Particulate Matter: Spotlight on Health Protection
Call to Order
Pledge of Allegiance
Public Comment
Approval of Minutes

Stan Hayes
Welcome Remarks

Jack Broadbent
Introduction

Jeff McKay
Jason Sacks, M.P.H.

- Senior Epidemiologist in the Center for Public Health & Environmental Assessment within U.S. EPA’s Office of Research and Development
- 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
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

### Planning
- Call for Information and Public Workshop: Feb. 2015

### Assessment
- Integrated Science Assessment (ISA):
  - Final ISA: Dec. 2019
- Policy Assessment (PA): Sep. 2019

### 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](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
# Draft PM ISA Health Effects: Causality Determinations

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

<table>
<thead>
<tr>
<th>Health Outcome</th>
<th>ISA</th>
<th>Current PM Draft ISA</th>
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<tbody>
<tr>
<td>Indicator</td>
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<td>PM&lt;sub&gt;2.5&lt;/sub&gt;</td>
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<td><strong>Respiratory</strong></td>
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<tr>
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<td>Long-term exposure</td>
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<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Long-term exposure</td>
<td></td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
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<td>*</td>
</tr>
<tr>
<td></td>
<td>Long-term exposure</td>
<td>*</td>
</tr>
<tr>
<td><strong>Nervous System</strong></td>
<td>Short-term exposure</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Long-term exposure</td>
<td>*</td>
</tr>
<tr>
<td><strong>Reproductive</strong></td>
<td>Male/Female Reproduction and Fertility</td>
<td>Long-term exposure</td>
</tr>
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<td><strong>Pregnancy and Birth Outcomes</strong></td>
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<td><strong>Cancer</strong></td>
<td>Long-term exposure</td>
<td>*</td>
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<tr>
<td><strong>Mortality</strong></td>
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<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Long-term exposure</td>
<td></td>
</tr>
</tbody>
</table>

* = 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

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**Figure 5-2.** Summary of associations between short-term PM$_{2.5}$ exposures and asthma hospital admissions for a 10 μg/m$^3$ increase in 24-hour average PM$_{2.5}$ concentrations.

Red = recent studies;  
Black = U.S. study evaluated in the 2009 PM ISA
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)
  o Epidemiologic evidence:
    ▪ Consistent evidence for cognitive decline/impairment and decreased brain volume
    ▪ Limited evidence for neurodegeneration (e.g., Alzheimer’s disease and dementia)
  o 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
  o 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)**
  o Epidemiologic evidence:
    ▪ Limited evidence for effects on cognitive development in children
  o 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
  o 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.

Working Draft: Do Not Cite or Quote
• 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**: 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
PM ISA Team

NCEA Team
Jason Sacks (Assessment Lead)
Barbara Buckley (Deputy Lead)
Michelle Angrish
Renee Beardslee**†
Adam Benson†
James Brown
Evan Coffman
Elizabeth Chan**
Allen Davis
Steve Dutton
Brooke Hemming
Erin Hines
Ellen Kirrane
Dennis Kotchmar
Meredith Lassiter
Vijay Limaye##†
Tom Long
Tom Luben
April Maxwell†
Joseph McDonald***

Steve McDow
Ihab Mikati†
Jennifer Nichols
Molini Patel†
Rob Pinder†
Joseph Pinto++
Kristen Rappazzo
Jennifer RichmondBryant
Lindsay Stanek#
Michael Stewart
Chris Weaver

Health & Environmental Effects Assessment Division
John Vandenberg, Director
Steve Dutton, Associate Director
Jane Ellen Simmons, Branch Chief

NCEA Management (Retired/Previously Acting)
Debra Walsh, Deputy Director (Retired)
Reeder Sams, Deputy Directory (Acting)
Andrew Hotchkiss, Branch Chief (Acting)
Alan Vette, Branch Chief (Acting)
Jennifer Richmond-Bryant, Branch Chief (Acting)
Tara Greaver, Branch Chief (Acting)
Jennifer Nichols, Branch Chief (Acting)

Technical Support
Marieka Boyd
Ryan Jones
Connie Meacham++
Shane Thacker

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

* ORISE
** Postdoctoral Fellow
*** NRMRL/OTAQ
# NERL
## Region 5
+ OAQPS
++ Retired
† Separated
Supplemental Materials
Evidence indicates there is no causal relationship 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 insufficient to conclude that there is a causal relationship with relevant pollutant exposures (e.g., because of exposure misclassification or other biases in studies) or that there is insufficient evidence from studies in which chance, confounding, and other biases could be ruled out. For example: (1) controlled human exposure studies are not consistent in failing to show an effect at any level of exposure; and (2) observational studies that cannot be explained by other lines 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 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 health outcome and/or at least one high-quality toxicological or small- to medium-scale field studies (e.g., animal studies or mode of action information) are limited or inconsistent; (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 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. Generally, the determination is based on multiple high-quality studies conducted by multiple research groups.

Evidence is suggestive but limited.

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

Evidence is of insufficient evidence (e.g., animal studies or mode of action information) to support the determination.

Evidence is insufficient to conclude that there is a causal relationship 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 indicates there is no causal relationship with relevant pollutant exposures. Several adequate studies show that human beings are not exposed to pollutant concentrations that are sufficiently different from background levels to show an effect at any level of exposure. Evidence indicates that a causal relationship exists with relevant pollutant exposures. A multiple hemichronic health outcome is consistent in showing a relationship with relevant pollutant exposures but is limited, and 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 or small-scale field studies (e.g., animal studies or mode of action information) are limited or inconsistent. Generally, the determination is based on multiple high-quality studies conducted by multiple research groups.

Evidence is suggestive but limited.

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

Evidence is of insufficient evidence (e.g., animal studies or mode of action information) to support the determination.

Evidence is insufficient to conclude that there is a causal relationship 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.

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Evidence indicates there is no causal relationship with relevant pollutant exposures. Several adequate studies show that human beings are not exposed to pollutant concentrations that are sufficiently different from background levels to show an effect at any level of exposure.

Evidence is of insufficient evidence (e.g., animal studies or mode of action information) to support the determination.
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.

<table>
<thead>
<tr>
<th>Study</th>
<th>Location</th>
<th>Lag</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>†Lee et al. (2015)a</td>
<td>3 Southeast states, U.S.</td>
<td>0-1</td>
<td>Cardiovascular CHF MI Stroke</td>
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<td>†Dai et al. (2014)</td>
<td>75 U.S. cities</td>
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<td>Cardiovascular MI Stroke</td>
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<tr>
<td>†Samoli et al. (2013)</td>
<td>10 European Med cities</td>
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<td>Cardiovascular Cardiac CHF</td>
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<tr>
<td>†Samoli et al. (2014)</td>
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<td>0-1</td>
<td>Cardiovascular Cardiac CHF Cerebrovascular Acute Coronary Events Arrhythmias</td>
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<td>9 French cities</td>
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<td>†Milojevic et al. (2014)</td>
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<tr>
<td>†Shah et al. (2015)</td>
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<td>---</td>
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<tr>
<td>†Wang et al. (2014)</td>
<td>Meta-analysis</td>
<td>---</td>
<td>Stroke</td>
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</tbody>
</table>

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 $\mu$g/m$^3$ increase in 24-hour average PM$_{2.5}$ concentrations observed in multicity studies and meta-analyses.
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.

**Figure 11-1. Summary of associations between short-term PM$_{2.5}$ exposure and total (nonaccidental) mortality in multiplicity studies for a 10 µg/m$^3$ increase in 24-hour average concentrations.**

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

<table>
<thead>
<tr>
<th>Reference</th>
<th>Cohort</th>
<th>Notes</th>
<th>Years</th>
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<td>tPope et al. 2014</td>
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<td>CA Cancer Prev</td>
<td></td>
<td>1973-2002</td>
<td>23.4</td>
</tr>
</tbody>
</table>
• **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.
Figure 6-15. Distribution of total (nonaccidental) mortality associations for short-term PM$_{2.5}$ and PM$_{2.5}$ components exposure.
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

<table>
<thead>
<tr>
<th>Welfare Effect</th>
<th>ISA</th>
<th>Current PM Draft ISA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>PM</td>
</tr>
<tr>
<td>Visibility</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Climate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Materials</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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

* = new determination or change in causality determination from 2009 PM ISA
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)
  o Long-term visibility improvements throughout the U.S as PM concentrations have decreased
  o Regional and seasonal patterns in atmospheric visibility parallel PM concentration patterns
  o More evidence supporting the relationship between visibility and PM composition

• Climate Effects (Causal)
  o New evidence provides greater specificity about radiative forcing
  o Increased understanding of additional climate impacts driven by PM radiative effects
  o Improved characterization of key sources of uncertainty particularly with response to PM-cloud interactions

• Materials Effects (Causal)
  o New information for glass and metals including modeling of glass soiling
  o Progress in the development of quantitative dose-response relationships and damage functions for materials in addition to stone, including glass and metals
  o Quantitative research on PM impacts on energy yield from photovoltaic systems
# At-Risk Framework Description

<table>
<thead>
<tr>
<th>Classification</th>
<th>Health Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adequate evidence</td>
<td>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.</td>
</tr>
<tr>
<td>Suggestive evidence</td>
<td>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.</td>
</tr>
<tr>
<td>Inadequate evidence</td>
<td>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.</td>
</tr>
<tr>
<td>Evidence of no effect</td>
<td>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.</td>
</tr>
</tbody>
</table>

Excerpt from Preamble to ISAs
Michael Kleinman, Ph.D.

• UC Irvine Professor of Environmental Toxicology
• Co-Director of the Air Pollution Health Effects Laboratory in the Department of Community and Environmental Medicine
• Adjunct Professor in College of Medicine
• Serves on the Air District Advisory Council
• Ph.D. in Environmental Health Sciences from New York University
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)
  • Carboxyls (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)

Smaller droplet size → clouds last longer → increase albedo → less precipitation

Black carbon

Sulfate organic carbon

NMVOCs, CO₂, CH₄ → OH + NOₓ

T↑

T↑

T↓

Pollutant sources

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

http://www.epa.gov/blackcarbon/basic.html
1 in 6 deaths, worldwide, is attributable to Pollution
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.
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

When you denude the UFP

ultrafines
less oxygenated
(to denuder)

\[
\frac{m/z 44 \text{ (CO}_2^+)}{m/z 55 \text{ (C}_4\text{H}_7^+)} \approx 0.4
\]

larger particles
oxygenated

\[
\frac{m/z 44 \text{ (CO}_2^+)}{m/z 55 \text{ (C}_4\text{H}_7^+)} \approx 4
\]

- Quasi-ultrafines
- Accumulation mode

<table>
<thead>
<tr>
<th>Temperature, °C</th>
<th>Ambient</th>
<th>Denuded</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>42%</td>
<td></td>
</tr>
<tr>
<td>100</td>
<td>47%</td>
<td></td>
</tr>
<tr>
<td>200</td>
<td>66%</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Temperature, °C</th>
<th>HMW PAHs</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>14%</td>
</tr>
<tr>
<td>100</td>
<td>53%</td>
</tr>
<tr>
<td>200</td>
<td>81%</td>
</tr>
</tbody>
</table>

- DTT activity, nmol/min/m³
- Concentration, pg/m³
Removing the Organic Constituents From Ambient UFP Blocks CV Effects

![Graphs showing changes in various parameters over weeks of exposure to different conditions.]

- **A** shows changes in a parameter labeled LF (% change from baseline) over weeks of exposure to CAP, Air, and deCAP conditions.
- **B** depicts changes in another parameter labeled HF (% change from baseline) with similar exposure conditions.
- **C** illustrates changes in a parameter labeled LV (% change from baseline) over weeks of exposure.
- **D** presents changes in a parameter labeled TGF (% change from baseline) with exposure conditions.

### Table of Results

<table>
<thead>
<tr>
<th>Condition</th>
<th>Plaque Size (% area of plaque in total lumen CS)</th>
<th>Lipid Accumulation (% area of lipid in total tissue CS)</th>
<th>Lipid Peroxidation (nM MDA/mg protein)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>14.5 ± 6.3</td>
<td>5.1 ± 3.3</td>
<td>134 ± 29</td>
</tr>
<tr>
<td>CAP</td>
<td>29.9 ± 10.0*</td>
<td>8.9 ± 2.4*</td>
<td>218 ± 32*, **</td>
</tr>
<tr>
<td>deCAP</td>
<td>2.3 ± 0.7</td>
<td>2.2 ± 0.3</td>
<td>141 ± 17</td>
</tr>
</tbody>
</table>

*Significant difference from baseline
**Significant difference from Air

---

**Aortic Arch 2x**
- **Air**
- **Denuded**
- **Undenuded**
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.

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

- Moving the AMS is a group effort!
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$_{10-2.5}$: Road dust, crustal, and biological material
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
Ambient Particulate Air Pollution and Daily Mortality in 652 Cities
Fine-Particulate Air Pollution and Life Expectancy in the United States

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
Inequality Curve

Cumulative share of population ranked by racial-ethnic or socioeconomic position

Cumulative share of environmental hazard

Ranking: most disadvantaged  most advantaged

Cumulative Risk

• People of color and low SES have
  – Greater exposures to outdoor particulate 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$_{2.5}$** 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)

**All Cardiovascular Causes**

- **Relative Risk**
  - Light
  - Medium
  - Heavy

**Slide credit:** Wayne Cascio

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

H. Christopher Frey
frey@ncsu.edu

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 and Public Review

- Draft IRP
- Final IRP

TIME

- 1st Draft ISA
- 2nd Draft ISA
- Final ISA

- REA Plan
- 1st Draft REA
- 2nd Draft REA
- Final REA

- 1st Draft PA
- 2nd Draft PA
- Final PA

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

• CASAC is split 4-2:
  – Four recommend keeping all current standards (primary PM2.5, coarse PM, secondary PM2.5) as is.
  – Rationales offered for keeping the annual primary PM2.5 standard:
    » “beta” coefficients used in the risk assessment are not causal coefficients
    » Exposures in recent studies are “estimated”
    » Temperature has not been properly accounted for
    » The concentration-response slopes from new studies are approximately the same as from old studies, so there’s nothing new here
    » EPA should have informed the CASAC of an acceptable risk level

I listened for both days. I can’t recall any of these four acknowledging anything learned from new studies

There Should be 26 People at This Table, Not 6 (one is EPA staff)
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

Panel report at ucsusa.org/pmpanel
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 $10 \ \mu g/m^3$ to $8 \ \mu g/m^3$ (versus $12 \ \mu g/m^3$ now).
- The **24-hour** standard should be $30 \ \mu g/m^3$ to $25 \ \mu g/m^3$ (versus $35 \ \mu g/m^3$ 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/m$^3$ 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/m$^3$ or lower.
- The Panel finds that there is not sufficient scientific certainty below 8 µg/m$^3$ 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/m³ 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

- Workshop on science-policy issues
- Integrated Review Plan (IRP): timeline and key policy-relevant issues and scientific questions
  - Integrated Science Assessment (ISA): evaluation and synthesis of most policy-relevant studies
  - Risk/Exposure Assessment (REA): quantitative assessment, as warranted, focused on key results, observations, and uncertainties
  - REA Planning Document
  - Clean Air Scientific Advisory Committee (CASAC) review
  - Public comment
  - Policy Assessment (PA): staff analysis of policy options based on integration and interpretation of information in the ISA and REA
  - EPA proposed decisions on standards
  - Interagency review
  - Agency decision making and draft proposal notice
  - Interagency review
  - EPA final decisions on standards
  - Public hearings and comments on proposal
  - Agency decision making and draft final notice
Generic “Full” NAAQS Science Review from CASAC and Public Perspective

<table>
<thead>
<tr>
<th>CASAC Meeting*</th>
<th>Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 &amp; 2</td>
<td>Draft Integrated Review Plan</td>
</tr>
<tr>
<td>3 &amp; 4</td>
<td>1st Draft Integrated Science Assessment</td>
</tr>
<tr>
<td></td>
<td>Risk &amp; Exposure Assessment Plan</td>
</tr>
<tr>
<td>5 &amp; 6</td>
<td>2nd Draft Integrated Science Assessment</td>
</tr>
<tr>
<td></td>
<td>1st Draft Risk &amp; Exposure Assessments</td>
</tr>
<tr>
<td>7 &amp; 8</td>
<td>2nd Draft Risk &amp; Exposure Assessments</td>
</tr>
<tr>
<td></td>
<td>1st Draft Policy Assessment</td>
</tr>
<tr>
<td>9 &amp; 10</td>
<td>2nd Draft Policy Assessment</td>
</tr>
</tbody>
</table>

*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

#### CASAC Meeting*  
<table>
<thead>
<tr>
<th>Meeting</th>
<th>Topic</th>
<th>2016 Before P/W</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 &amp; 2</td>
<td>Draft Integrated Review Plan</td>
<td></td>
</tr>
<tr>
<td>3 &amp; 4</td>
<td>1st Draft Integrated Science Assessment</td>
<td></td>
</tr>
<tr>
<td>5 &amp; 6</td>
<td>1st Draft Policy Assessment</td>
<td></td>
</tr>
</tbody>
</table>

*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 O₃ Reviews

“Pool” of 12
May only interact with CASAC in writing

Written questions from CASAC

Written answers from “Pool”

No Iteration

No Interactive Deliberation Within Pool Or With CASAC
Typical Pre-Pruitt/Wheeler CASAC for PM and O₃ 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 ($\text{PM}_{10}$ as an indicator for $\text{PM}_{10-2.5}$)
  – 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 $\text{PM}_{2.5}$ standard.
  – Should move to $\text{PM}_{10-2.5}$ as the indicator in the next review.
Major Findings: Visibility

- Welfare (Secondary) Standards
  - Current annual standard has no effect (15 \( \mu g/m^3 \) vs. 12 \( \mu g/m^3 \) 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 needed for health studies and for baselines
Break
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
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
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
Population Concentration-Response Relationships

Incidence of Effect vs Concentration

Background
Variability Underlying Concentration Response Observations

Variable Risk at a Given Dose

Increasing Risk

Variable Concentration with Location

High: Low ~ 5:1

Median

Sarah Vogel svogel@edf.org
Chemical Stressor

Background Exposure (Endogenous and Exogenous)

Susceptibility: Health & Disease Status, Genetics, Age, Sex

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

<table>
<thead>
<tr>
<th></th>
<th>Individual level</th>
<th>Population Level</th>
</tr>
</thead>
</table>
| 1. An individual's: Nonlinear  
The population: Linear | ![Individual Level Graph](image1) | ![Population Level Graph](image2) |
|   | Probability of Effect | Fraction of Population Affected |
|   | Background, Concentration | Concentration |
| 2. An individual's: Nonlinear  
The population: Nonlinear | ![Individual Level Graph](image3) | ![Population Level Graph](image4) |
|   | Probability of Effect | Fraction of Population Affected |
|   | Background, Concentration | Concentration |
| 3. An individual's: Linear  
The population: Linear | ![Individual Level Graph](image5) | ![Population Level Graph](image6) |
|   | Probability of Effect | Fraction of Population Affected |
|   | Concentration | Concentration |
• 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
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

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

<table>
<thead>
<tr>
<th>Source</th>
<th>Marker Constituents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biomass</td>
<td>EC, OC, K</td>
</tr>
<tr>
<td>Secondary Ammonium Nitrate</td>
<td>NO$_3^-$, NH$_4^+$</td>
</tr>
<tr>
<td>Secondary Ammonium Sulfate</td>
<td>SO$_4^{2-}$, NH$_4^+$</td>
</tr>
<tr>
<td>Resuspended Soil</td>
<td>Al, Si, Ca, Fe, Ti</td>
</tr>
<tr>
<td>Vehicular Emissions</td>
<td>EC, OC, Fe, Cu, Zn</td>
</tr>
</tbody>
</table>
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
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 Ambient 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*

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Averaging Time</th>
<th>Primary/Secondary</th>
<th>Level</th>
<th>Form</th>
<th>Decisions in 2012 Review</th>
</tr>
</thead>
<tbody>
<tr>
<td>PM$_{2.5}$</td>
<td>Annual</td>
<td>Primary</td>
<td>12.0 µg/m³</td>
<td>Annual arithmetic mean, averaged over 3 years</td>
<td>Revised level from 15 to 12 µg/m³**</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Secondary</td>
<td>15.0 µg/m³</td>
<td></td>
<td>Retained**</td>
</tr>
<tr>
<td></td>
<td>24-hour</td>
<td>Primary and Secondary</td>
<td>35 µg/m³</td>
<td>98th percentile, averaged over 3 years</td>
<td>Retained</td>
</tr>
<tr>
<td>PM$_{10}$</td>
<td>24-hour</td>
<td>Primary and Secondary</td>
<td>150 µg/m³</td>
<td>Not to be exceeded more than once per year on average over a 3-year period</td>
<td>Retained</td>
</tr>
</tbody>
</table>

*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

**Planning:** Identified new scientific information, policy-relevant issues
- Call for Information
- Workshop
- Planning Document

**Assessment:** Scientific evidence, risk information, potential policy implications for standards (indicator, averaging time, form, level)
- Integrated Science Assessment – final in Dec 2019
- Policy Assessment – final in Jan 2020

**Rulemaking:** Agency decision making, interagency review and public comments process
- Proposed Decision – Spring 2020
- Final Decision – Dec 2020

**Clean Air Scientific Advisory Committee (CASAC) review**
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 µg/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 98$^{th}$ 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)

*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 $> 8.0$ µg/m$^3$.
- In most studies with data available, 75% of exposures (or deaths) are at predicted ambient PM$_{2.5}$ concentrations $> 6.0$ µg/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 25$^{th}$ (filled) and 10$^{th}$ (open) percentiles of exposures or deaths.
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.

* 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 $\mu$g/m$^3$; alternative 24-hour standard with a level of 30 $\mu$g/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 $\geq$ 30 years: ~60M)
- 30 annual-controlling (population $\geq$ 30 years: ~50M)
- 11 daily-controlling (population $\geq$ 30 years: ~4M)
- 6 mixed (population $\geq$ 30 years: ~5M)
## Summary of Risk Estimates

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

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Study</th>
<th>Air quality simulation approach*</th>
<th>Current Standard Absolute Risk (12/35 µg/m³)</th>
<th>CS (12/35) % of baseline**</th>
<th>Alternative Standard Absolute Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Alternative Annual (10 µg/m³)</td>
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<tr>
<td><strong>Long-term exposure related mortality</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic Heart Disease</td>
<td>Jerrett 2016</td>
<td>Pri-PM</td>
<td>16,500 (12,600-20,300)</td>
<td>14.1</td>
<td>14,400 (11,000-17,700)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>16,800 (12,800-20,500)</td>
<td>14.3</td>
<td>14,200 (10,900-17,500)</td>
</tr>
<tr>
<td></td>
<td>Pope 2015</td>
<td>Pri-PM</td>
<td>15,600 (11,600-19,400)</td>
<td>13.3</td>
<td>13,600 (10,100-17,000)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>15,800 (11,800-19,600)</td>
<td>13.4</td>
<td>13,400 (9,870-16,700)</td>
</tr>
<tr>
<td>All-cause</td>
<td>Di 2017</td>
<td>Pri-PM</td>
<td>46,200 (45,000-47,500)</td>
<td>8.4</td>
<td>40,300 (39,200-41,400)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>46,900 (45,600-48,200)</td>
<td>8.8</td>
<td>39,700 (38,600-40,800)</td>
</tr>
<tr>
<td></td>
<td>Pope 2015</td>
<td>Pri-PM</td>
<td>51,300 (41,000-51,400)</td>
<td>7.1</td>
<td>44,700 (35,700-53,500)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>52,100 (41,600-52,300)</td>
<td>7.2</td>
<td>44,000 (35,100-52,700)</td>
</tr>
<tr>
<td></td>
<td>Thurston 2015</td>
<td>Pri-PM</td>
<td>13,500 (2,360-24,200)</td>
<td>3.2</td>
<td>11,700 (2,050-21,100)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>13,700 (2,400-24,600)</td>
<td>3.2</td>
<td>11,500 (2,010-20,700)</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>Turner 2016</td>
<td>Pri-PM</td>
<td>3,890 (1,240-6,360)</td>
<td>8.9</td>
<td>3,390 (1,080-5,560)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>3,950 (1,250-6,460)</td>
<td>9.1</td>
<td>3,330 (1,060-5,470)</td>
</tr>
<tr>
<td><strong>Short-term exposure related mortality</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cause</td>
<td>Baxter 2017</td>
<td>Pri-PM</td>
<td>2,490 (983-4,000)</td>
<td>0.4</td>
<td>2,160 (850-3,460)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>2,530 (998-4,060)</td>
<td>0.4</td>
<td>2,120 (837-3,400)</td>
</tr>
<tr>
<td>Ito 2013</td>
<td></td>
<td>Pri-PM</td>
<td>1,180 (-16,2,370)</td>
<td>0.2</td>
<td>1,020 (-14,2,050)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>1,200 (-16,2,400)</td>
<td>0.2</td>
<td>1,000 (-14,2,020)</td>
</tr>
<tr>
<td>Zanobetti 2014</td>
<td></td>
<td>Pri-PM</td>
<td>3,810 (2,530-5,080)</td>
<td>0.7</td>
<td>3,300 (2,190-4,400)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sec-PM</td>
<td>3,870 (2,570-5,160)</td>
<td>0.7</td>
<td>3,250 (2,160-4,330)</td>
</tr>
</tbody>
</table>

* Pri-PM (primary PM-based modeling approach), Sec-PM (secondary PM-based modeling approach)
** CS denotes the current standard.
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)
• 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 $\sim$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 $\geq$ ~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

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 98$^{th}$ 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 98$^{th}$ percentile values below 10 and 25 $\mu$g m$^{-3}$, respectively.
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

<table>
<thead>
<tr>
<th>Exposure Duration</th>
<th>Outcome</th>
<th>2009 ISA Conclusion</th>
<th>2018 Draft ISA Conclusion</th>
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</thead>
<tbody>
<tr>
<td>Long-Term</td>
<td>Mortality</td>
<td>Causal</td>
<td>Causal</td>
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<tr>
<td></td>
<td>Cardiovascular</td>
<td>Causal</td>
<td>Causal</td>
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<tr>
<td></td>
<td>Respiratory</td>
<td>Likely to be causal</td>
<td>Likely to be causal</td>
</tr>
<tr>
<td></td>
<td>Cancer</td>
<td>Suggestive</td>
<td>Likely to be causal</td>
</tr>
<tr>
<td></td>
<td>Nervous System</td>
<td>None</td>
<td>Likely to be causal</td>
</tr>
<tr>
<td>Short-Term</td>
<td>Mortality</td>
<td>Causal</td>
<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Cardiovascular</td>
<td>Causal</td>
<td>Causal</td>
</tr>
<tr>
<td></td>
<td>Respiratory</td>
<td>Likely to be causal</td>
<td>Likely to be causal</td>
</tr>
</tbody>
</table>
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
Particulate Matter: Spotlight on Health Protection
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
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

- 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)
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
For any location, we can use the sub-totals to draw pie charts 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

* Contributed by modeled "present-day" emissions from existing local sources. Impacts from sources outside West Oakland not included.
Targets and Source Contributions for Diesel PM

Targets:

2025 – Today’s *average* residential neighborhood

2030 – Today’s *cleanest* residential neighborhood

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

DRAFT 2019-08-16
Targets and Source Contributions for PM$_{2.5}$

**Targets:**

2025 – Today’s *average* residential neighborhood

2030 – Today’s *cleanest* residential neighborhood

*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.
Impact Per Ton: \( \text{PM}_{2.5} \) in West Oakland

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

**PM$_{2.5}$**
- 1.7 µg/m$^3$

**Diesel PM**
- 0.3 µg/m$^3$

<table>
<thead>
<tr>
<th>Local model – mapped impacts*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regional model (minus West Oakland)</td>
</tr>
</tbody>
</table>

*Construction, residential woodburning, and restaurants not modeled
Thank you
Break
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
Adjournment
Particulate Matter:
Spotlight on Health Protection