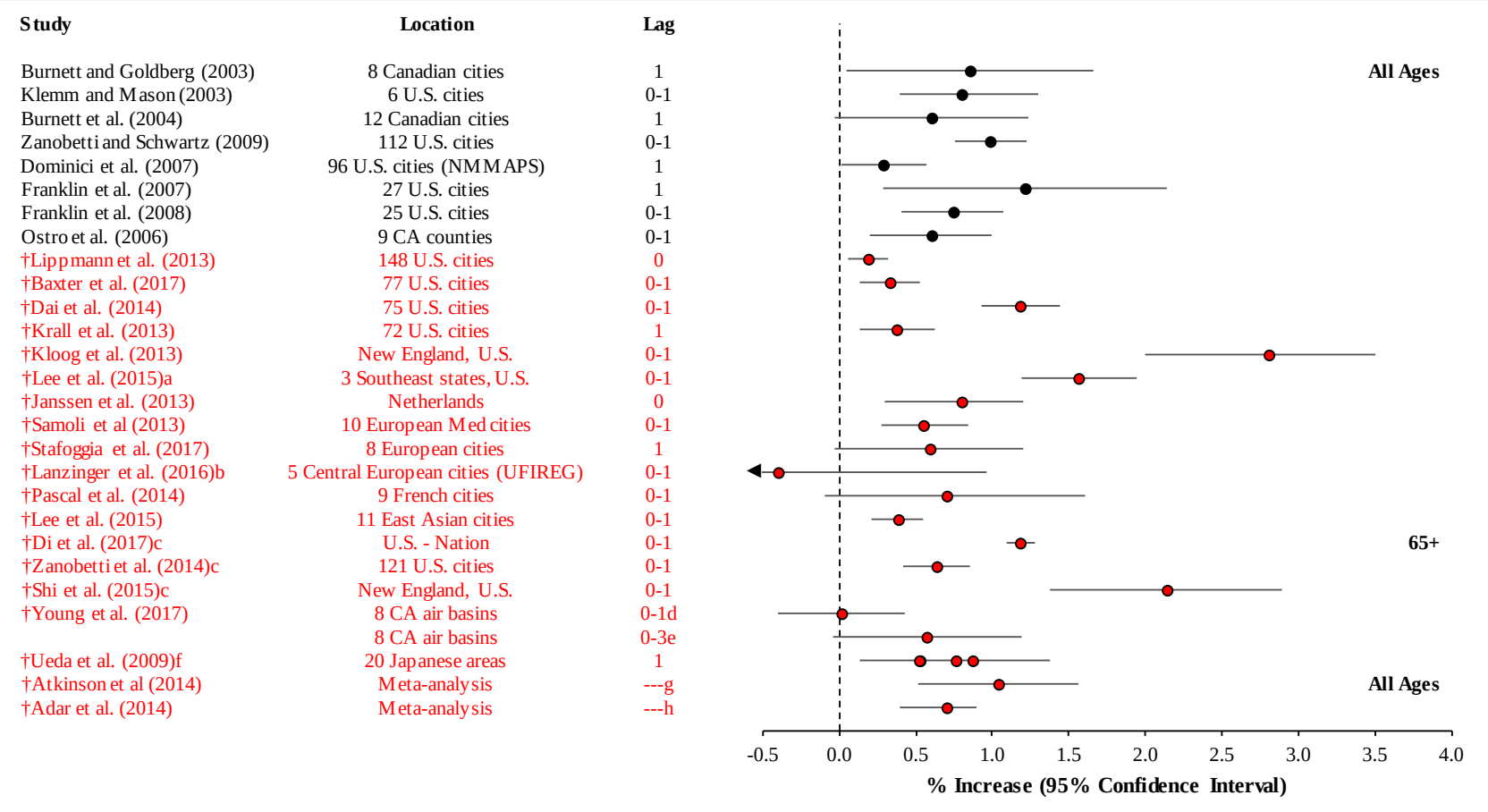


Note: Red = recent studies; Black = studies evaluated in the 2009 PM ISA

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.

Mortality – Short-term $PM_{2.5}$ Exposure

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

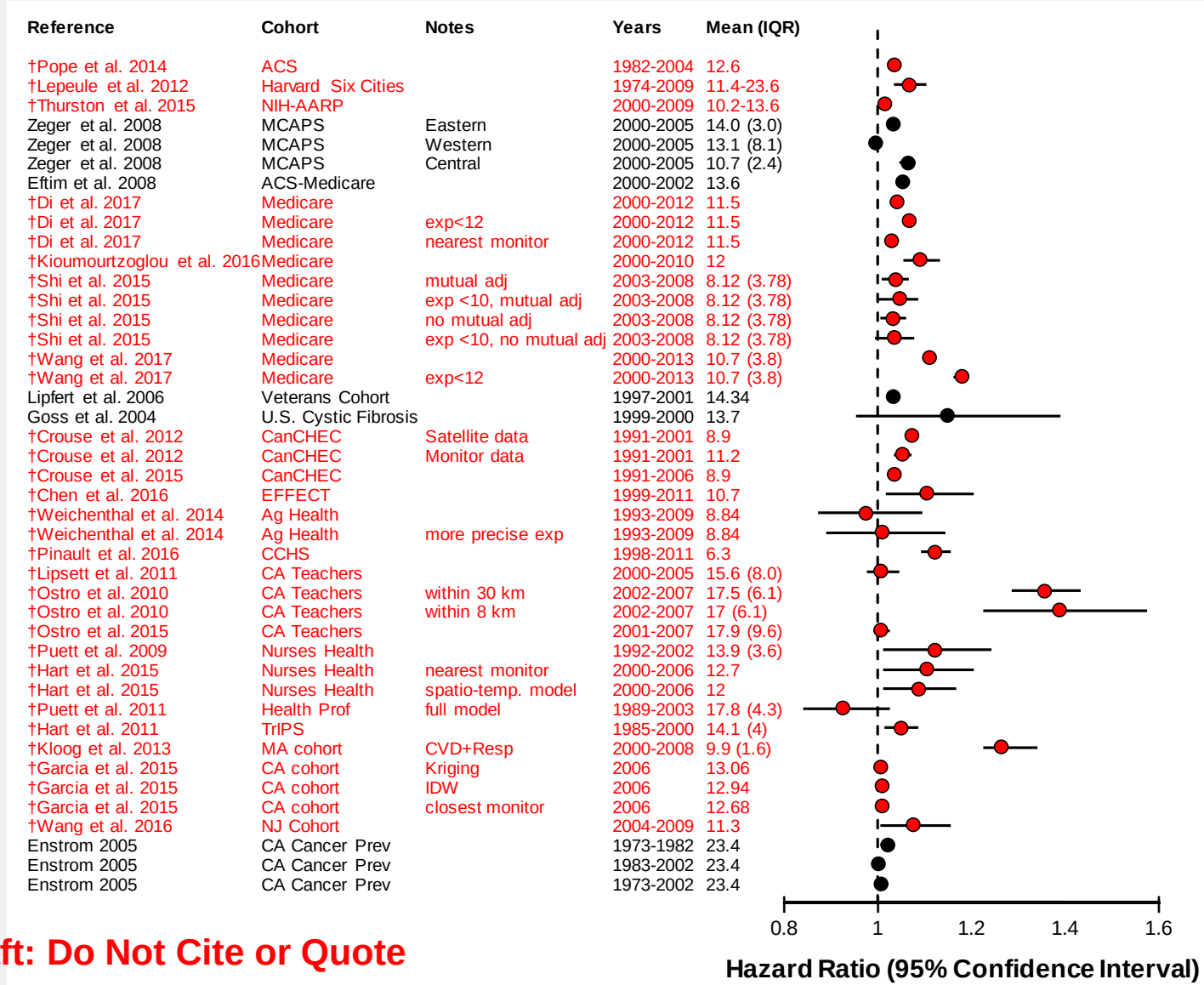
Mortality – Long-term $PM_{2.5}$ Exposure

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

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

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



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

$PM_{2.5}$ Components and Respiratory Effects

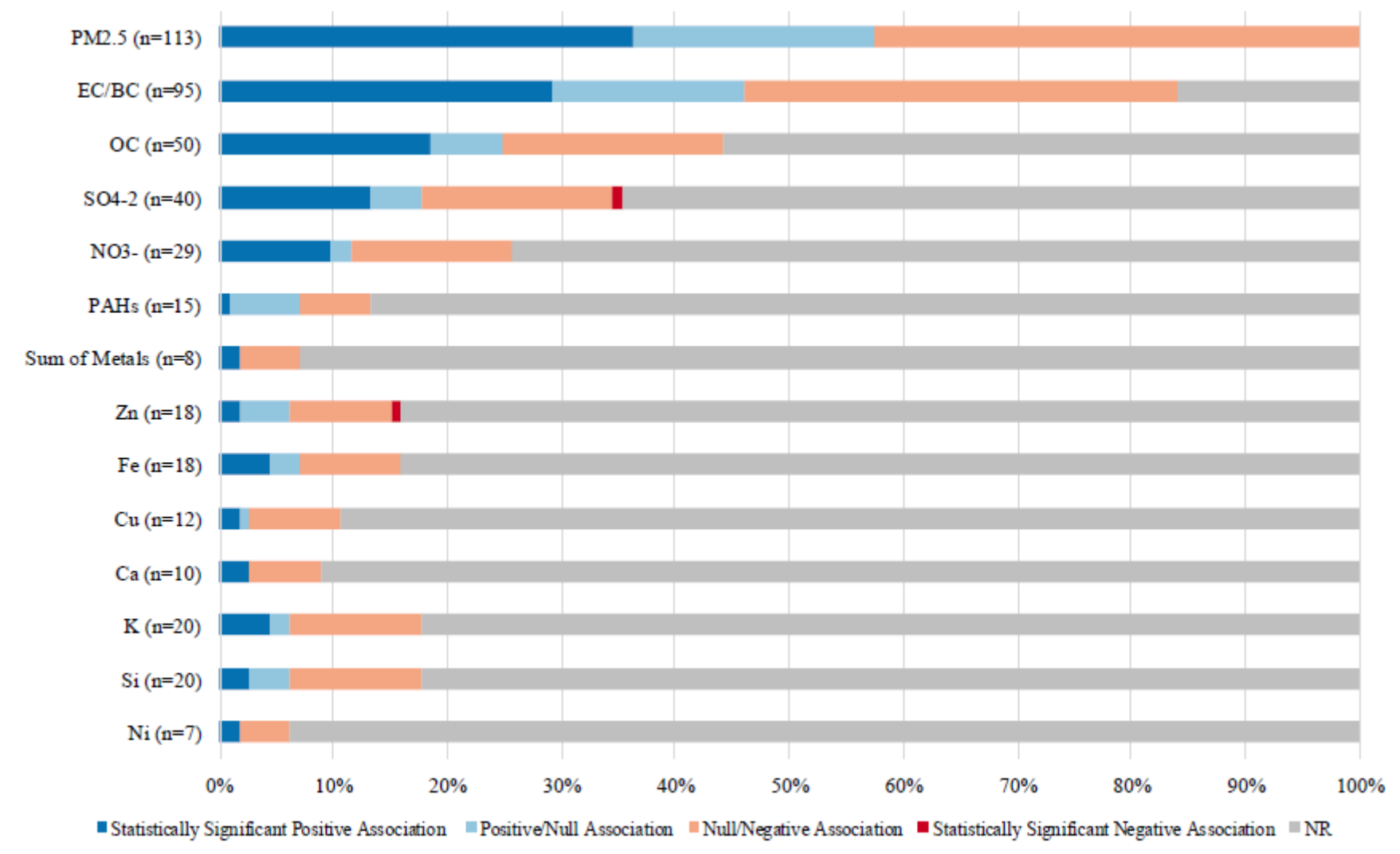


Figure 5-25. Distribution of associations for all respiratory effects and short-term $PM_{2.5}$ mass and $PM_{2.5}$ components exposure.

$PM_{2.5}$ Components and Mortality

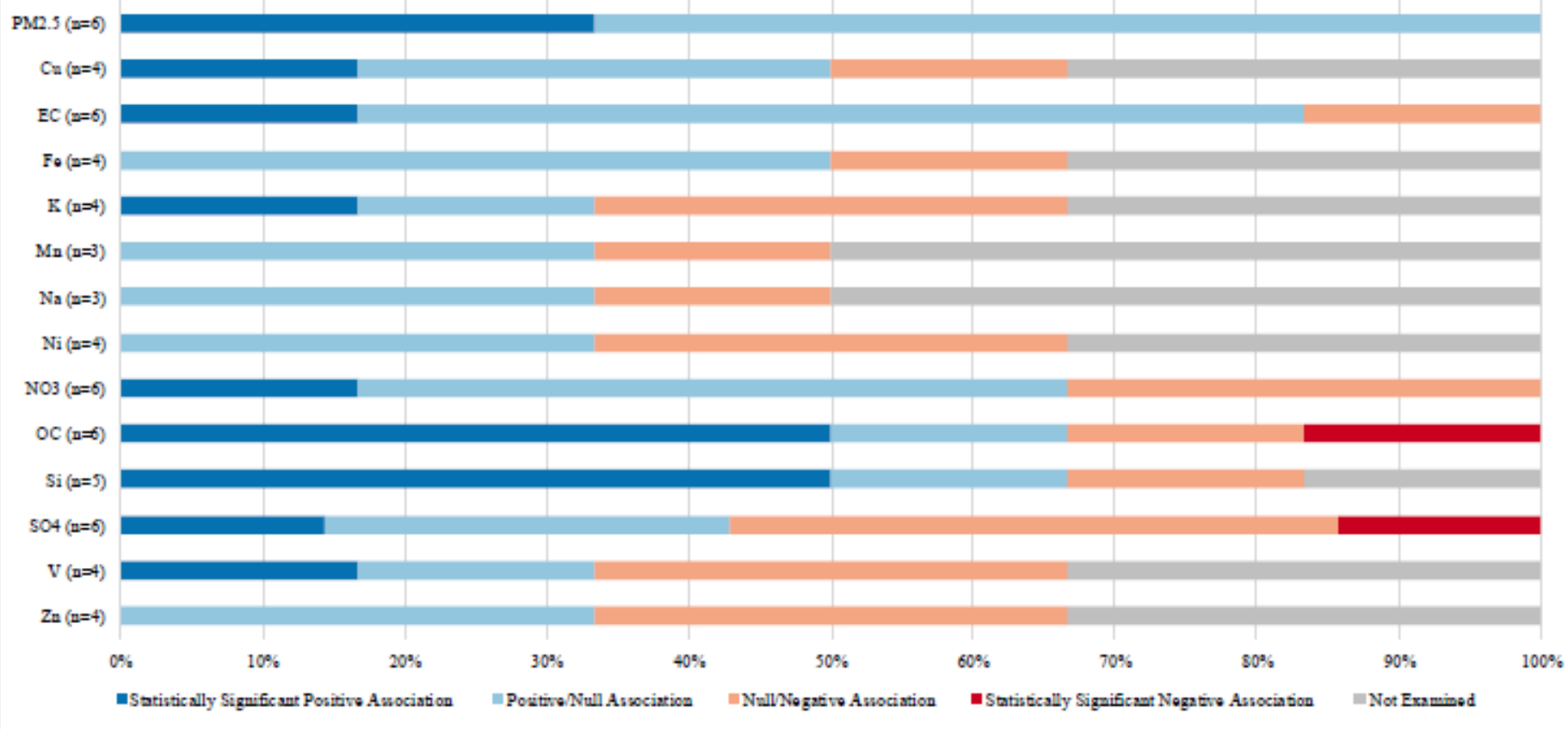
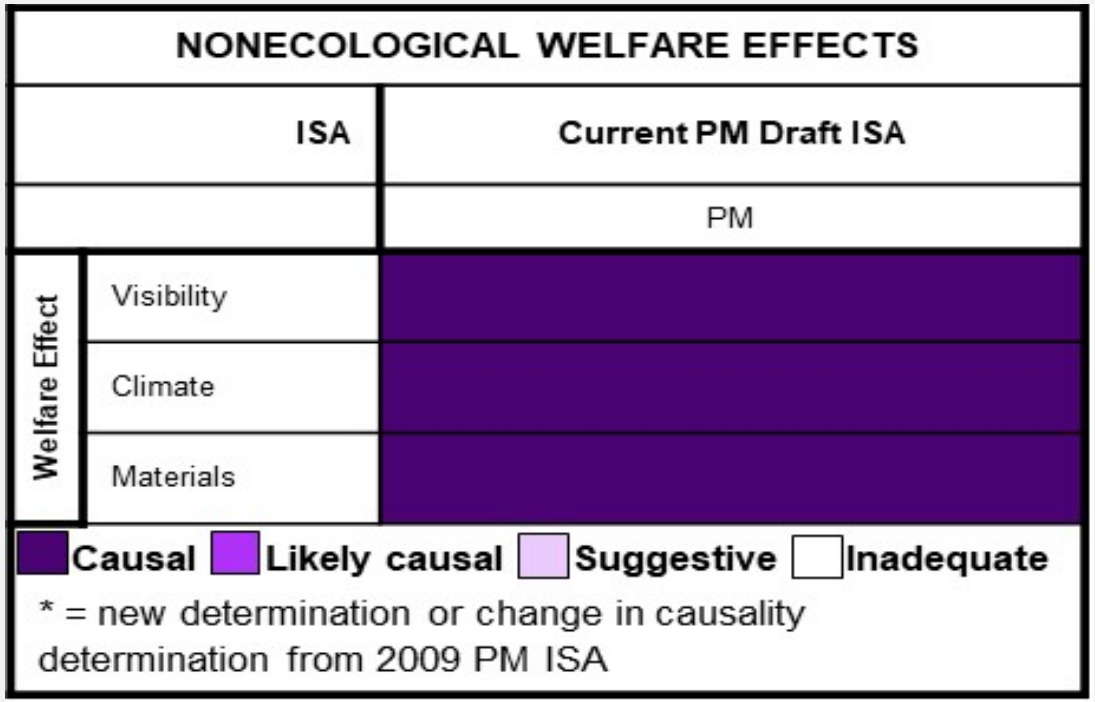


Figure 6-15. Distribution of total (nonaccidental) mortality associations for short-term $PM_{2.5}$ and $PM_{2.5}$ components exposure.

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

Draft PM ISA
Welfare Effects: Causality Determinations



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

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

Particulate Matter:
Spotlight on Health Protection

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PARTICULATE MATTER: A COMPLEX MIXTURE THAT AFFECTS HEALTH

Michael T. Kleinman

With the help of David Herman, Rebecca Johnson, Lisa Wingen and a lot of other people

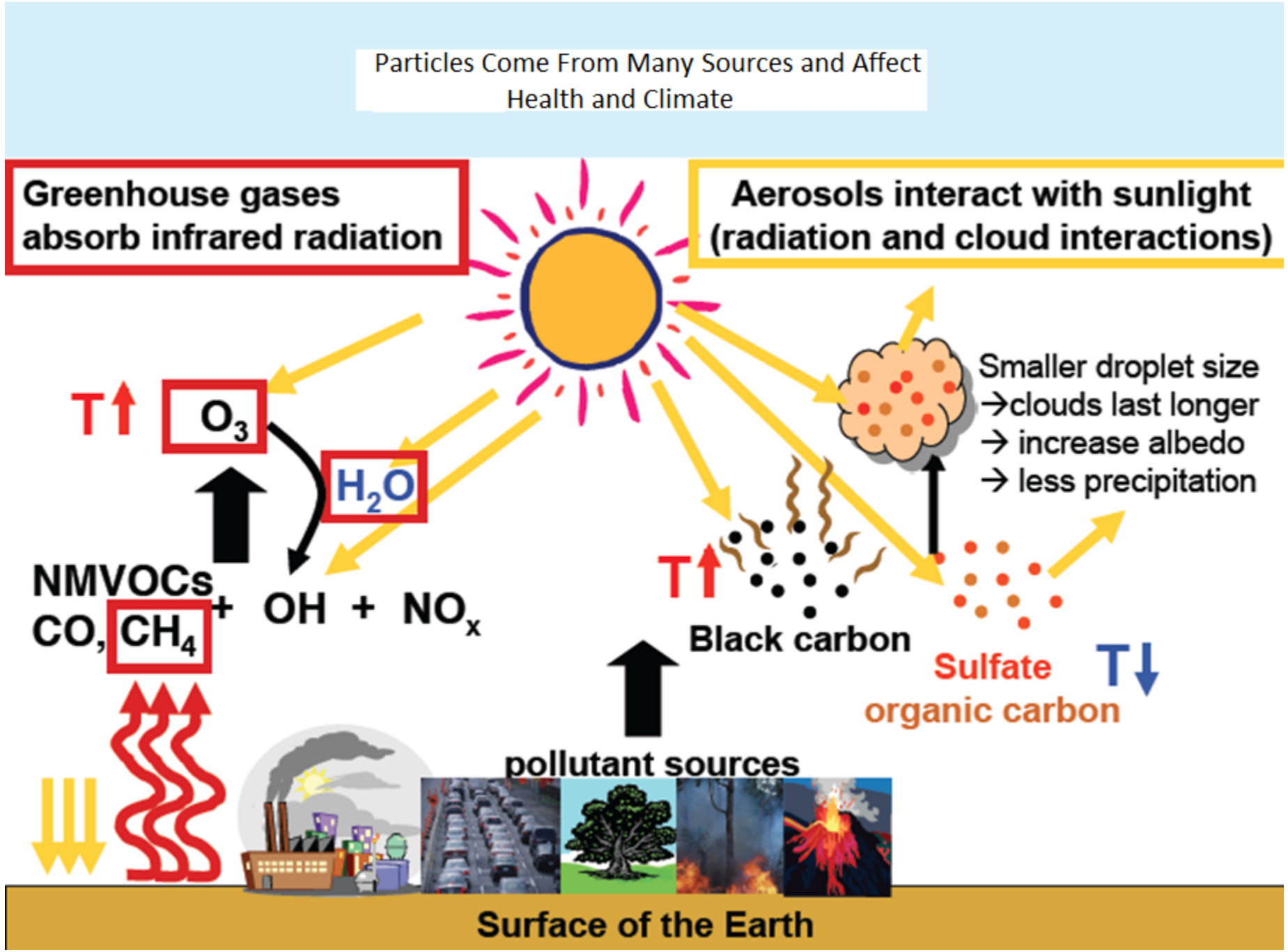
University of California, Irvine

Overall Goal of this Presentation is to Address These Questions

- Why are some species of PM more dangerous than others?
- How does PM affect health?
- Do ultrafine particles (UFPs) have a special role?

What are the health-relevant components of urban air?

- Emissions from power plants, motor vehicles, dust.
- Pollutants gases:
  - Ozone and NO_2 are major problems in California.
  - SO_2 and organic vapors are also important.
- Particles or Particulate Matter (PM):
  - Particles are associated with increased heart-related deaths during air pollution episodes.
  - Toxicology studies show that PM2.5 accelerates the development of atherosclerosis.
  - The strongest associations with human heart-related illness and death are with PM.
  - PM composition includes toxic organic and inorganic chemicals
- Combustion sources generate fine and ultrafine PM often coated with toxic substances.
  - Polycyclic Aromatic Hydrocarbons (PAHs)
  - Carbonyls (acrolein, formaldehyde)
  - Quinones


Particles Come From Many Sources and Affect Health and Climate
Greenhouse gases absorb infrared radiation
Aerosols interact with sunlight (radiation and cloud interactions)
T↑
O_3
H_2O
NMVOCs
CO, CH_4
+ OH + NO_x
Smaller droplet size
→clouds last longer
→ increase albedo
→ less precipitation
T↑
Black carbon
Sulfate
organic carbon
T↓
pollutant sources
Surface of the Earth

Fine (PM2.5) and ultrafine particles (UFP) are the most biologically active

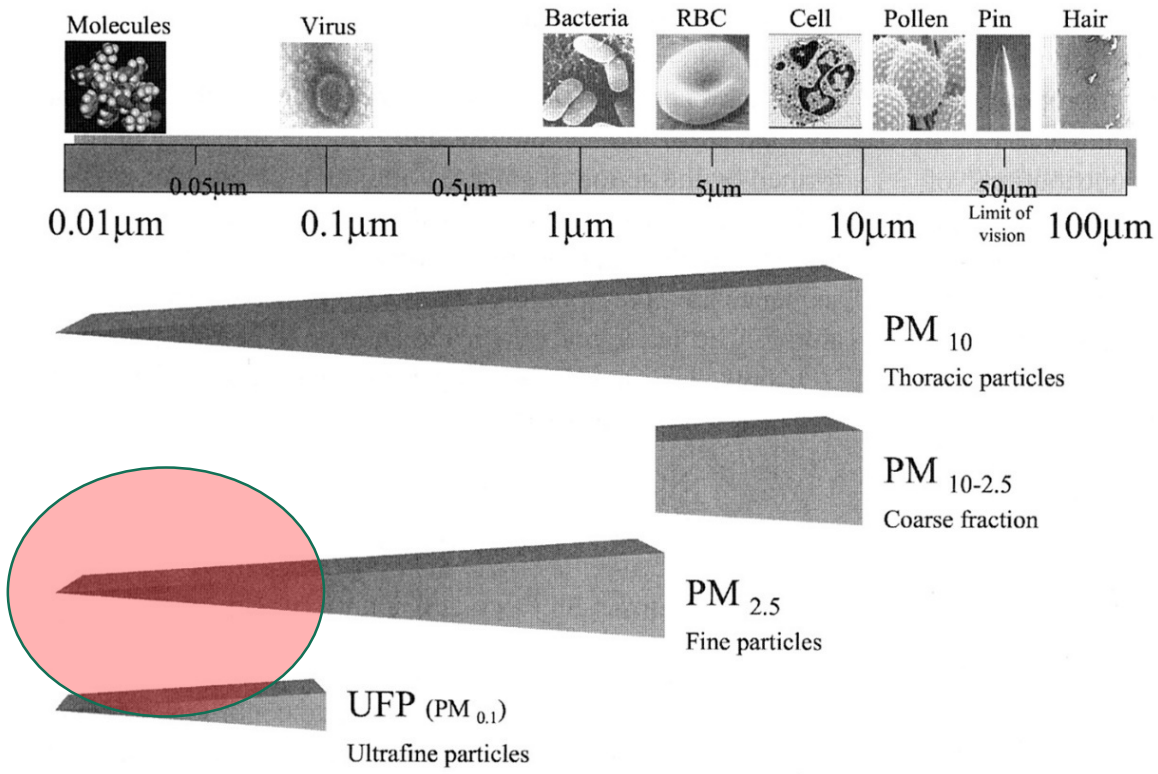


Figure 1. Particulate matter air pollution size distribution.

Combustion Sources Produce Toxic Air Contaminants

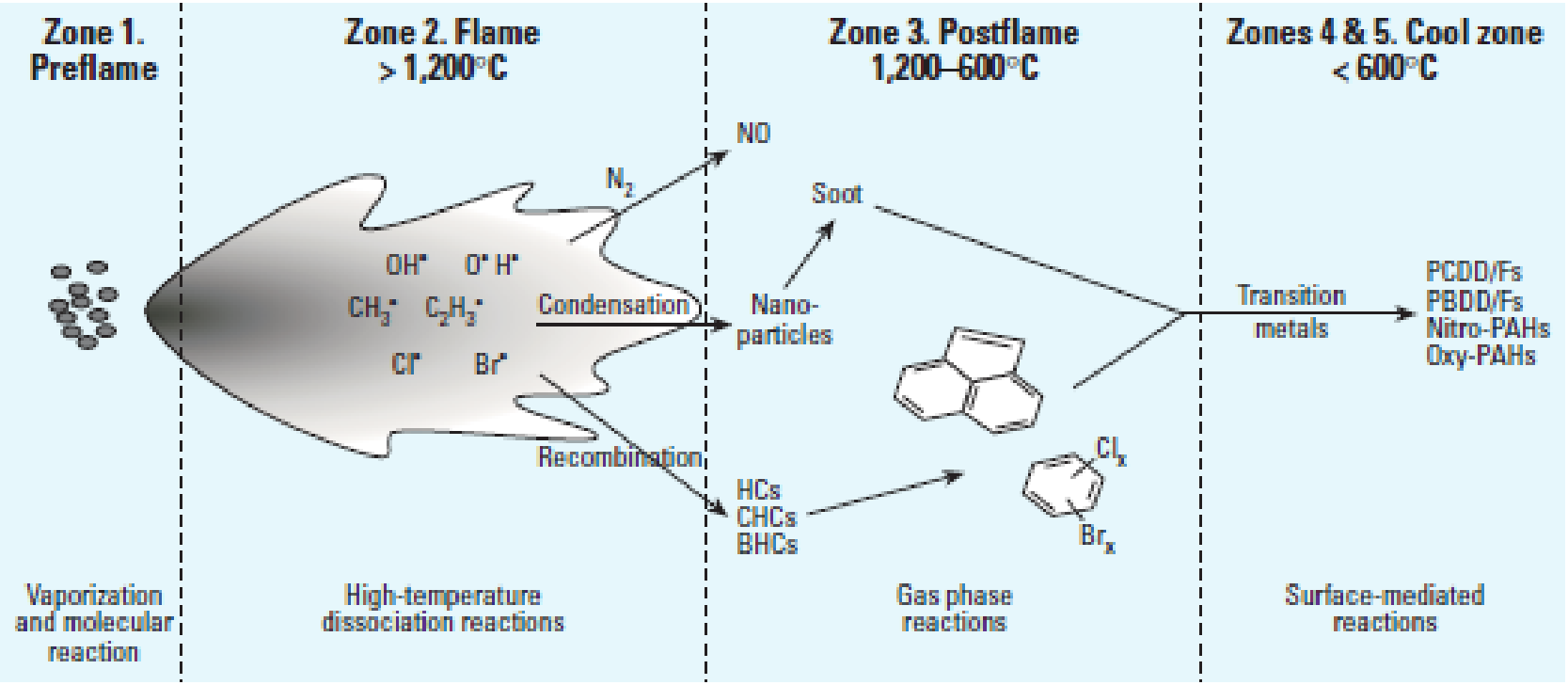


Figure 1. Combustor reaction zones. Zone 1, preflame, fuel zone; zone 2, high-temperature, flame zone; zone 3, postflame, thermal zone; zone 4, gas-quench, cool zone; zone 5, surface-catalysis, cool zone. PBDD/Fs, polybrominated dibenzo-*p*-dioxins and dibenzofurans. Reaction products from upstream zones pass through downstream zones and undergo chemical modifications, resulting in formation of new pollutants. Zone 2 controls formation of many "traditional" pollutants (e.g., carbon monoxide, sulfur oxides, and nitrogen oxides). Zones 3 and 4 control formation of gas-phase organic pollutants. Zone 5 is a major source of PCDD/Fs and is increasingly recognized as a source of other pollutants previously thought to originate in zones 1–4.

Origin and Health Impacts of Emissions of Toxic By-Products and Fine Particles from Combustion and Thermal Treatment of Hazardous Wastes and Materials

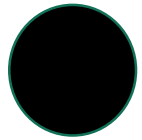
Stephania A. Cormier,[1] Slawo Lomnicki,[2] Wayne Backes,[3] and Barry Dellinger[2]

[1]Department of Biological Science, and [2]Department of Chemistry, Louisiana State University, Baton Rouge, Louisiana, USA; [3]Department of Pharmacology, Louisiana State University Health Sciences Center, Baton Rouge, Louisiana, USA

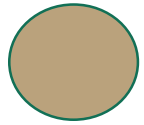
PM2.5 and UFP From Combustion Sources is a Mixture of Solid and Liquid Droplets that we call “SOOT”

- Black carbon (BC) is a major component of “soot”, a complex light-absorbing mixture that comprised of a mixture of Elemental Carbon (EC) and Particulate Organic Carbon (OC).
- BC is the most strongly light-absorbing component of particulate matter (PM), and is formed by the incomplete combustion of fossil fuels, biofuels, and biomass.
- BC is emitted directly into the atmosphere in the form of fine particles ($PM_{2.5}$) and ultrafine particles ($PM_{0.1}$). These are also considered nanoparticles.
- BC is the most effective form of PM, by mass, at absorbing solar energy: per unit of mass in the atmosphere, BC can absorb a million times more energy than carbon dioxide (CO_2).
- Organic carbon aerosols are a significant absorber of solar radiation. The absorbing part of organic aerosols is referred to as "brown" carbon (BrC).

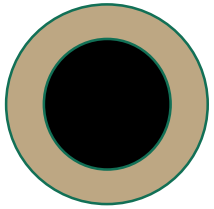
EC



OC + BrC



BC



http://www.epa.gov/blackcarbon/basic.html

1 in 6 deaths, worldwide, is attributable to Pollution

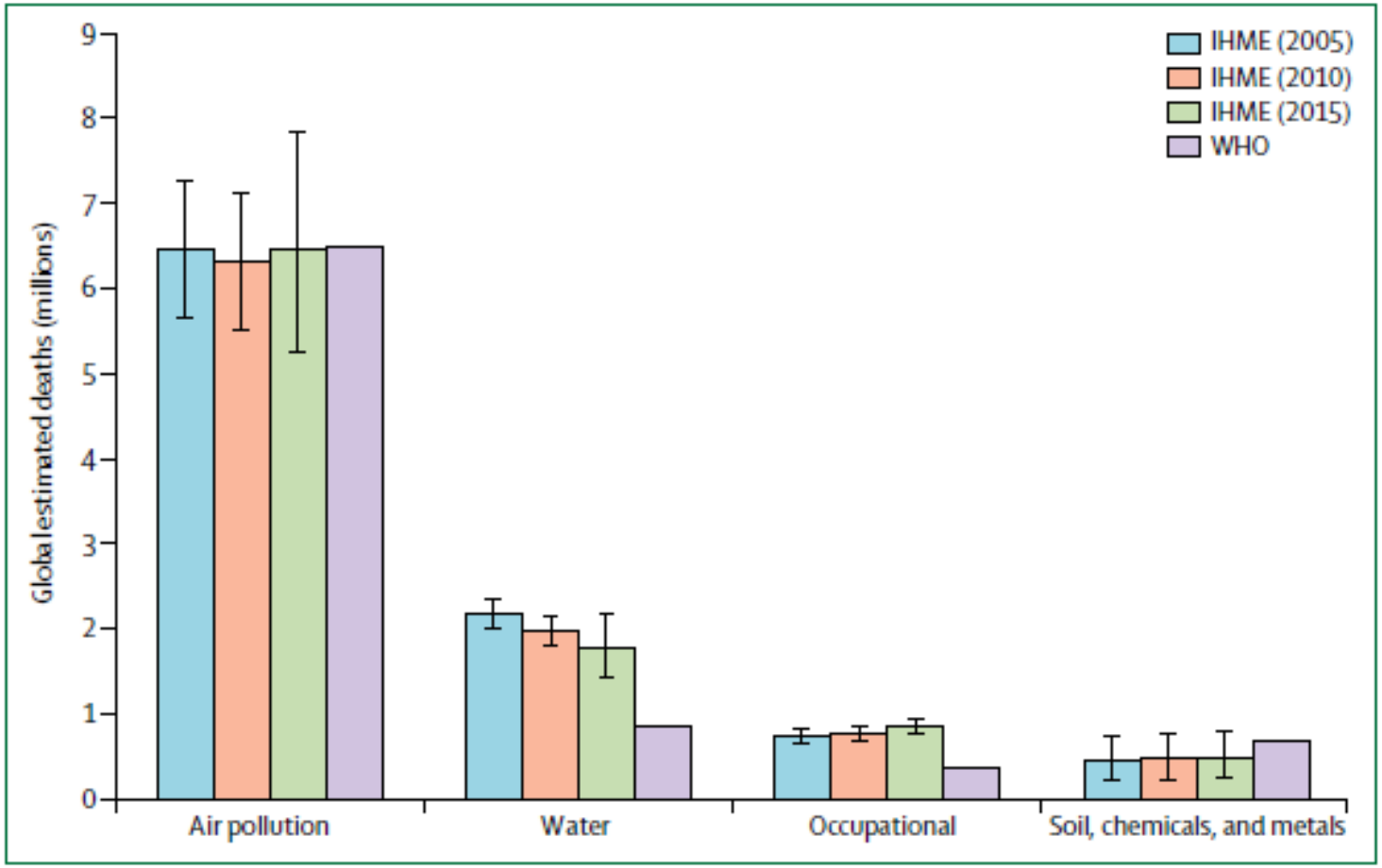


Figure 4: **Global estimated deaths (millions) by pollution risk factor, 2005–15**
Using data from the GBD study[42] and WHO.[99] IHME=Institute for Health Metrics and Evaluation.

Air Pollution Contributes to Multiple Diseases

The *Lancet* Commission on pollution and health, Lancet, October 2017

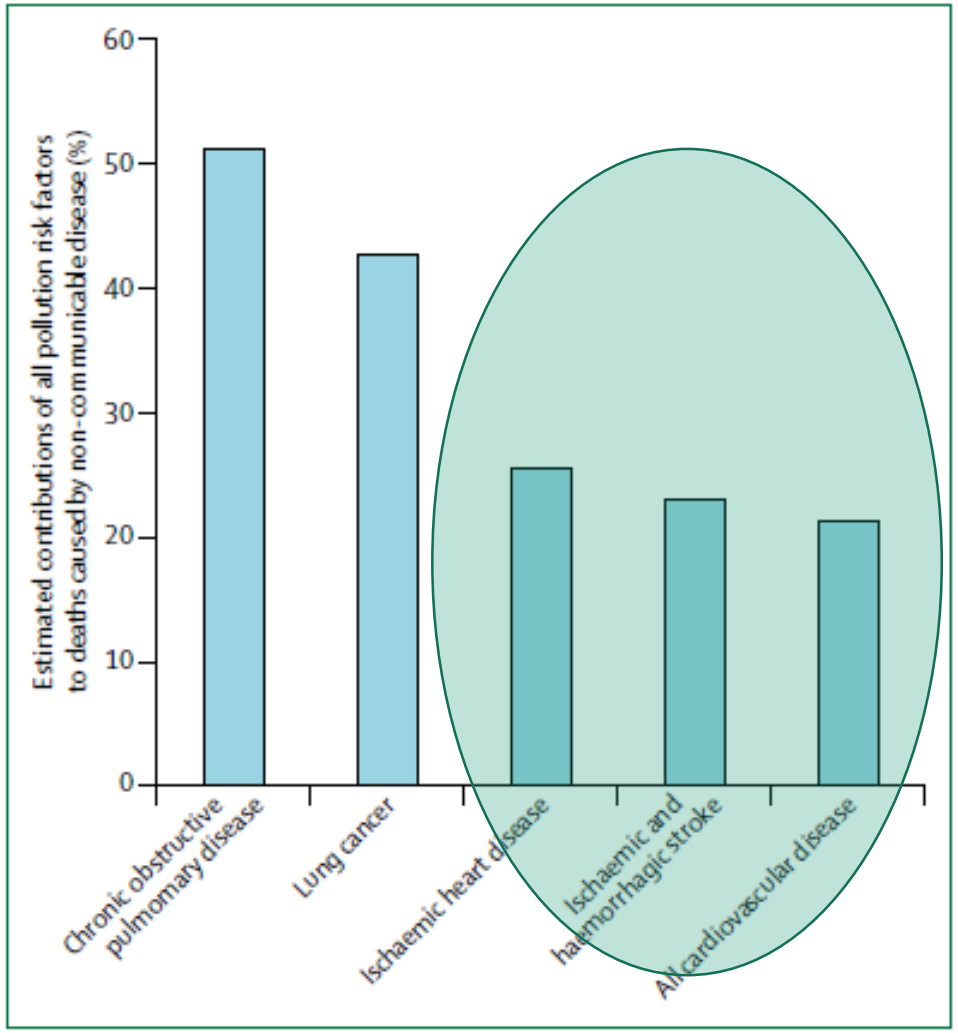
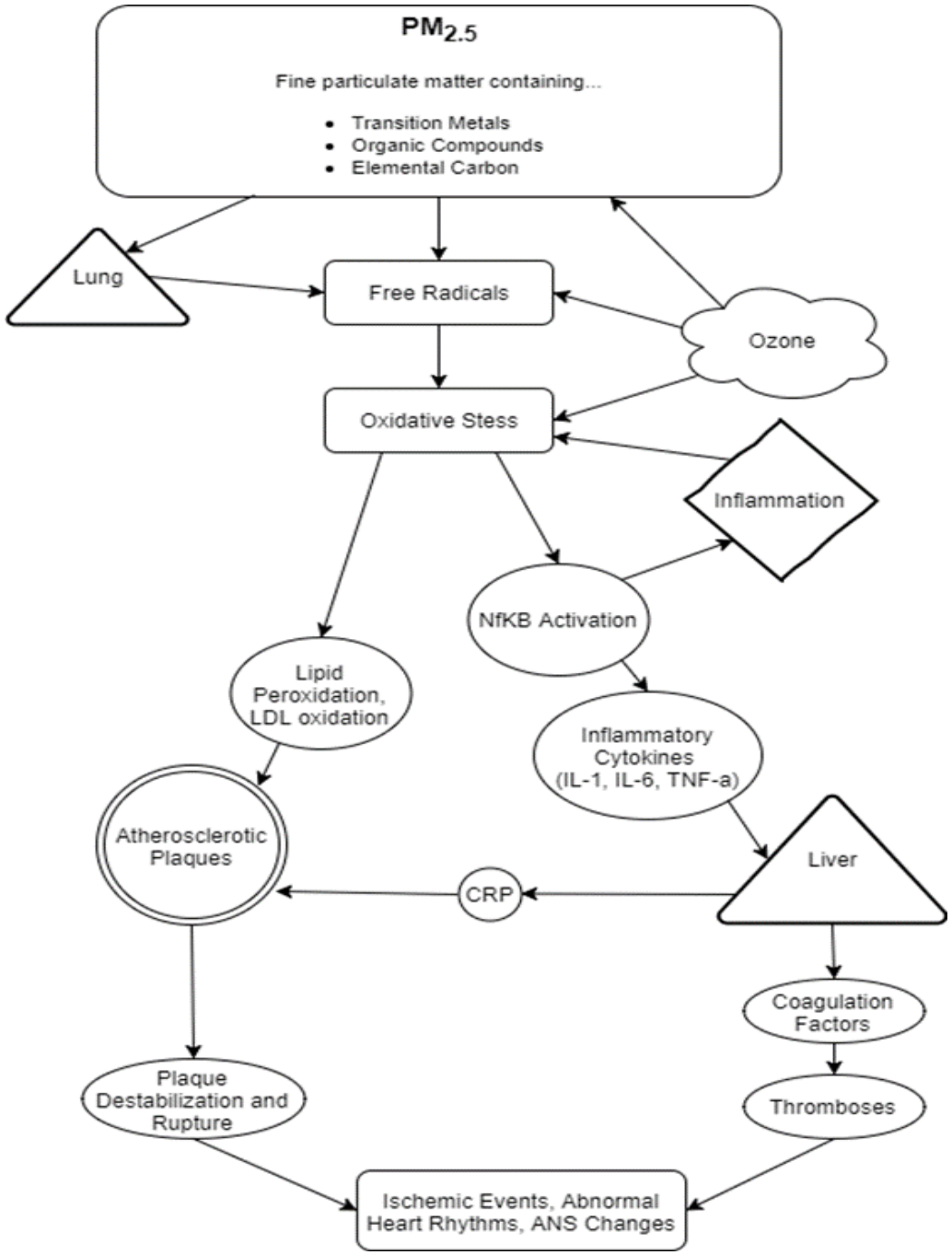


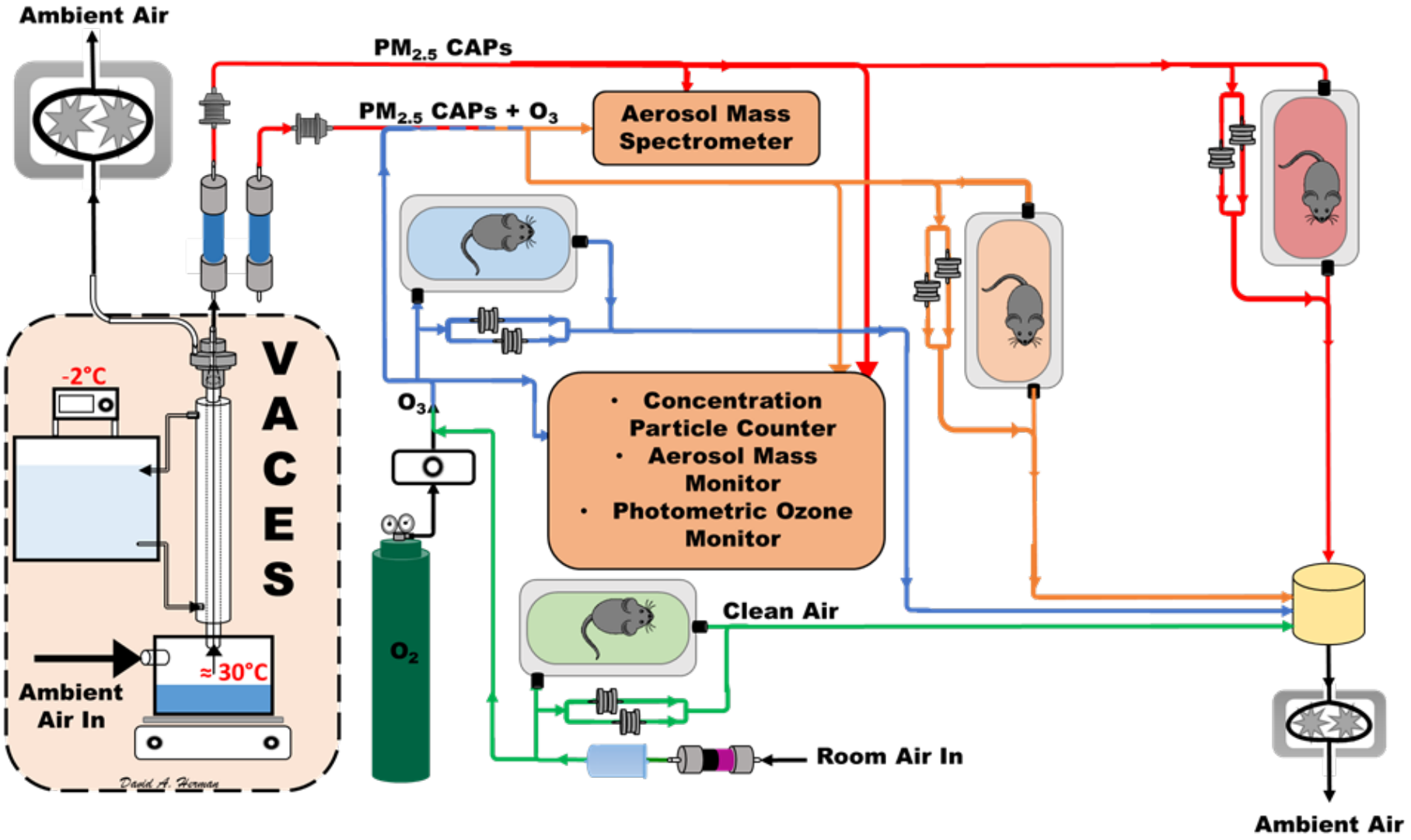
Figure 6: **Estimated contributions of all pollution risk factors to deaths caused by non-communicable diseases, 2015**

GBD Study, 2016.[42]

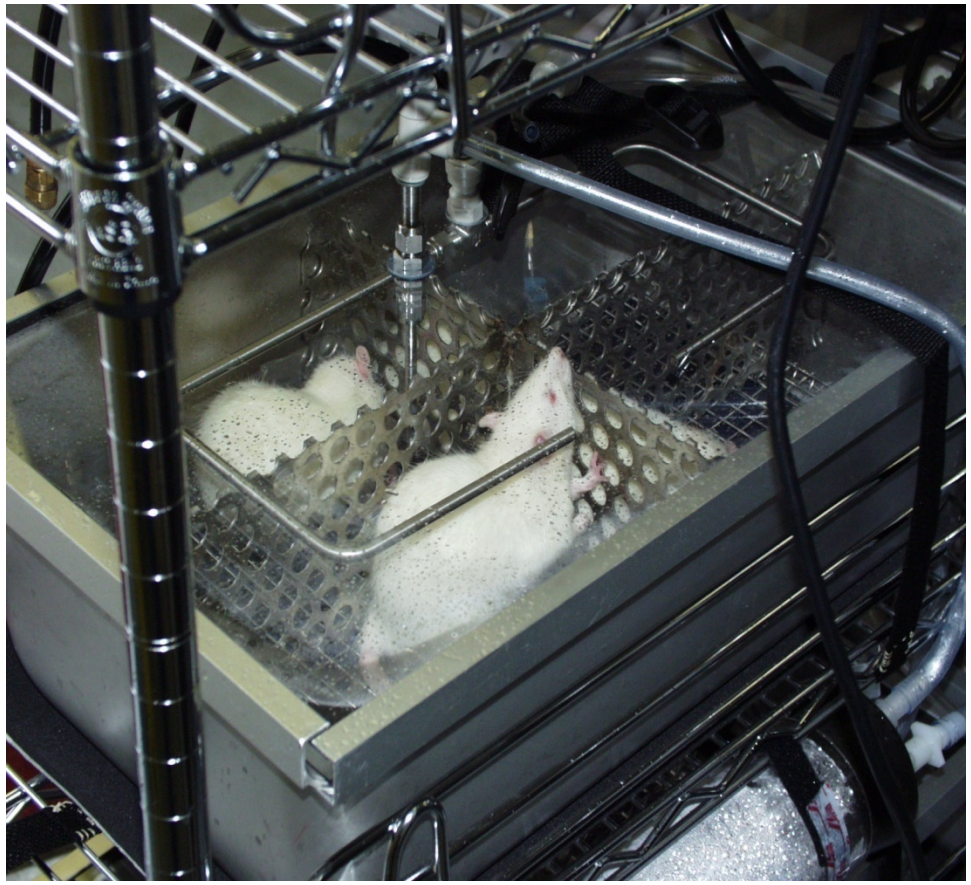
A Mechanistic Framework for PM2.5 Effects Leading to Cardiovascular Disease



We can examine the health effects of specific pollutants using controlled exposures and help understand the mechanisms by which PM causes or worsens cardiovascular diseases.



Rats or Mice Can
Be Exposed to
Purified Air or CAPs
in Sealed Chambers



The Sealed Chambers
Can Be Placed Onto
Racks to Facilitate
Transport

ECG and Blood Pressure Telemetry Devices can be Implanted to provide physiology data before, during and after exposures.

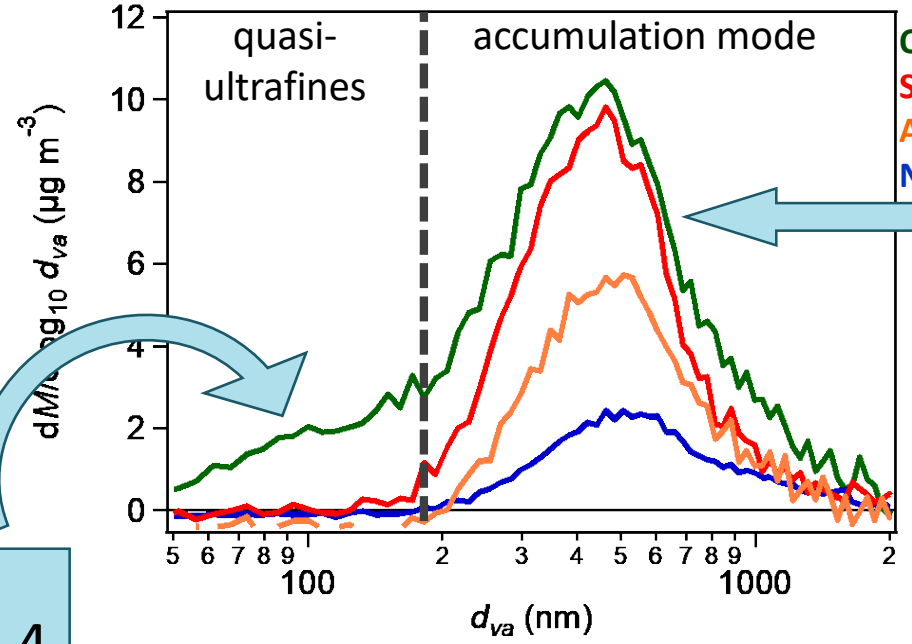
Exposure Protocol

- ApoE-/- mice were surgically implanted with ECG telemetry devices.
- Mice were exposed 5 hr per day (8AM to 1 PM) 4 days per week for 8 weeks at UC Irvine and were housed in filtered air-supplied caging systems between exposures.
- ECG data were monitored during exposures and while the mice were in housing (21 hr / day).
- All animal protocols were approved by the Institutional Animal Care and Use Committee.

What Happens When You Denude Quasi-Ultrafine CAPs (d_p < 180 nm)?

- Particle number and mass are reduced.
- Refractory constituents, such as heavy metals and elemental carbon, were only marginally affected by heating.
- Labile species such as total and water soluble organic carbon and PAHs showed progressive loss in concentration with increase in TD temperature.

Health-related characteristics of Ultrafine PM



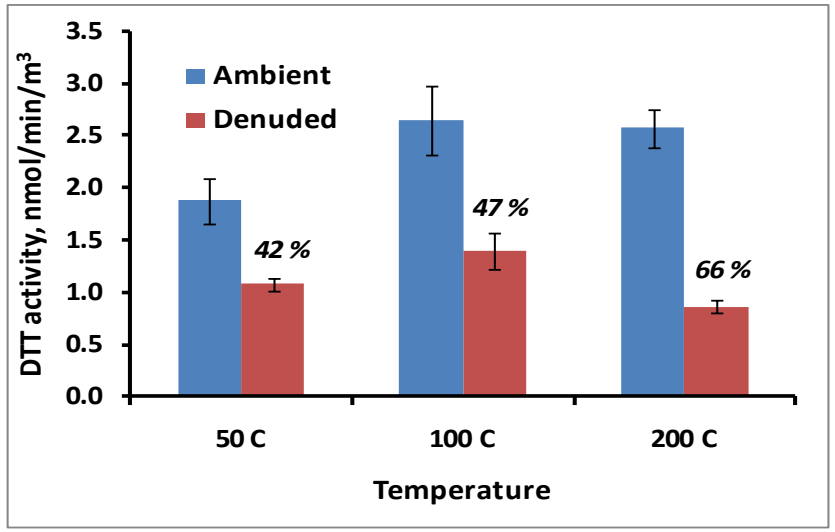
ultrafines
less oxygenated
(to denuder)

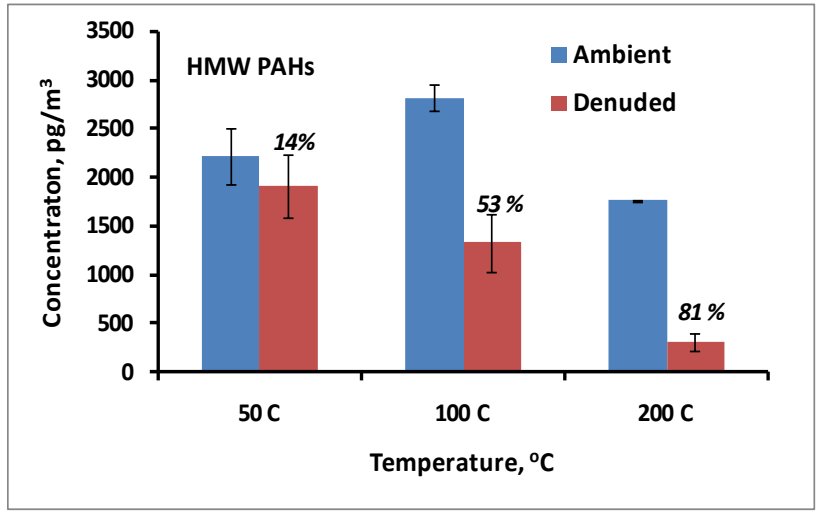
$$\frac{m/z\ 44\ (CO_2^+)}{m/z\ 55\ (C_4H_7^+)} \cong 0.4$$

larger particles
oxygenated

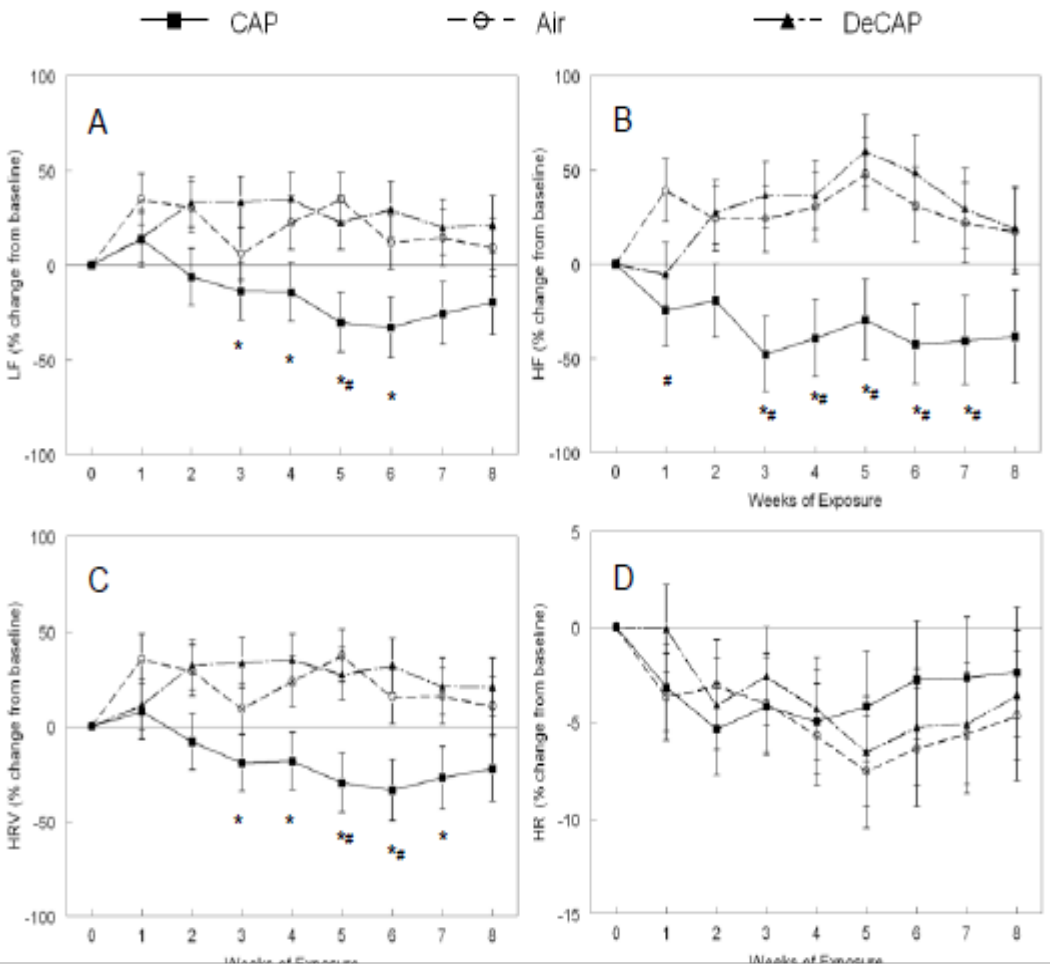
$$\frac{m/z\ 44\ (CO_2^+)}{m/z\ 55\ (C_4H_7^+)} \cong 4$$

When you denude the UFP

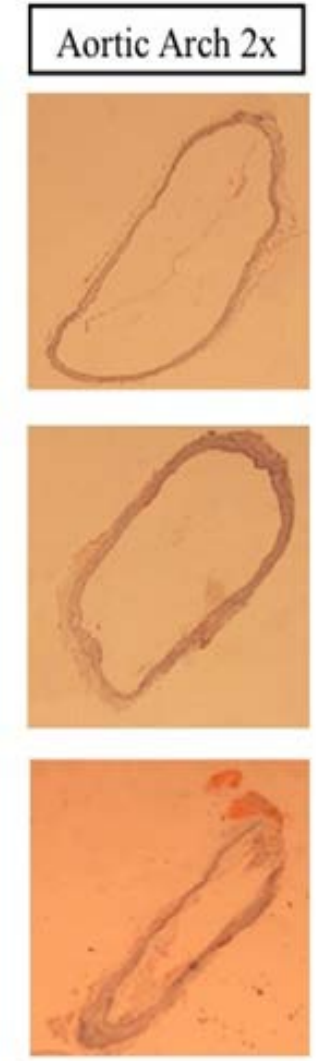


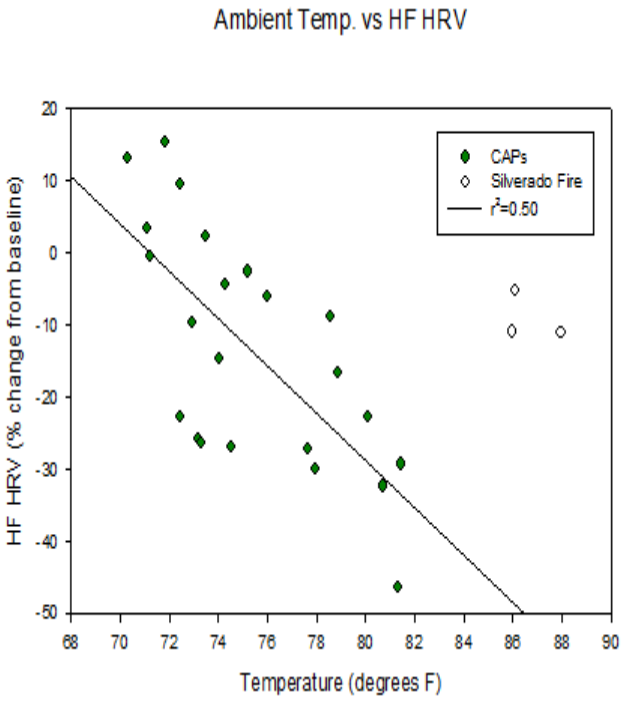


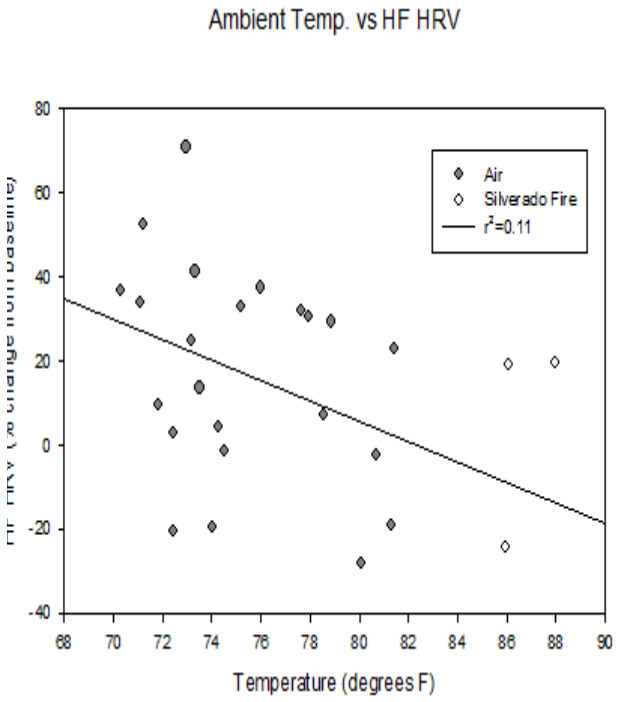
Removing the Organic Constituents From Ambient UFP Blocks CV Effects

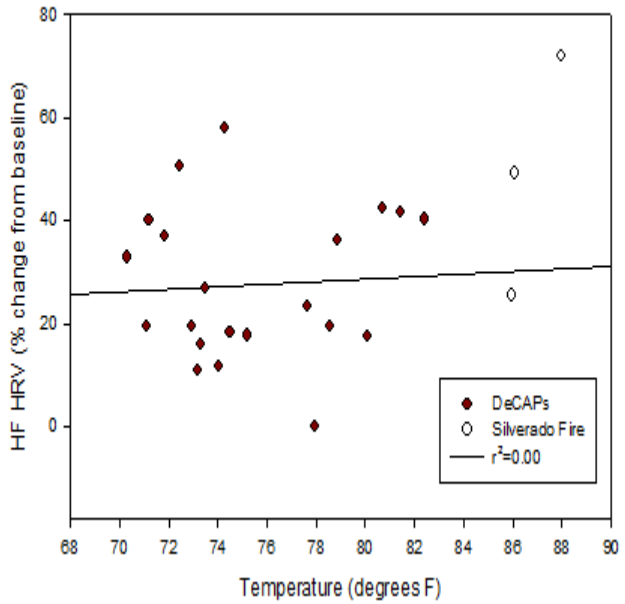


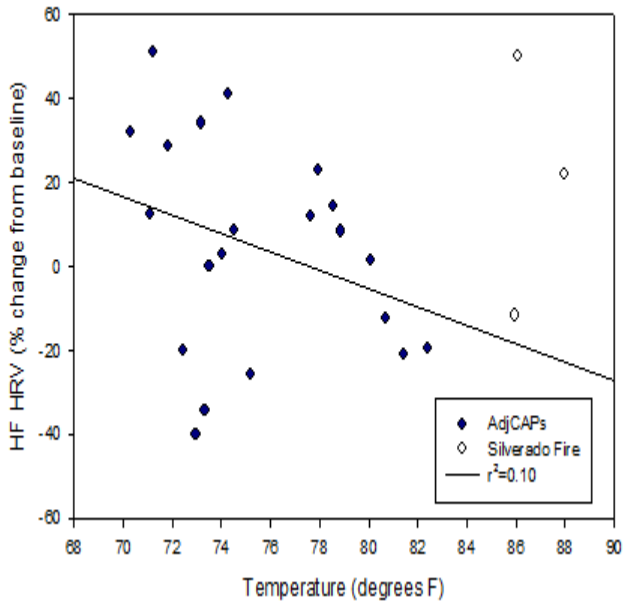
	Air	CAP	deCAP
Plaque Size (% area of plaque in of total lumen CS)	14.5 ± 6.3	29.9 ± 10.0*	2.3 ± 0.5
Lipid Accumulation (% area of lipid in total tissue CS)	5.1 ± 3.3	8.9 ± 2.4,#	2.2 ± 0.3
Lipid Peroxidation (nM MDA/mg protein)	134 ± 29	218 ± 32*,**	141 ± 17











These data show an association between ambient temperature and toxicity measured using heart rate variability (HRV).

The composition of the particles, which determines particle toxicity, is a function of atmospheric chemical reactivity, which is dependent on temperature and photochemical processes.

Conclusions

- PM exposures can exacerbate lung disease, heart disease and cancer
- UFP and PM2.5 contain toxic components and carcinogens
- Children, elderly and Individuals with pre-existing lung and heart conditions are at elevated risk
- The human studies and the toxicology studies support the premise that PM can be mechanistically and causally linked to cardiovascular health effects.

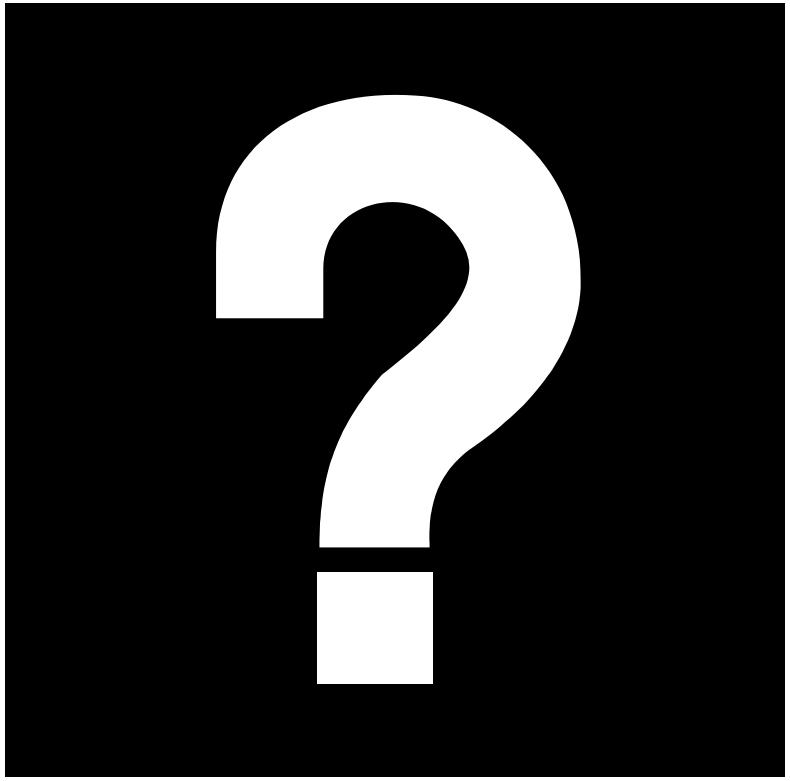
Funding Sources

- Research using advanced instrumentation (AMS and SMPS) was through AirUCI and funded by the National Science Foundation

Moving the AMS is a group effort!

Health studies at are currently sponsored by the California Air Resources Board, the South Coast Air Quality Management District and the NIEHS

Questions and Discussion



Particulate Matter:
Spotlight on Health Protection

John R. Balmes, M.D.

- Professor of Medicine at UC San Francisco
- Professor of Environmental Health Sciences in the School of Public Health at UC Berkeley
- Director of the Northern California Center for Occupational and Environmental Health
- Authored over 300 papers on occupational and environmental health-related topics
- Physician Member of the California Air Resources Board

Particulate Matter Health Effects: What Do We Know and What Do We Still Need to Know?

John R. Balmes, MD

University of California,

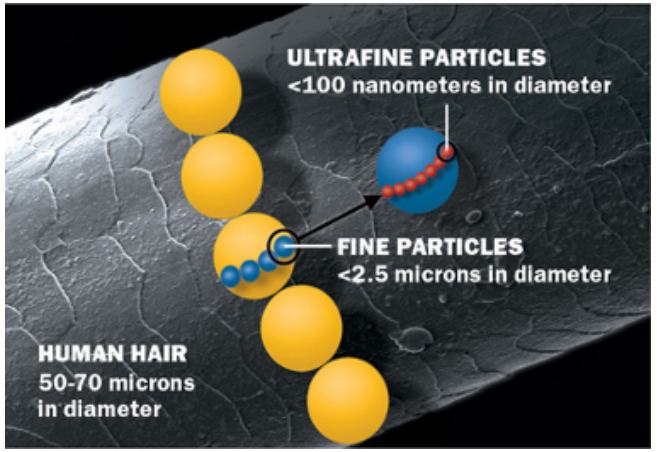
San Francisco and Berkeley

Outline

- Particulate Pollution
  - What Do We Know
  - New Evidence
- Exposure Inequality
  - Cumulative Risk
- Wildfire PM
  - Cardiovascular Risk

Ambient Particulate Matter (PM)

- PM is a mixture, including particles of differing origin (combustion, crustal, biological) and varying size.
- Multiple sources
  - Ultrafines ($PM_{<0.1}$): Fuel (including biomass) combustion
  - $PM_{2.5}$: Fuel (including biomass) combustion
  - $PM_{10\text{-}2.5}$: Road dust, crustal, and biological material



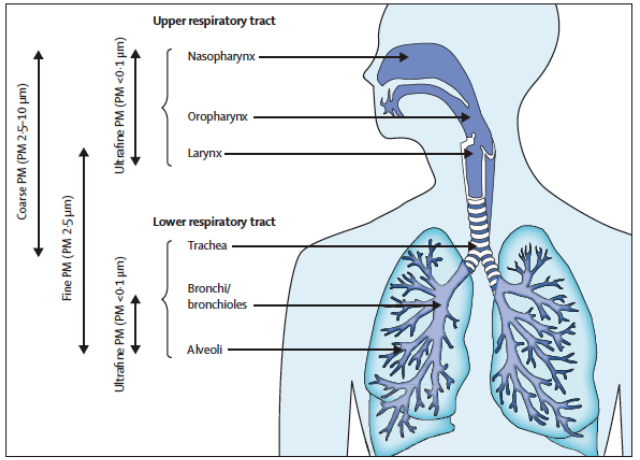


Figure 4: Compartmental deposition of particulate matter

Particulate Matter: Health Effects

- Asthma
  - Exacerbation
  - New-onset
- Decreased lung function growth
- Mortality
  - Ischemic heart disease
- Lung cancer

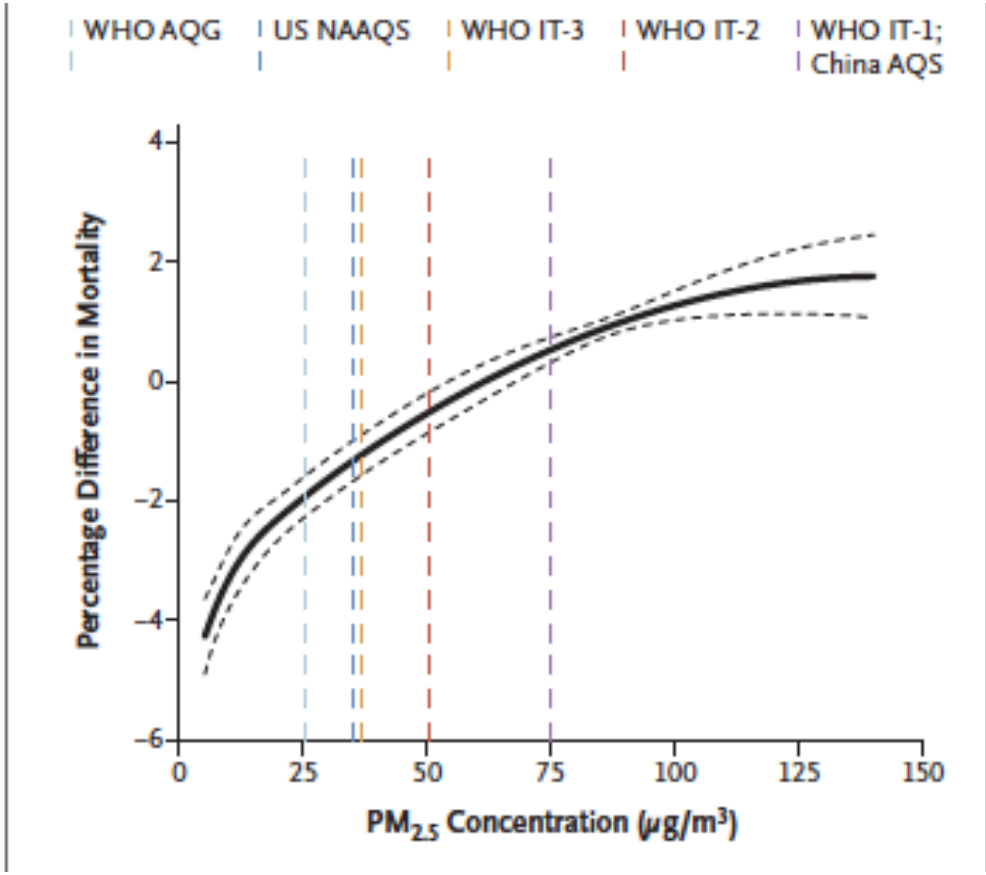
Key Questions

- Are current PM standards sufficiently protective?
  -- No margin of safety
- How has the PM health evidence been strengthened?
  – New evidence of mortality effect at levels below the current NAAQS

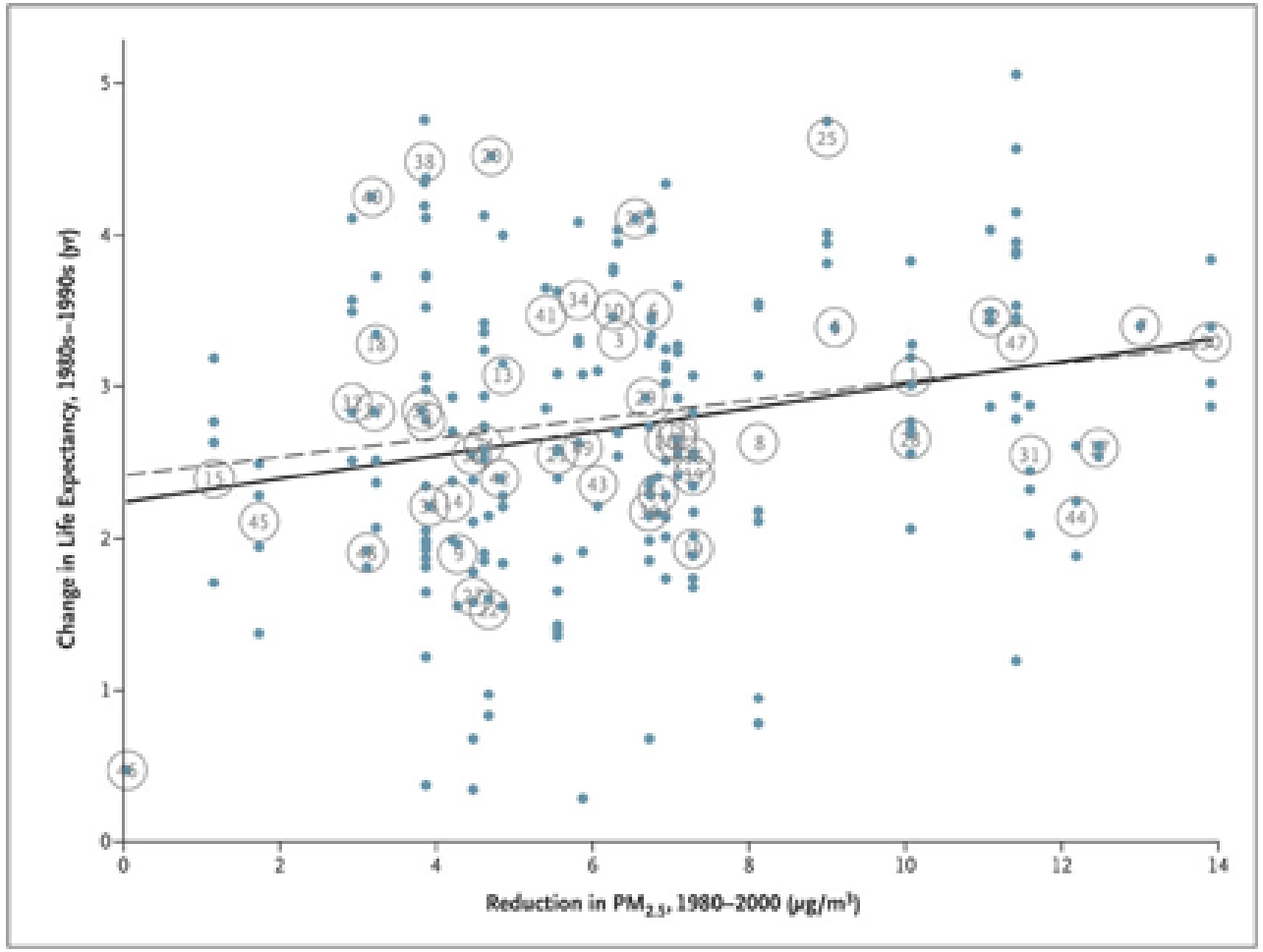
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 AUGUST 22, 2019 VOL. 381 NO. 8

Ambient Particulate Air Pollution and Daily Mortality in 652 Cities



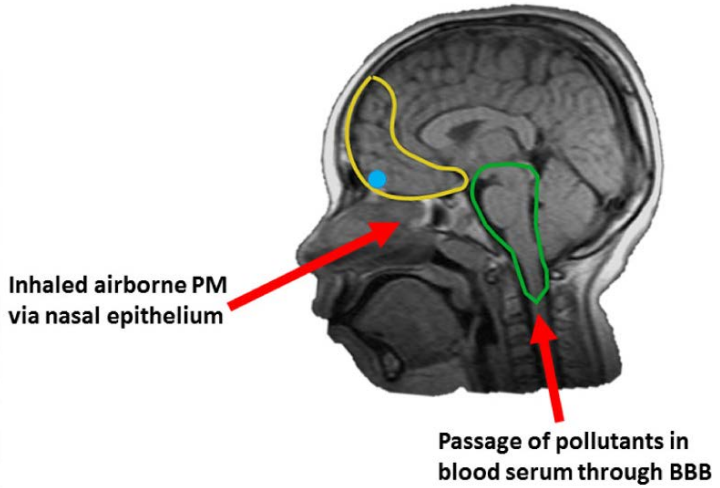
Fine-Particulate Air Pollution and Life Expectancy in the United States



Pope et al. N Engl J Med 2009;360:376-386.

Key Questions

- What new health effects are now recognized?
  - Adverse birth outcomes
  - Metabolic effects
  - Neurological effects



What is role of ultrafine particles (UFP)?

- UFP (PM<0.1µm) are generated both as primary emissions from combustion processes and as secondary products of atmospheric chemistry
- Toxicological studies suggest UFP are a high-risk hazard, but epidemiological data are sparse because there is no monitoring network

Key Questions

- Are there “new” sensitive groups?
  - Children
  - People of color and low SES
- How should we account for spatial scale of effects (i.e., regional versus local-scale impacts, including proximity to major sources)?

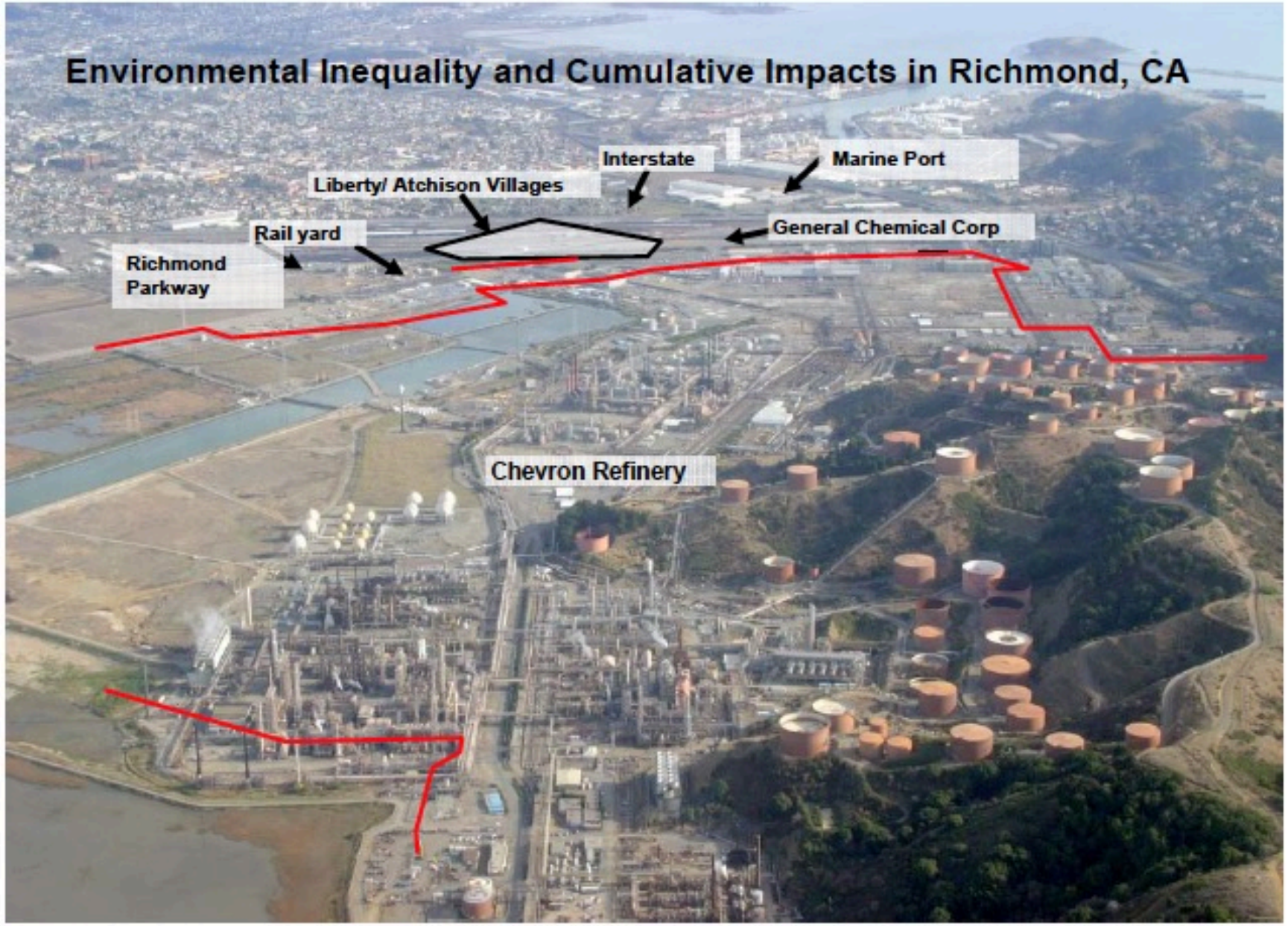
Demographics of Children Living Near Freeways

- Children of color 3x more likely to live near high traffic density in California

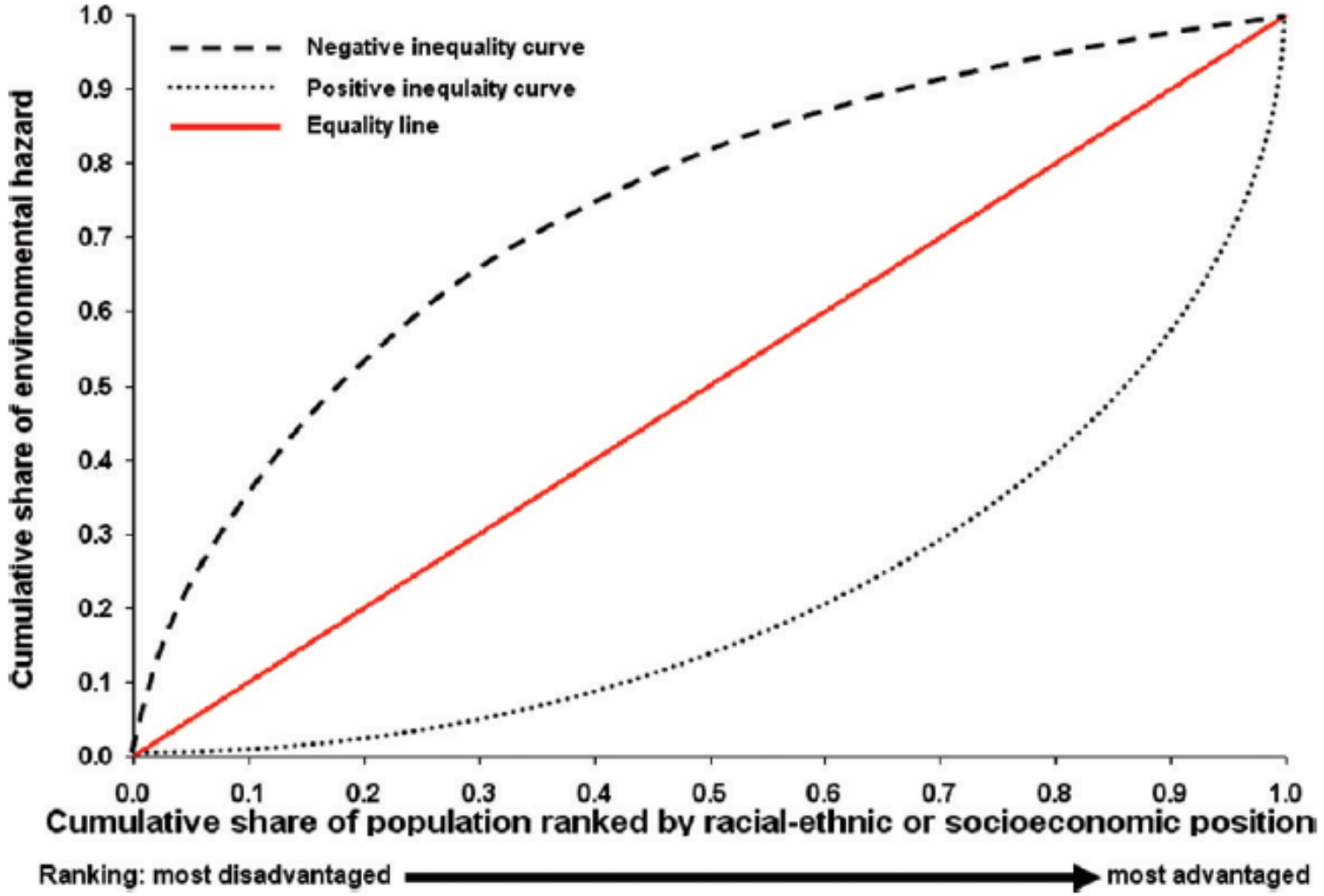
Gunier et al., California Dept of Health Services, 2003

- Schools near busy roads have a disproportionate number of children who are economically disadvantaged and non-white

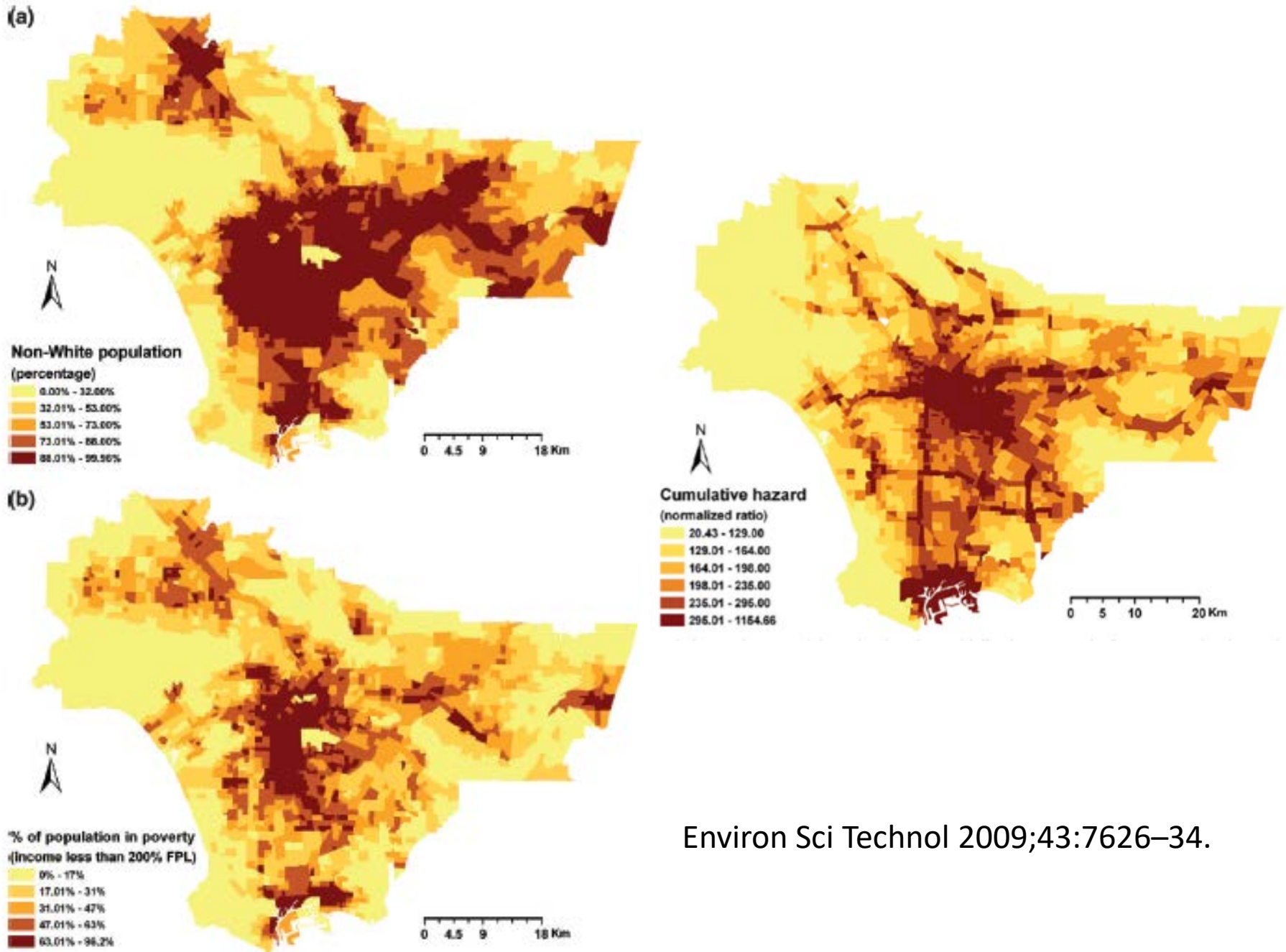
RS Green et al, Environ Health Perspect 2004;112:61.


Environmental Inequality and Cumulative Impacts in Richmond, CA
Interstate
Marine Port
Liberty/ Atchison Villages
Rail yard
General Chemical Corp
Richmond Parkway
Chevron Refinery

Inequality Curve



Environ Sci Technol 2009;43:7626–34.



Environ Sci Technol 2009;43:7626–34.

Cumulative Risk

- People of color and low SES have
  - Greater exposures to outdoor partculate pollution
  - Disproportionate proximity to polluting land uses and toxic emissions
- Poor communities have more health-damaging factors and less health-promoting amenities
  - Less access to healthy food and health care
  - Less green space and recreational programs
  - Poor quality housing and greater violence

Key Questions

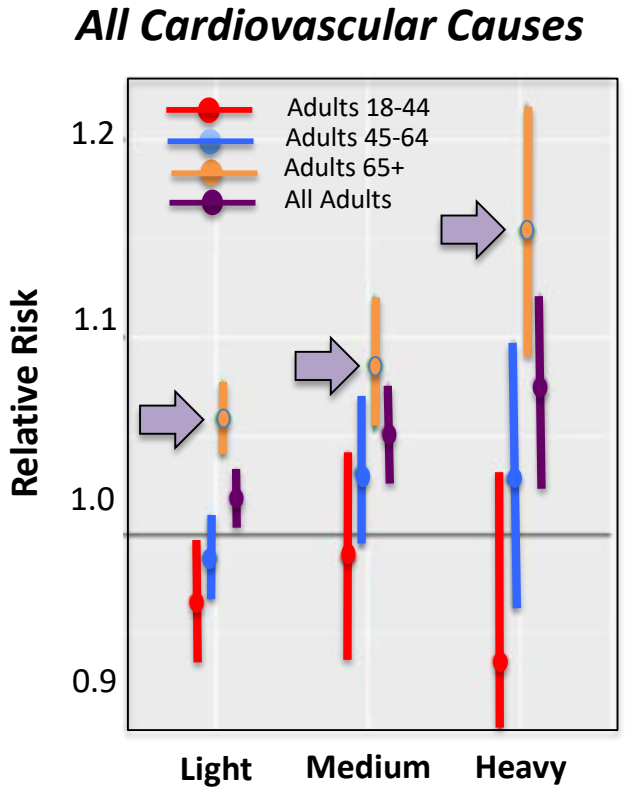
- What are health impacts of high-concentration acute events (e.g., wildfires)? How should we compare them to day-to-day PM impacts?

Clear evidence of an association between wildfire smoke and respiratory health

- Asthma exacerbations significantly associated with higher wildfire smoke ***in nearly every study***
- Exacerbations of chronic obstructive pulmonary disease (COPD) significantly associated with higher wildfire smoke in most studies
- Growing evidence of a link between wildfire smoke and respiratory infections (pneumonia, bronchitis)

Wildfire-$PM_{2.5}$ Increases Heart Attack & Stroke

- ***Wildfire-$PM_{2.5}$ associated with heart attacks and strokes for all adults, particularly for those over 65 years old***
- ***Increase in risk the day after exposure:***
  - All cardiovascular, 12%
  - Heart attack, 42%
  - Heart failure, 16%
  - Stroke, 22%
  - All respiratory causes, 18%
  - Abnormal heart rhythm, 24%
    (on the same day as exposure)



Wettstein Z, Hoshiko S, Cascio WE, Rappold AG et al. JAHA April 11, 2018

Slide credit: Wayne Cascio

Thank you

Particulate Matter:
Spotlight on Health Protection

H. Christopher Frey, Ph.D., F. A&WMA, F. SRA

- Glenn E. Futrell Distinguished University Professor of Environmental Engineering in the Department of Civil, Construction, and Environmental Engineering at North Carolina State University
- Adjunct professor in the Division of the Environment and Sustainability at the Hong Kong University of Science and Technology
- Fellow of the Air & Waste Management Association and of the Society for Risk Analysis
- Ph.D. in Engineering and Public Policy from Carnegie Mellon

Recent Developments in the Scientific Review of the National Ambient Air Quality Standards for Particulate Matter

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NC STATE UNIVERSITY

Department of Civil, Construction & Environmental Engineering
North Carolina State University
Raleigh, NC 27695

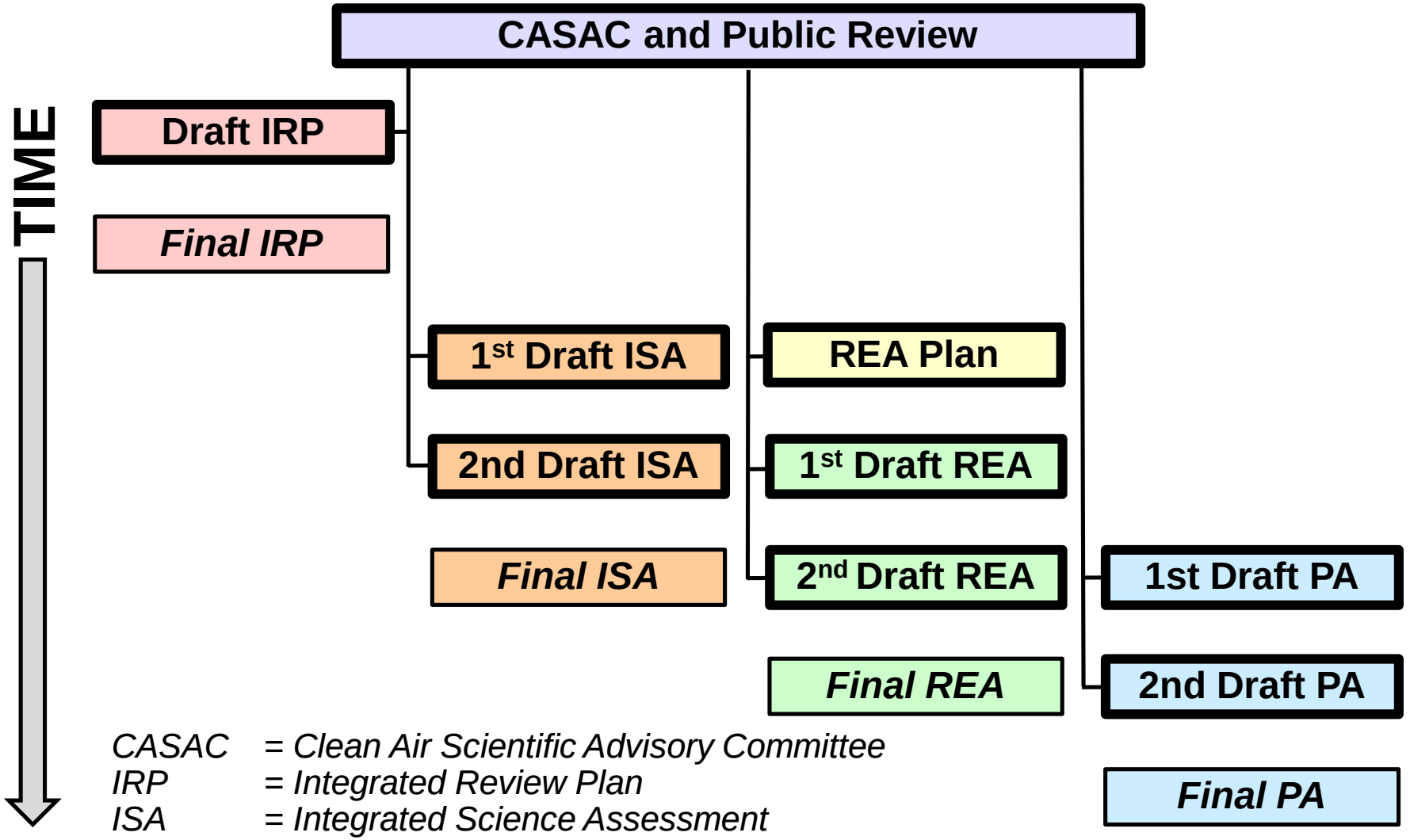
Presented at:
Particulate Matter: Spotlight on Health Protection
Bay Area Air Quality Management District
San Francisco, CA

October 28, 2019

Key Points

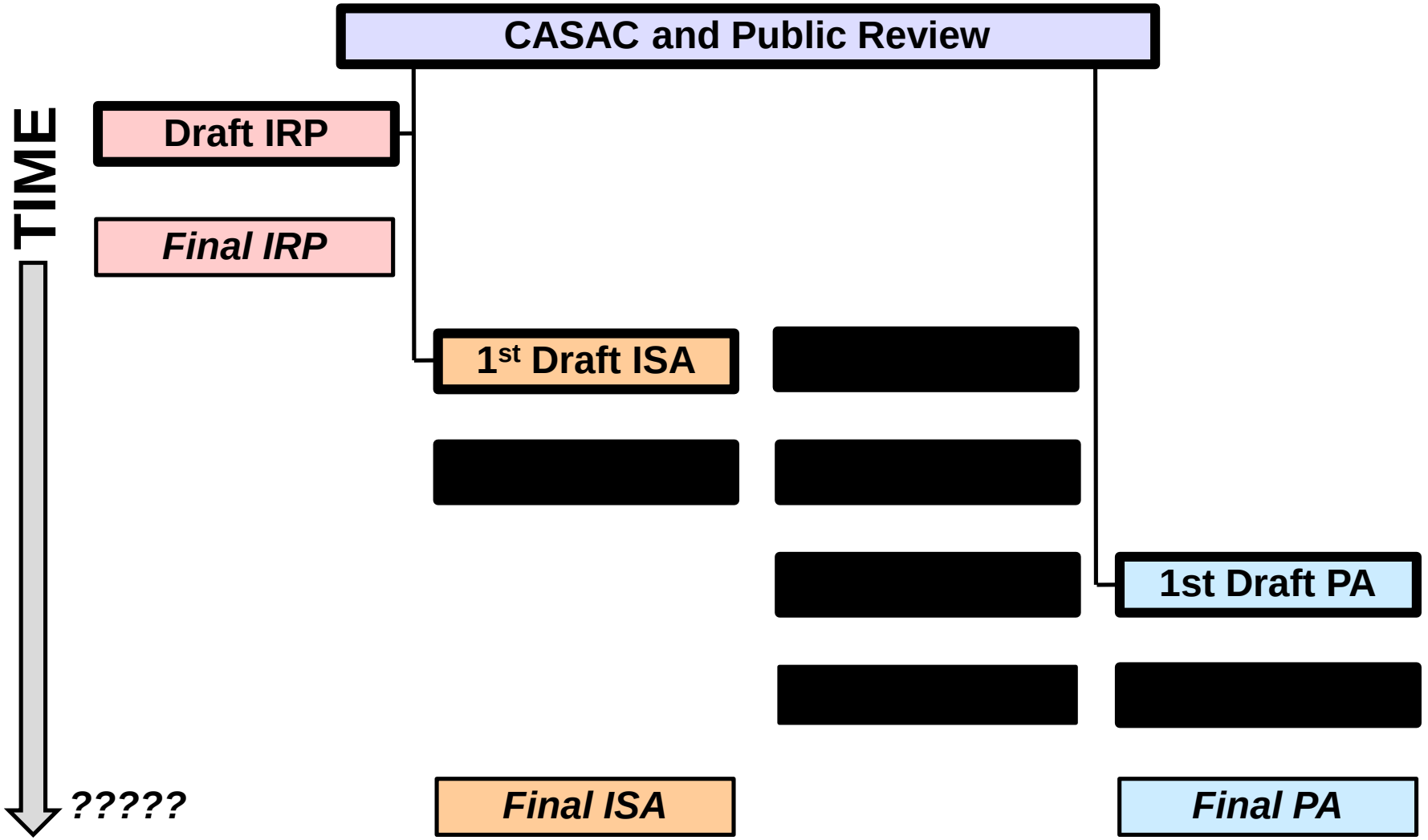
- The National Ambient Air Quality Standard (NAAQS) Science Review Process Worked Well Until 2017
- EPA Administrators Pruitt and Wheeler Have Broken the Process
- Particulate Matter Science Review By the EPA Clean Air Scientific Advisory Committee (CASAC) is Highly Deficient: Appropriate to Look Elsewhere
- Disbanded CASAC PM Review Panel Reconvened Itself
- Key Findings of the Independent Particulate Matter Review Panel

Generic "Full" National Ambient Air Quality Standard (NAAQS) Science Review from Document Perspective

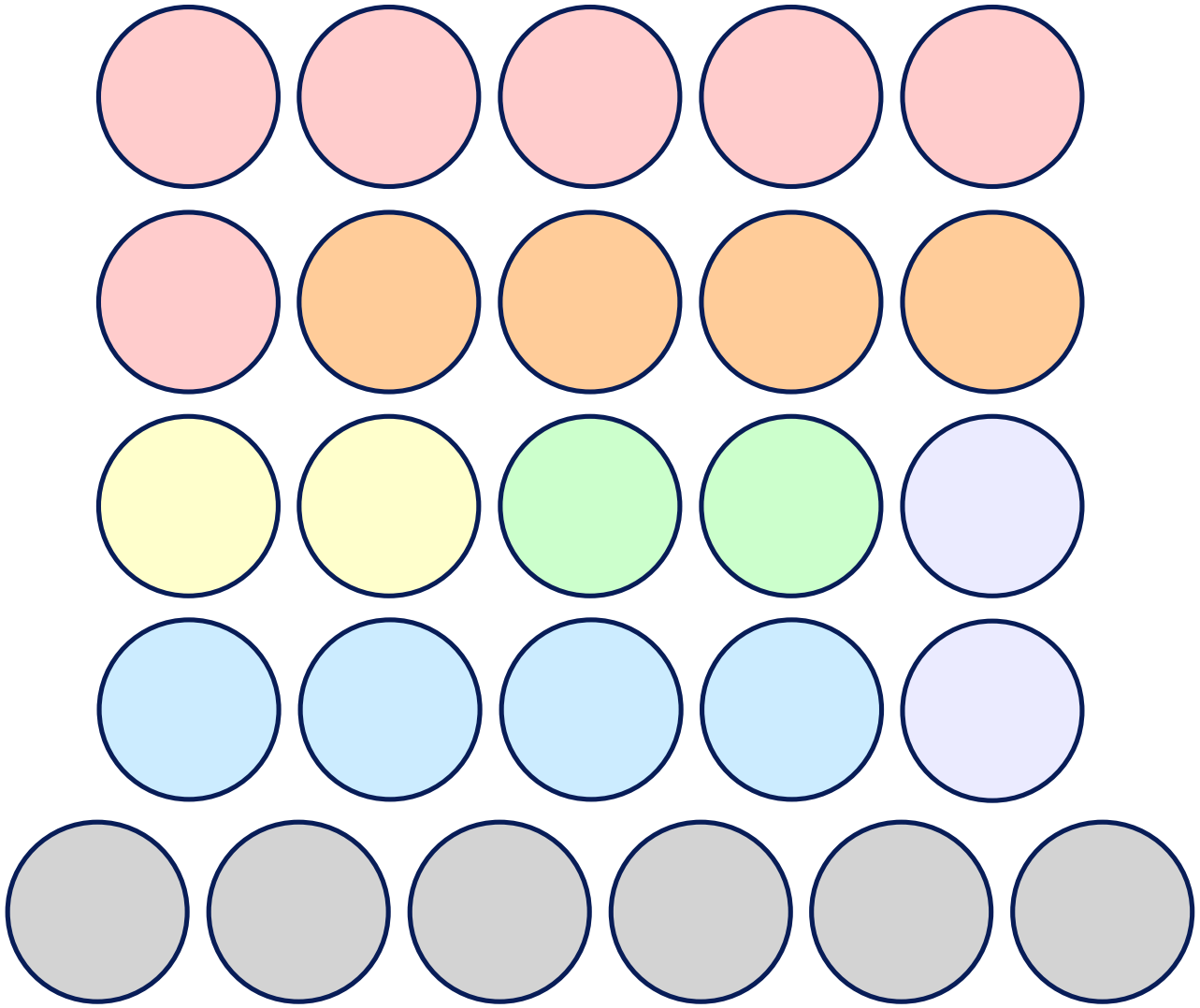


CASAC = Clean Air Scientific Advisory Committee
IRP = Integrated Review Plan
ISA = Integrated Science Assessment
REA = Risk and Exposure Assessment
PA = Policy Assessment

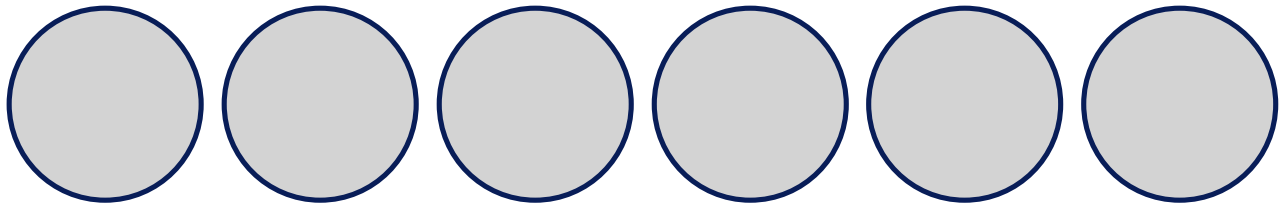
Pruitt/Wheeler (P/W) Particulate Matter NAAQS Science Review from Document Perspective



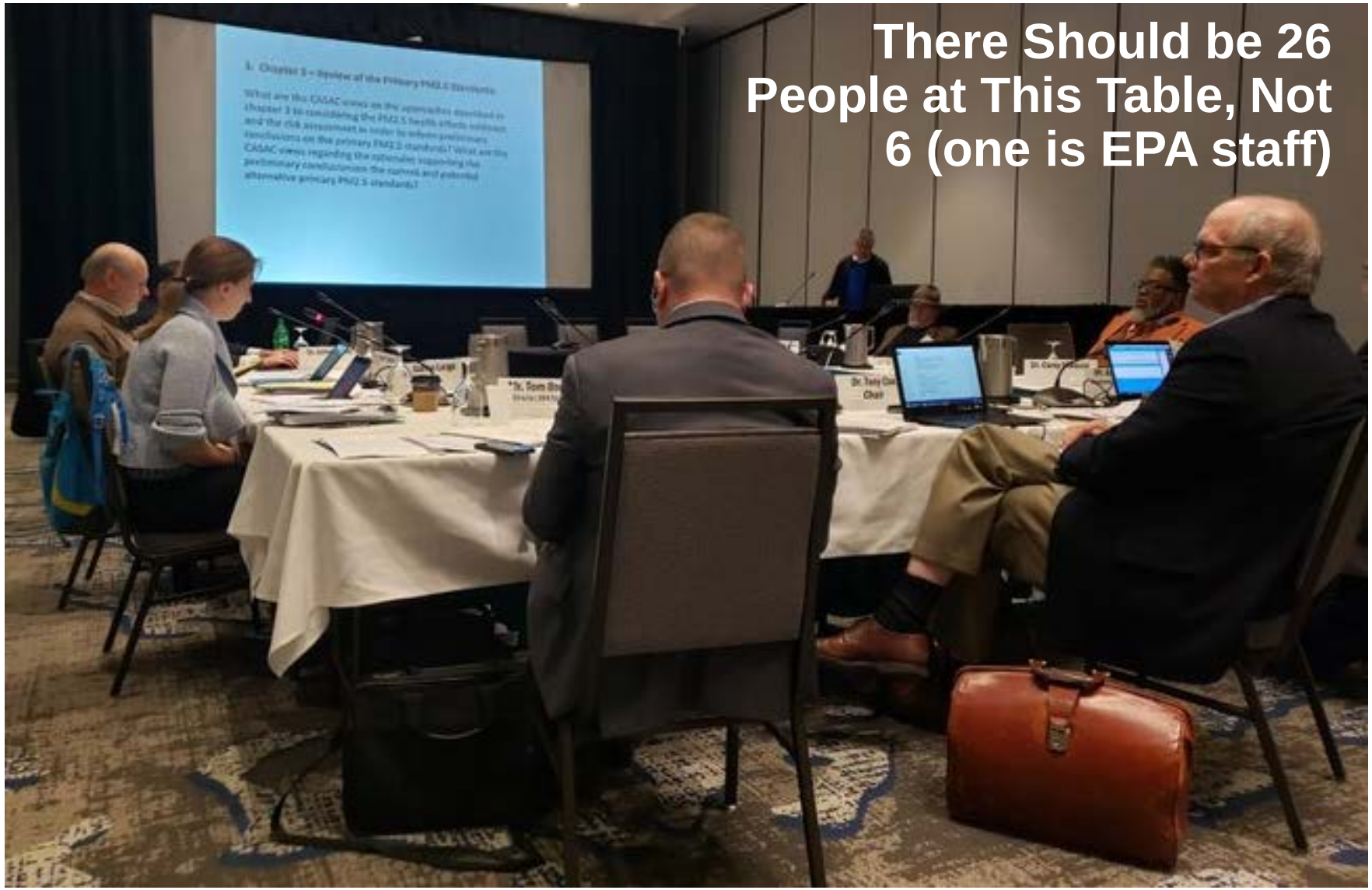
2015 EPA CASAC Particulate Matter Review Panel (26)



Pruitt/Wheeler EPA CASAC Particulate Matter Review Panel (6 last week, 7 by statute)



The Latest from CASAC, as of 2:25 pm Friday, October 25, 2019



The Latest from CASAC, as of 2:25 pm Friday, October 25, 2019

- CASAC is split 4-2:
  - Four recommend keeping all current standards (primary $PM_{2.5}$, coarse PM, secondary $PM_{2.5}$) as is.
  - Rationales offered for keeping the annual primary $PM_{2.5}$ standard are ill-informed or inappropriate, given the state of the science, lack of needed expertise and obvious lack of understanding of the statutory mandate of the Clean Air Act.

Independent Particulate Matter Review Panel

- Formerly the CASAC PM Review Panel
- Disbanded October 10, 2018
- Met October 10, 2019 to October 11, 2019 in Crystal City, VA
- Follow-up Teleconference October 18, 2019 to finalize report

Independent Particulate Matter Review Panel

- **Dr. H. Christopher Frey**, Chair, North Carolina State University
- **Dr. Peter Adams**, Carnegie Mellon University
- **Dr. John L. Adgate**, Colorado School of Public Health
- **Mr. George Allen**, NESCAUM
- **Dr. John Balmes**, University of California at San Francisco
- **Dr. Kevin Boyle**, Virginia Tech
- **Dr. Judith Chow**, Desert Research Institute
- **Dr. Douglas W. Dockery**, Harvard T.H. Chan School of Public Health
- **Mr. Dirk Felton**, NY State Dept. of Environmental Conservation
- **Dr. Terry Gordon**, New York University School of Medicine
- **Dr. Jack Harkema**, Michigan State University
- **Dr. Joel Kaufman**, University of Washington
- **Dr. Patrick Kinney**, Boston University School of Public Health
- **Dr. Michael T. Kleinman**, University of California at Irvine
- **Dr. Rob McConnell**, University of Southern California
- **Mr. Richard Poirot**, Independent Consultant
- **Dr. Lianne Sheppard**, University of Washington
- **Dr. Jeremy Sarnat**, Rollins School of Public Health, Emory University
- **Dr. Barbara Turpin**, University of North Carolina at Chapel Hill
- **Dr. Ronald Wyzga**, Retired, Electric Power Research Institute

Independent Particulate Matter Review Panel

- Followed the same process and procedures as we did formerly as the CASAC PM Review Panel
- Developed a letter to the EPA Administrator and Consensus Responses to EPA Charge Questions on the Draft Policy Assessment
- Submitted our report to CASAC, the docket, and the Administrator
- **ucsusa.org/pmpanel**

Acknowledgment of EPA Staff

- The Panel finds that the EPA staff in the Office of Air Quality Planning and Standards have undertaken a good faith effort to produce a first draft of the PA.
- This draft was produced under extenuating, unprecedented, and inappropriate constraints.
- **The Panel commends the staff for this effort.**

Causality Determinations

- The **weight of evidence framework for causality determination** that is applied by EPA **is an appropriate and well-vetted tool** for drawing causal conclusions.
- The epidemiologic **evidence**, supported by evidence from controlled human studies and toxicological studies, **supports the 'causal' and 'likely to be causal' determinations** that are the focus of the draft PA.
- "The epidemiologic **evidence** provides strong scientific **support for recommendations** regarding current and alternative standard levels."
- **Arguments to retain the current primary $PM_{2.5}$ standards** "would require disregard of the epidemiological evidence," and "are not scientifically justified and **are specious**."

Major Findings: Fine Particle Standards

- The current primary fine particle ($PM_{2.5}$) annual and 24-hour standards are **not protective of public health**.
- Retain current indicators, averaging times, and forms.
- The **annual** standard should **be 10 μg/m³ to 8 μg/m³** (versus 12 μg/m³ now).
- The **24-hour** standard should be **30 μg/m³ to 25 μg/m³** (versus 35 μg/m³ now).
- **Consistent epidemiological evidence** from multiple multi-city studies, **augmented with** evidence from **single-city studies**, at policy-relevant **ambient concentrations** in areas with design values at and **below the levels of the current standards.**
- **Supported** by research from **experimental models** in animals and humans and by **accountability studies**

Major Findings: Fine Particle Standards

- A motivation for strengthening the 24-hour $PM_{2.5}$ standard is high 24-hour to annual ratios related to **residential wood combustion** in some areas.
- Panel notes **growing frequency and severity of so-called "wildfires."**

Accounting for Limitations

- The Panel considered in detail uncertainties and limitations of available epidemiologic evidence, such as:
  - Use of linear, multipollutant models
  - Possibility that co-pollutants may be effect modifiers rather than confounders
  - Confounding by individual characteristics has been considered and evaluated
  - No rationale or empirical support for confounding by temperature in annual studies
- Consistency among **multiple multicity models**, for which there is variability in relative ambient **mixtures of co-pollutants**, **population demographics**, **climatic zones**, and distributions of **housing characteristics**, supports the **robustness** of their results.

Recommended Range for Annual $PM_{2.5}$ Standard

- At **10 µg/m³** there is a **very high degree of scientific confidence** in the relationship between exposure to fine particles and **adverse effects**.
- The risk is **linear** with **no threshold** below the current standard down to an annual level of 8 µg/m³ or lower.
- The Panel finds that there is **not sufficient scientific certainty below 8 µg/m³ to support a lower recommendation.**

Other Issues: At Risk Groups

- Di et al. (2017a) chronic Medicare study shows that the relative risk for African Americans is three times higher than that of the entire population (hazard ratio of 1.21 per 10 μg/m^3 increase in $PM_{2.5}$).

BAAQMD's Questions

- Are current PM standards sufficiently protective? **Emphatic NO – definitely not for $PM_{2.5}$.**
- How has the PM health evidence been strengthened? **Better "exposure" models, much larger study populations at much lower levels than before.**
- What new health effects are now recognized? **Strengthening of some causality determinations, but largely the focus is still premature mortality, respiratory morbidity, and cardiovascular morbidity.**
- New endpoints like cancer and central nervous system effects? **Opinions differ.**
- New sensitive groups, like children and lower socioeconomic status, SES, populations? **Growing recognition of "at risk" groups.**
- Are all types of PM equal? **Probably not.** Or, are some more dangerous than others? **Probably. But, more work needed. No components are as yet 'exonerated.'**
- How severe are PM health risks? **Premature mortality is severe.**
- What additional health benefits can be achieved by further reducing PM to below current standards? **Difficult to quantify with certainty but on the order of tens of thousands of deaths nationally.**

BAAQMD's Questions

- How important are short-term PM events, like wildfires? **Not well-known scientifically but of concern for potential or anticipated effects**. **Research recommended.**
- How should we weight them in comparison with ongoing day-to-day PM levels? **No simple answer. Depends… can they be controlled? If so, how? Via a state implementation plan? And would you slap non-attainment on an area just devastated by a wildfire?**
- How important are ultrafine particles, UFPs? **Current evidence of adverse effects is generally weak but there is concern for potential or anticipated effects. Need more monitoring to support more epidemiological studies**. **Panel recommends a UFP FRM for this purpose.**
- Should we consider more than just PM mass? *(meaning particle number concentration?)* **In research, absolutely. In regulation, too soon, unless one takes a very precautionary, highly risk-averse decision approach.**
- Which is most protective, an annual average target or a 24-hour average one? Or, a sub-daily average? **For most parts of the country, annual can offer protection also for 24-hour averages. For other parts, not so. Panel comments on this. Health data on sub-daily is too limited as yet to support a standard at the national level, but Panel has recommendations to look at this further**.

Next Steps

- CASAC will release its draft report on the draft PM Policy Assessment within a few weeks.
- CASAC will meet on December 3, 2019 to review and likely finalize its report to the Administrator
- Opportunity for public comment in writing beforehand and oral comment at the meeting.
- *CASAC will review the draft ISA and draft PA for Ozone at the Dec 3-6, 2019 meeting*.

Key Points

- The NAAQS Science Review Process Worked Well Until 2017
- EPA Administrators Pruitt and Wheeler Have Broken the Process
- Particulate Matter Science Review By CASAC is Highly Deficient: Appropriate to Look Elsewhere
- Disbanded CASAC PM Review Panel Reconvened Itself
- Key Findings of the Independent Particulate Matter Review Panel

Acknowledgments

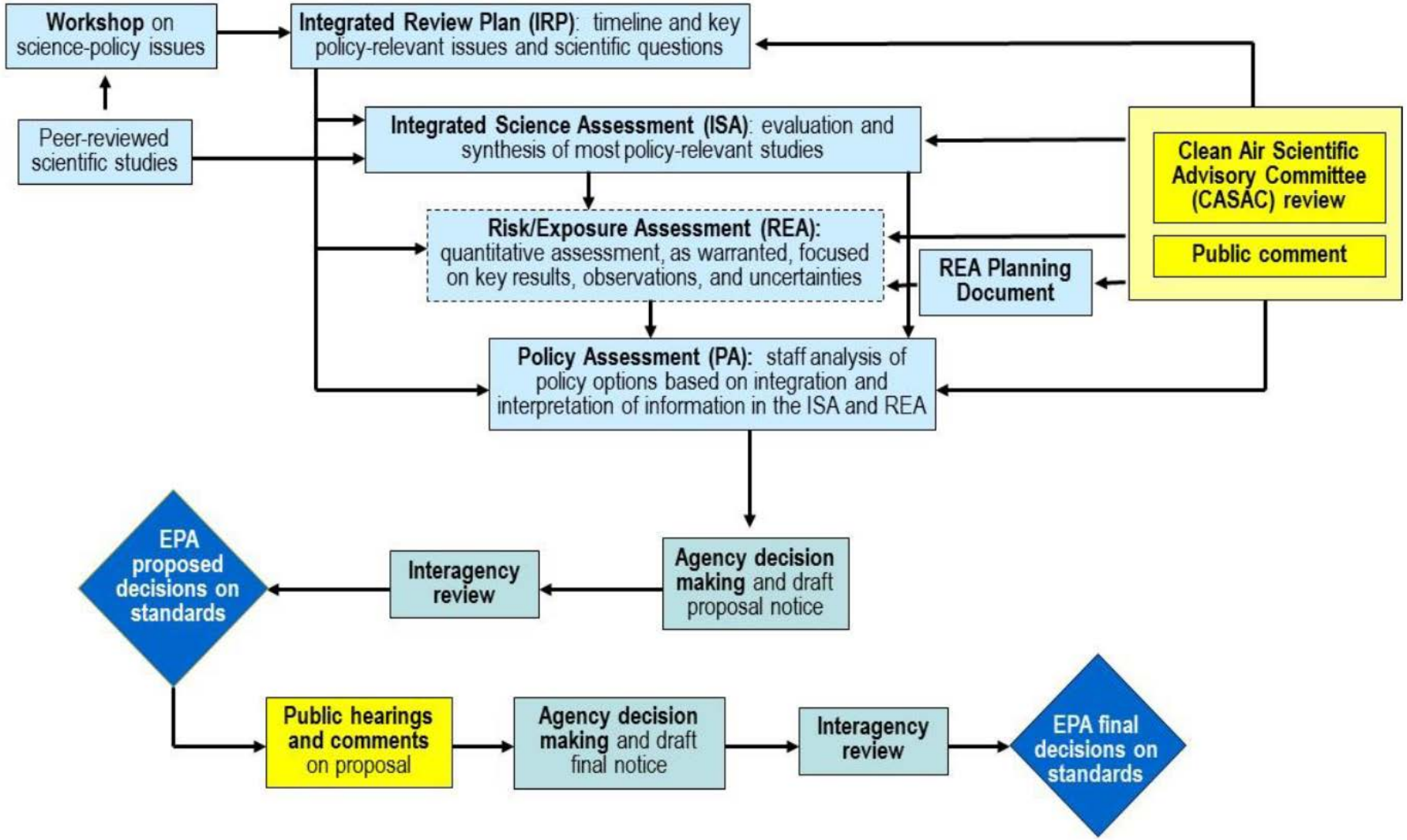
- Members of the Independent Particulate Matter Review Panel.
- Union of Concerned Scientists hosted the October 2019 meetings of the Panel. Special thank you to Dr. Gretchen Goldman.
- Mr. Chris Zarba acted in the role of a designated officer for the panel.
- Mr. John Bachmann and Mr. Steven Silverman provided technical and legal clarifications, respectively.
- This presentation has not been reviewed or approved by anyone. The author is solely responsible for its content.

frey@ncsu.edu

Report of the Independent Particulate Matter Review Panel is at:

ucsusa.org/pmpanel

Overview of EPA's Process for Reviewing National Ambient Air Quality Standards, 2016

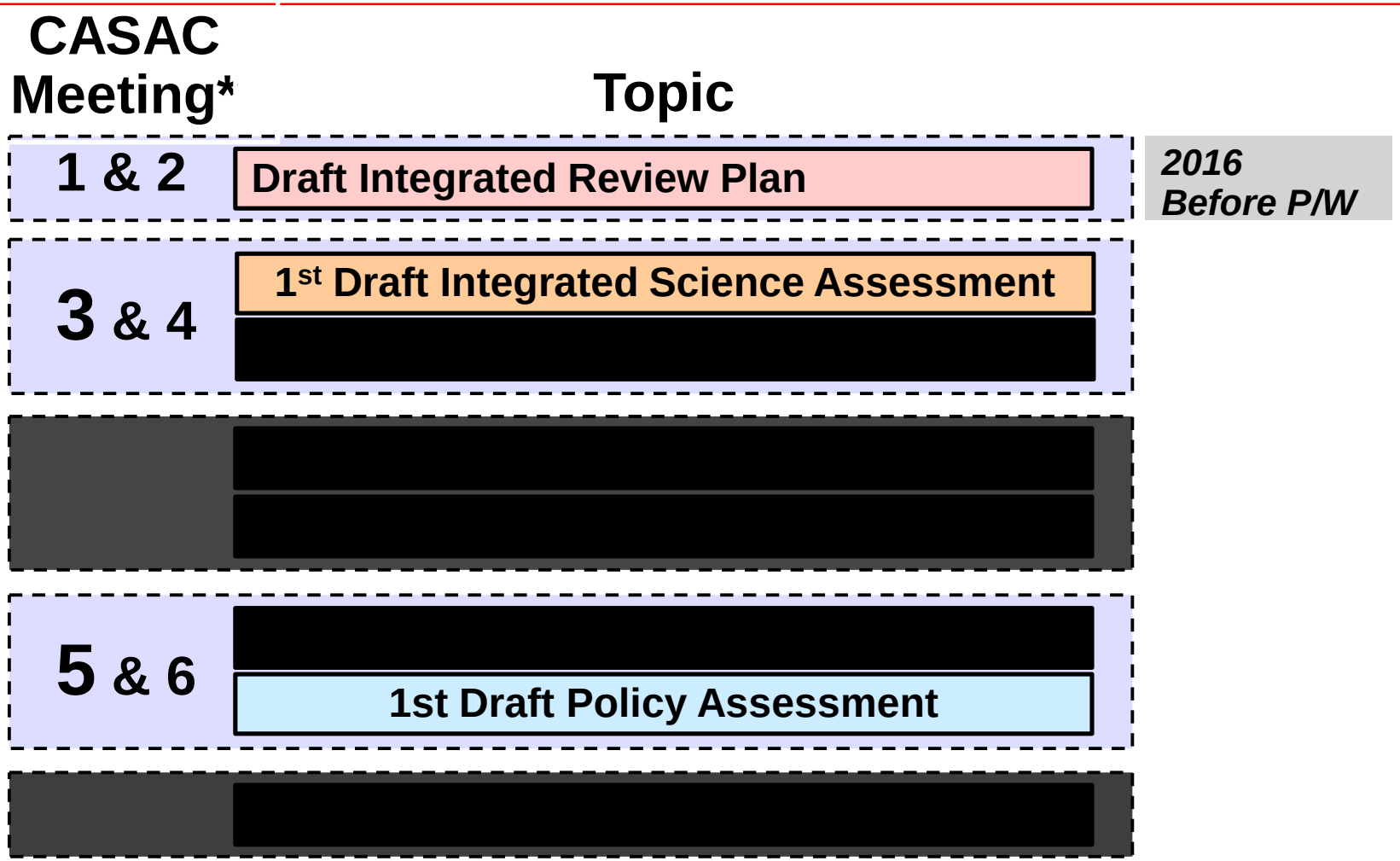


Generic "Full" NAAQS Science Review from CASAC and Public Perspective

CASAC Meeting*	Topic
1 & 2	Draft Integrated Review Plan
3 & 4	1st Draft Integrated Science Assessment Risk & Exposure Assessment Plan
5 & 6	2nd Draft Integrated Science Assessment 1st Draft Risk & Exposure Assessments
7 & 8	2nd Draft Risk & Exposure Assessments 1st Draft Policy Assessment
9 & 10	2nd Draft Policy Assessment

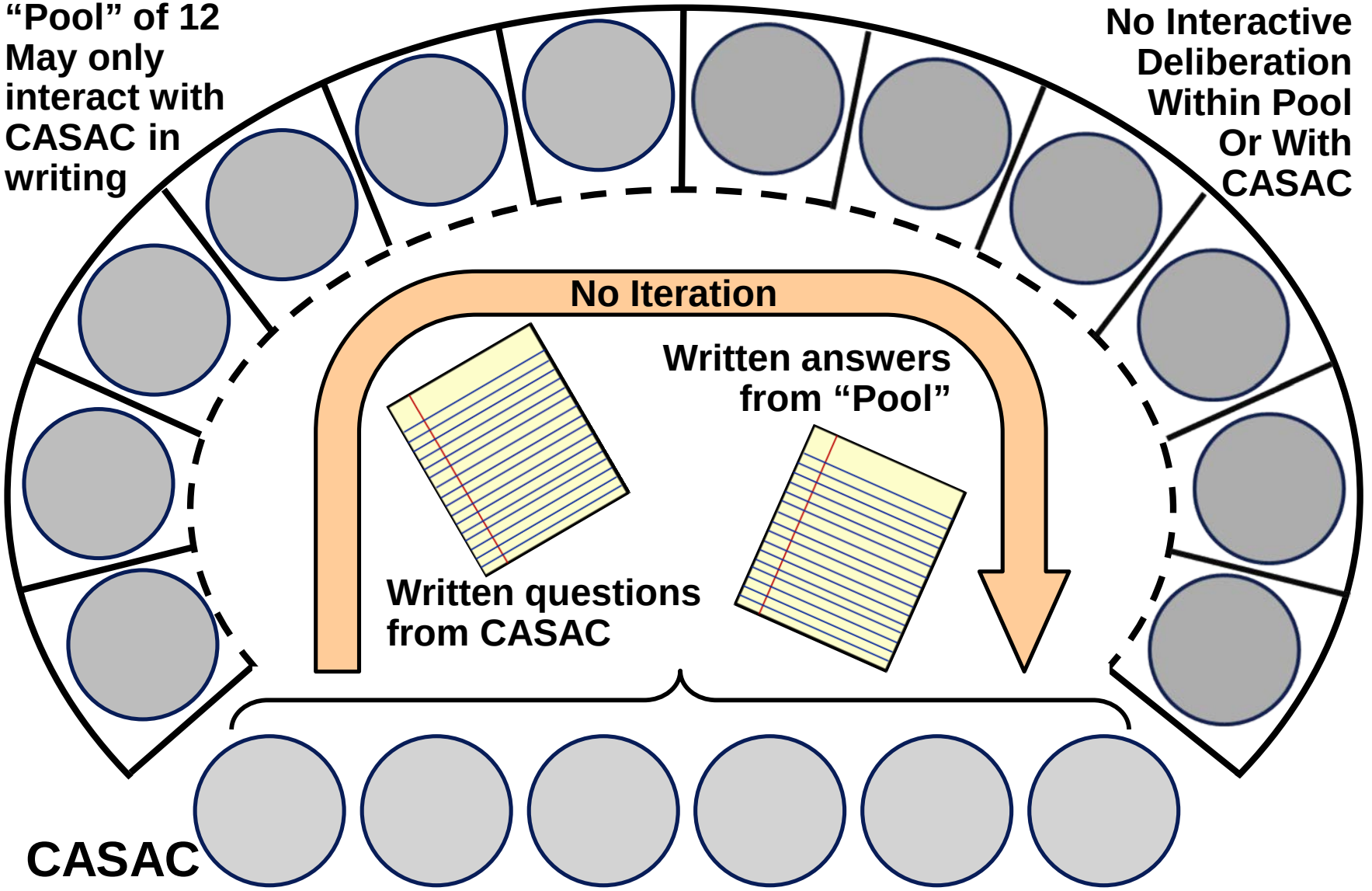
*Meetings 1, 2, 4, 6, 8, 10 by teleconference; Meetings 3, 5, 7, 9 face-to-face
Public Comment at EVERY meeting (10 opportunities)

Pruitt/Wheeler (P/W) Particulate Matter NAAQS Science Review from CASAC and Public Perspective

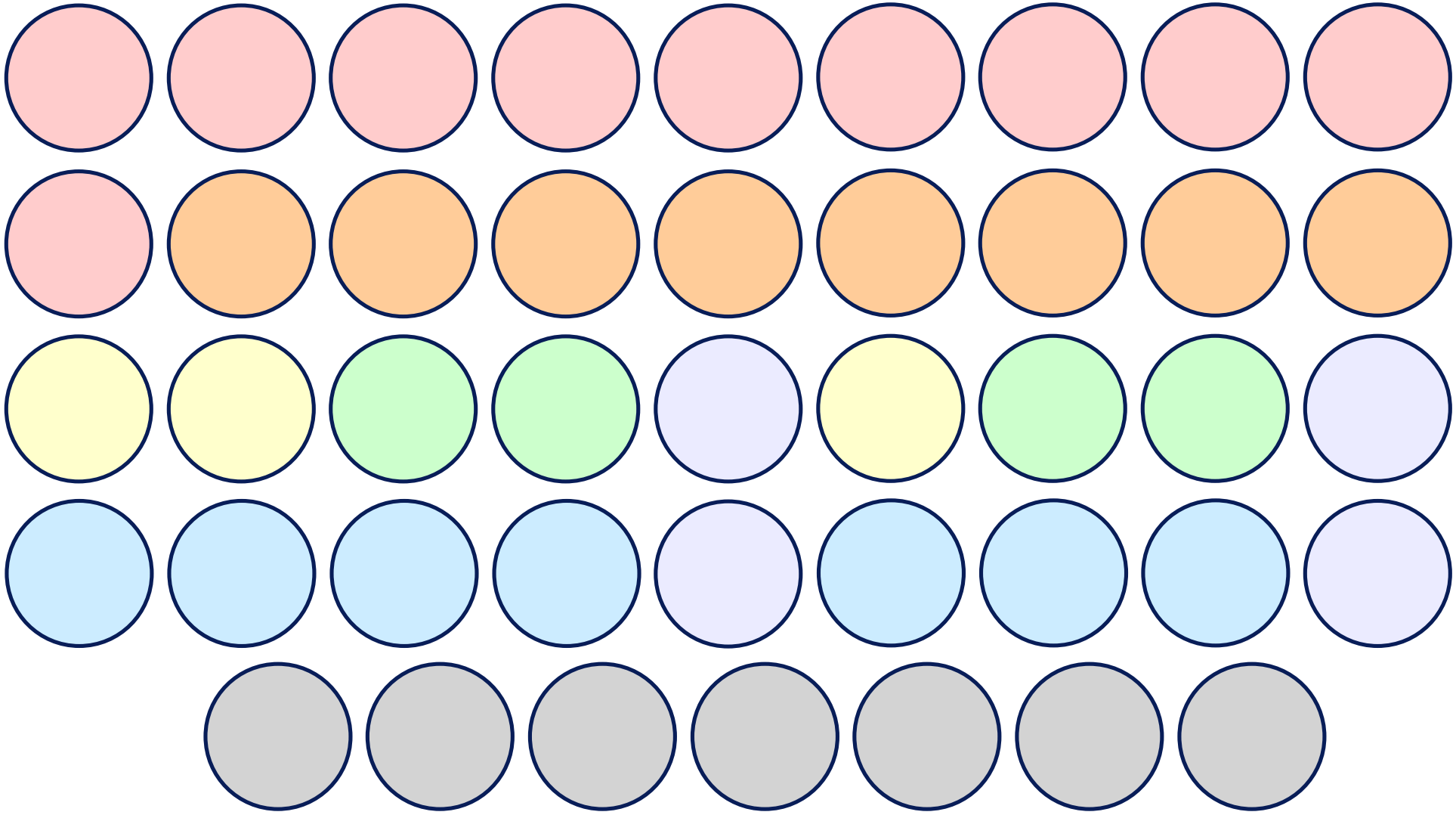


*Meetings 1, 2, 4, 6 by teleconference; Meetings 3, 5 face-to-face
Public Comment at EVERY meeting (6 opportunities) [Only 4 in P/W era]

Wheeler Ad Hoc "Pool" of External Consultants for PM and O_3 Reviews



Typical Pre-Pruitt/Wheeler CASAC for PM and O_3 Reviews: CASAC Augmented with PM and O3 Panels



Report of the Independent Particulate Matter Review Panel

- ucsusa.org/pmpanel
- 11 page letter (5 pages of text)
- Attachment A: Panel Roster (2 pages)
- Attachment B: Consensus Responses (43 pages)
- Attachment C: Individual Member Comments (117 pages)
- Attachment D: History, Membership Criteria, and Administrative Procedures of the Panel
- Attachment E: Panel Member Biosketches

Major Findings: Coarse PM

- Coarse PM (PM_{10} as an indicator for $PM_{10-2.5}$)
  - Retain current indicator, form, and averaging time (24-hour)
  - Current level of protection should at least be maintained
  - Need to revise downward with downward revision of 24-hour $PM_{2.5}$ standard.
  - Should move to $PM_{10-2.5}$ as the indicator in the next review.

Major Findings: Visibility

- Welfare (Secondary) Standards
  - Current annual standard has no effect (15 μg/m^3 vs. 12 μg/m^3 for primary $PM_{2.5}$ standard.
  - Annual should at least match primary annual.
  - 24-hour standard is not adequate to protect against visibility effects
  - A second draft of the PA should identify and analyze alternatives
  - Panel offers recommendations regarding alternative indicators, averaging times, forms, and levels to be considered.

Process Issues (Overview, Examples)

- Since 2017, the Panel finds that the EPA has made unwarranted changes to the CASAC and the NAAQS review process.
- Detailed recommendations to reverse the unwarranted changes are in the consensus responses.
- A second draft of the ISA should be reviewed by CASAC and the public, and the ISA should be finalized, prior to release of a second external review draft of the PA
- The CASAC PM Review Panel should be reappointed to provide CASAC with the expertise it needs.

New Federal Reference Methods Needed

- The Panel recommends that Federal Reference Methods be developed for Ultrafine Particles and Black Carbon
- FRMs for UFP and BC should be deployed to collect data need for health studies and for baselines

Break

Particulate Matter:
Spotlight on Health Protection




Advisory Council Discussion with Health Effects Panel

Discussion Questions

Are current PM standards sufficiently health protective?
Are some species of PM more dangerous than others?
What is role of ultrafine particles (UFPs)?
How should air quality targets be set? Should form of target expand to account for more than just mass?
How should we include draft PM ISA's new "likely-causal" health endpoints (nervous system effects, cancer) and new more sensitive populations (children, lower socio-economic status)?
What are health impacts of high-concentration acute events (e.g., wildfires)? How should we compare them to day-to-day PM impacts?

Lunch

Keynote – Gina McCarthy

Particulate Matter:
Spotlight on Health Protection

Particulate Matter:
Spotlight on Health Protection

Gina McCarthy

- Former EPA Administrator
- Finalized the Clean Power Plan and the Clean Water Rule
- Professor of the Practice of Public Health in the Department of Environmental Health at Harvard T.H. Chan School of Public Health
- Director of the Center for Climate, Health, and the Global Environmental
- Member of the Board of Directors of the Energy Foundation and Ceres
- M.Sc. in Environmental Health Engineering, Planning and Policy from Tuft's University

Particulate Matter:

Spotlight on Health Protection




Exposure and Risk

Lauren Zeise, Ph.D.

- Appointed by Gov. Brown as Director of the California Office of Environmental Health Hazard Assessment in December 2016
- Former Chief of the cancer unit at the California Department of Health Services
- Leading role in OEHHA's development of CalEnviroScreen
- Co-led the team that developed the hazard trait regulation for California's Safer Consumer Products program
- Member, fellow, former editor, and former councilor of the Society for Risk Analysis
- 2008 recipient of the Society's Outstanding Risk Practitioner Award
- Ph.D. from Harvard University

Exposure and Risk Panel
Particulate Matter: Spotlight on Health

Bay Area Air Quality Management District

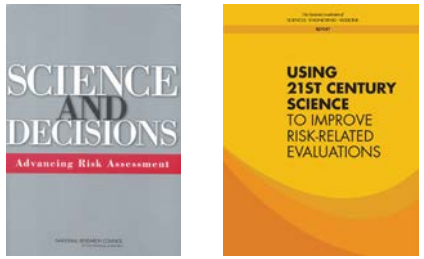
October 28, 2019

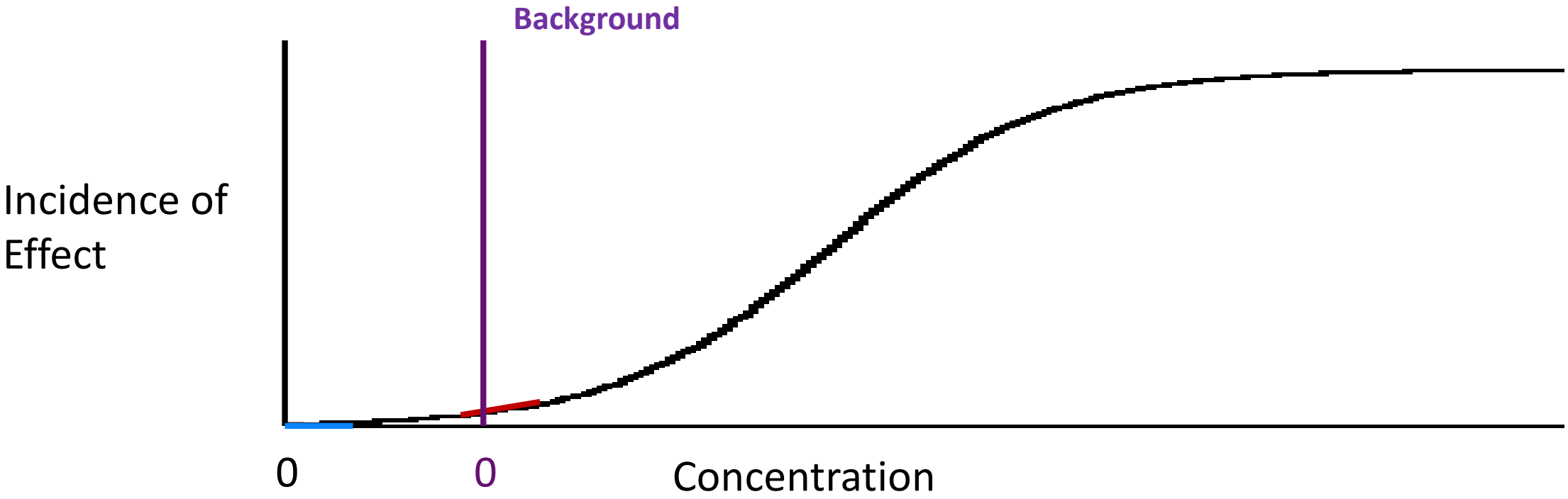
Lauren Zeise

California Environmental Protection Agency

Office of Environmental Health Hazard Assessment

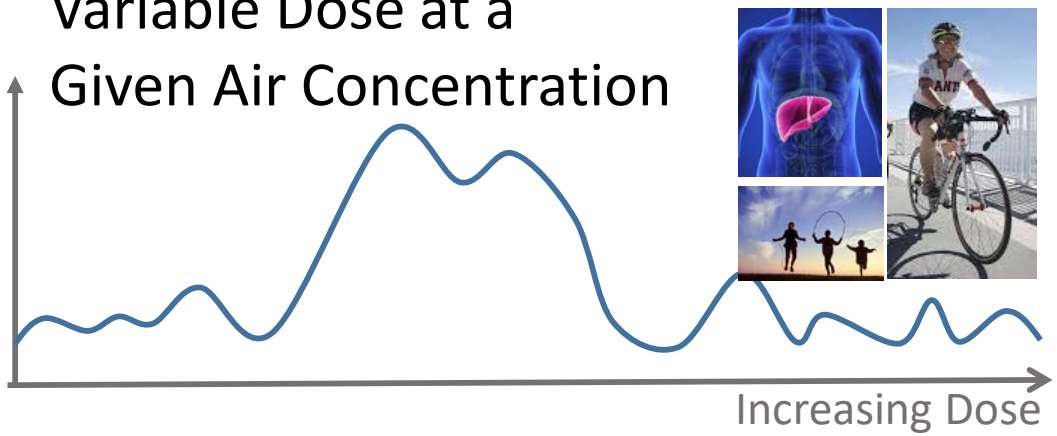
Population Concentration-Response Relationships



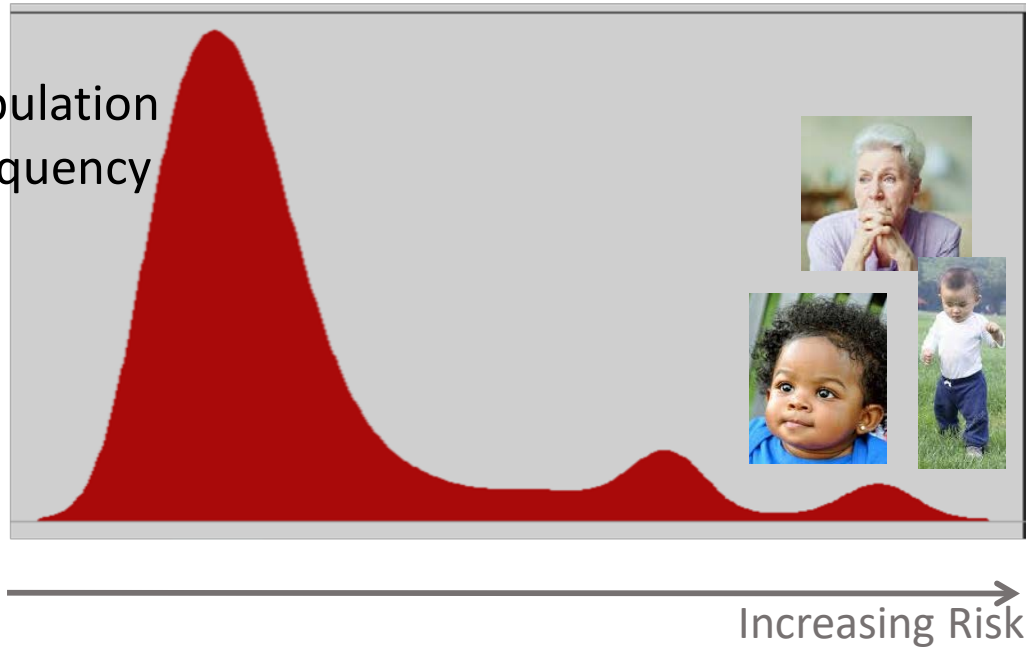


Variability Underlying Concentration Response Observations

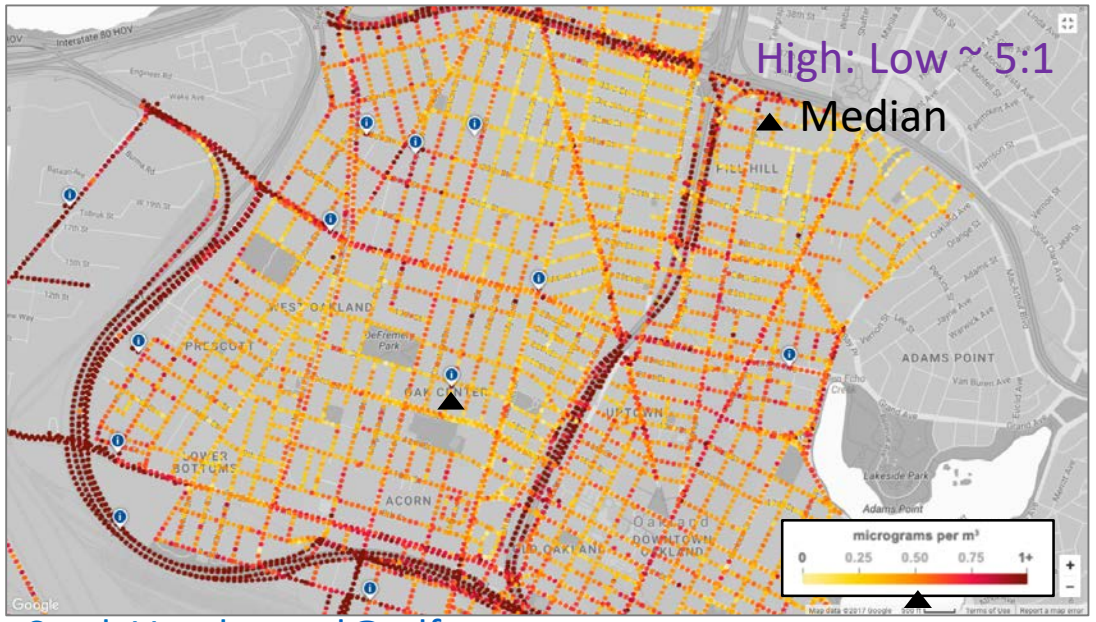
Variable Dose at a Given Air Concentration



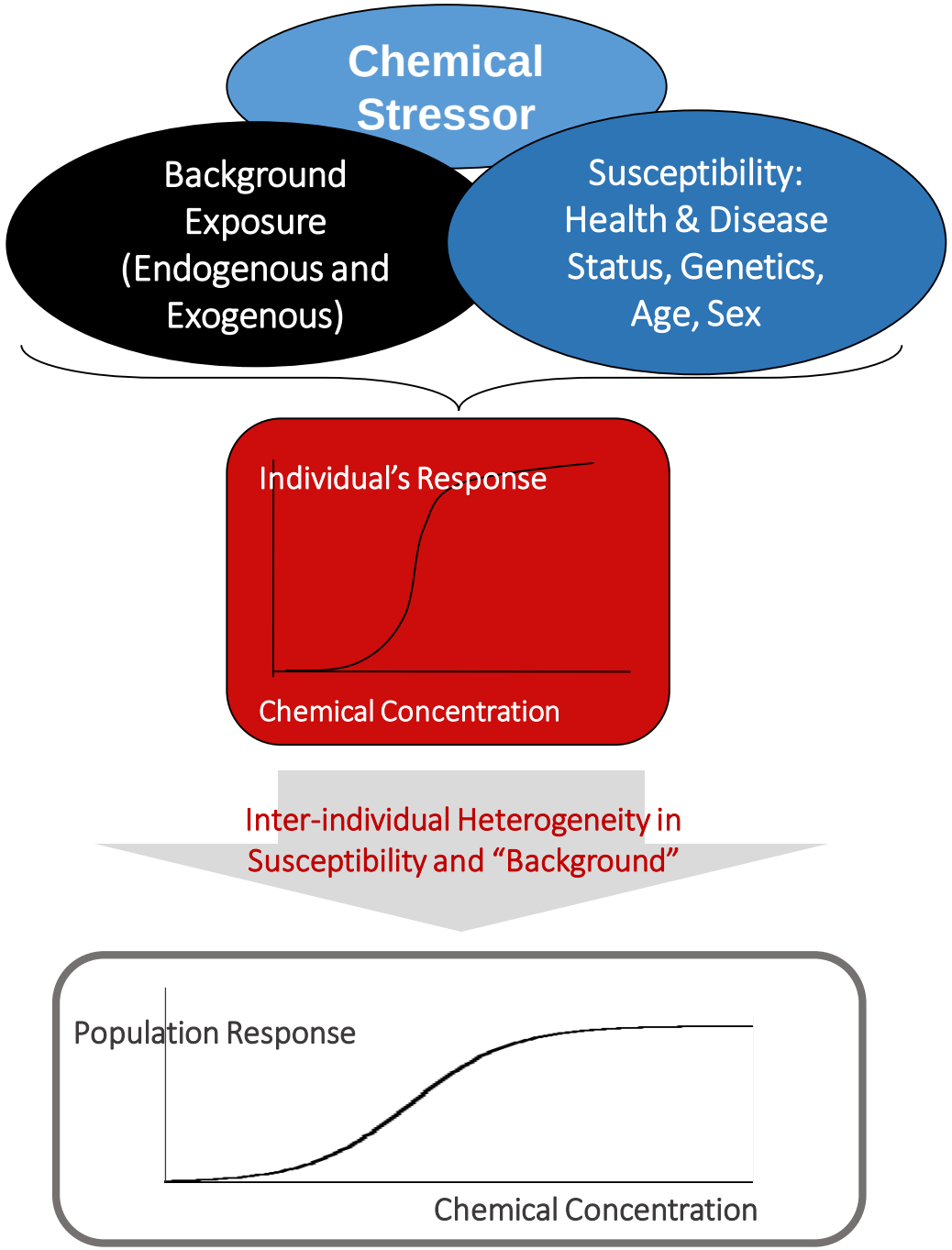
Variable Risk at a Given Dose



Variable Concentration with Location



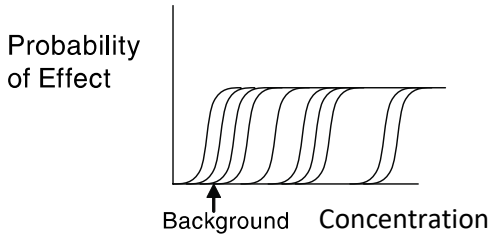
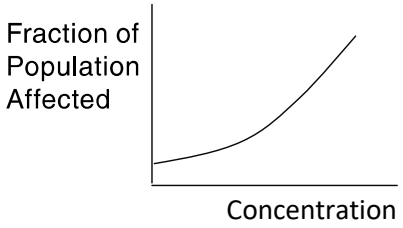
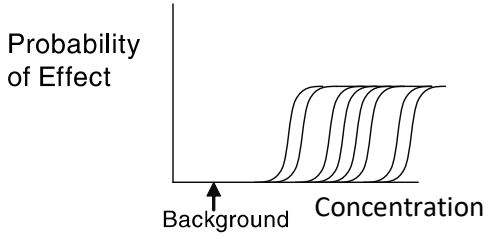
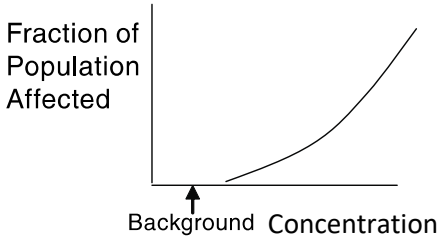
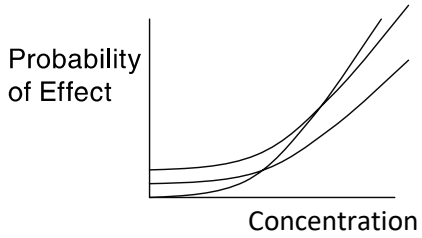
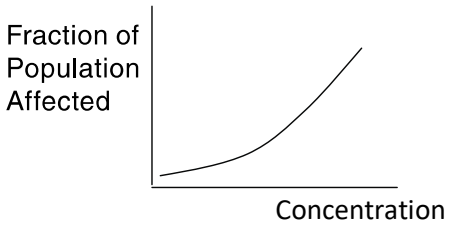
Sarah Vogel svogel@edf.org



Considerations for Interventions

- Risk determined by individual's biologic make-up, health status, endogenous and exogenous exposures that affect toxic chemical process
- Differences among people in these factors affect the shape of the concentration response curve

Individual vs Population Concentration-Response

	Individual level	Population Level
1. An individual's: Nonlinear The population: Linear		
2. An individual's: Nonlinear The population: Nonlinear		
3. An individual's: Linear The population: Linear		

- Measure exposures to diesel exhaust in East Bay community residents
  - Biomonitoring – urine (1-Nitropyrene metabolites)
  - Dust in home
  - Indoor Air (1-Nitropyrene, Black carbon with real-time sensor)
- Measure in child-parent pairs to evaluate exposure patterns within family and across ages
- Collect urine & air samples at two time points to look at seasonal differences
  - 25 families: one urine sample at end of 4 day periods
  - 15 families: daily urine samples x 4 days
- Collect information related to sources and activities
  - Exposure questionnaire
  - GPS data loggers – every 5 minutes
  - Activity diaries

BIOMONITORING CALIFORNIA

cerch Center for Environmental Research & Children's Health

OEHHA

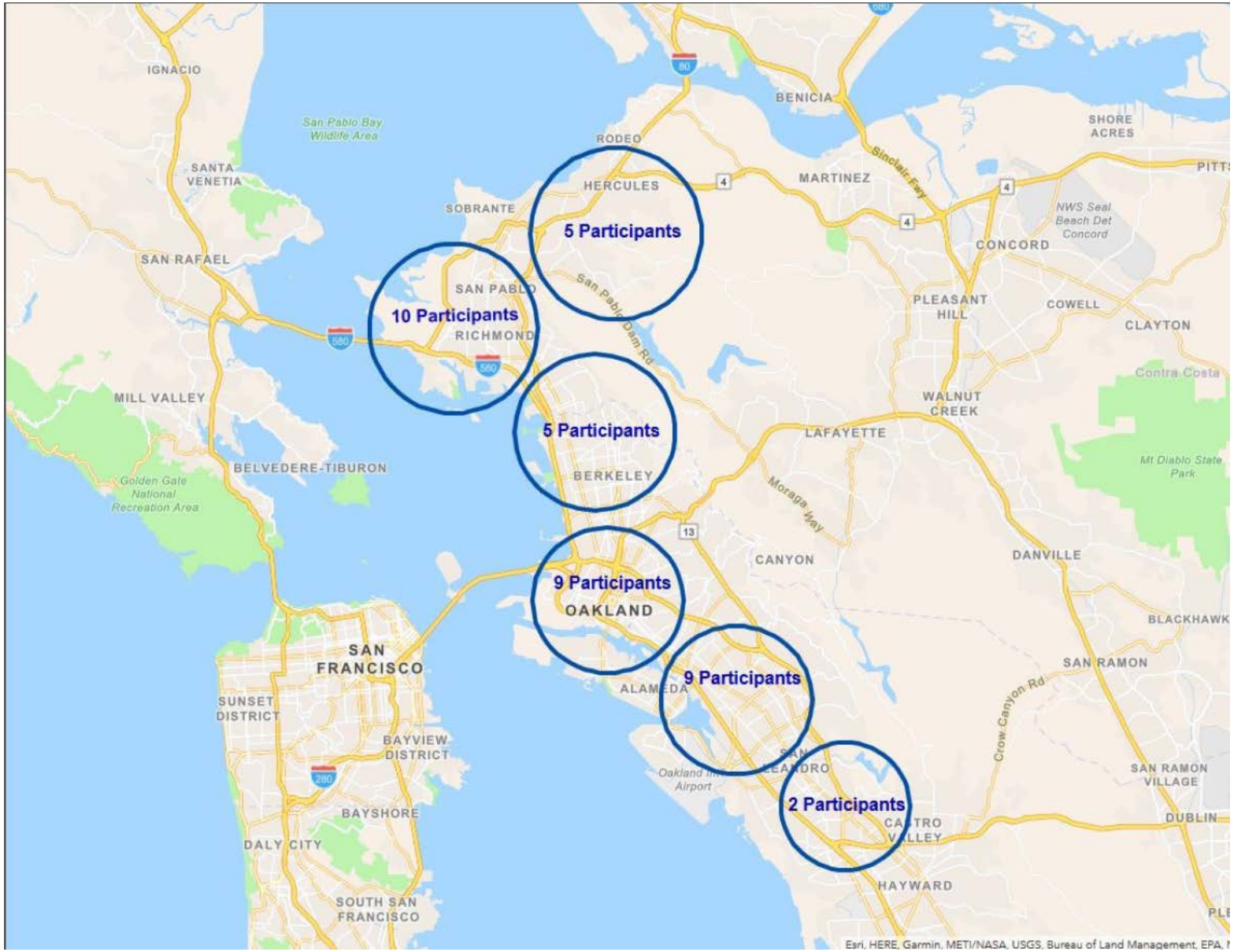
Berkeley UNIVERSITY OF CALIFORNIA

CDPH California Department of PublicHealth

UNIVERSITY of WASHINGTON

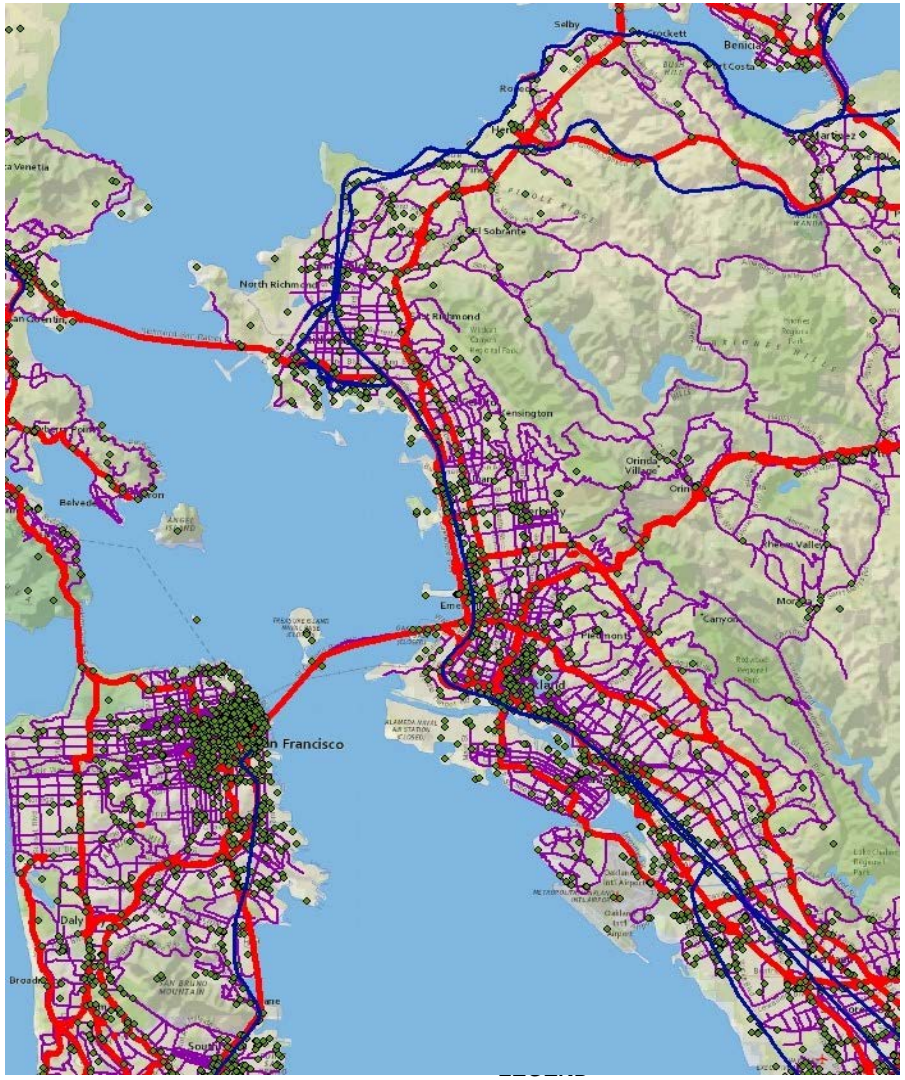
EBDEP Participant Locations

- East Bay
- Neighborhoods with a range of diesel exhaust exposure, based on:
  - CalEnviroScreen's diesel particulate matter indicator (based on CARB data)
  - Diesel truck traffic patterns
  - Local air pollution mapping



GIS Diesel Source Layers and Maps

- Permitted stationary emission sources (BAAQMD)
- Railway lines and railway road crossings
- Caltrans Truck Network
- Caltrans Bottlenecks (highway congestion)
- AC Transit and Amtrak bus routes and stops
- Major roads
- Industrial land use zoning maps (county)
- Highway Performance Monitoring System traffic data
- California ports



Complementary Pilot Air Quality Study

- Measure ambient air concentrations of black carbon and selected PAHs in areas of Richmond relevant to EBDEP
- Conduct field sampling for several days during periods of moderate and high pollution
- Analyze results to:
  - Compare levels across location and time
  - Examine patterns for possible clues on sources

Berkeley UNIVERSITY OF CALIFORNIA

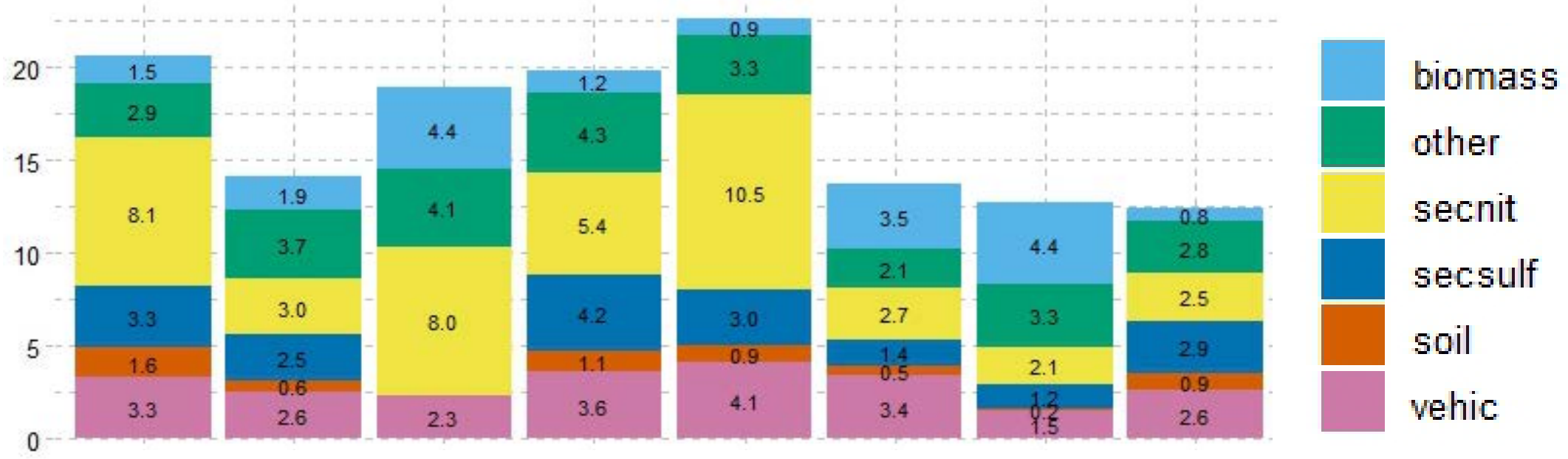
Principal Investigator: Betsey Noth, UC Berkeley
OEHHA funded

OEHHA Biomonitoring to Support AB 617

- Directly measure exposure to a chemical(s) of concern
- Establish baseline exposures prior to reduction efforts
- Examine exposures associated with a specific source(s) in the community, and/or
- Evaluate the effectiveness of exposure reduction efforts

Estimated $PM_{2.5}$ Source Contribution by Monitoring Site

Annual Average $PM_{2.5}$ µg/m³



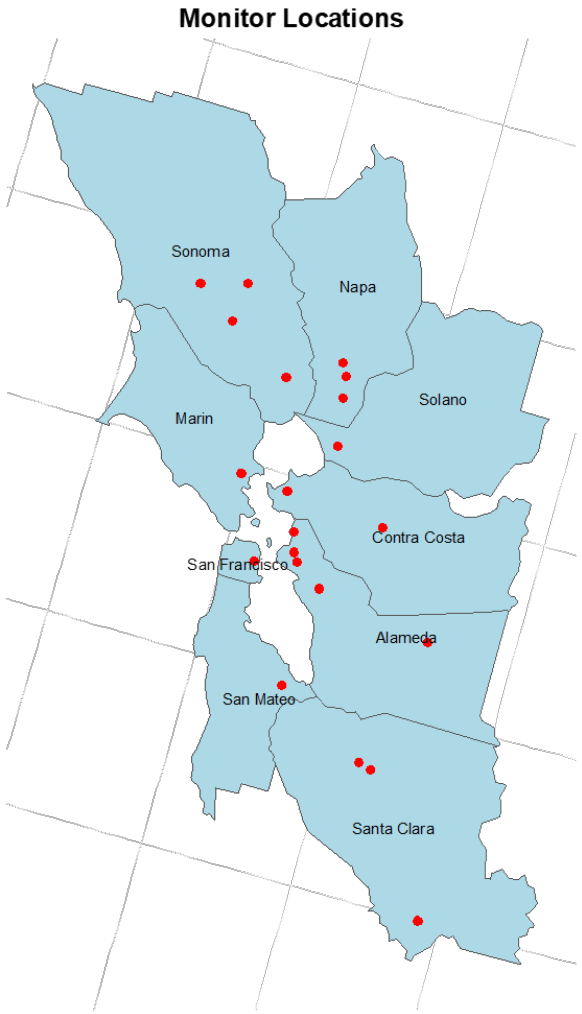
Secnit: Secondary Ammonium Nitrate
Secsulf: Secondary Ammonium Sulfate

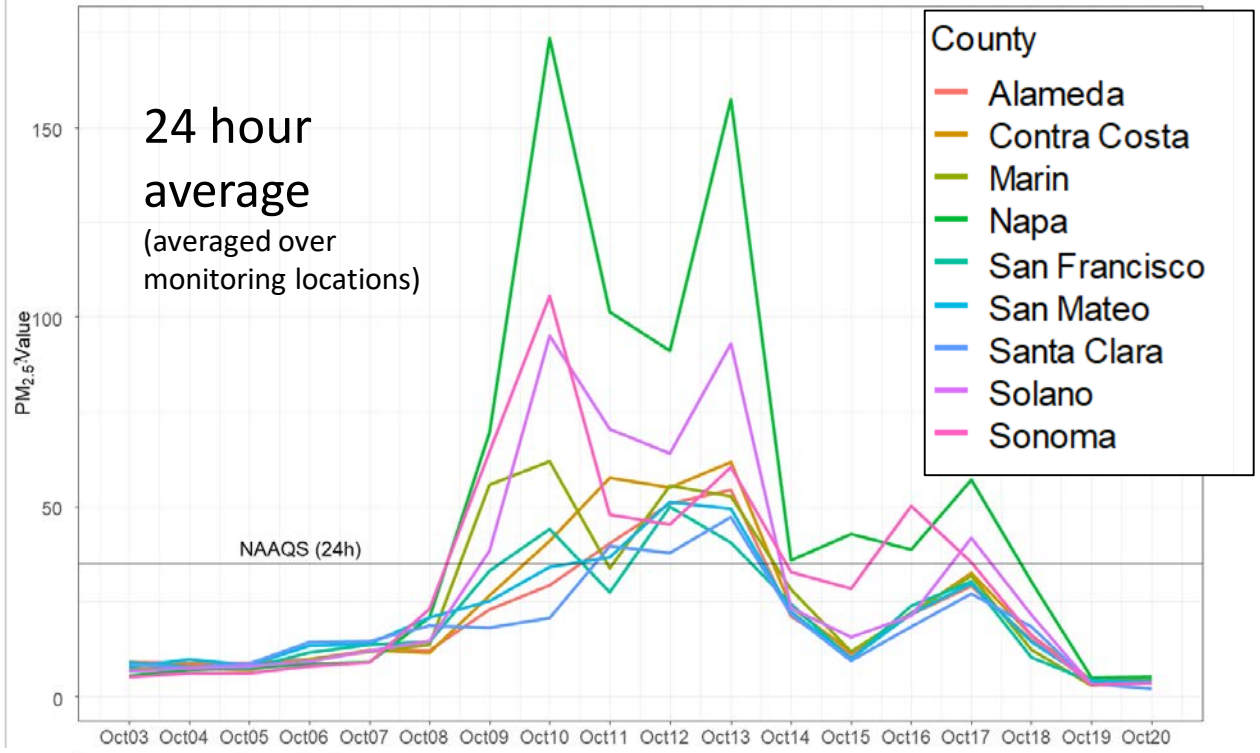
Source	Marker Constituents
Biomass	EC, OC, K
Secondary Ammonium Nitrate	NO_3^-, NH_4^+
Secondary Ammonium Sulfate	$SO_4^=$, NH_4^+
Resuspended Soil	Al, Si, Ca, Fe, Ti
Vehicular Emissions	EC, OC, Fe, Cu, Zn

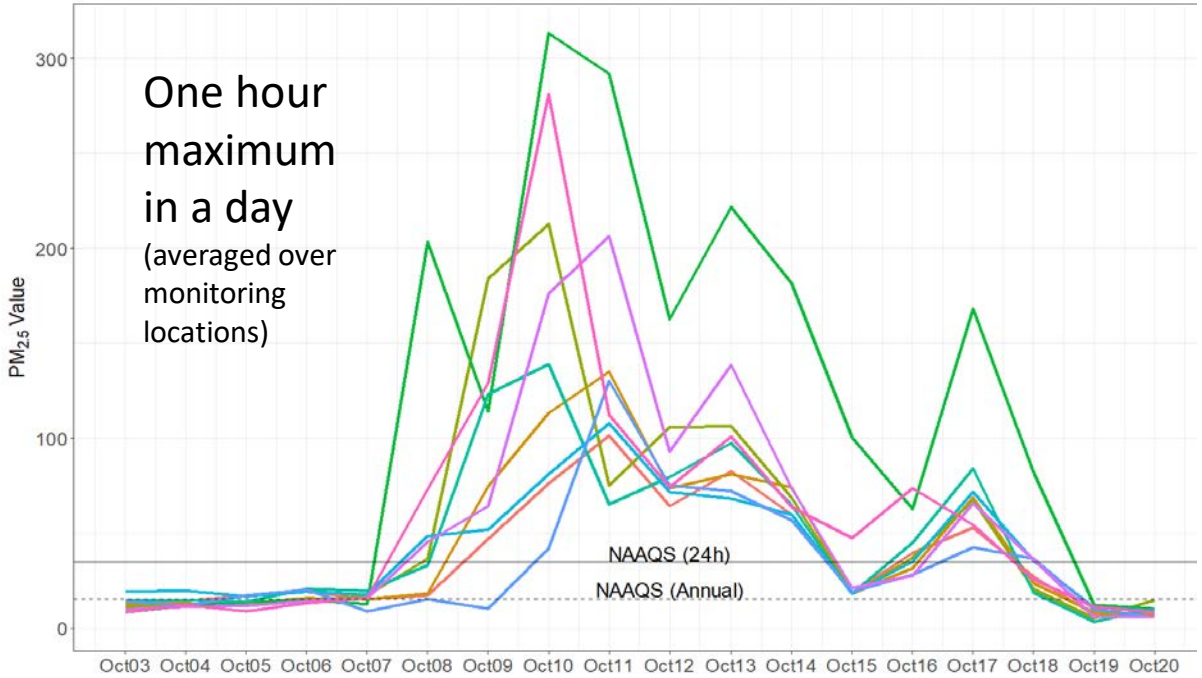
$PM_{2.5}$ in Bay Area During 2017 Napa Wildfire

Health Outcomes Being Investigated

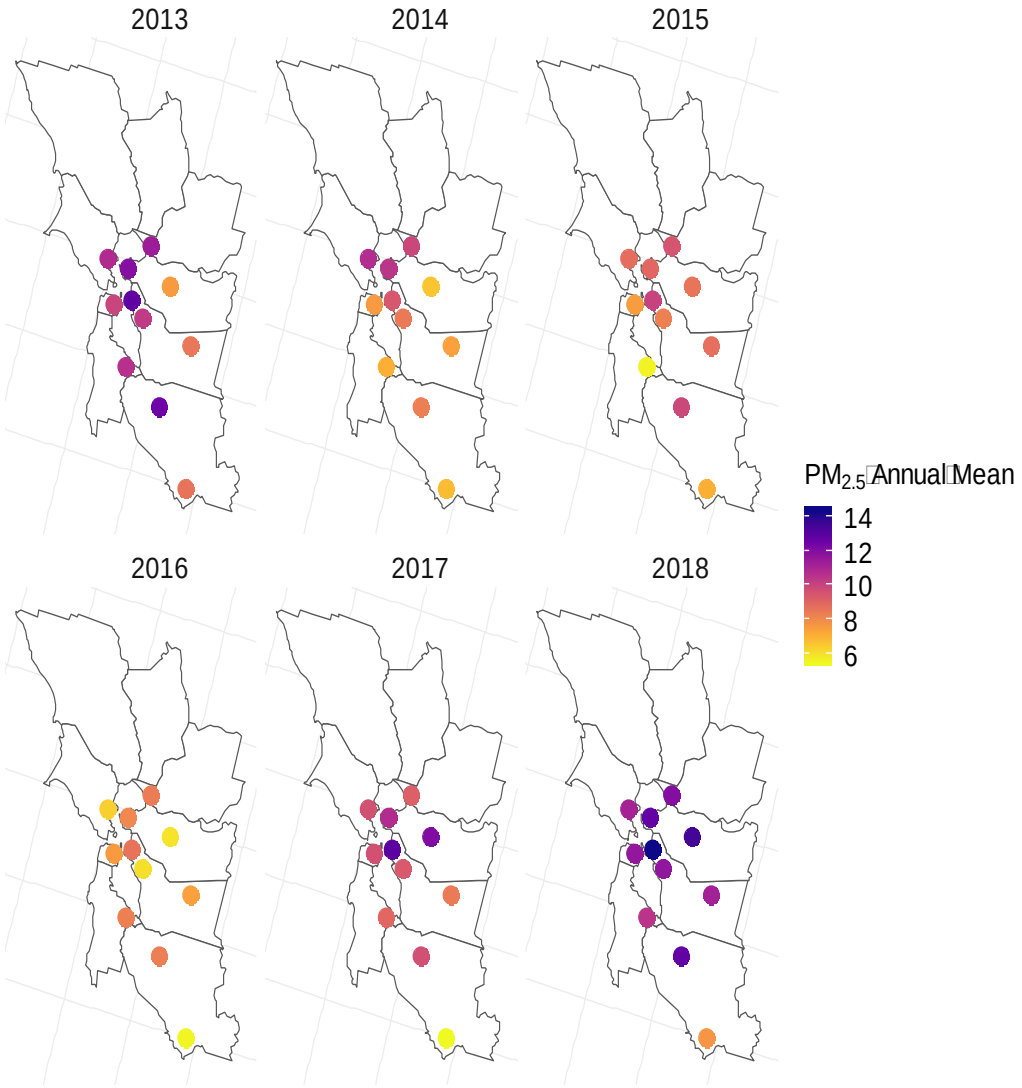
- Cardiovascular Disease
- Ischemic Heart Disease
- Acute Myocardial Infarction
- Dysrhythmia
- Cerebrovascular Disease
- Transient Ischemic Attack
- Peripheral Vascular Disease
- Diabetes
- Respiratory Disease
- Asthma/Wheeze
- Pneumonia
- Chronic Lower Respiratory Disease
- Acute Upper Respiratory Infection
- Mental/Behavioral Disorders



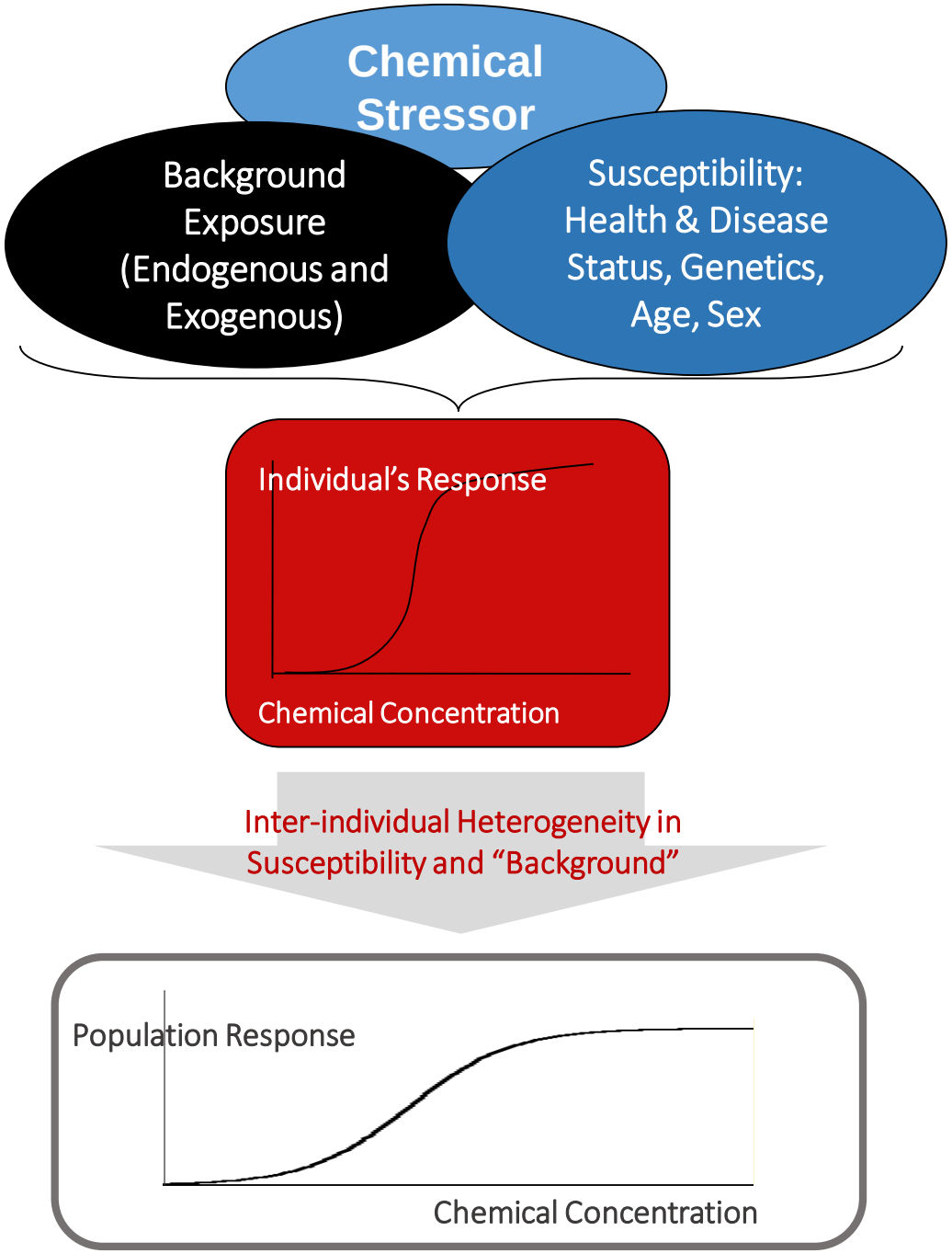




Wildfire Affects Annual Average of $PM_{2.5}$



- Wildfire PM adds to underlying "baseline"
- Monitor in West Oakland:
  - 2017: 12.9 µg/m^3
  - 2018: 14.4 µg/m^3


Chemical Stressor
Background Exposure (Endogenous and Exogenous)
Susceptibility: Health & Disease Status, Genetics, Age, Sex
Individual's Response
Chemical Concentration
Inter-individual Heterogeneity in Susceptibility and "Background"
Population Response
Chemical Concentration

Acknowledgements

- OEHHA Community Health and Environmental Impacts Section: Rupa Basu, Keita Ebisu, et al.
- OEHHA Safer Alternatives Assessment and Biomonitoring Section: Sara Hoover, Russ Bartlett, Duyen Kauffman et al.

Particulate Matter:
Spotlight on Health Protection

Bay Area Air Quality Management District

Julian Marshall, Ph.D.

- Kiely Endowed Professor of Environmental Engineering at University of Washington with a focus on air quality management
- Founded and runs the Grand Challenges Impact Lab, a UW study abroad program in Bangalore, India
- Associate Editor for Environmental Health Perspectives and Development Engineering
- Published over 100 peer-reviewed journal articles
- Ph.D. in Energy and Resources from UC Berkeley

Particulate Matter:
Spotlight on Health Protection

Scott Jenkins, Ph.D.

- Senior Environmental Health Scientist in EPA's Office of Air Quality Planning and Standards (OAQPS)
- Currently leading EPA’s review of the National Ambien Air Quality Standards (NAAQS) for Particulate Matter (PM)
- Howard Hughes Postdoctoral Research Fellow in the Department of Cell Biology at Duke University
- Ph.D. in Behavioral Neuroscience from the University of Alabama at Birmingham

REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR PARTICULATE MATTER

OVERVIEW OF THE DRAFT POLICY ASSESSMENT

Scott Jenkins
U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards

Presentation to the Bay Area Air Quality Management District

October 28, 2019

Outline of Presentation

- Overview of the standards, process and schedule
- Key information and analyses in draft Policy Assessment
- Preliminary conclusions on the primary $PM_{2.5}$ standards

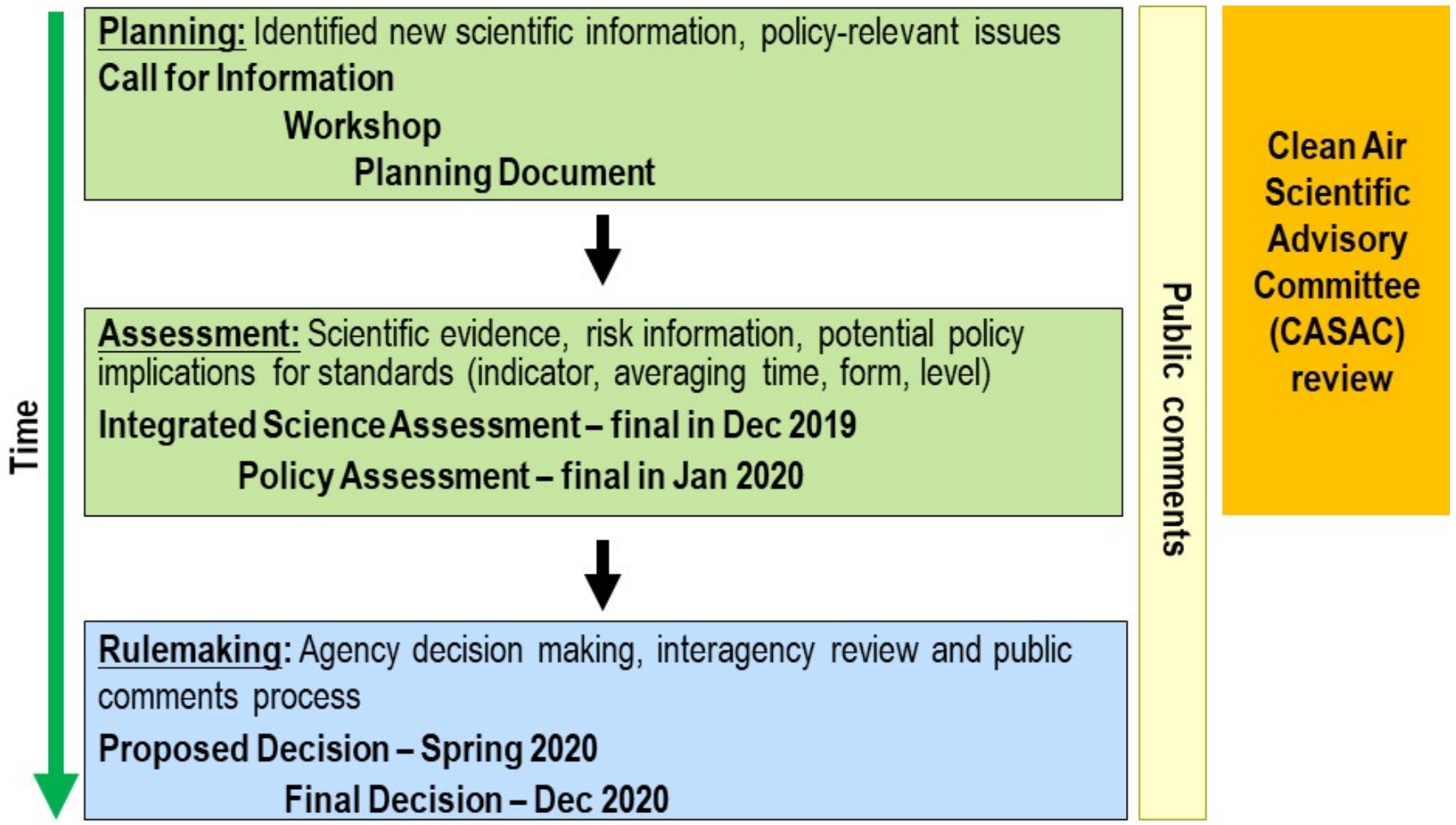
Current PM Standards Under Review

Current Standards – Last Review Completed in 2012*					Decisions in 2012 Review
Indicator	Averaging Time	Primary/Secondary	Level	Form	
$PM_{2.5}$	Annual	Primary	12.0 µg/m^3	Annual arithmetic mean, averaged over 3 years	Revised level from 15 to 12 µg/m^3**
		Secondary	15.0 µg/m^3		Retained**
	24-hour	Primary and Secondary	35 µg/m^3	98th percentile, averaged over 3 years	Retained
PM_{10}	24-hour	Primary and Secondary	150 µg/m^3	Not to be exceeded more than once per year on average over a 3-year period	Retained

*Prior to 2012, PM NAAQS were reviewed and revised several times – established in 1971 (total suspended particulate – TSP) and revised in 1987 (set PM_{10}), 1997 (set $PM_{2.5}$), 2006 (revised $PM_{2.5}$, PM_{10})

**EPA eliminated spatial averaging for the annual standards

Process and Anticipated Schedule for This Review of the PM NAAQS



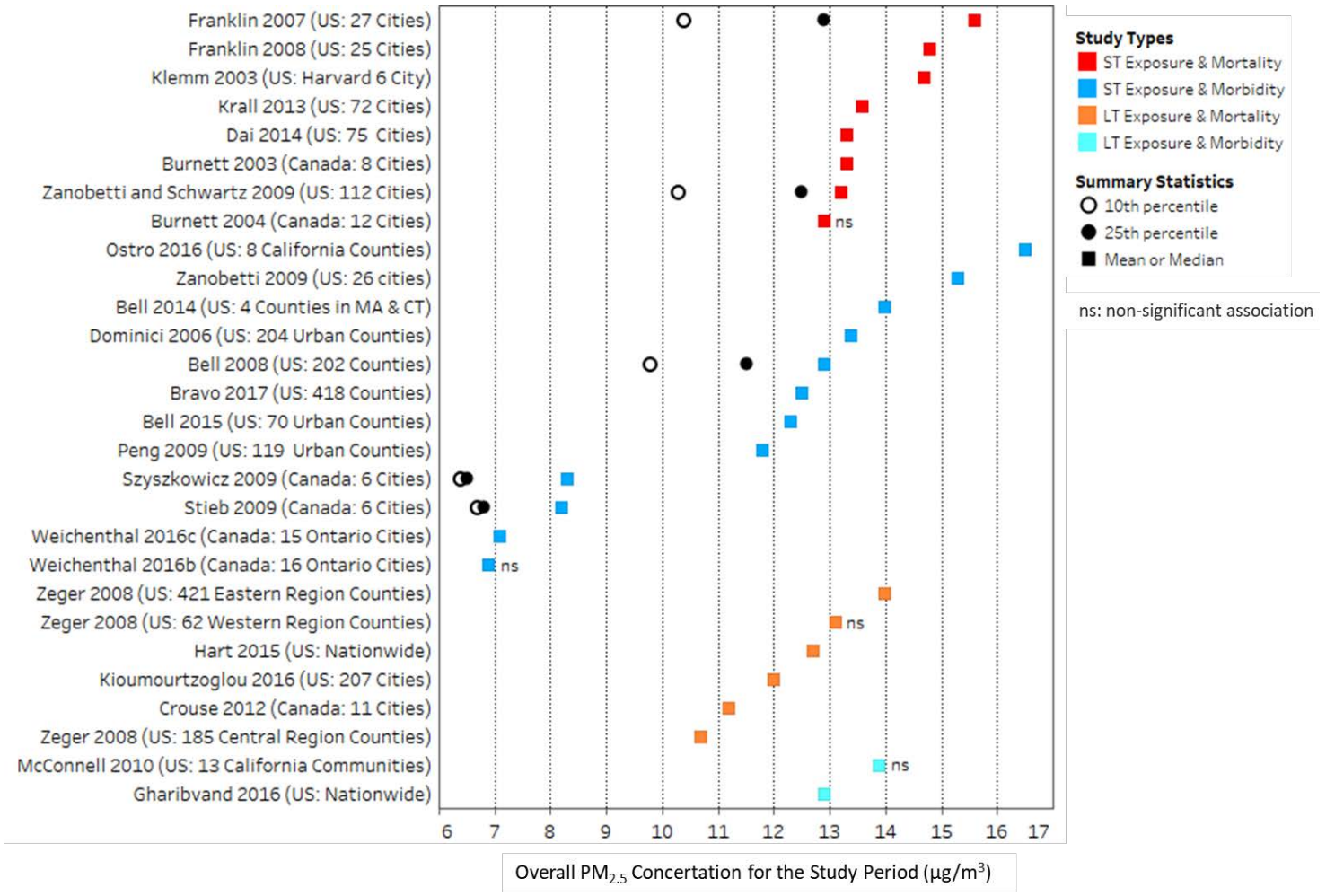
Evaluating Primary $PM_{2.5}$ Standards: Summary of Approach

- The **annual $PM_{2.5}$ standard** is viewed as the principle means of providing public health protection against the bulk of the distribution of short- and long-term $PM_{2.5}$ exposures
- In previous reviews, conclusions on the annual $PM_{2.5}$ standard have been informed by consideration of the $PM_{2.5}$ air quality distributions associated with mortality or morbidity in epidemiologic studies
  - The current level of 12.0 $\mu g/m^3$ was set below the overall means of the long- and short-term $PM_{2.5}$ exposure estimates in key studies
- In this review, the draft PA characterizes those distributions by identifying overall means of $PM_{2.5}$ exposure estimates, concentrations corresponding to the lower quartiles of data (when available), and study-area metrics similar to design values (pseudo-design values)
- The **24-hour $PM_{2.5}$ standard**, with its 98th percentile form, is viewed as a means of providing protection against short-term exposures to peak $PM_{2.5}$ concentrations, such as can occur in areas with strong contributions from local or seasonal sources, even when mean $PM_{2.5}$ concentrations remain relatively low
- Controlled human exposure studies provide evidence for health effects following single, short-term $PM_{2.5}$ exposures to concentrations that typically correspond to upper end of the $PM_{2.5}$ air quality distribution in the U.S. (i.e., “peak” concentrations – see additional slides)

$PM_{2.5}$ Concentrations in Epidemiologic Studies

- Overall mean concentrations reflect study averages of daily or annual $PM_{2.5}$ exposures – bulk of data generally occurs around overall means
- Key studies consistently reporting positive and statistically significant associations have overall mean $PM_{2.5}$ concentrations > 8.0 μg/m^3
- In studies with data available, 75% of health events occurred in areas with mean $PM_{2.5}$ concentrations ≥ 11.5 μg/m^3 (U.S. studies) or 6.5 μg/m^3 (Canadian studies)

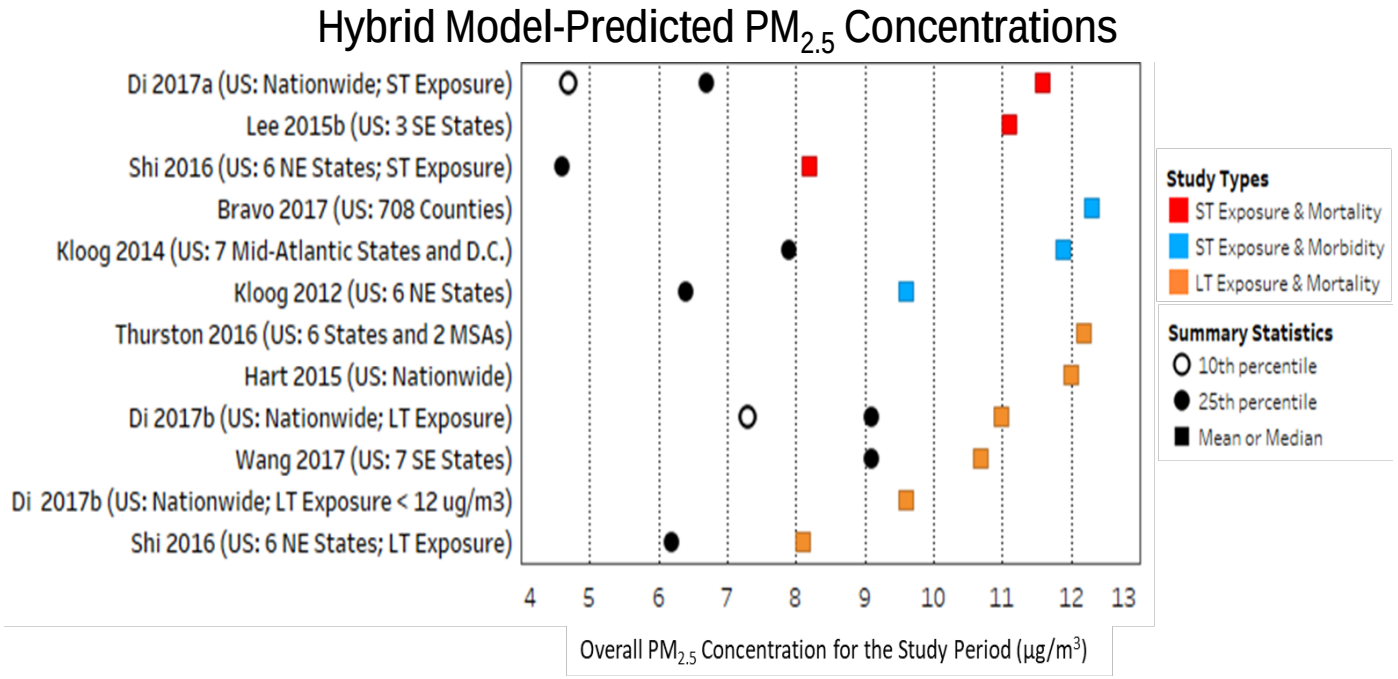
Monitored $PM_{2.5}$ concentrations*



*Colored squares reflect overall study-reported mean (or median) $PM_{2.5}$ concentrations. Circles reflect the mean $PM_{2.5}$ concentrations corresponding to the 25th (filled) and 10th (open) percentiles of health events.

$PM_{2.5}$ Concentrations in Epidemiologic Studies (Continued)

- Many new studies have used hybrid modeling approaches to estimate $PM_{2.5}$ exposures in monitored and unmonitored locations
- Approaches use information from multiple sources, potentially including satellites and models, in addition to ground-based monitors
- All of these key studies report positive and statistically significant associations and have overall mean $PM_{2.5}$ concentrations > 8.0 $\mu g/m^3$
- In most studies with data available, 75% of exposures (or deaths) are at predicted ambient $PM_{2.5}$ concentrations > 6.0 $\mu g/m^3$



Uncertainties in using this information to inform conclusions on standards include:

- Mean and lower quartile concentrations are not the same as those used by the EPA to compare with standard levels
- Studies have not identified a threshold concentration below which associations do not occur
- Hybrid model performance varies by location, with factors contributing to poorer performance (e.g., sparse monitoring) often coinciding with relatively low ambient $PM_{2.5}$ concentrations

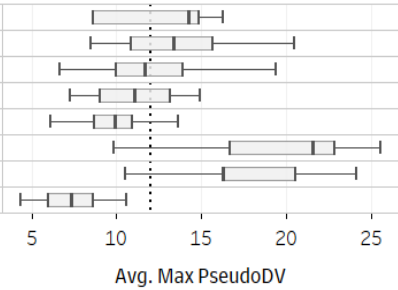
*Colored squares reflect overall study-reported mean $PM_{2.5}$ concentrations. Circles reflect the mean $PM_{2.5}$ concentrations corresponding to the 25th (filled) and 10th (open) percentiles of exposures or deaths.

$PM_{2.5}$ Annual Pseudo-Design Values in Locations of Key Studies

- The draft PA also identifies monitor-based metrics – similar to design values – in study locations (annual and 24-hr pseudo-design values)
- For most of the 29 key studies evaluated, ≥ about 25% of study area health events/populations were in locations that generally would have met both standards during study periods
- For 9 key studies, > 50% of study area health events/populations were in such locations
- For 4 key studies, > 75% of study area health events/populations were in such locations
- Uncertainties include:
  - Many studies examine a mix of locations and time periods meeting and violating standards
  - Values are not available in unmonitored areas
  - Values do not reflect current near-road monitoring requirements

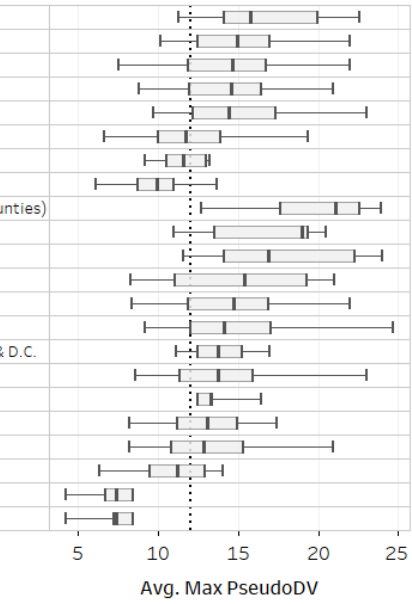
Long-term exposure studies

Country	Endpoint Group	Citation	Study Years	Geographic Areas
U.S.	Mortality	Lepeule et al., 2012*	2001-2009	6 U.S. Cities
		Kiomourtzoglou et al., 2016*	2000-2010	207 U.S. Cities
		Di et al., 2017b*	2000-2012	U.S. Nationwide
		Wang et al., 2017*	2000-2013	7 SE U.S. States
		Shi et al., 2016*	2003-2008	6 NE U.S. States
	Morbidity	Urman et al., 2014*	2002-2007	8 CA Counties
		Mcconnell et al., 2010	2003-2005	13 CA Communities
Canada	Mortality	Pinault et al., 2016*	2000-2011	Multicity



Short-term exposure studies

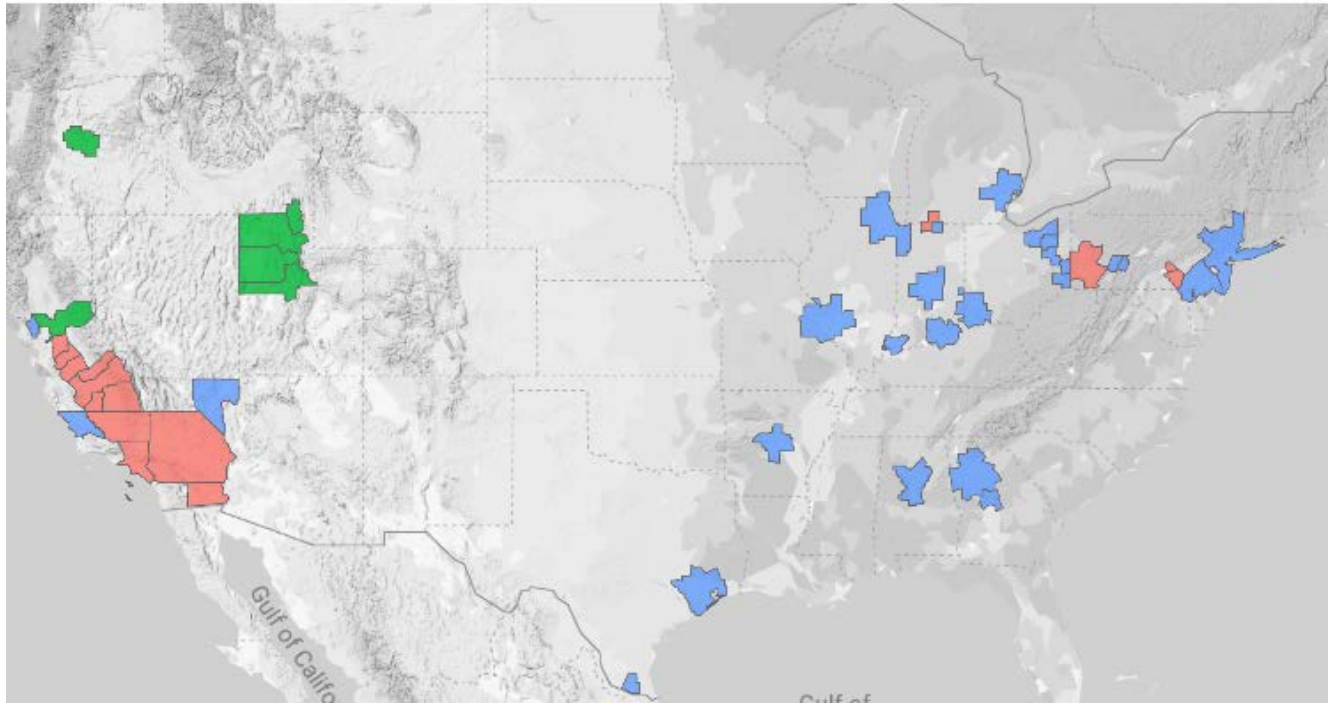
Coun..	Endpoint Group	Citation	Study Years	Geographic Areas
U.S.	Mortality	Franklin et al., 2008*	2000-2005	25 U.S. Cities
		Dai et al., 2014*	2000-2006	75 U.S. Cities
		Baxter et al., 2017*	2001-2005	77 U.S. Cities
		Zanobetti et al., 2014*	1999-2010	121 U.S. Cities
		Zanobetti and Schwartz, 2009*	1999-2005	112 U.S. Cities
		Di et al., 2017a*	2000-2012	U.S. Nationwide
		Lee et al., 2015b*	2007-2011	3 SE U.S. States
		Shi et al., 2016*	2003-2008	6 NE U.S. States
	Morbidity	Yap et al, 2013*	2000-2005	CA (Central & Southern Counties)
		Ostro et al., 2016*	2005-2009	8 CA Counties
		Zanobetti et al., 2009*	2000-2003	26 U.S. Cities
		Malig et al., 2013*	2005-2008	35 CA Counties
		Peng et al., 2009*	2000-2006	119 U.S. Urban Counties
		Dominici et al., 2006*	1999-2002	204 U.S. Urban Counties
		Kloog et al., 2014*	2000-2006	7 U.S. Mid-Atlantic States & D.C.
		Bell et al., 2008*	1999-2005	202 U.S. Urban Counties
		Bell et al., 2014*	2000-2004	4 U.S. Counties, MA & CT
		Bravo et al., 2017*	2002-2006	708 U.S. Counties
		Bell et al., 2015*	1999-2010	213 U.S. Urban Counties
		Kloog et al., 2012*	2000-2006	6 NE U.S. States
Canada	Morbidity	Weichenthal et al., 2016b	2004–2011	16 Ontario Cities
		Weichenthal et al., 2016c*	2004-2011	15 Ontario Cities



* Whiskers correspond to 5th and 95th percentiles, boxes correspond to 25th and 75th percentiles, central vertical lines correspond to 50th percentiles

$PM_{2.5}$ Risk Assessment

- Examined $PM_{2.5}$-associated mortality risk in 47 urban study areas
- Assessed current standards; alternative annual standards with levels of 11.0, 10.0, and 9.0 µg/m^3; alternative 24-hour standard with a level of 30 µg/m^3
- 2015 analysis year
- Examined two approaches to adjusting air quality
  - Focus on primary PM
  - Focus on secondary PM



47 urban study areas (population ≥ 30 years: ~60M)

- 30 annual-controlling (population ≥ 30 years: ~50M)
- 11 daily-controlling (population ≥ 30 years: ~4M)
- 6 mixed (population ≥ 30 years: ~5M)

Above 10 annual and 30 daily
Above 30 daily
Above 10 annual

Summary of Risk Estimates

Estimates of $PM_{2.5}$-associated deaths in the full set of 47 study areas

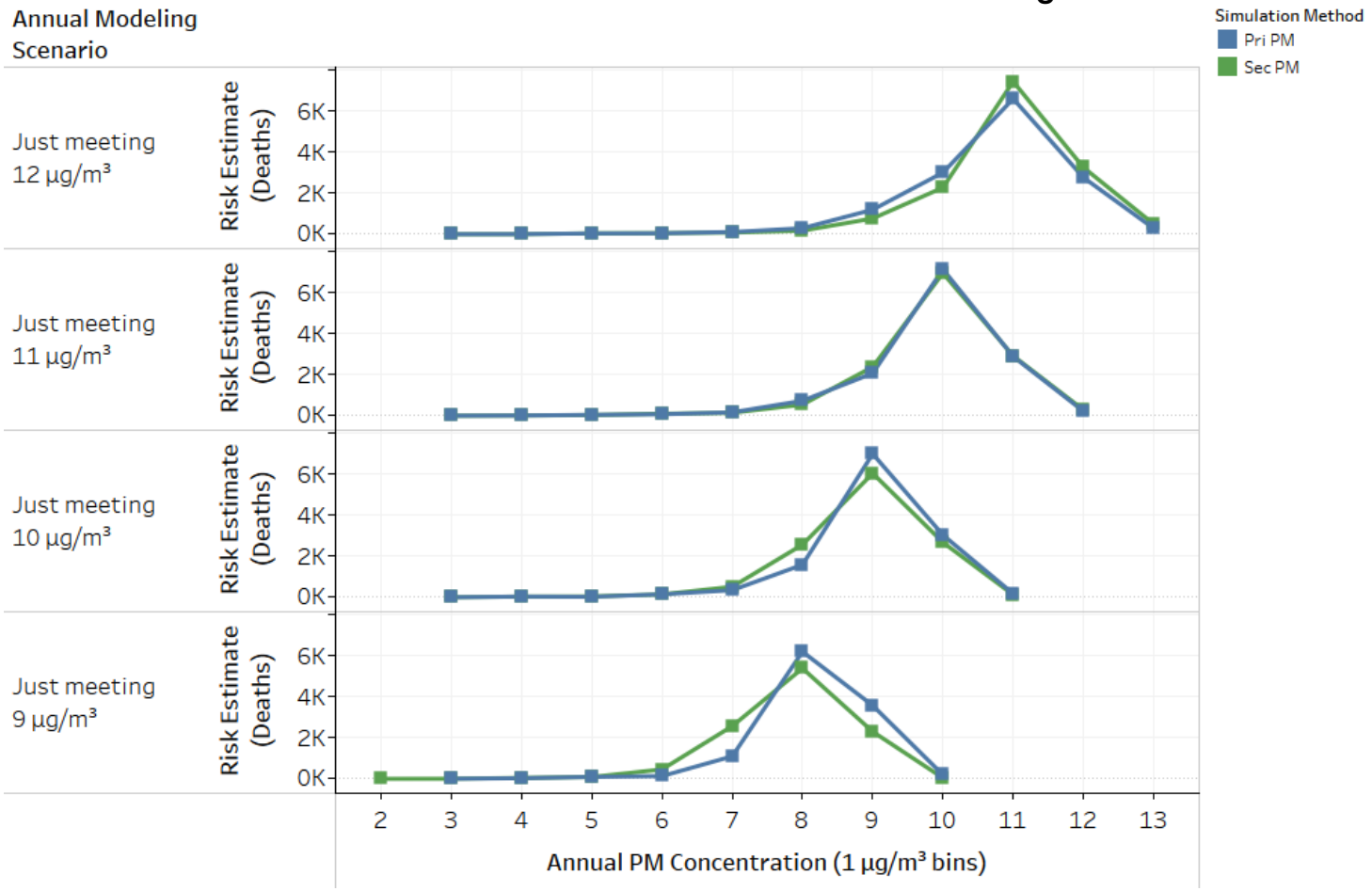
Endpoint	Study	Air quality simulation approach*	Current Standad Absolute Risk (12/35 µg/m^3)	CS (12/35) % of baseline**	Alternative Standard Absolute Risk	
					Alternative Annual (10 µg/m^3)	Alternative 24-hr (30 µg/m^3)
Long-term exposure related mortality						
Ischemic Heart Disease	Jerrett 2016	Pri-PM	16,500 (12,600-20,300)	14.1	14,400 (11,000-17,700)	16,400 (12,500-20,000)
		Sec-PM	16,800 (12,800-20,500)	14.3	14,200 (10,900-17,500)	16,500 (12,600-20,200)
	Pope 2015	Pri-PM	15,600 (11,600-19,400)	13.3	13,600 (10,100-17,000)	15,400 (11,500-19,200)
		Sec-PM	15,800 (11,800-19,600)	13.4	13,400 (9,970-16,700)	15,600 (11,600-19,400)
All-cause	Di 2017	Pri-PM	46,200 (45,000-47,500)	8.4	40,300 (39,200-41,400)	45,700 (44,500-47,000)
		Sec-PM	46,900 (45,600-48,200)	8.5	39,700 (38,600-40,800)	46,200 (44,900-47,500)
	Pope 2015	Pri-PM	51,300 (41,000-61,400)	7.1	44,700 (35,700-53,500)	50,700 (40,500-60,700)
		Sec-PM	52,100 (41,600-62,300)	7.2	44,000 (35,100-52,700)	51,300 (41,000-61,400)
	Thurston 2015	Pri-PM	13,500 (2,360-24,200)	3.2	11,700 (2,050-21,100)	13,300 (2,330-24,000)
		Sec-PM	13,700 (2,400-24,600)	3.2	11,500 (2,010-20,700)	13,500 (2,360-24,200)
Lung cancer	Turner 2016	Pri-PM	3,890 (1,240-6,360)	8.9	3,390 (1,080-5,560)	3,850 (1,230-6,300)
		Sec-PM	3,950 (1,260-6,460)	9.1	3,330 (1,060-5,470)	3,890 (1,240-6,370)
Short-term exposure related mortality						
All cause	Baxter 2017	Pri-PM	2,490 (983-4,000)	0.4	2,160 (850-3,460)	2,460 (970-3,950)
		Sec-PM	2,530 (998-4,060)	0.4	2,120 (837-3,400)	2,490 (982-3,990)
	Ito 2013	Pri-PM	1,180 (-16-2,370)	0.2	1,020 (-14-2,050)	1,160 (-16-2,340)
		Sec-PM	1,200 (-16-2,400)	0.2	1,000 (-14-2,020)	1,180 (-16-2,370)
	Zanobetti 2014	Pri-PM	3,810 (2,530-5,080)	0.7	3,300 (2,190-4,400)	3,760 (2,500-5,020)
		Sec-PM	3,870 (2,570-5,160)	0.7	3,250 (2,160-4,330)	3,810 (2,530-5,070)

* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)

** CS denotes the current standard.

Summary of Risk Estimates (Continued)

Distributions of estimated risks in the 30 study areas where the annual standard is controlling*



Uncertainty in risk estimates results from uncertainties in the underlying epidemiologic studies, in the air quality adjustments, and in the application of study and air quality information to develop quantitative estimates of $PM_{2.5}$-associated mortality risks

*Estimates of ischemic heart disease deaths associated with long-term $PM_{2.5}$ exposures for air quality adjusted to simulate "just meeting" the current and alternative primary standards (based on Jerrett et al., 2016)

Preliminary Conclusions on the Current Primary $PM_{2.5}$ Standards

- The available scientific information can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the current annual and 24-hour primary $PM_{2.5}$ standards
- Basis for this preliminary conclusion:
  - Long-standing body of health evidence, strengthened in this review, supporting relationships between $PM_{2.5}$ exposures and various outcomes, including mortality and serious morbidity effects
  - Recent U.S. and Canadian epidemiologic studies reporting positive and statistically significant health effect associations for $PM_{2.5}$ air quality likely to be allowed by the current standards
  - Analyses of pseudo-design values indicating substantial portions of study area health events/populations in locations with air quality likely to have met the current $PM_{2.5}$ standards
  - Risk assessment estimates that the current primary standards could allow thousands of $PM_{2.5}$-associated deaths per year – most at annual average $PM_{2.5}$ concentrations from 10 to 12 $\mu g/m^3$ (well within the range of overall mean concentrations in key epidemiologic studies)

Preliminary Conclusions on the Current Primary $PM_{2.5}$ Standards (Continued)

- In contrast, a conclusion that the current primary $PM_{2.5}$ standards do provide adequate health protection would place little weight on the epidemiologic evidence or the risk assessment
- Such a conclusion would place greater weight on uncertainties and limitations, including:
  - Increasing uncertainty in the biological pathways through which $PM_{2.5}$ exposures could cause serious health effects as the ambient concentrations being considered fall farther below the $PM_{2.5}$ exposure concentrations shown to cause effects in experimental studies
  - Increasing uncertainty in the potential public health impacts of air quality improvements as the ambient concentrations being considered fall farther below those present in accountability studies that document improving health with declining $PM_{2.5}$
    - Accountability studies evaluate air quality improvements with "starting" mean $PM_{2.5}$ concentrations (i.e., prior to the reductions evaluated) from ~13 to > 20 $\mu g/m^3$
  - Uncertainty in the risk assessment results from uncertainties in the underlying epidemiologic studies, in the air quality adjustments, and in the application of study and air quality information to develop quantitative estimates of $PM_{2.5}$-associated mortality risks

Preliminary Conclusions on the Annual Standard Level

- If consideration is given to revising the primary $PM_{2.5}$ standards to increase public health protection, it would be appropriate to focus on lowering the level of the annual standard
- Support for particular levels depends on the weight placed on various aspects of the science and uncertainties
- For example, a level as low as 10.0 $\mu g/m^3$ could be considered if weight is placed on:
  - Setting a standard to maintain mean $PM_{2.5}$ concentrations below those in most key U.S. epidemiologic studies
  - Setting the standard level at or below the pseudo-design values corresponding to about the 50^{th} percentiles of study area health event/populations in key U.S. studies
  - Setting a standard estimated to reduce $PM_{2.5}$-associated health risks, such that a substantial portion of the risk reduction is estimated at annual average $PM_{2.5}$ concentrations ≥ ~8 $\mu g/m^3$
- A level below 10.0 $\mu g/m^3$, potentially as low as 8.0 $\mu g/m^3$, could be supported to the extent more weight is placed on $PM_{2.5}$ health effect associations and estimated risks at lower concentrations and less weight is placed on uncertainties at lower concentrations

Preliminary Conclusions on the 24-Hour Standard Level

- Purpose of the 24-hour standard is to provide protection against the short-term exposures to peak $PM_{2.5}$ concentrations, such as those that can occur in areas with strong contributions from local or seasonal sources even when overall mean concentrations remain relatively low
- In considering potential support for additional protection against short-term exposures to "peak" concentrations, we focus on the evidence from key epidemiologic studies and human clinical studies
  - Key epidemiologic studies do not indicate that $PM_{2.5}$ health effect associations are driven disproportionately by peak concentrations
  - Human clinical studies report effects following single short-term $PM_{2.5}$ exposures, but these studies generally examine exposures well above those measured in areas meeting the current standards
- Thus, the evidence provides little support for the need to provide additional protection against short-term peak concentrations in areas meeting the current 24-hour standard and the current, or revised (i.e., with a lower level), annual standard

Additional Information

Two-Hour $PM_{2.5}$ Concentrations

- In human clinical studies, statistically significant effects on one or more indicators of cardiovascular function are often, though not always, reported following 2-hour exposures to average $PM_{2.5}$ concentrations at and above about 120 µg/m^3
- There is less consistent evidence for effects following exposures to lower concentrations

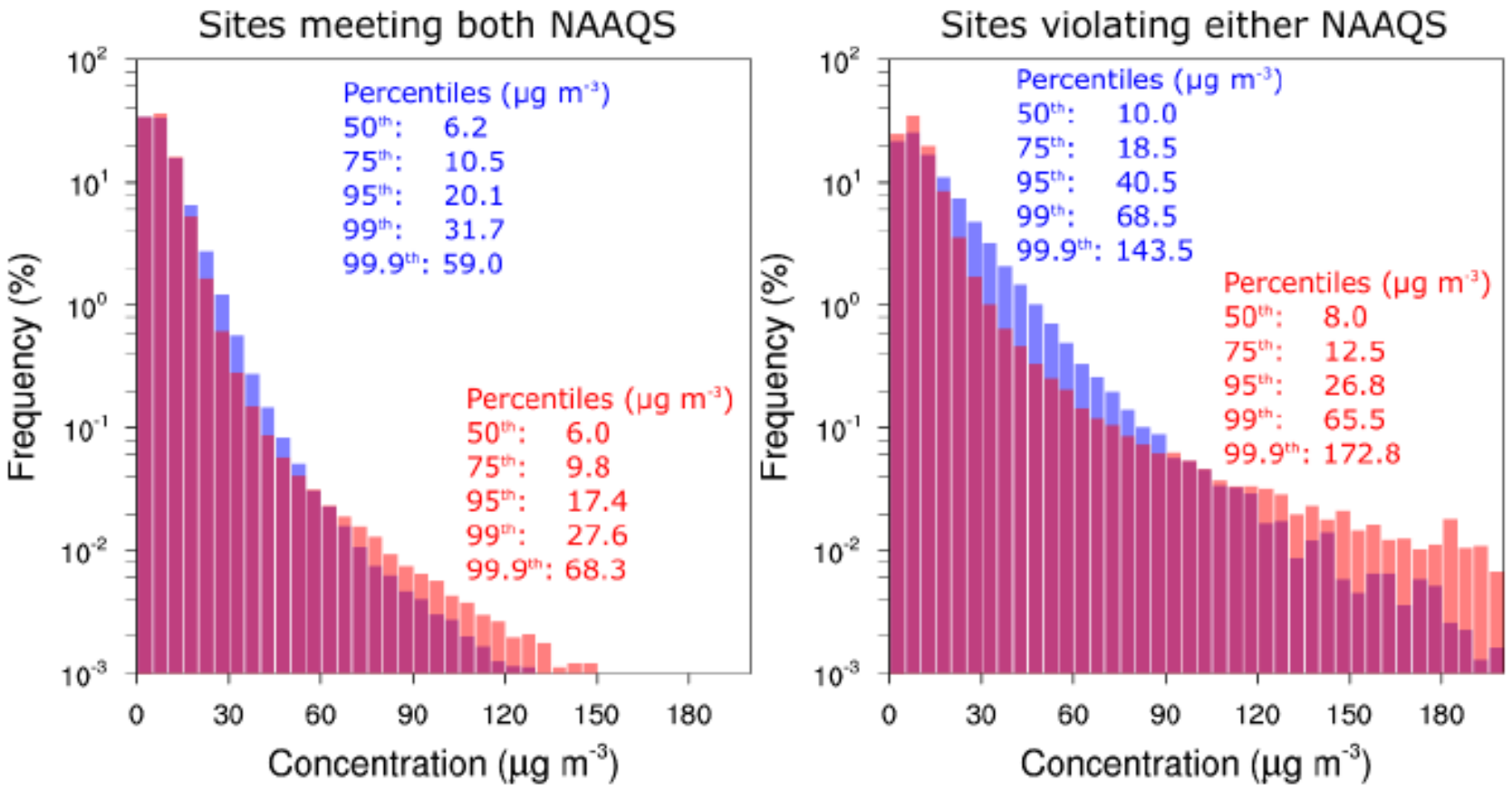
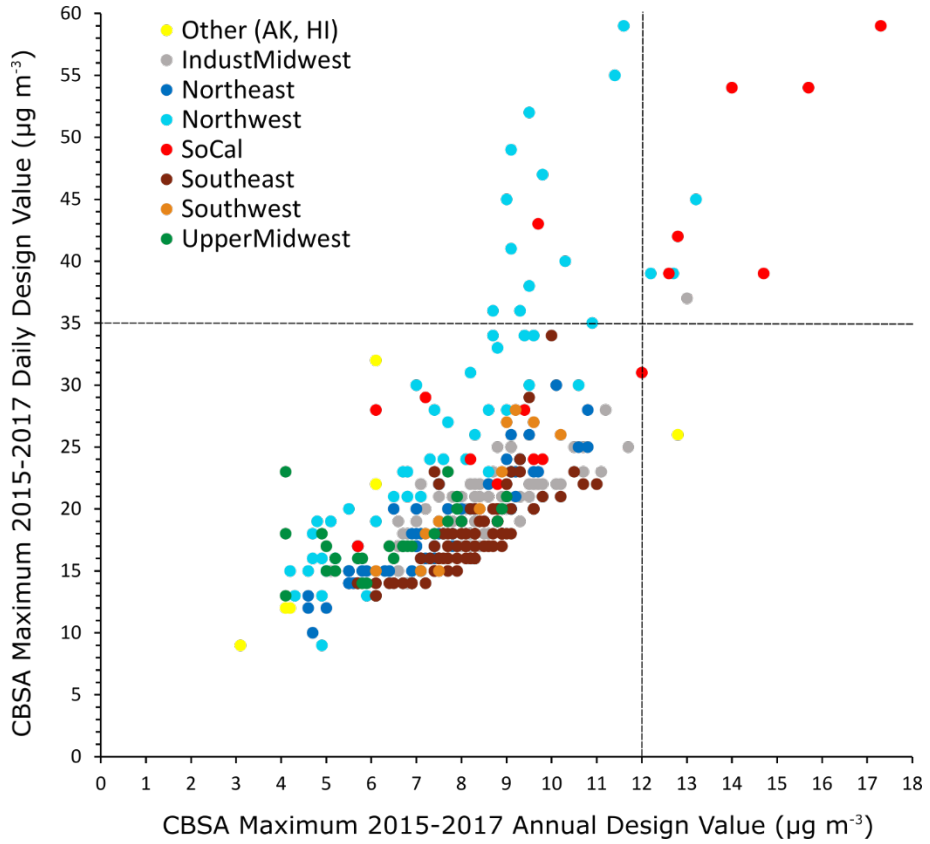


Figure 2-14.Frequency distribution of 2015-2017 2-hour averages for sites meeting or violating the annual $PM_{2.5}$ NAAQS for October to March (blue) and April to September (red).

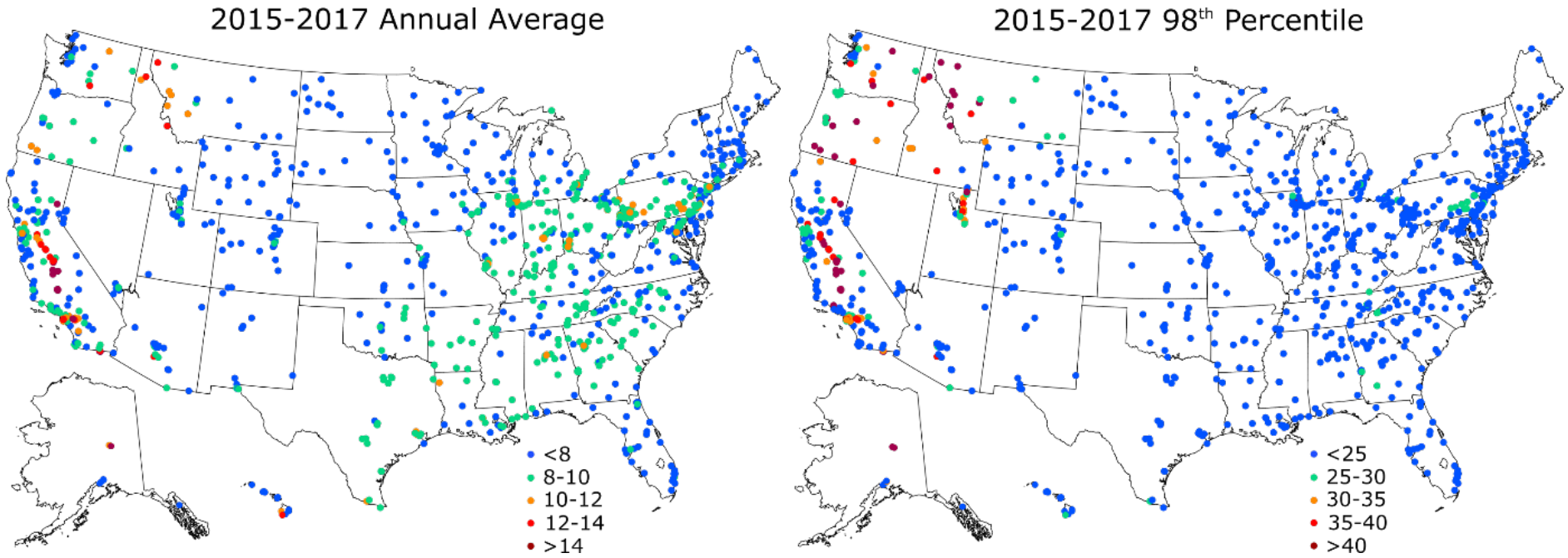
Annual and 24-Hour DVs



Draft PA Figure 2-11

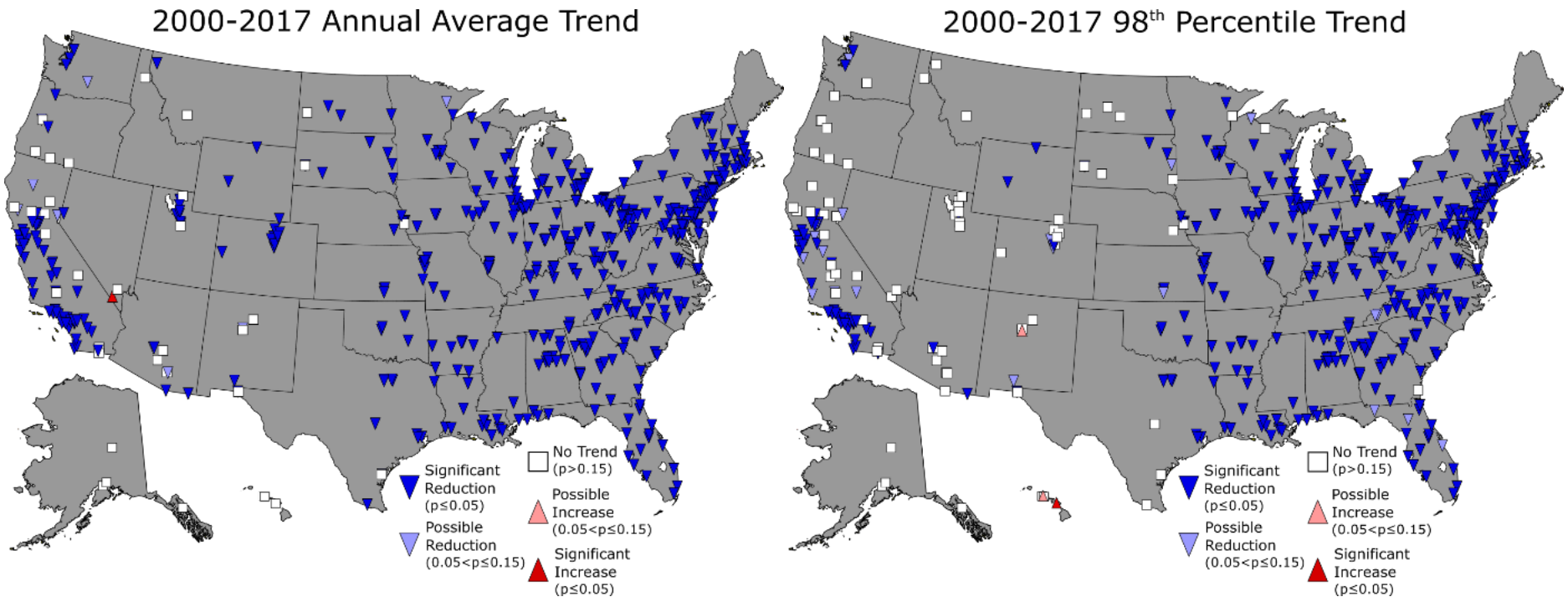
It is likely that some of the annual and daily design values above are impacted by potential exceptional events associated with wildfire smoke that have yet to be removed from the calculations.

$PM_{2.5}$: Recent Concentrations



- Highest annual average and 98th percentile $PM_{2.5}$ concentrations are in California
- Fires in the Northwest were frequent during the 2015-2017 period
- Most Eastern sites had annual average and 98th percentile values below 10 and 25 µg m^{-3}, respectively

$PM_{2.5}$ Trends



- The annual average and 98th percentile values have decreased over much of the Eastern US since 2000
- In the Western US, many sites have had no trend in the 98th percentile values in part because of the impact of meteorology and wildfires

Key $PM_{2.5}$-Related Health Outcomes Considered in the Draft PA

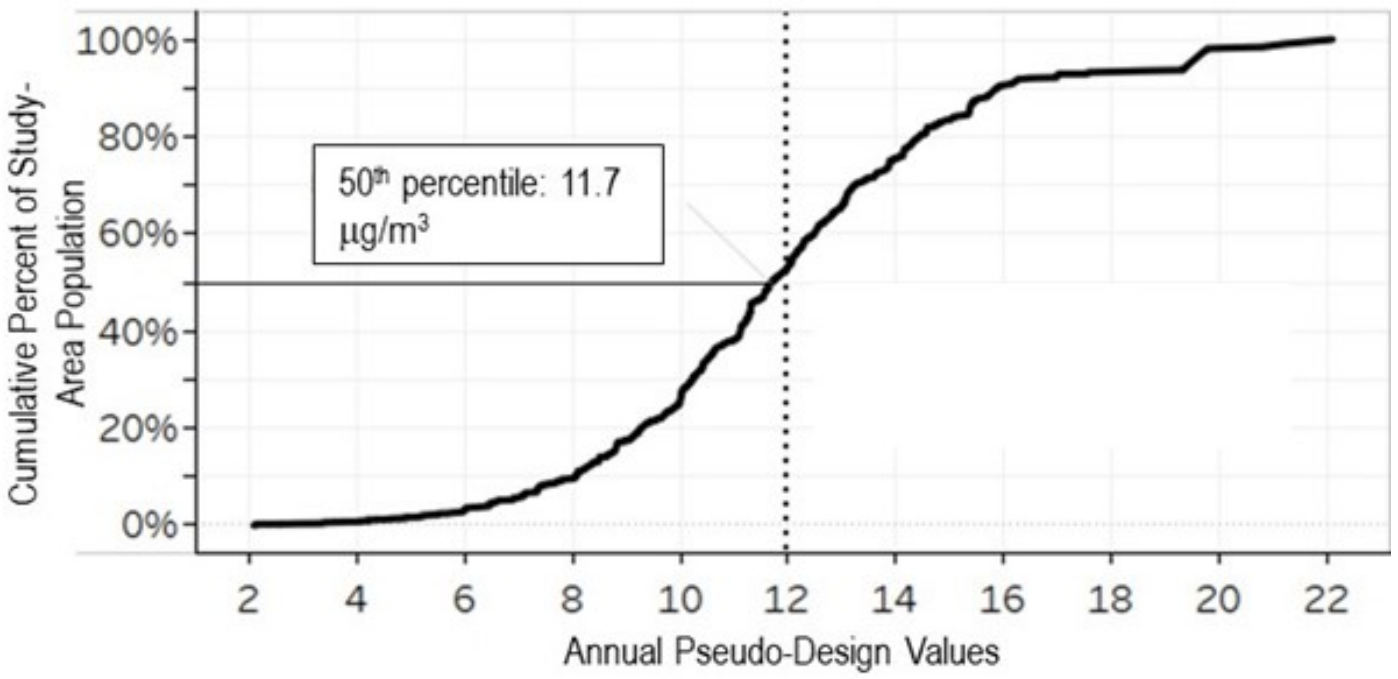
Exposure Duration	Outcome	2009 ISA Conclusion	2018 Draft ISA Conclusion
Long-Term	Mortality	Causal	Causal
	Cardiovascular	Causal	Causal
	Respiratory	Likely to be causal	Likely to be causal
	Cancer	Suggestive	Likely to be causal
	Nervous System	None	Likely to be causal
Short-Term	Mortality	Causal	Causal
	Cardiovascular	Causal	Causal
	Respiratory	Likely to be causal	Likely to be causal

Calculation of $PM_{2.5}$ Pseudo-Design Values

Approach

- Identify study areas (counties/cities) with sufficient monitoring data to calculate pseudo-design values
- For each monitored area and each 3-yr period of the study, identify the highest monitored $PM_{2.5}$ value
- For each monitored area, calculate the study-period average of these highest values
- Link study locations to study populations or health events
- Arrange study locations by ascending pseudo-design values
- Identify the cumulative percent of population or health events in study locations with various pseudo-design values

Example for Di et al. (2017)



Particulate Matter:
Spotlight on Health Protection

Bay Area Air Quality Management District

Phil Martien, Ph.D.

- Director of the Assessment, Inventory, & Modeling Division at the Bay Area Air Quality Management District
- Leading role in the Technical Assessment of AB617's West Oakland Community Action Plan
- Leading role in the Technical Assessment of the Air District's 2017 Clean Air Plan: Spare the Air, Cool the Climate
- Leading role in the Air District's Community Air Risk Evaluation Program
- Ph.D. from UC Berkeley

Targeting Particulate Matter: West Oakland Community Emissions Reduction Program

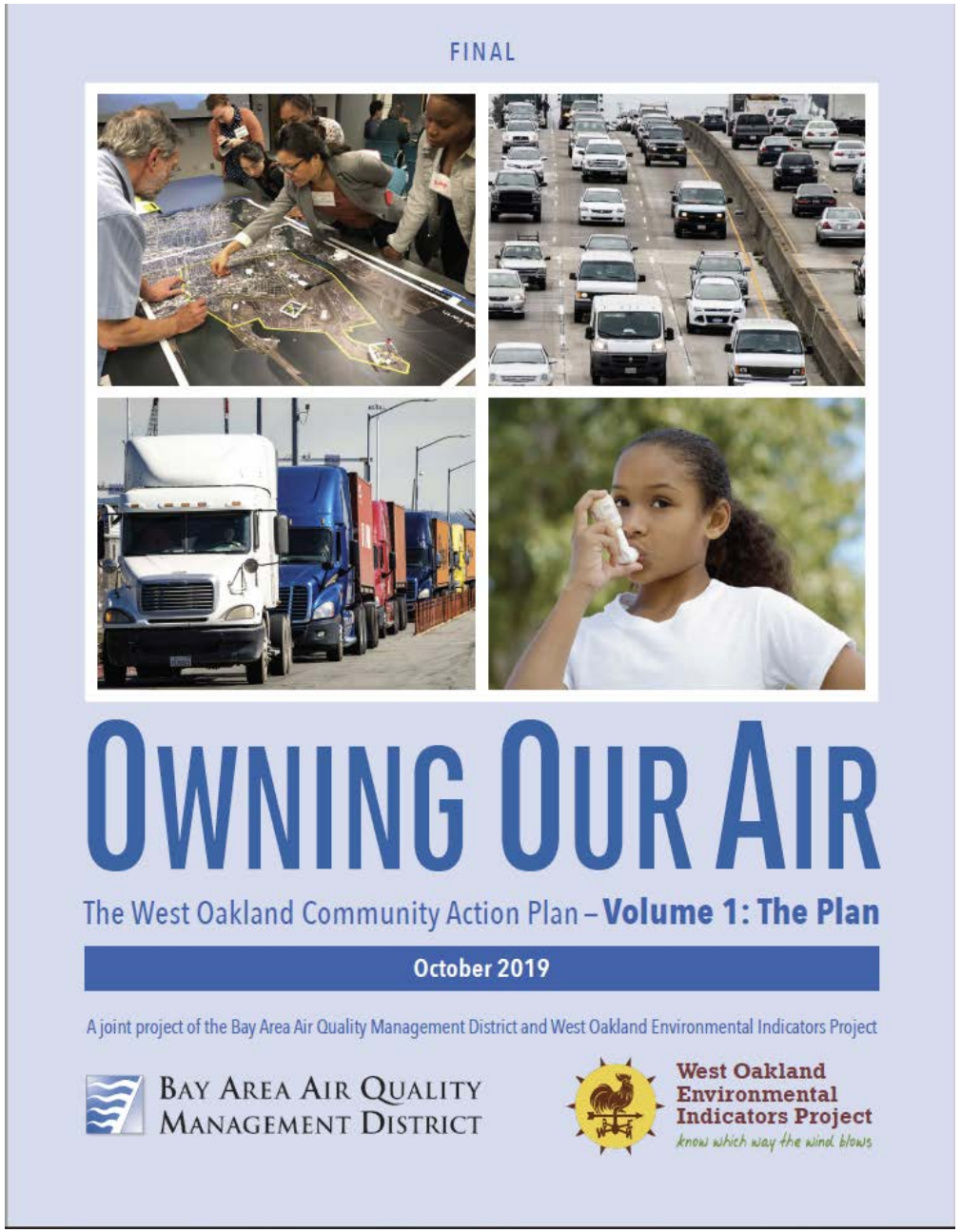
Phil Martien, PhD
Bay Area Air Quality Management District
Particulate Matter: Spotlight on Health Protection
October 28, 2019

Bay Area Air Quality Management District

West Oakland Environmental Indicators Project
know which way the wind blows

Acknowledgements

- Bay Area Air Quality Management District
- West Oakland Environmental Indicators Project
- West Oakland Steering Committee
- California Air Resources Board



Assessment of Particulate Matter (PM) in West Oakland

- **Motivation**
  - Implementing Assembly Bill (AB) 617: West Oakland Community Emissions Reduction Program
- **Modeling-based assessment approach**
- **Findings**
  - Source contributions to impacts
  - Equity-based targets
  - Effective emission reduction measures

West Oakland

Motivation

Implementing AB 617

- Address environmental justice concerns: higher air pollution in some communities
- Key mandates:
  - Local air districts to partner with community groups
  - Identify top sources of community impacts
  - Develop and implement plans to reduce emissions

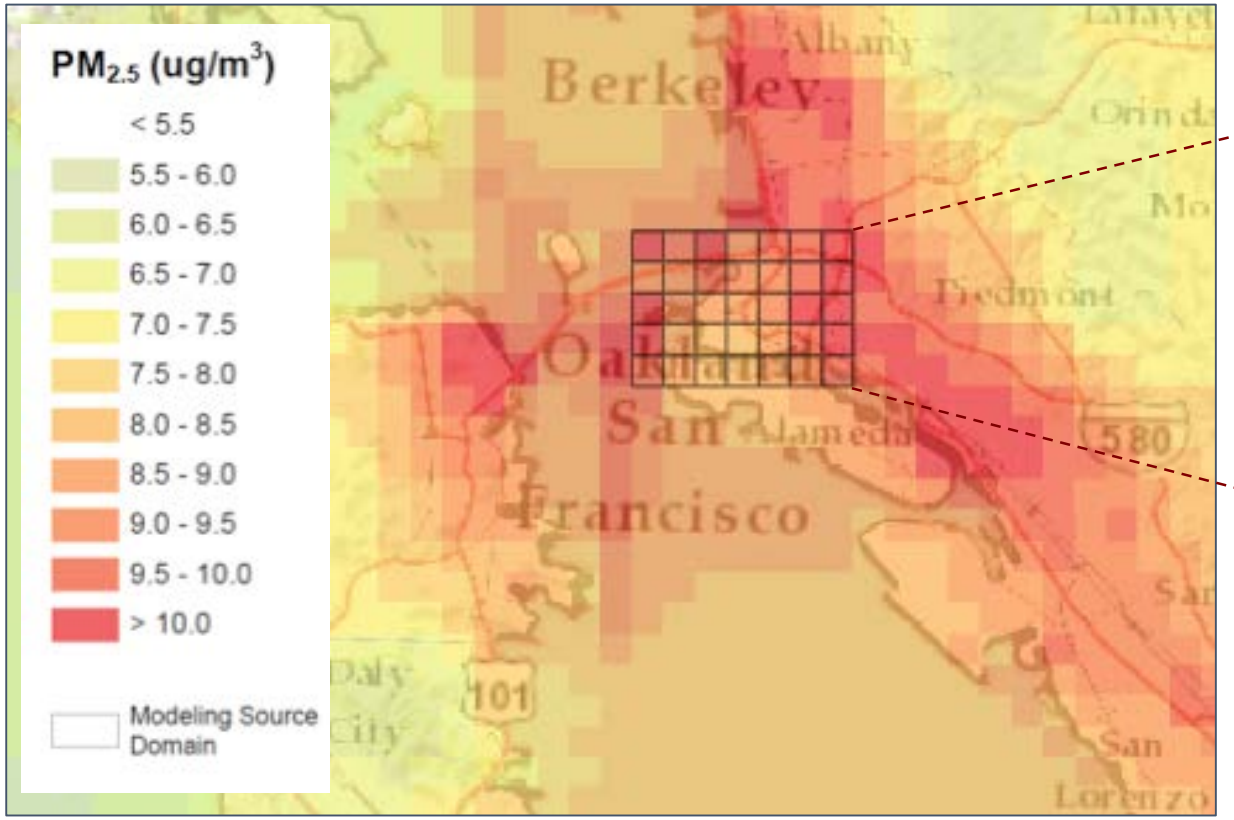
West Oakland: Year 1 Community Emissions Reduction Plan

West Oakland Environmental Indicators Project
know which way the wind blows

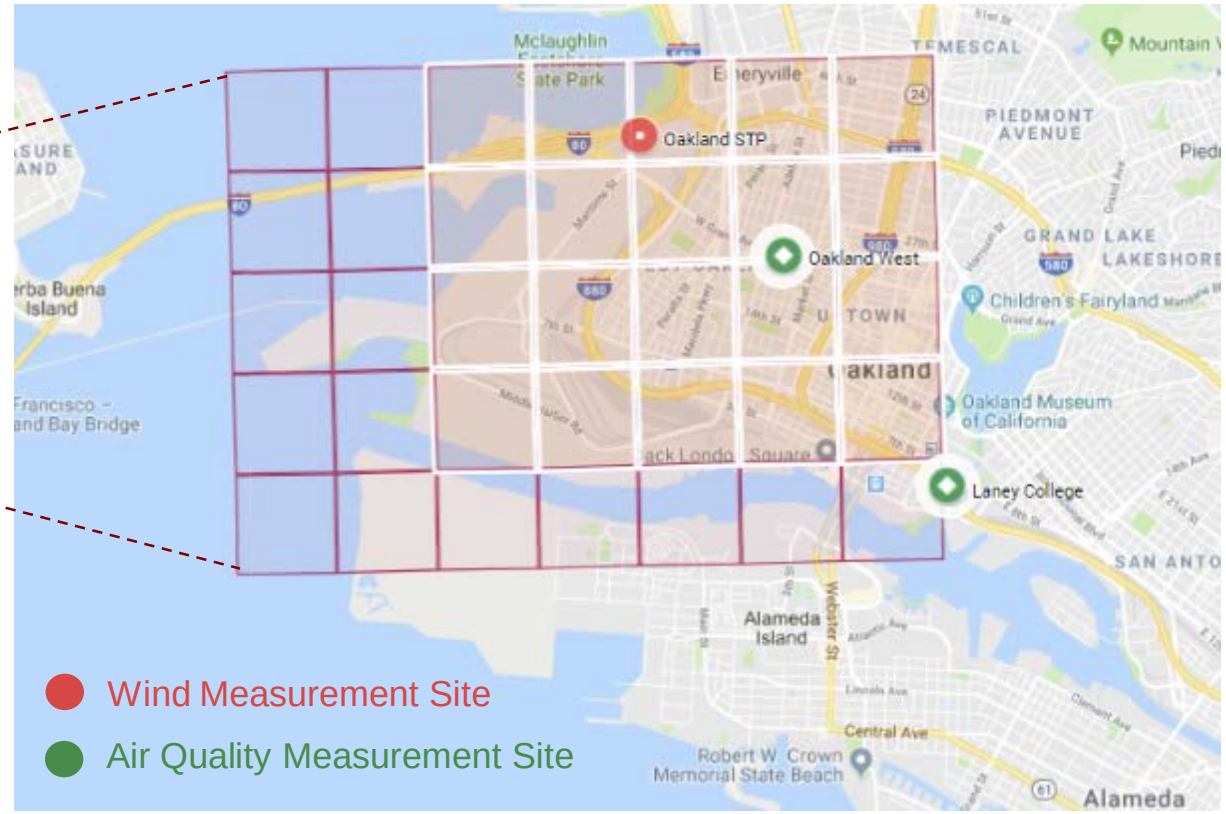
- Established partner: WOEIP has decades of experience
- High mobile-source emissions
  - Adjacent to the Port of Oakland
  - Surrounded by the I-880, I-80, I-580, and I-980 freeways
  - Industrial sources
- High health burdens and socio-economic vulnerabilities

Assessment Approach

Regional-Scale and Community-Scale Modeling (2017)



Regional-scale modeling: covers the Bay Area



Local-scale modeling: covers West Oakland, including impacts in receptor area (white) from sources in source area (red)

Community-Scale Modeling

Pollutants

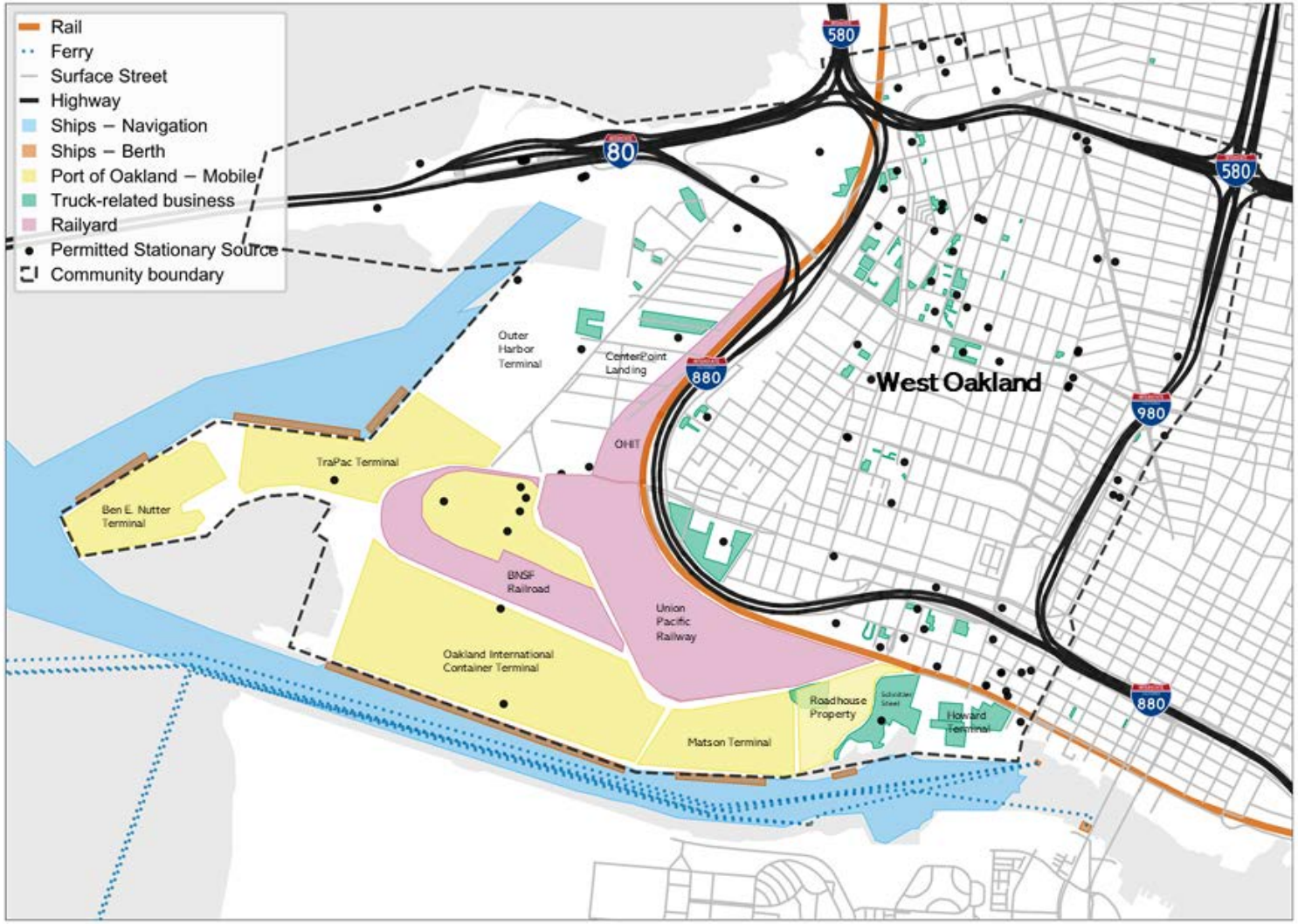
- $PM_{2.5}$
- Diesel PM
- Air toxics (cancer risk)

Sources modeled

- Port of Oakland and marine
- Railyards and trains
- Vehicles on freeways, streets
- Truck-related businesses
- Permitted stationary sources

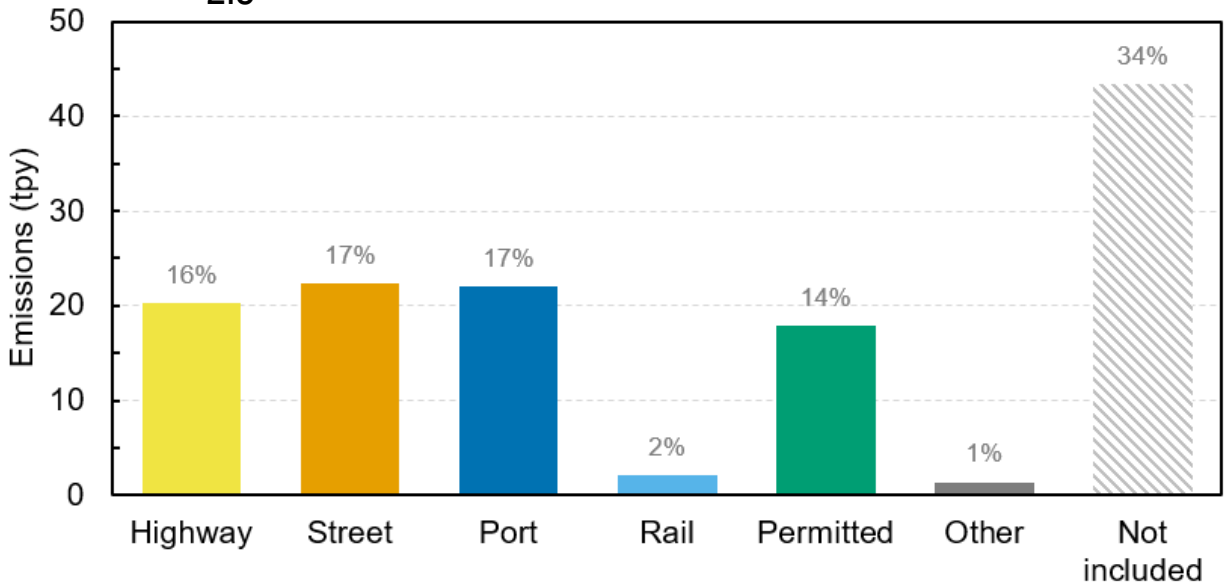
Not modeled

- Construction, residential woodburning, and restaurants

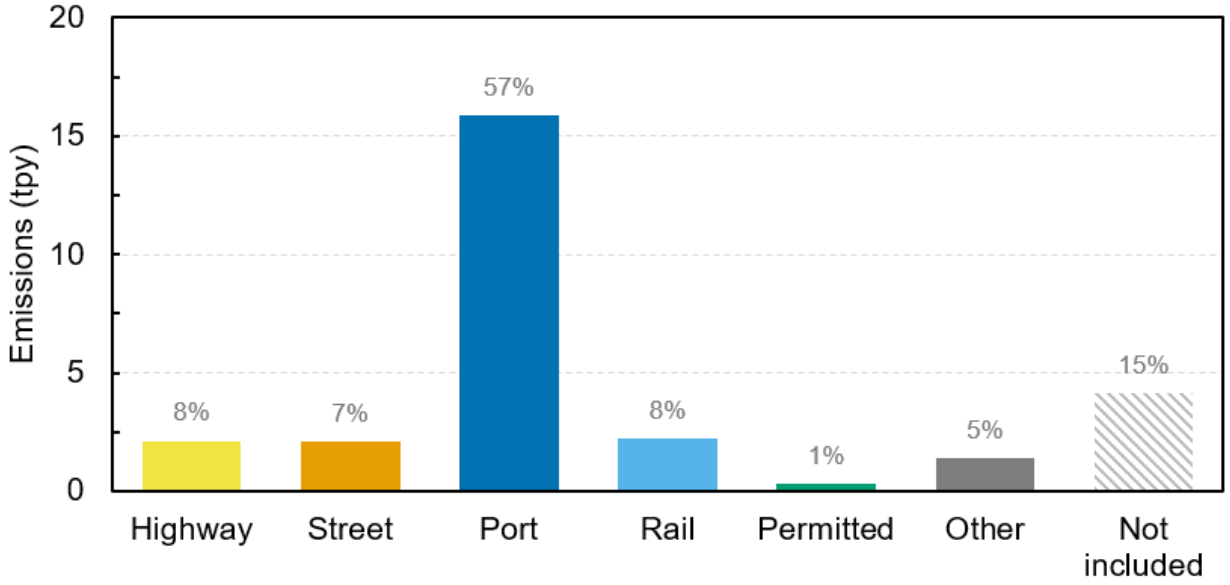


West Oakland Emissions by Source Category (2017)

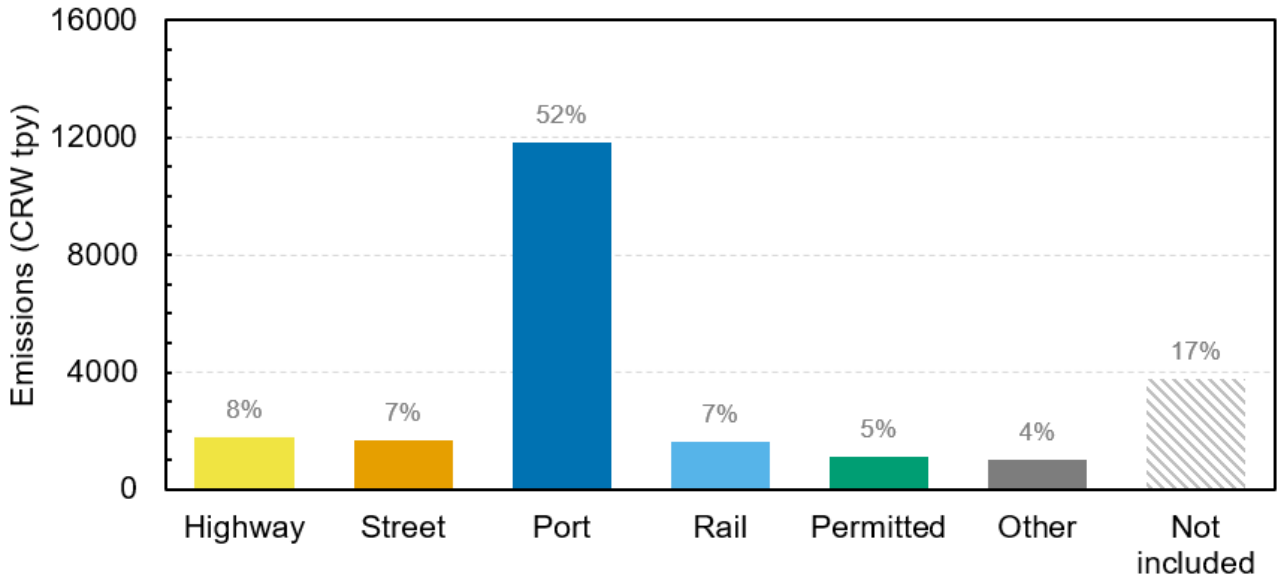
(a) $PM_{2.5}$



(b) Diesel PM



(c) Cancer risk-weighted toxics



Impact Varies by Location

Local Impact Zones

Local Impact Zones

1. Lower bottom/West Prescott
2. Third Street
3. Seventh Street
4. Acorn
5. Upper Adeline
6. Clawson
7. West Grand and San Pablo

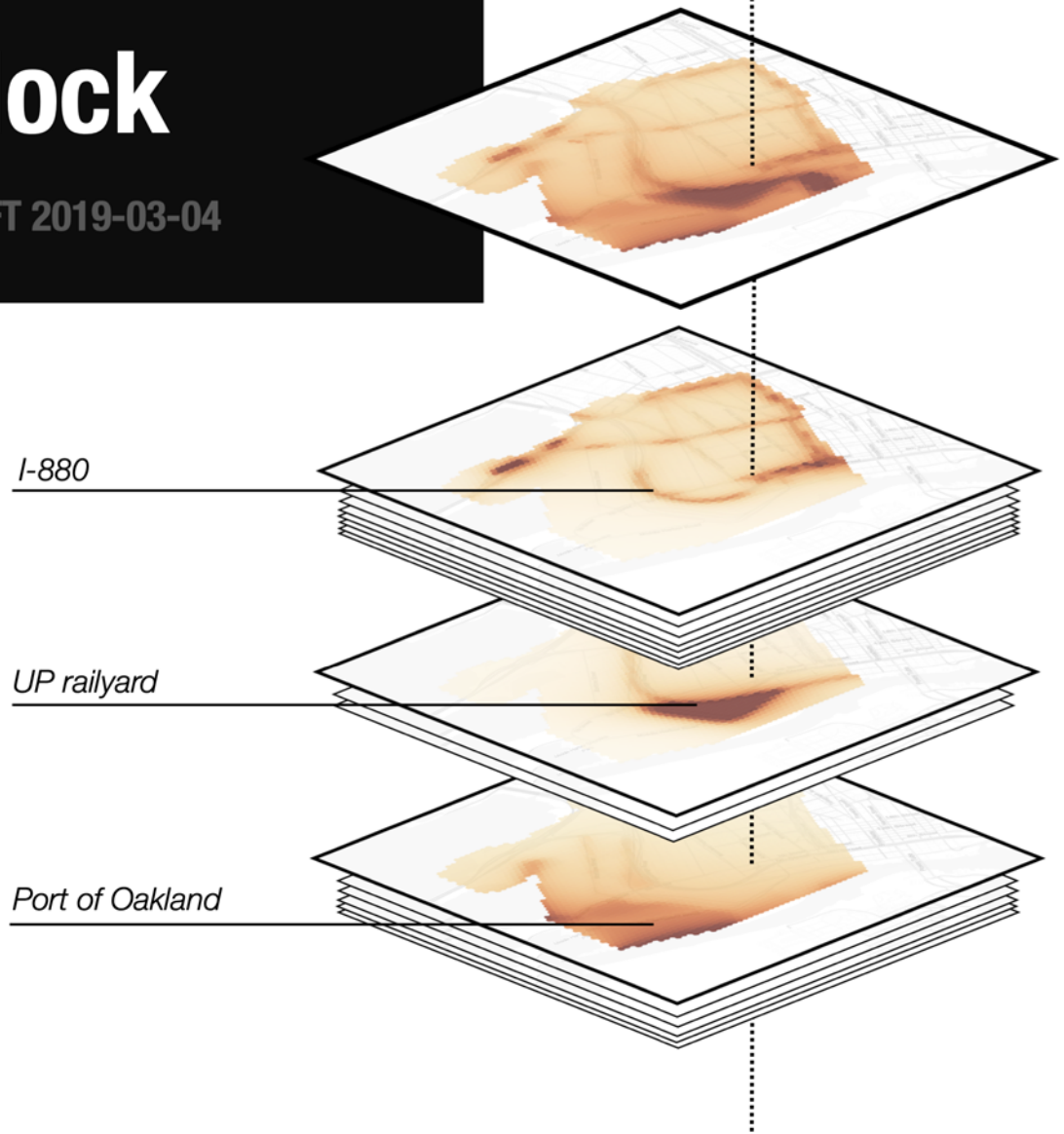
Black Carbon above Median (Env. Def. Fund, 2019-01-13)

Impact Zones
on Census Blocks

Source Apportionment

Block by Block

DRAFT 2019-03-04

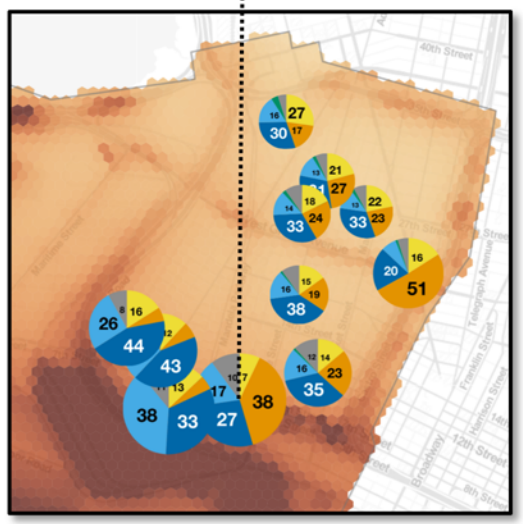


Grand total of modeled impacts from local sources

Sub-total from trucks, cars, and other vehicles on streets and highways

Sub-total from locomotive engines and railyards

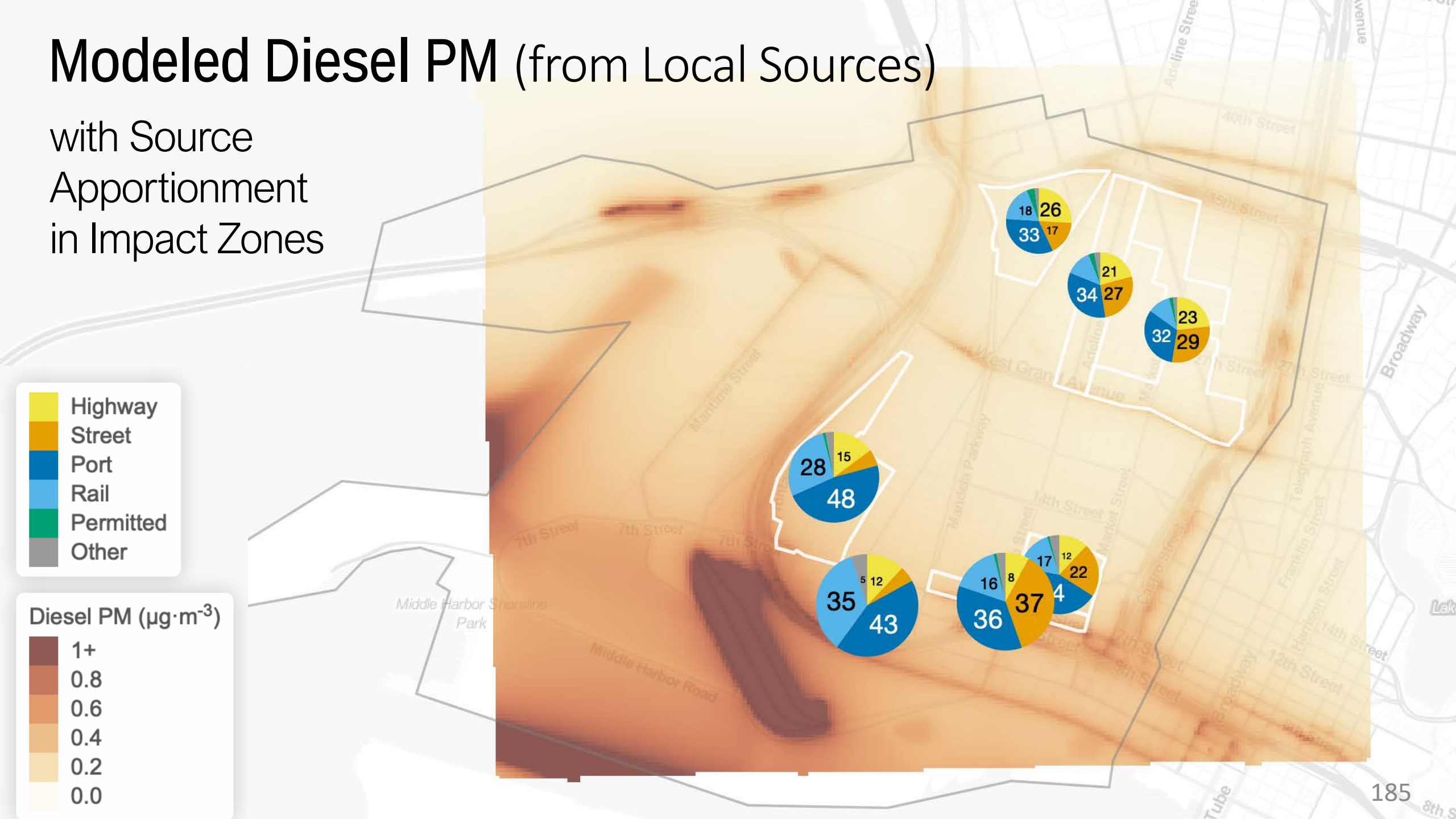
Sub-total from harbor craft, ocean-going vessels, drayage, cargo handling, etc.



For any location, we can use the sub-totals to draw piecharts showing the relative impacts of sources A, B, C, etc.

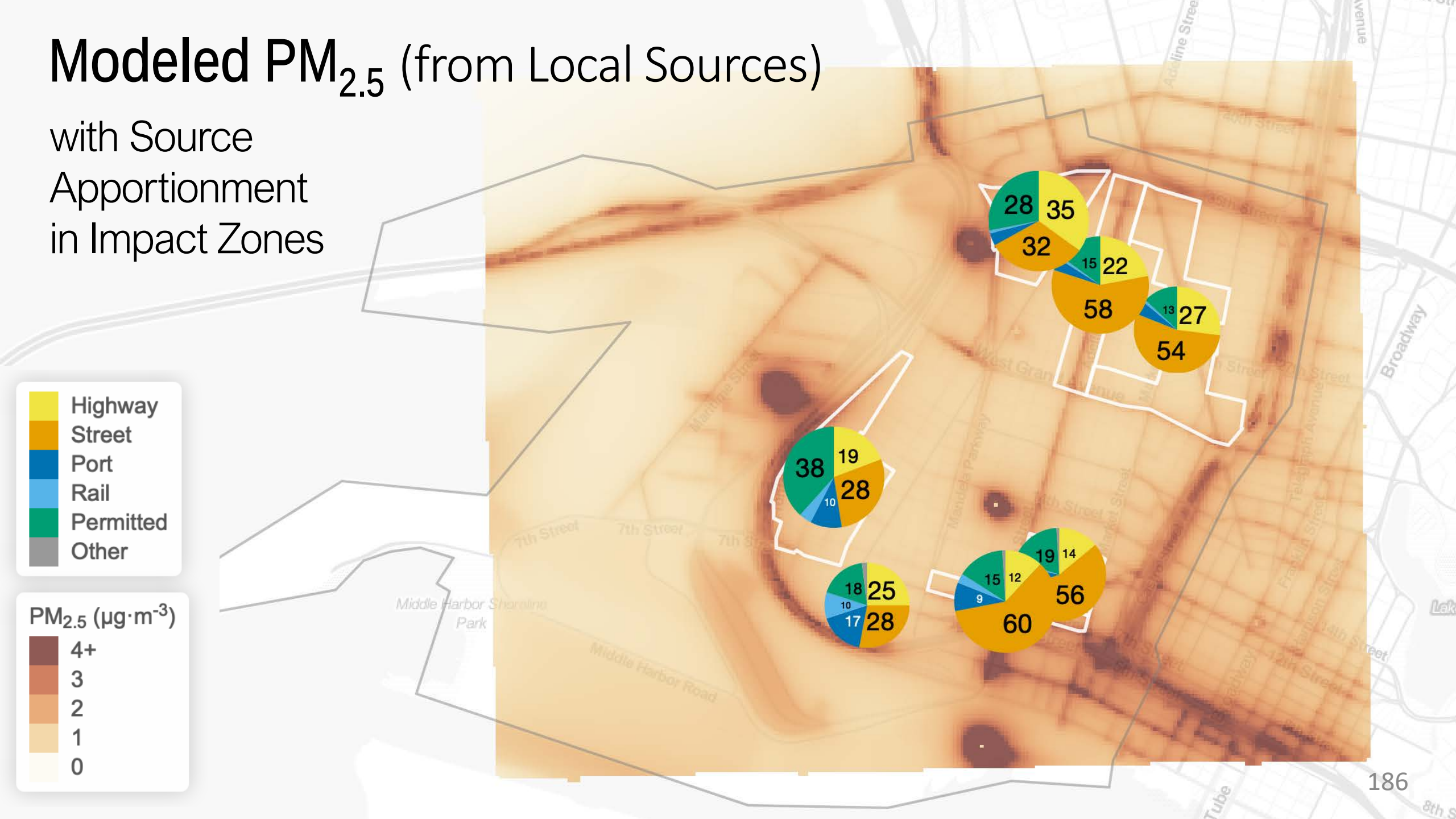
Modeled Diesel PM (from Local Sources)

with Source Apportionment in Impact Zones



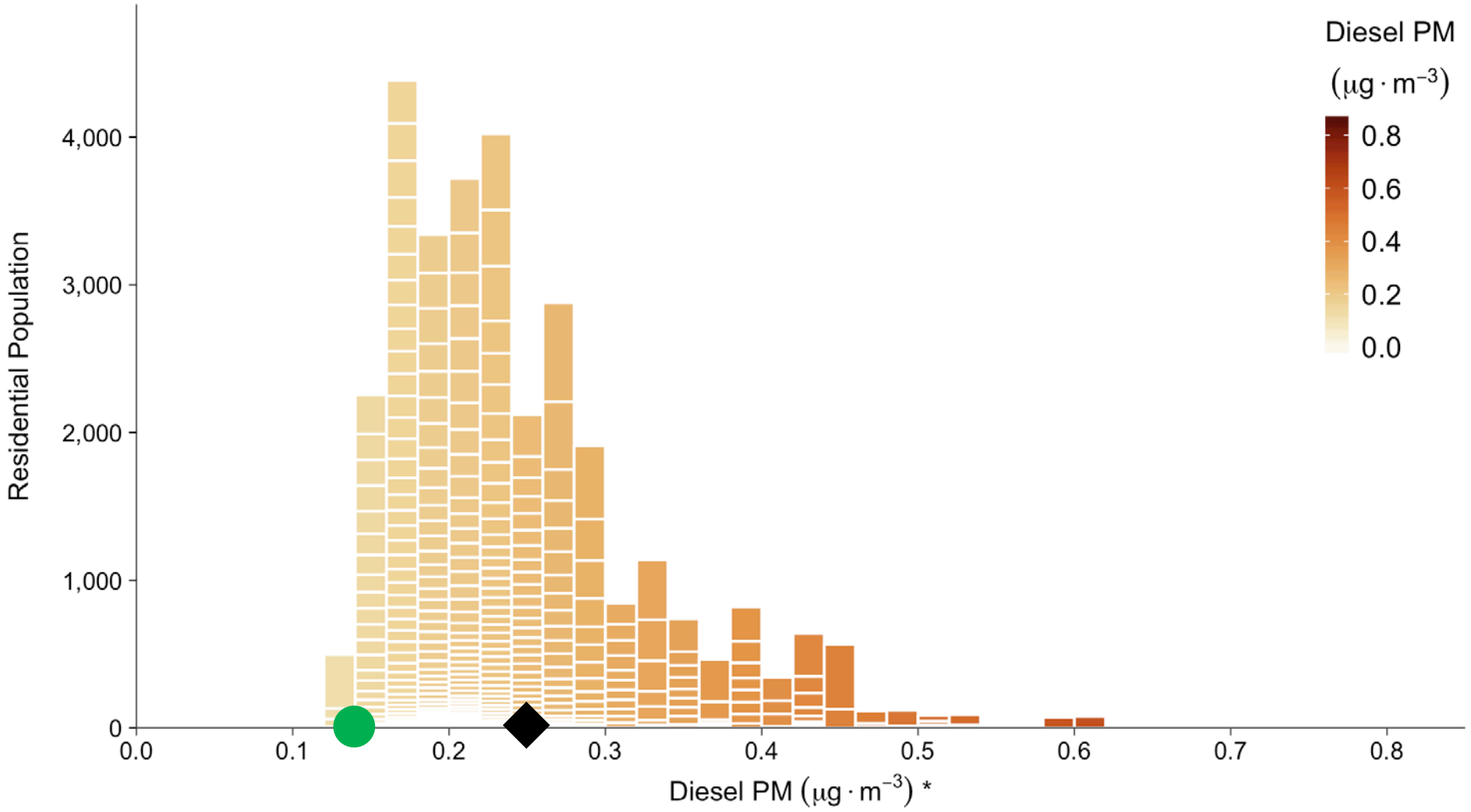
Modeled $PM_{2.5}$ (from Local Sources)

with Source Apportionment in Impact Zones



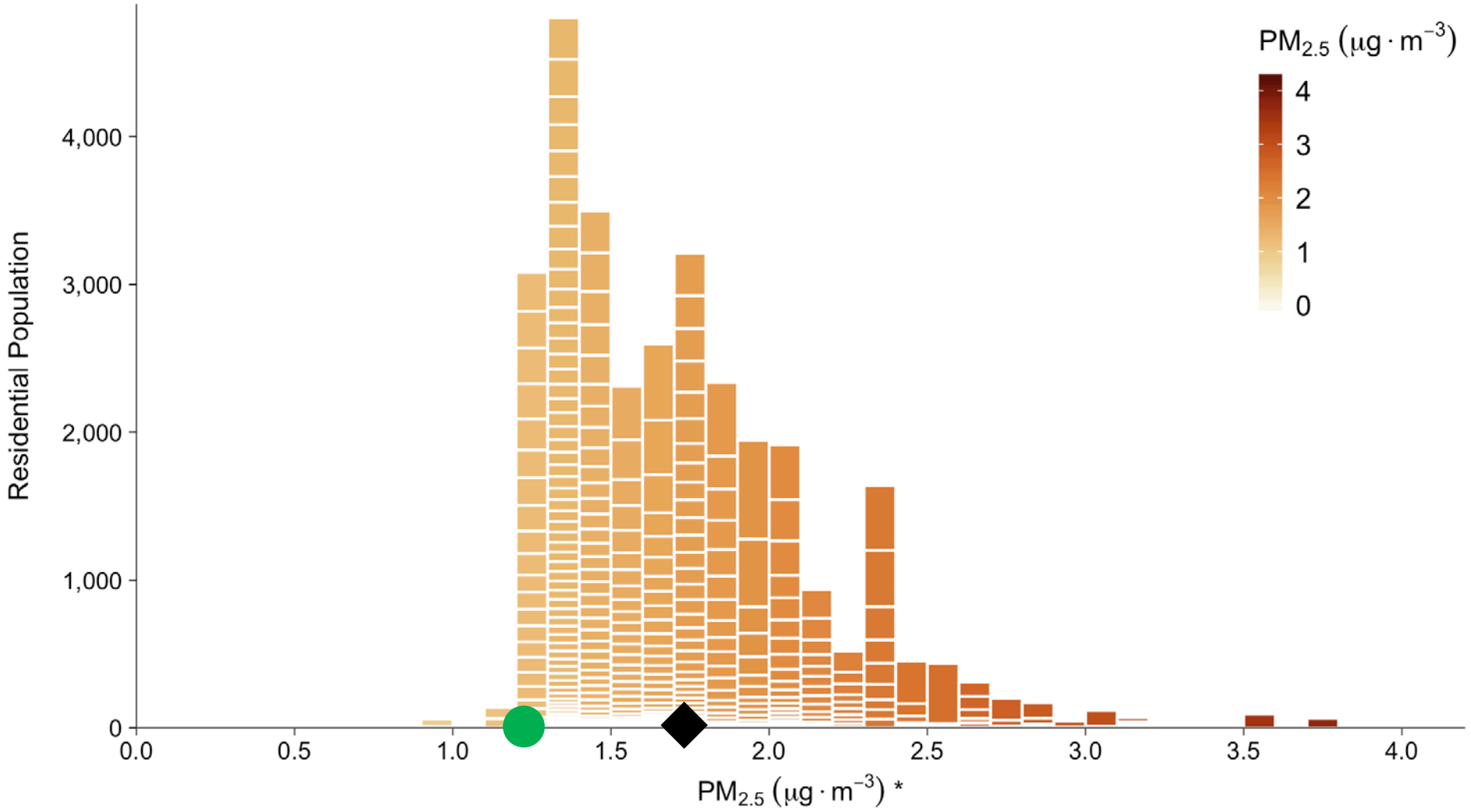
Equity-Based Targets

Unequal Impacts: Diesel PM Across West Oakland



* Contributed by modeled "present-day" emissions from existing local sources. Impacts from sources outside West Oakland not included.

Unequal Impacts: $PM_{2.5}$ Across West Oakland



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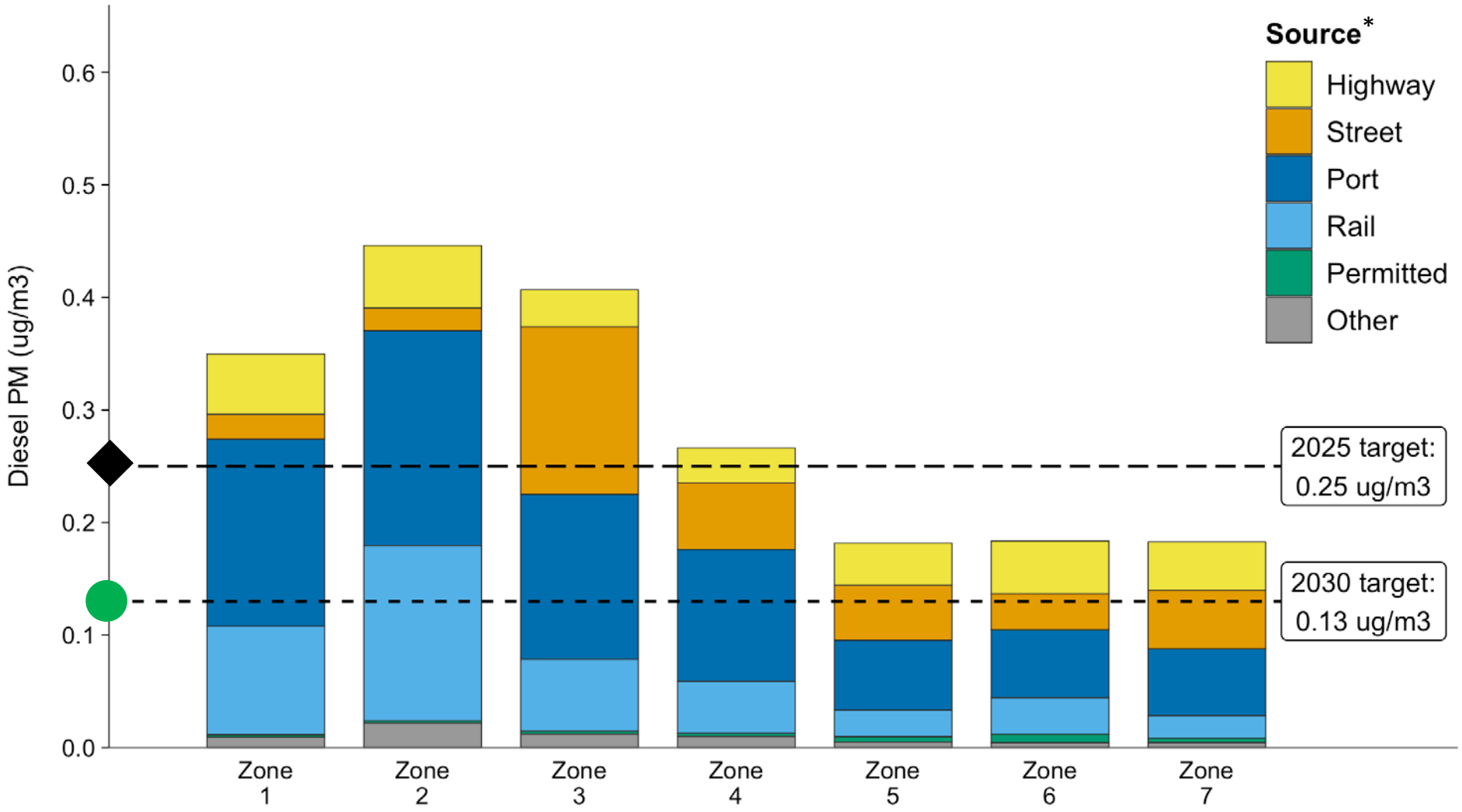
Targets and Source Contributions for Diesel PM

Targets:

2025 – Today's *average* residential neighborhood

2030 – Today's *cleanest* residential neighborhood



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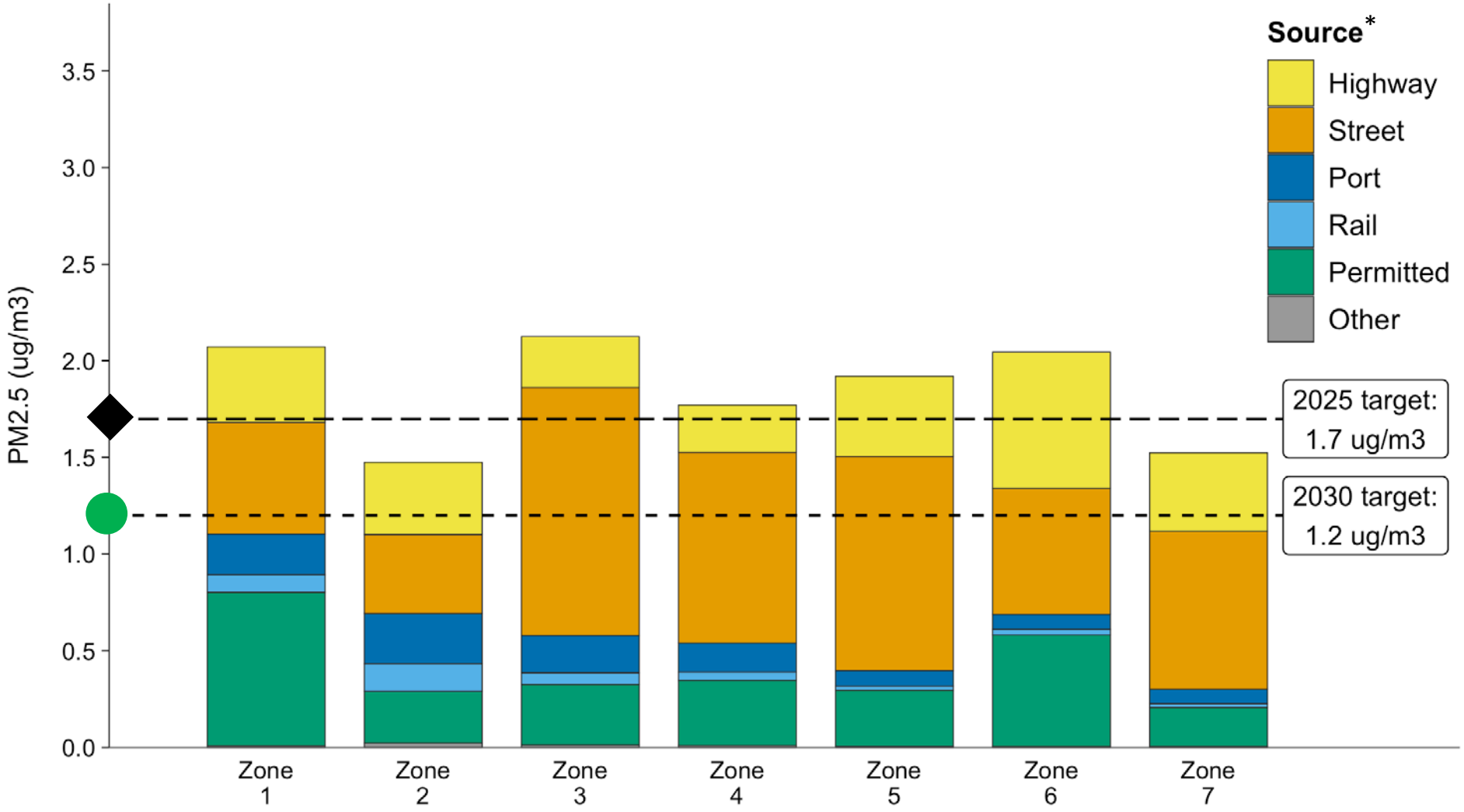


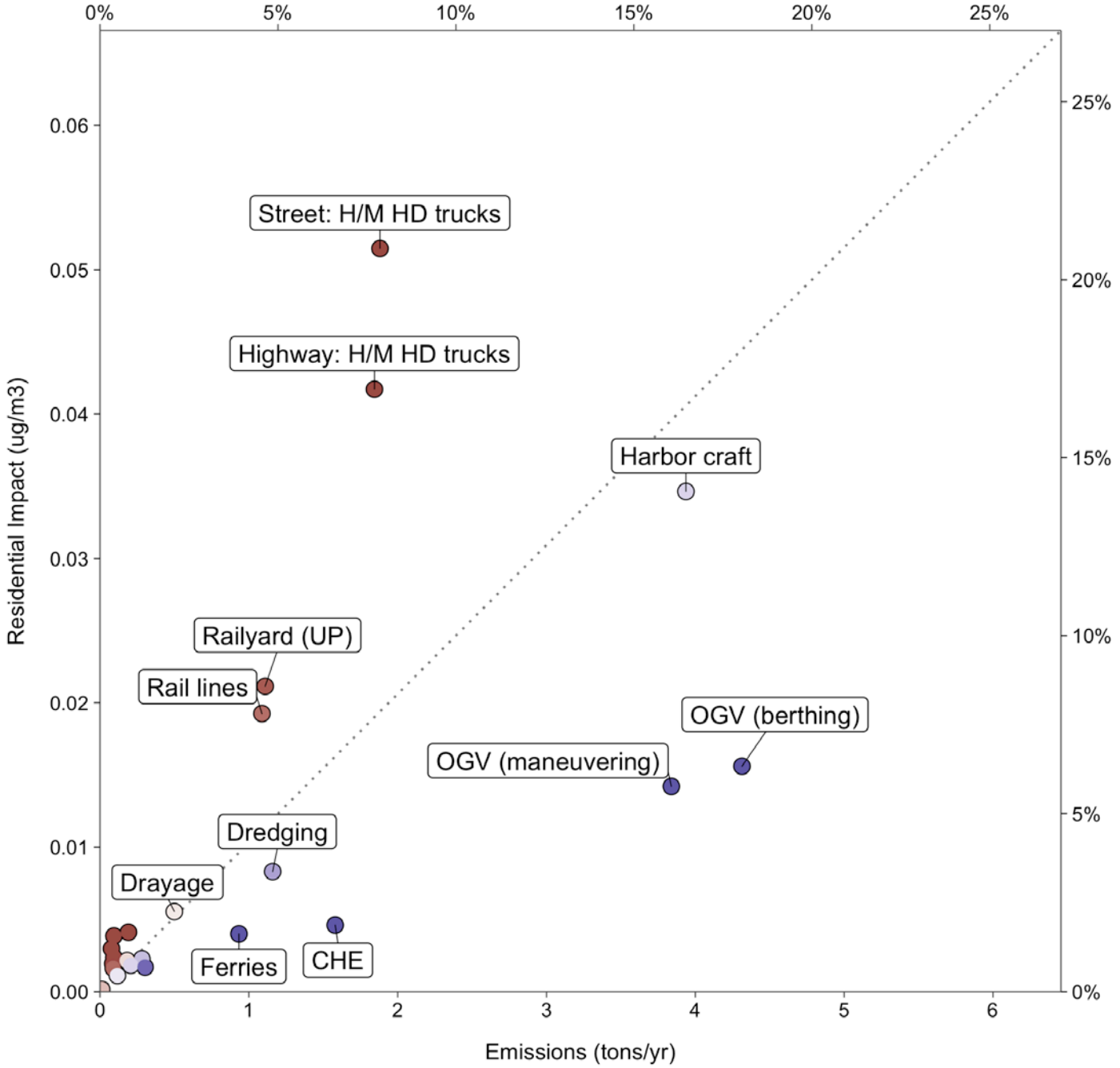
* Contributed by emissions from modeled local sources. Impacts from sources outside West Oakland not included.

Impact Per Ton Varies by Source

What Moves the Needle?

Impact Per Ton: Diesel PM in West Oakland

Circles are modeled local sources.
Red is more impact.
Blue is less impact.
Percentages are shares of modeled impact.



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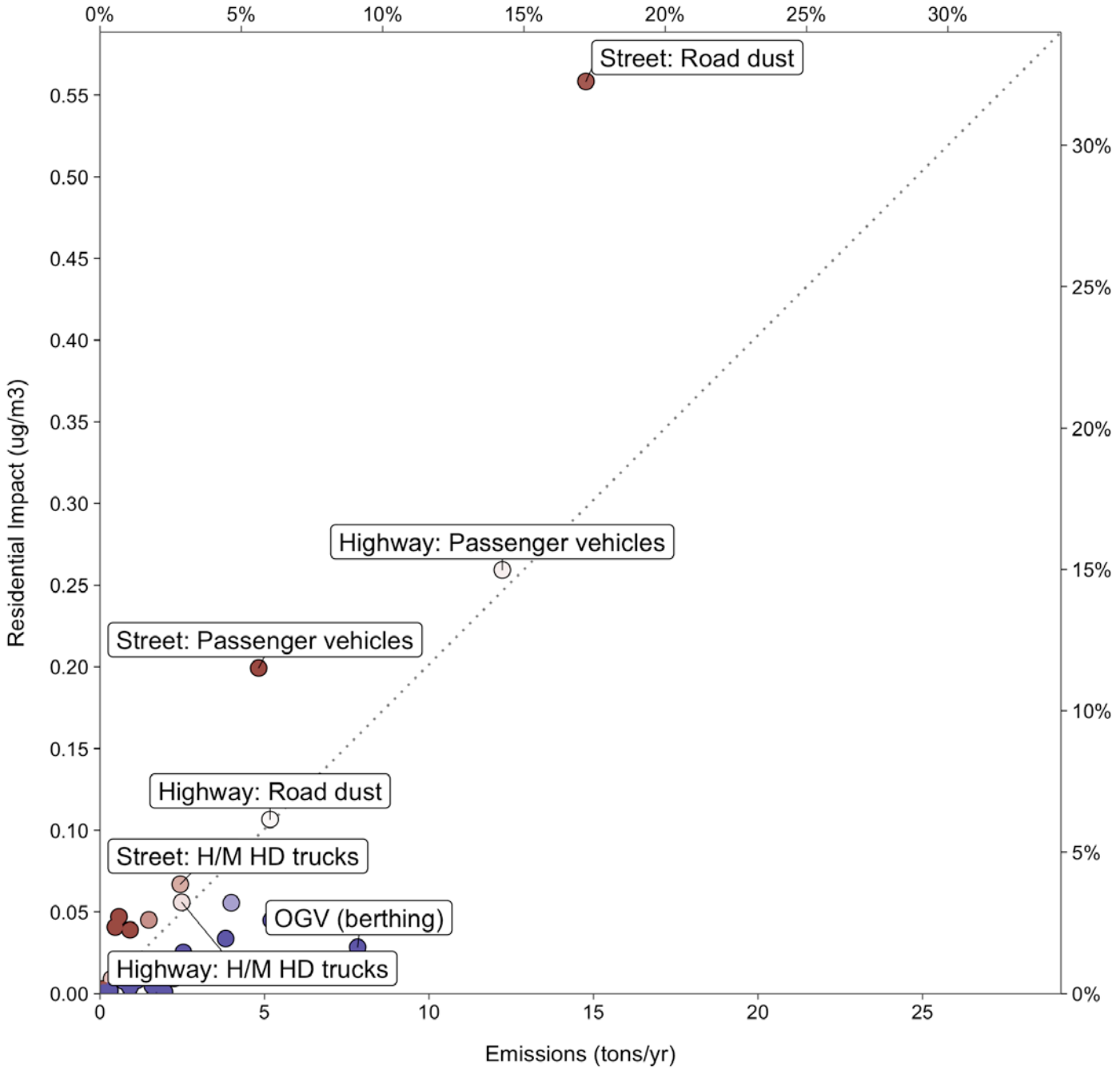
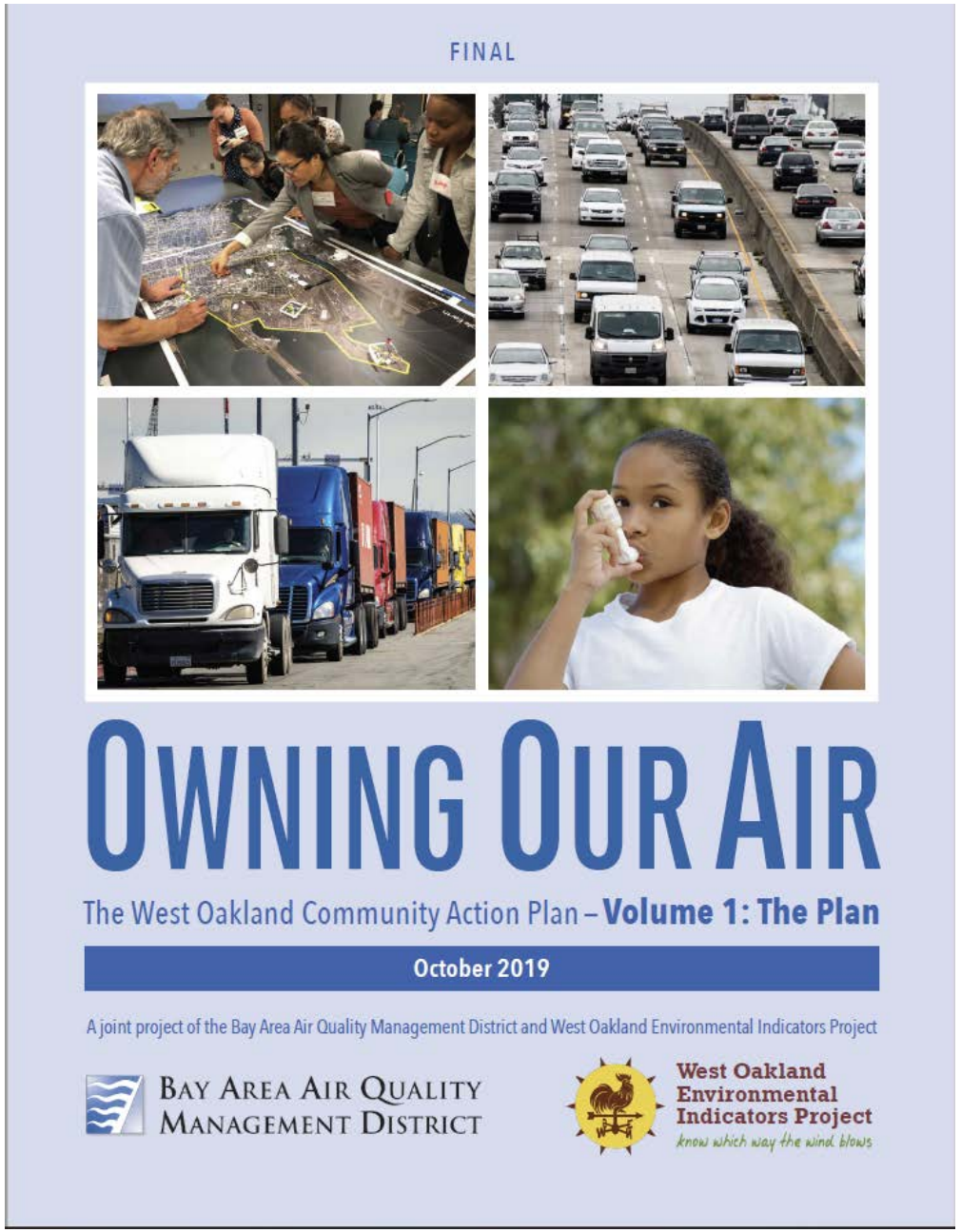


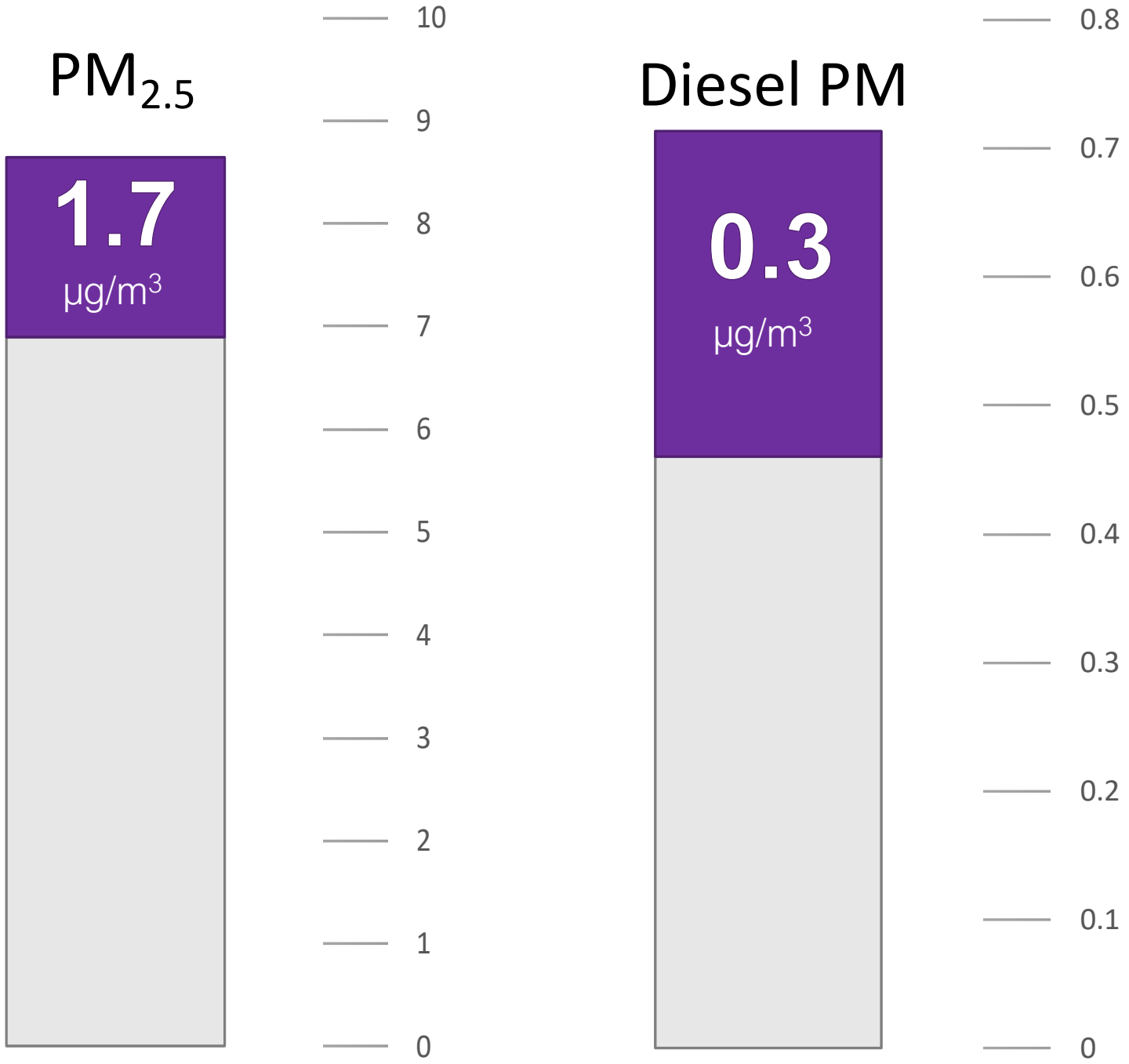
More Information

- baaqmd.gov/communityhealth/community-health-protection-program/
- woeip.org/
- arb.ca.gov/ourwork/programs/community-air-protection-program
- pmartien@baaqmd.gov



Extra Slides

How Much is Local?



Local vs. Regional

- Local model – mapped impacts*
- Regional model (minus West Oakland)

*Construction, residential woodburning, and restaurants not modeled

Thank you

Break

Particulate Matter:
Spotlight on Health Protection




Advisory Council Discussion with Exposure and Risk Panel

Discussion Questions

What are major sources of PM in the Bay Area?

What PM levels exist in Bay Area? What health risks do they pose?

How much additional health benefit can be achieved?

How should we account for spatial scale of effects (i.e., regional versus local-scale impacts, including proximity to major sources)?

How should we determine which measures would most move public health needle?

Advisory Council Deliberation

Particulate Matter:
Spotlight on Health Protection

Adjournment

Particulate Matter:
Spotlight on Health Protection

Particulate Matter:
Spotlight on Health Protection

Bay Area Air Quality Management District