GEORGIA
DEPARTMENT OF NATURAL RESOURCES
ENVIRONMENTAL PROTECTION DIVISION

Ozone and PM NAAQS Reviews

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NACAA Criteria Pollutants Committee
Monthly Conference Call
December 9, 2019

OUTLINE

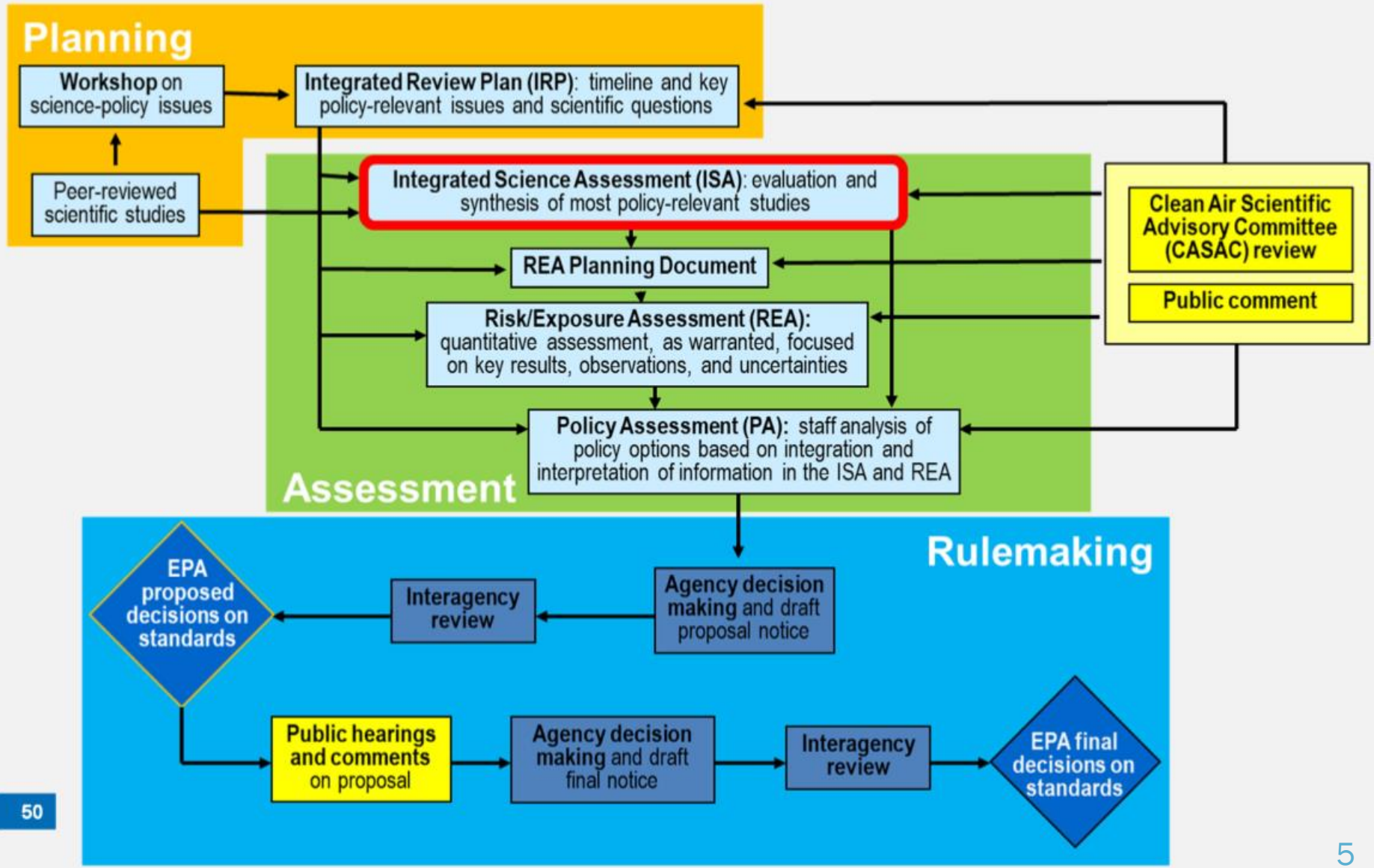
- NAAQS Review Process
- PM NAAQS Review
- Ozone NAAQS Review
- Additional Information

NAAQS REVIEW PROCESS

Introduction and Statutory Requirements

- EPA sets national ambient air quality standards (NAAQS) for six pollutants
  - Ground-level ozone
  - Carbon monoxide
  - Nitrogen dioxide
  - Particulate matter
  - Lead
  - Sulfur dioxide
- Sections 108 and 109 of the Clean Air Act govern the establishment, review, and revision (as appropriate) of NAAQS, including:
  - **Primary (health-based) standards** which in the "judgment of the Administrator" are "requisite to protect the public health", including at-risk populations, with an "adequate margin of safety"
  - **Secondary (welfare-based) standards** which in the "judgment of the Administrator" are "requisite to protect the public welfare from any known or anticipated adverse effects"
- The law requires EPA to review the scientific information and NAAQS for each criteria pollutant every five years, and to obtain advice from the Clean Air Scientific Advisory Committee (CASAC) on each review.
- Court decisions provide additional guidance on aspects of EPA decision-making
  - EPA is required to engage in "reasoned decision making" to translate scientific evidence into standards
  - EPA may not consider cost in setting standards; however, cost is considered in developing control strategies to meet the standards (implementation phase)

Overview of the NAAQS Review Process



Initiation of Expedited Review (May 2018 memo)

May 9, 2018 memo from the EPA Administrator:

- Directed the initiation of an expedited review of the PM NAAQS, targeting completion by the end of 2020
  - Also specified expedited review of NAAQS for ozone
- Identified ways to streamline the review process (e.g., increased focus on policy-relevant information and avoiding multiple drafts of documents)
- Identified standardized set of charge questions for CASAC including:
  - General charge questions for NAAQS reviews, to be supplemented with more detailed requests as necessary
  - Two additional charge questions that may elicit information not relevant to the standard-setting process.
    - EPA may consider an appropriate mechanism, including after receiving CASAC's final advice on the standards, to facilitate robust feedback on these topics

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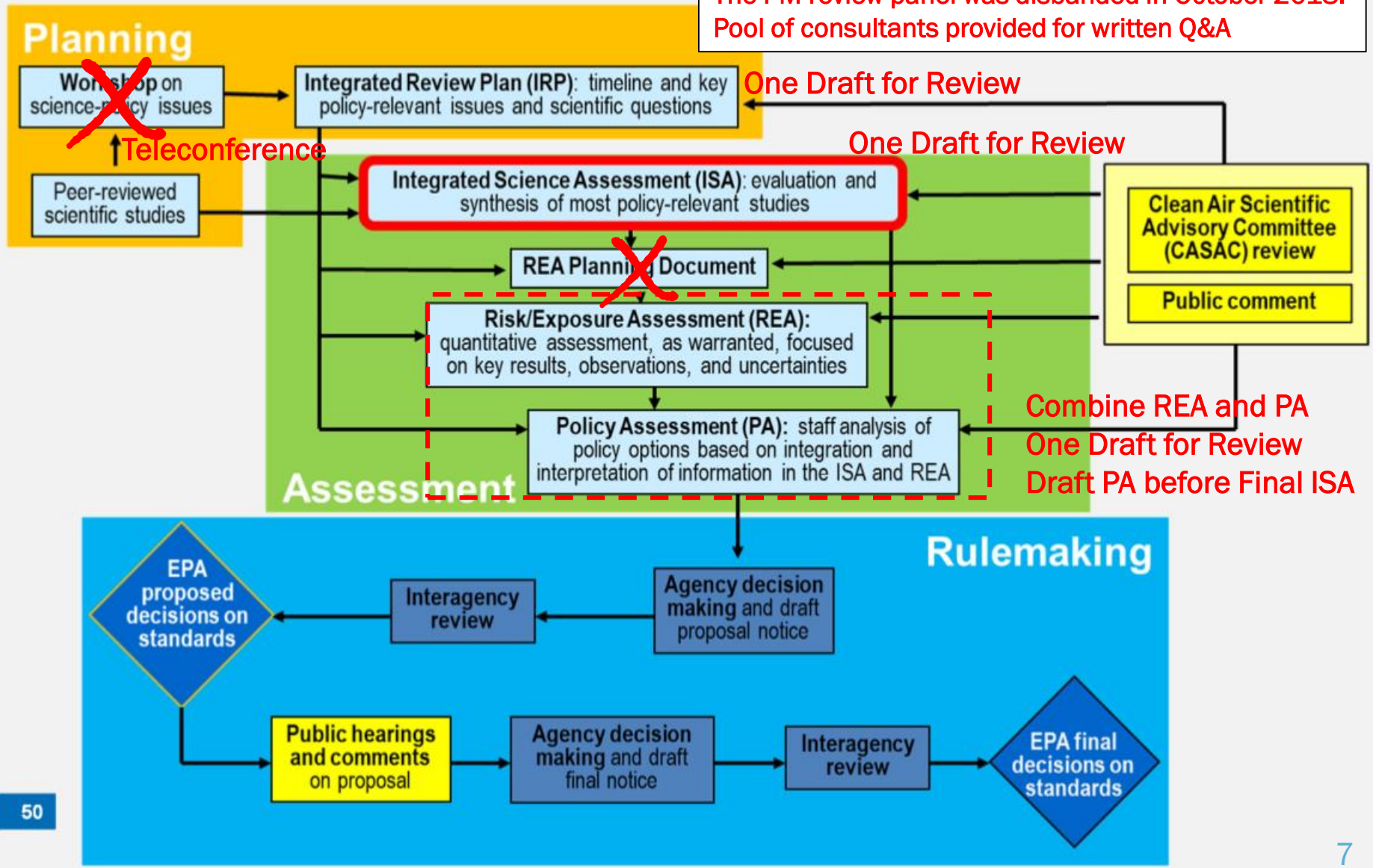
Streamline NAAQS Review Process

Overview of the NAAQS Review Process

No ozone review panel was formed.
The PM review panel was disbanded in October 2018.
Pool of consultants provided for written Q&A



CPHEA/ORD and OAQPS/OAR Interactions: NAAQS Review

CPHEA/ORD	NAAQS Activity	OAQPS/OAR
Co-lead development of workshop	**Workshop on science-policy issues (ORD/OAR)**	Co-lead development of workshop
Author – Chapter on ISA	**Integrated Review Plan (ORD/OAR)**	Author of other chapters (e.g., REA, PA)
Lead development	**Integrated Science Assessment (ORD)**	Review draft materials with focus on identifying areas where clarification is needed
Review draft materials and provide comments on interpretation of science	**Risk/Exposure Assessment (OAR)**	Lead development
Review draft materials and provide comments on interpretation of science	**Policy Assessment (OAR)**	Lead development
Provide technical and scientific support	**Rule-making materials (OAR)**	Lead development

Statutory Requirements: CASAC

- Section 109(d)(2) addresses the appointment and advisory functions of an independent scientific review committee
- Section 109(d)(2)(B) provides that, at 5-year intervals, this committee "shall complete a review of the criteria…and the national primary and secondary ambient air quality standards…and shall recommend to the Administrator any new…standards and revisions of existing criteria and standards as may be appropriate…".
- Section 109(d)(2)(C) reads: "Such committee shall also

  (i) advise the Administrator of areas in which additional knowledge is required to appraise the adequacy and basis of existing, new, or revised national ambient air quality standards,

  (ii) describe the research efforts necessary to provide the required information,

  (iii) advise the Administrator on the relative contribution to air pollution concentrations of natural as well as anthropogenic activity, and

  (iv) advise the Administrator of any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance of such national ambient air quality standards.

CASAC DELIVERABLES

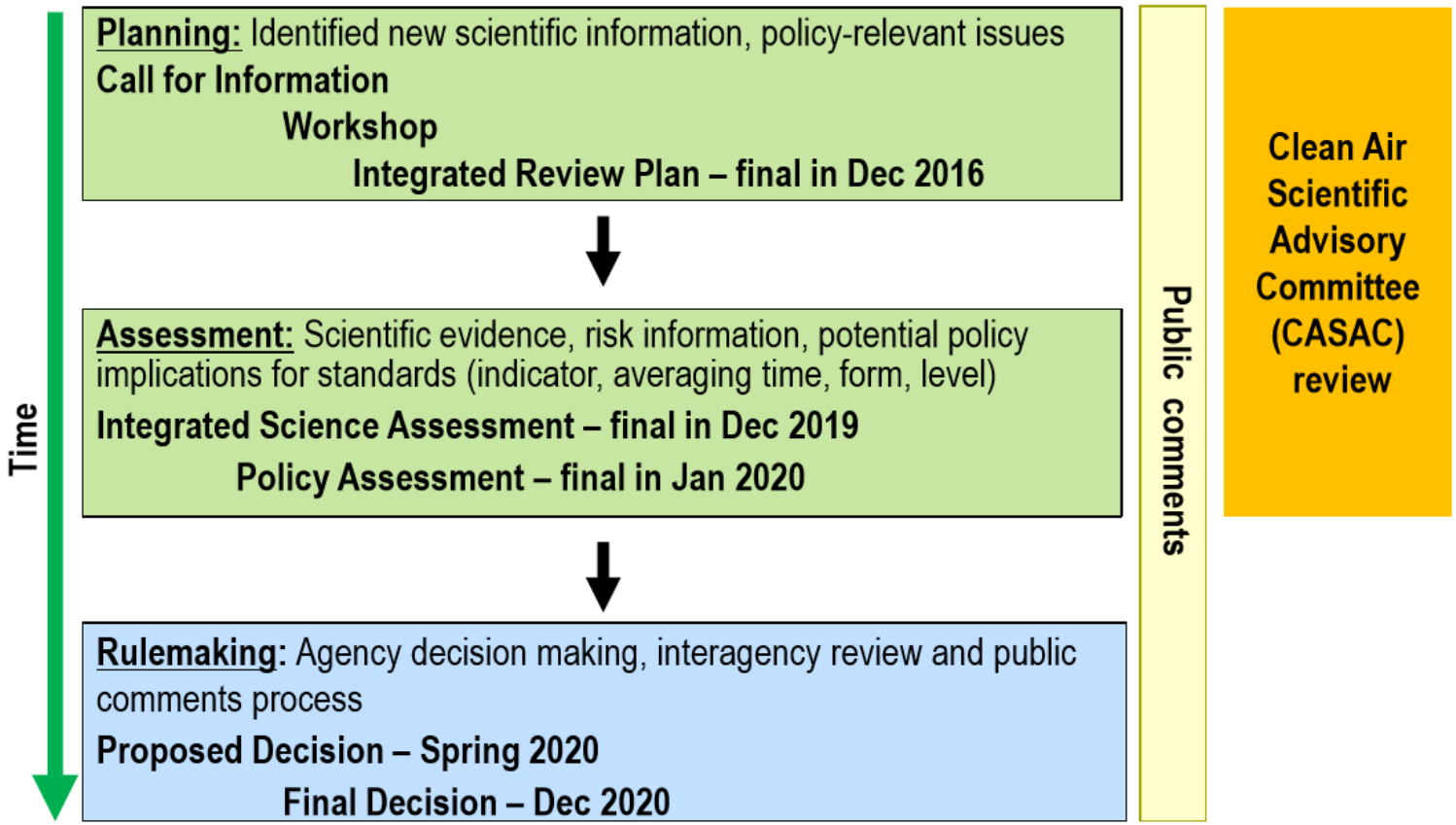
- **Integrated Review Plan (IRP)**
  - Letter to EPA Administrator, Individual CASAC Comments
- **Integrated Science Assessment (ISA)**
  - Letter to EPA Administrator, Consensus Response to Charge Questions, Individual CASAC Comments
- **REA Planning Document**
  - Letter to EPA Administrator, Individual CASAC Comments
- **Risk/Exposure Assessment (REA)**
  - Letter to EPA Administrator, Consensus Response to Charge Questions, Individual CASAC Comments
- **Policy Assessment (PA)**
  - Letter to EPA Administrator, Consensus Response to Charge Questions, Individual CASAC Comments

ADMINISTRATOR DECISION

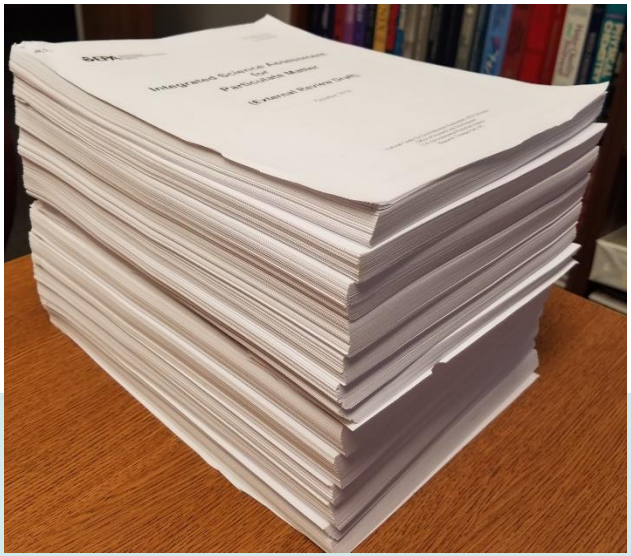
- Section 109(b)(1) defines primary standards as ones "the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health."
- The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels.
- What is an "acceptable" risk?

PM NAAQS REVIEW

Process and Schedule for This Review of the PM NAAQS



PM ISA REVIEW



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U.S. Environmental Protection Agency Clean Air Scientific Advisory Committee (CASAC) Public Meeting

Review of the Integrated Science Assessment for Particulate Matter
External Review Draft

National Center for Environmental Assessment
Office of Research and Development
Washington, DC, December 12-13, 2018

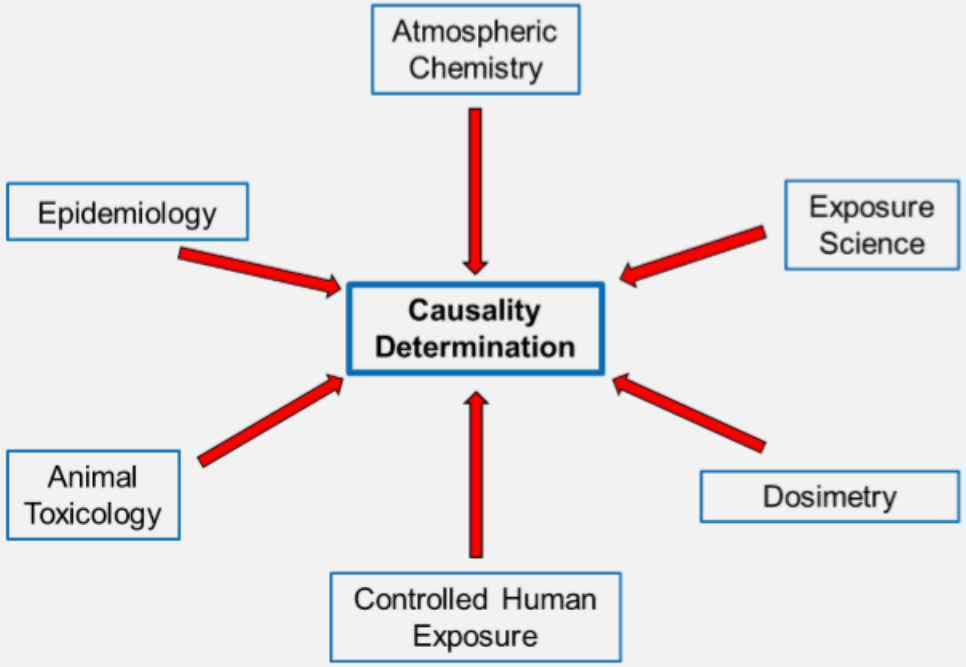
Weight-of-Evidence Approach for Causality Determinations for Health and Welfare Effects

- Provides transparency through structured framework
- Developed and applied in ISAs for all criteria pollutants
- Emphasizes synthesis of evidence across scientific disciplines (e.g., controlled human exposure, epidemiologic, and toxicological studies)
- Five categories based on overall weight-of-evidence:
  - Causal relationship
  - Likely to be a causal relationship
  - Suggestive of, but not sufficient to infer, a causal relationship
  - Inadequate to infer the presence or absence of a causal relationship
  - Not likely to be a causal relationship
- ISA Preamble describes this framework
  - Preamble is now stand-alone document (http://www.epa.gov/isa)
- CASAC reviewed the Agency's causal framework **~13 times** by **~90** CASAC charter and ad hoc panel members in the process of reviewing ISAs from 2008 – 2015; its use was supported in all ISAs

Evaluation of the Scientific Evidence

- Organize relevant literature for broad health outcome categories
- Evaluate studies, characterize results, extract relevant data
- Integrate evidence across disciplines for health outcome categories
- Develop causality determinations using established framework
- Evaluate evidence for populations potentially at increased risk
- Consideration of evidence spans many scientific disciplines from source to effect:

  - Atmospheric chemistry
  - Exposure
  - Controlled human exposure studies
  - Epidemiologic studies
  - Animal toxicologic studies
  - At-risk populations/lifestages



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

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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) [illegible] been shown to result in health effects in studies in which chance, confounding, and other biases could be ruled out with [illegible] (1) controlled human exposure studies that demonstrate consistent effects, or (2) observational studies that cannot be expl[illegible] 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 [illegible] in which chance, confounding, and other biases could be ruled out with reasonable confidence. Controlled exposure studies (laboratory [illegible] studies) provide the strongest evidence for causality, but the scope of inference may be limited. Generally, the [illegible] 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 re[illegible]. For example: (1) observational studies show an association, but copollutant exposures are difficult to address and/or other li[illegible] 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, [illegible] other biases are minimized but uncertainties remain. For example, field studies show a relationship, but suspected interacting factors [illegible] 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-quali[illegible] 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 avail[illegible] 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 [illegible] The available studies are of insufficient quality, consistency, or statistical power to permit a conclusion regarding the presence [illegible] effect.
Not likely to be a causal relationship	Evidence indicates there is no causal relationship with relevant pollutant exposures. Several adequate studies, co[illegible] of exposure that human beings are known to encounter and considering at-risk populations and lifestages, are mutually consistent in [illegible] any level of exposure.	Evidence indicates there is no causal relationship with relevant pollutant exposures. Several adequate studies examining relationships with relevant exposures are consistent in failing to show an effect at any level of exposure.

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

Multiple, high-quality studies
Important uncertainties remain

Evidence is suggestive but limited

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

Multiple studies show no effect across exposure concentrations

Contents of the Draft PM ISA

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

Executive Summary

Chapter 1. Integrated Synthesis

Chapter 2. Sources, Atmospheric Chemistry, and Ambient Concentrations

Chapter 3. Exposure to Ambient PM

Chapter 4. Dosimetry of PM

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

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

Chapter 13. Welfare Effects

EPA United States Environmental Protection Agency

Draft PM ISA
Health Effects: Causality Determinations

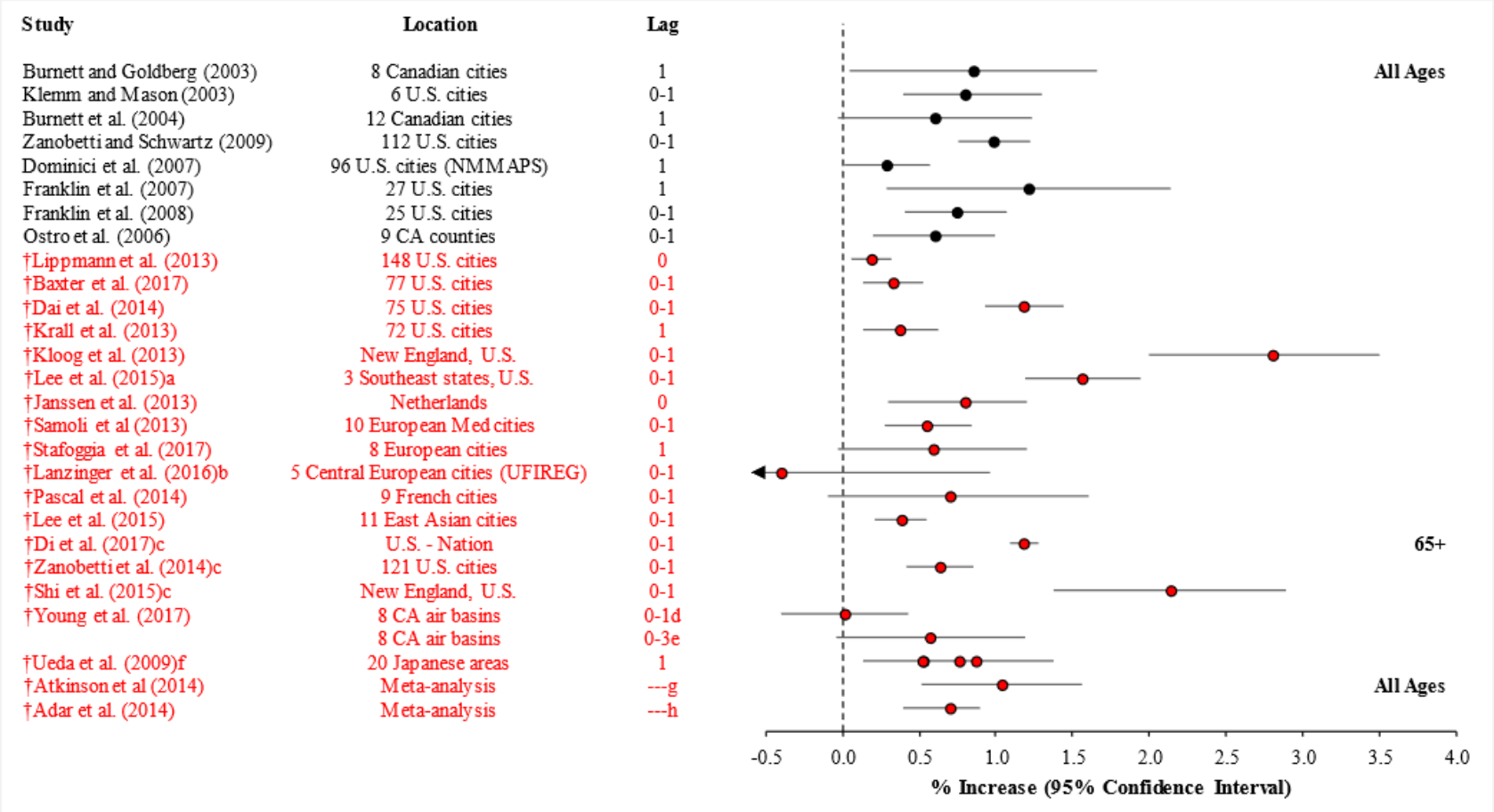
HUMAN HEALTH EFFECTS					
		ISA	Current PM Draft ISA		
		Indicator	$PM_{2.5}$	$PM_{10-2.5}$	UFP
Health Outcome	Respiratory	Short-term exposure			
		Long-term exposure			
	Cardiovascular	Short-term exposure			
		Long-term exposure		*	
	Metabolic	Short-term exposure	*	*	*
		Long-term exposure	*	*	*
	Nervous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

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

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



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

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

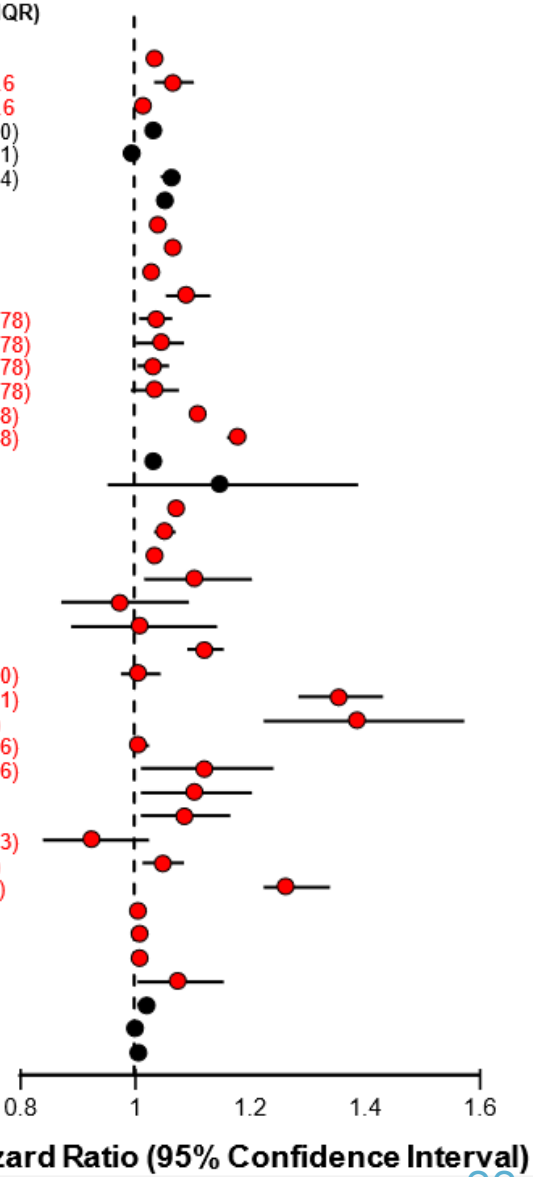
Mortality – Long-term $PM_{2.5}$ Exposure (Chapter 11) (Causal)

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

Reference	Cohort	Notes	Years	Mean (IQR)
†Pope et al. 2014	ACS		1982-2004	12.6
†Lepeule et al. 2012	Harvard Six Cities		1974-2009	11.4-23.6
†Thurston et al. 2015	NIH-AARP		2000-2009	10.2-13.6
Zeger et al. 2008	MCAPS	Eastern	2000-2005	14.0 (3.0)
Zeger et al. 2008	MCAPS	Western	2000-2005	13.1 (8.1)
Zeger et al. 2008	MCAPS	Central	2000-2005	10.7 (2.4)
Eftim et al. 2008	ACS-Medicare		2000-2002	13.6
†Di et al. 2017	Medicare		2000-2012	11.5
†Di et al. 2017	Medicare	exp<12	2000-2012	11.5
†Di et al. 2017	Medicare	nearest monitor	2000-2012	11.5
†Kioumourtzoglou et al. 2016	Medicare		2000-2010	12
†Shi et al. 2015	Medicare	mutual adj	2003-2008	8.12 (3.78)
†Shi et al. 2015	Medicare	exp <10, mutual adj	2003-2008	8.12 (3.78)
†Shi et al. 2015	Medicare	no mutual adj	2003-2008	8.12 (3.78)
†Shi et al. 2015	Medicare	exp <10, no mutual adj	2003-2008	8.12 (3.78)
†Wang et al. 2017	Medicare		2000-2013	10.7 (3.8)
†Wang et al. 2017	Medicare	exp<12	2000-2013	10.7 (3.8)
Lipfert et al. 2006	Veterans Cohort		1997-2001	14.34
Goss et al. 2004	U.S. Cystic Fibrosis		1999-2000	13.7
†Crouse et al. 2012	CanCHEC	Satellite data	1991-2001	8.9
†Crouse et al. 2012	CanCHEC	Monitor data	1991-2001	11.2
†Crouse et al. 2015	CanCHEC		1991-2006	8.9
†Chen et al. 2016	EFFECT		1999-2011	10.7
†Weichenthal et al. 2014	Ag Health		1993-2009	8.84
†Weichenthal et al. 2014	Ag Health	more precise exp	1993-2009	8.84
†Pinault et al. 2016	CCHS		1998-2011	6.3
†Lipsett et al. 2011	CA Teachers		2000-2005	15.6 (8.0)
†Ostro et al. 2010	CA Teachers	within 30 km	2002-2007	17.5 (6.1)
†Ostro et al. 2010	CA Teachers	within 8 km	2002-2007	17 (6.1)
†Ostro et al. 2015	CA Teachers		2001-2007	17.9 (9.6)
†Puett et al. 2009	Nurses Health		1992-2002	13.9 (3.6)
†Hart et al. 2015	Nurses Health	nearest monitor	2000-2006	12.7
†Hart et al. 2015	Nurses Health	spatio-temp. model	2000-2006	12
†Puett et al. 2011	Health Prof	full model	1989-2003	17.8 (4.3)
†Hart et al. 2011	TrIPS		1985-2000	14.1 (4)
†Kloog et al. 2013	MA cohort	CVD+Resp	2000-2008	9.9 (1.6)
†Garcia et al. 2015	CA cohort	Kriging	2006	13.06
†Garcia et al. 2015	CA cohort	IDW	2006	12.94
†Garcia et al. 2015	CA cohort	closest monitor	2006	12.68
†Wang et al. 2016	NJ Cohort		2004-2009	11.3
Enstrom 2005	CA Cancer Prev		1973-1982	23.4
Enstrom 2005	CA Cancer Prev		1983-2002	23.4
Enstrom 2005	CA Cancer Prev		1973-2002	23.4



Draft PM ISA
Welfare Effects: Causality Determinations

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

Causal Likely causal Suggestive Inadequate

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

CASAC LETTER ON PM ISA (4/11/19)

- "The need for substantial revisions to the Draft ISA to provide clearer definitions, and technical details and methods in order to enable meaningful independent scientific review leads to the following two process recommendations:
  1. The CASAC recommends development of a Second Draft ISA for CASAC review.
  2. The CASAC recommends that the EPA reappoint the previous CASAC PM panel (or appoint a panel with similar expertise)… The panel should be appointed in time to review the Second Draft ISA."
- "The CASAC finds that the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal association between longterm $PM_{2.5}$ exposure and nervous system effects; between long-term UFP exposure and nervous system effects; or between long-term $PM_{2.5}$ exposure and cancer."

PM PA REVIEW

EPA
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REVIEW OF THE NATIONAL AMBIENT AIR QUALITY STANDARDS FOR PARTICULATE MATTER

OVERVIEW OF THE DRAFT POLICY ASSESSMENT

Presentation to the Clean Air Scientific Advisory Committee

October 24, 2019

Primary $PM_{2.5}$: Summary of Approach

- The draft PA considers what the available scientific evidence and quantitative risk information may indicate regarding the annual and 24-hour $PM_{2.5}$ standards – focus is on "causal" or "likely to be causal" $PM_{2.5}$-related health outcomes

Annual $PM_{2.5}$ standard

- Generally viewed as the principle means of providing public health protection against "typical" daily and annual $PM_{2.5}$ exposures
- In previous reviews, conclusions on the annual $PM_{2.5}$ standard have been largely 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 epidemiologic studies reporting health effect associations
- In this review, the draft PA characterizes the $PM_{2.5}$ air quality distributions in key studies (i.e., overall means, lower quartiles) and identifies study-area $PM_{2.5}$ metrics similar to design values (pseudo-design values)
- Similar to previous reviews, the PA also provides quantitative estimates of health risks that would be allowed by the current and various alternative standards

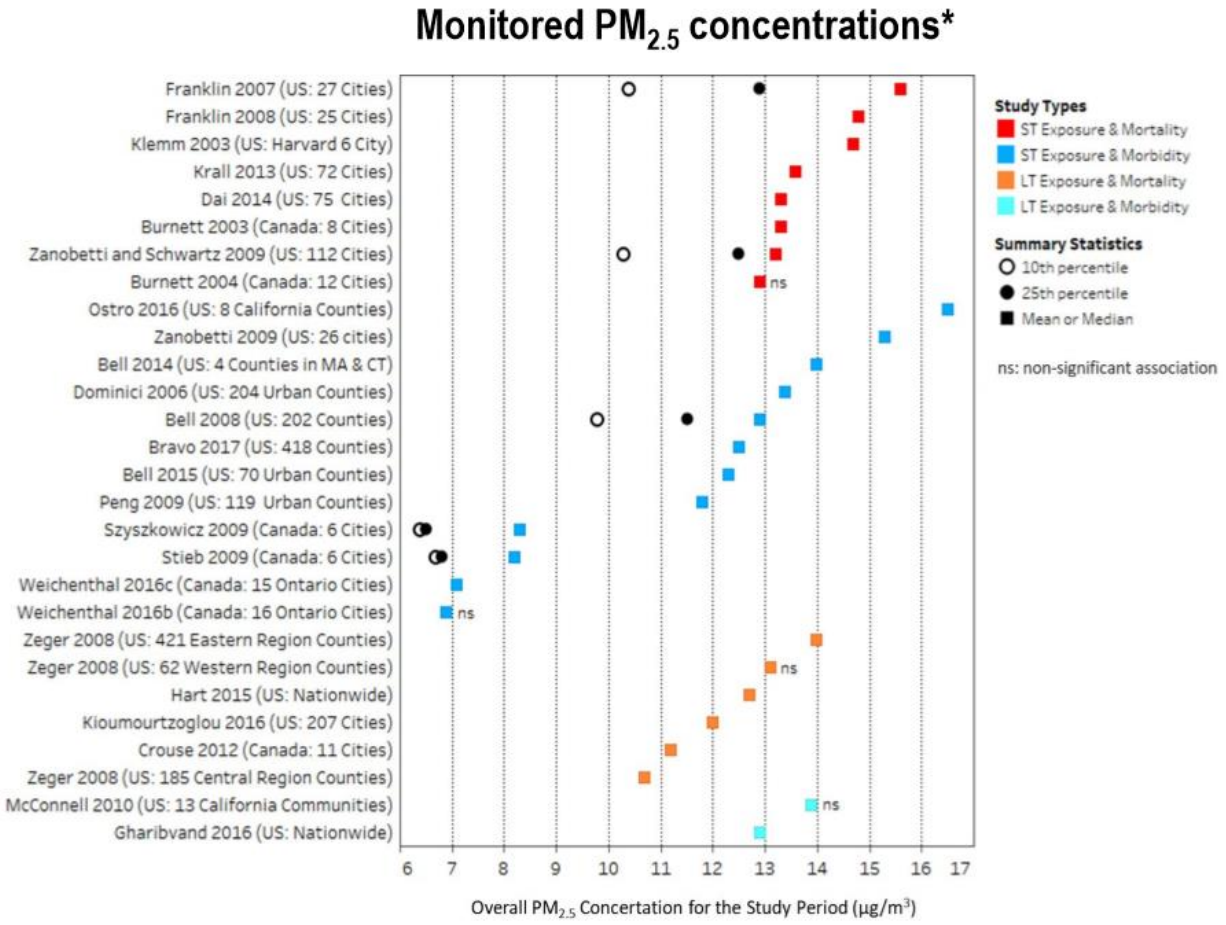
Primary $PM_{2.5}$: Summary of Approach (cont)

24-hour $PM_{2.5}$ standard (98^{th} percentile form)

- Generally viewed as a means of providing protection against the 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 annual average $PM_{2.5}$ concentrations remain relatively low
- Focus is on controlled human exposure studies, which provide evidence for health effects following single, short-term exposures (e.g., 2 hours) to $PM_{2.5}$ concentrations corresponding to the peak of the air quality distribution (e.g., at or above 120 ug/m^3)
- The $PM_{2.5}$ epidemiologic evidence is less informative regarding the health effects that can result following exposures to atypical, peak $PM_{2.5}$ concentrations
- Air quality and risk assessment analyses can inform the relationship between the annual and 24-hr standards

$PM_{2.5}$ Concentrations in Epidemiologic Studies

- Overall mean concentrations reflect study averages of daily or annual $PM_{2.5}$ exposure estimates – bulk of data generally occurs around overall means
- Key studies that consistently report positive and statistically significant associations have overall mean $PM_{2.5}$ concentrations > 8.0 μg/m³
- In studies with data available, 75% of health events occurred in areas with mean $PM_{2.5}$ concentrations ≥ 11.5 μg/m³ (U.S. studies) or 6.5 μg/m³ (Canadian studies)



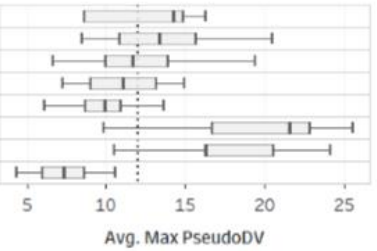
*Colored squares reflect overall study-reported mean (or median) $PM_{2.5}$ concentrations. Circles reflect the mean $PM_{2.5}$ concentrations corresponding to the 25th (filled) and 10th (open) percentiles of health events.

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

- For most key studies, about 25% or more of study area health events/populations were in locations that generally would have met both standards during study periods
- For 9 key studies (of the 29 evaluated), more than 50% of study area health events/populations were in such locations
- For 4 key studies, more than 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 data from currently required near-road monitors

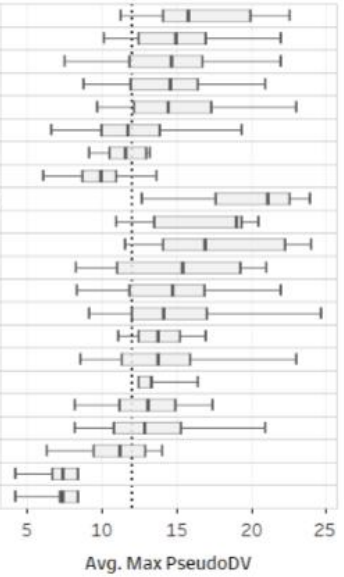
Long-term exposure studies

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

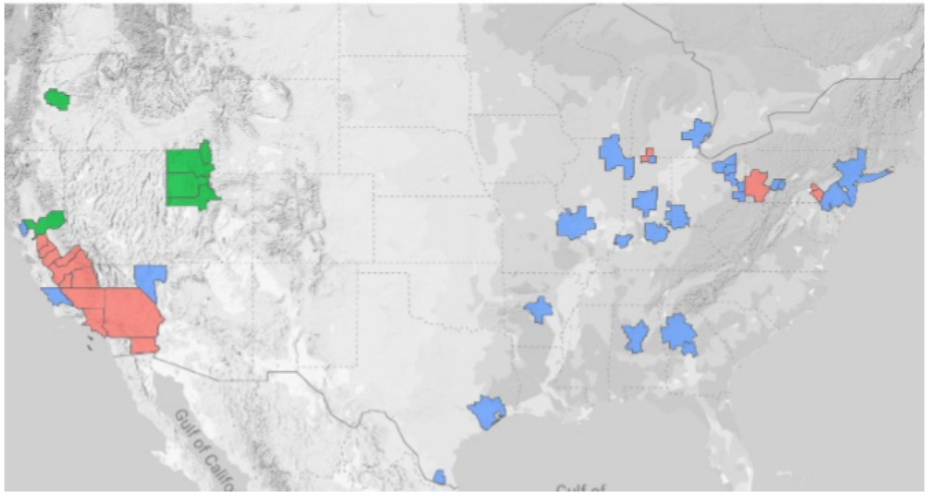


* Whiskers correspond to 5th and 95th percentiles, boxes correspond to 25th and 75th percentiles, central vertical lines correspond to 50th percentiles

$PM_{2.5}$ Risk Assessment – Background and Approach

- To inform conclusions regarding the primary $PM_{2.5}$ standards that are "requisite" to protect the public health, it is important to consider the health risks that would be allowed under those standards
- The risk assessment combines concentration-response functions with $PM_{2.5}$ air quality scenarios of interest, baseline health incidence data, and population demographic information
- The risk assessment evaluates air quality adjusted to simulate "just meeting" the current standards; alternative annual standards with levels of 11.0, 10.0, and 9.0 µg/m^3; and alternative 24-hour standard with a level of 30 µg/m^3 (analysis year is 2015)

In **selecting study areas**, the draft PA focuses on areas with relatively dense ambient monitoring networks; areas that represent a variety of U.S. regions and that include a substantial portion of the U.S. population; and areas for which downward air quality adjustments, or relatively small upward adjustments, are required

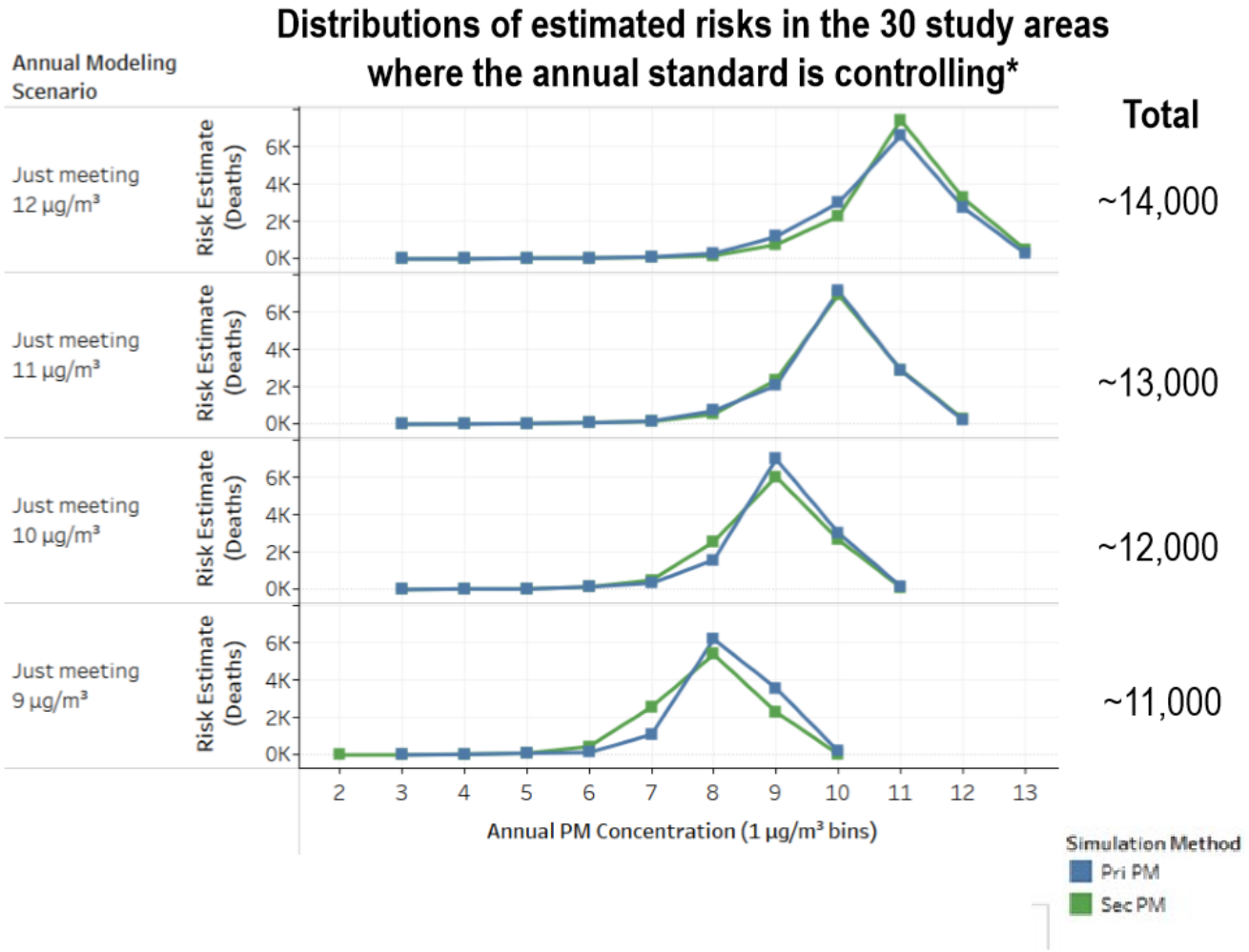


47 urban study areas (population ≥ 30 years: ~60M)
- 30 annual-controlling (population ≥ 30 years: ~50M)
- 11 daily-controlling (population ≥ 30 years: ~4M)
- 6 mixed (population ≥ 30 years: ~5M)

$PM_{2.5}$ Risk Assessment – Background and Approach (Continued)

- Concentration-response functions are from U.S. multicity studies examining total mortality (all-cause and non-accidental), ischemic heart disease mortality, and lung cancer mortality associated with long-term $PM_{2.5}$ exposures and total mortality associated with short-term $PM_{2.5}$ exposures
- Model-based approach to adjusting $PM_{2.5}$ air quality combines CMAQ-modeled surfaces with ambient monitoring data to generate ambient $PM_{2.5}$ estimates for 2015 on a grid with 12-km horizontal resolution
- Two strategies are used to adjusting air quality to the current standards and to potential alternatives with levels of 10.0 $\mu g/m^3$ (annual) and 30 $\mu g/m^3$ (24-hour)
  - Focus on adjusting direct emissions (pri-PM)
  - Focus on adjusting precursor emissions to simulate changes in secondarily formed $PM_{2.5}$ (sec-PM)
- Linear interpolation and extrapolation were used to simulate just meeting additional alternative annual standard levels (9.0 and 11.0 $\mu g/m^3$)

Summary of $PM_{2.5}$ Risk Estimates (Continued)



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 primary $PM_{2.5}$ standards
- Basis for this preliminary conclusion:
  - Long-standing body of health evidence, strengthened in this review, supporting relationships between short- and long-term $PM_{2.5}$ 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:
  - Uncertainty in the biological pathways through which $PM_{2.5}$ exposures could cause serious health effects at typical ambient concentrations, given that experimental studies showing effects generally examine exposures to much higher $PM_{2.5}$ concentrations
  - 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 Level of the Annual $PM_{2.5}$ Standard

- 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 $\mu g/m^3$

Preliminary Conclusions on the Level of the Annual $PM_{2.5}$ Standard (Continued)

- 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 greater weight is placed on the importance of $PM_{2.5}$ health effect associations and estimated risks at lower concentrations, as indicated by the following:
  - The few key studies with overall mean $PM_{2.5}$ concentrations below 8.0 $\mu g/m^3$
  - The ambient $PM_{2.5}$ concentrations somewhat below overall means (e.g., corresponding the lower quartiles) in the broader body of key studies
  - Annual pseudo-design values for the smaller number of key studies conducted in Canada, which tend to be somewhat lower than those in the U.S.
  - Annual pseudo-design values corresponding to 25th percentiles of study area populations or health events for the broader body of key studies
  - The potential public health importance of the additional reductions in $PM_{2.5}$-associated health risks estimated for a level of 9.0 $\mu g/m^3$ and the potential for continued reductions at lower standard levels
- A decision to set the level below 10.0 $\mu g/m^3$ would place less weight on the limitations in the evidence that contribute to greater uncertainty at lower concentrations

Preliminary Conclusions on the Level of the 24-Hour $PM_{2.5}$ Standard

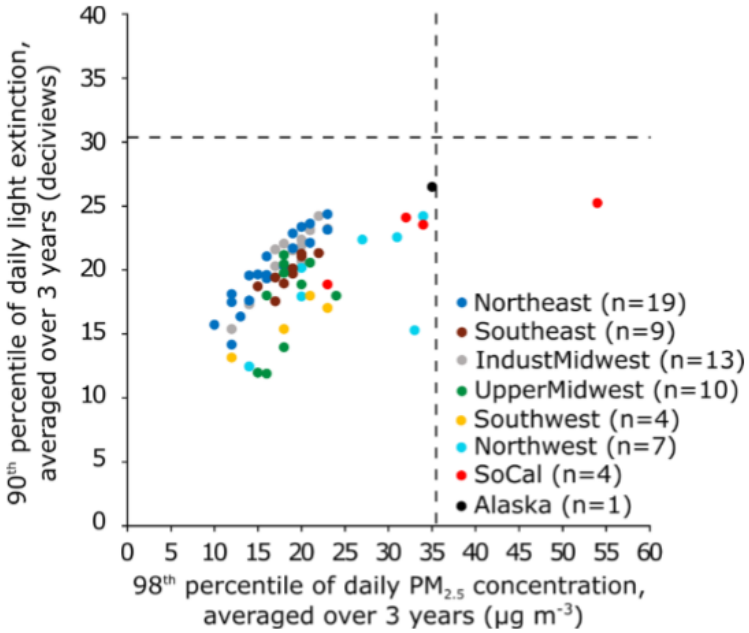
- The evidence provides little support for the need to provide additional protection against short-term peak concentrations in areas meeting the current standards
  - The currently available epidemiologic evidence does not indicate that $PM_{2.5}$ health effect associations are driven disproportionately by peak concentrations
  - Human clinical studies report effects following single short-term $PM_{2.5}$ exposures, but most examine concentrations well-above those typically measured in areas meeting the current standards
- Lowering the level of the 24-hour standard (in conjunction with its current 98th percentile form) could be considered in order to reduce the "typical" short- and long-term $PM_{2.5}$ exposures corresponding to the middle portion of the air quality distribution
- However, compared to lowering the level of the annual standard, there would be greater uncertainty in the effectiveness of using the 24-hour standard to achieve national-scale reductions in typical $PM_{2.5}$ exposures

Primary PM_{10} Standard

- The purpose of the PM_{10} standard is to protect against $PM_{10-2.5}$ exposures – therefore, the draft PA focuses on the evidence for $PM_{10-2.5}$-related health effects
- Recent epidemiologic studies reporting positive associations between $PM_{10-2.5}$ exposures and mortality or morbidity have expanded and strengthened the evidence for some outcome categories
- However, remaining uncertainties result in the draft ISA conclusions that the strongest evidence for $PM_{10-2.5}$-related effects is "suggestive of, but not sufficient to infer, causal relationships"
  - Lack of systematic evaluation/comparison of exposure estimation methods
  - Limited examination of copollutant models, with some showing attenuation
  - Limited experimental evidence to support biological plausibility
- Drawing from this evidence, the draft PA reaches the preliminary conclusions that:
  - While the available evidence supports maintaining a PM_{10} standard to provide some measure of protection against $PM_{10-2.5}$ exposures, uncertainties lead to questions regarding the potential public health implications of revising the existing PM_{10} standard
  - The available evidence does not call into question the adequacy of the public health protection afforded by the current primary PM_{10} standard, and thus, supports consideration of retaining that standard without revision

Secondary PM: Summary of Quantitative Information for Visibility Impairment

- Consistent with the last review, the draft PA evaluates visual air quality in terms of the 3-year visibility metric, based on recent air quality
  - 30 deciviews (dv) is the target protection level identified in the last review based on studies of public preferences of acceptable levels of visibility impairment; there is no new information available in this review regarding public preferences of acceptable levels of visibility impairment
- New information:
  - Recent air quality data (2015-2017)
  - 67 geographically distributed areas
  - Spatially refined relative humidity data
  - Estimated $PM_{2.5}$ light extinction using three versions of the IMPROVE equation
  - Additional coarse PM monitoring data
- Findings are consistent with the last review, in that the 3-year visibility metric was no higher than 30 dv in areas that meet the current 24-hour $PM_{2.5}$ standard (average of 20 dv across 67 sites)



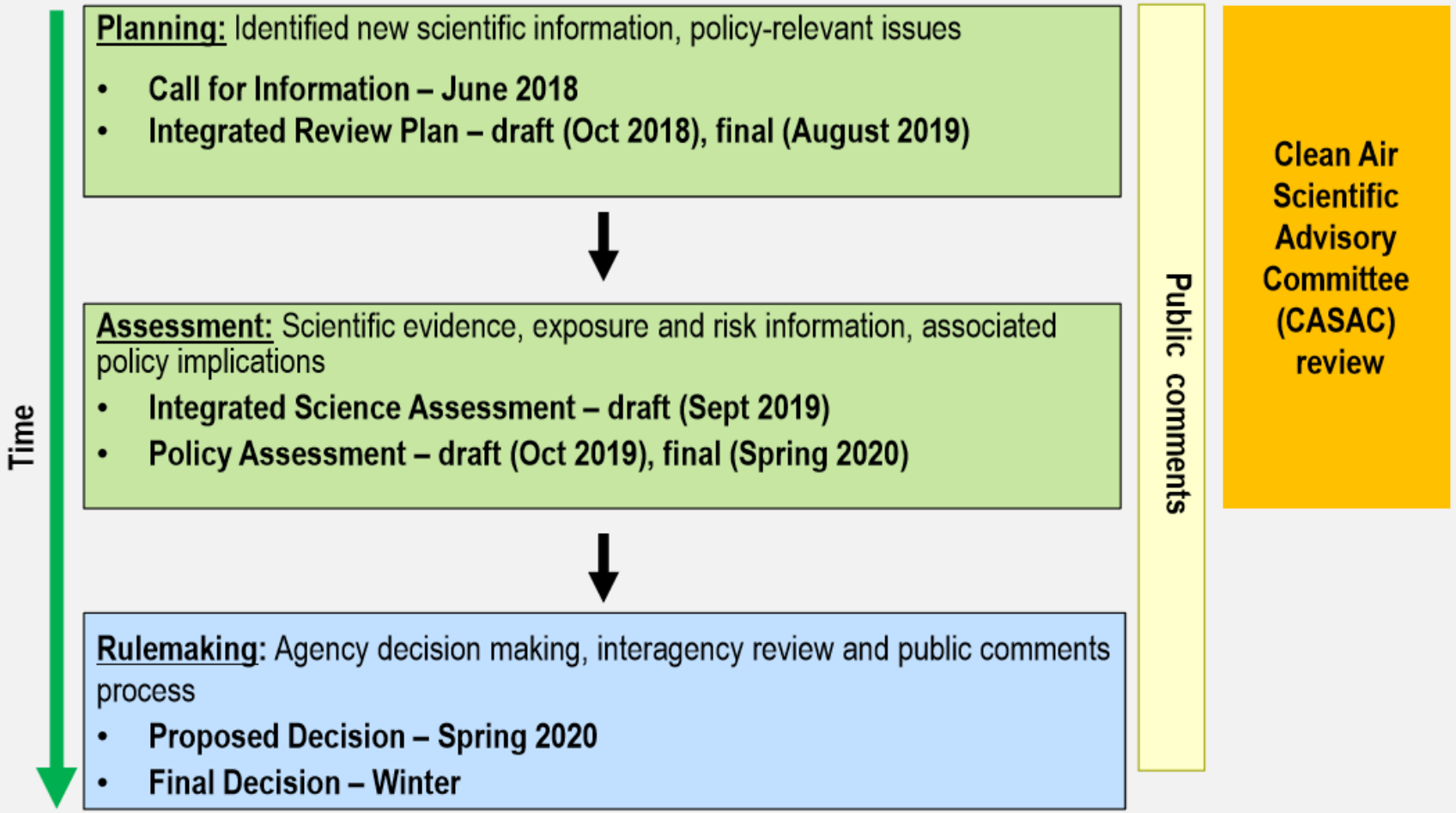
Note: For the figure above, light extinction was calculated using the original IMPROVE equation, consistent with the methods used in the last review

Secondary PM: Preliminary Conclusions

- Scientific evidence for PM-related visibility impairment, climate effects, and materials effects that is newly available in this review is consistent with evidence base in last review, including uncertainties associated with that evidence
- Quantitative analyses for visibility impairment suggest that those areas meeting the current secondary 24-hour $PM_{2.5}$ standard are also meeting the target level of protection (i.e. 30 dv)
- Drawing from this information, the draft PA reaches the preliminary conclusion that the available evidence and quantitative information, including uncertainties, do not call into question the adequacy of protection provided by the current secondary PM standards, and thus, support consideration of retaining the current secondary standards, without revision

OZONE NAAQS REVIEW

Process and Schedule for this Review of the Ozone NAAQS



OZONE ISA REVIEW

EPA
United States
Environmental Protection
Agency

U.S. Environmental Protection Agency Clean Air Scientific Advisory Committee (CASAC) Public Meeting

Review of the Integrated Science Assessment for Ozone

(External Review Draft)

Center for Public Health and Environmental Assessment

Office of Research and Development

December 4, 2019

EPA United States Environmental Protection Agency

Purpose and Contents of ISA

- **Purpose:** To identify, evaluate, and communicate the scientific information representing the "air quality criteria" per Section 108; Make causality determinations for health and welfare effects; Serves as the scientific foundation for the NAAQS
- **Contents of the Ozone ISA:**

  Preface: Legislative Requirements, History

  Executive Summary

  Integrated Synthesis

  Appendix 1: Atmospheric Source, Chemistry, Meteorology, Trends, and Background Ozone

  Appendix 2: Exposure to Ambient Ozone

  Appendix 3-7: Health Effects- Respiratory, Cardiovascular, Metabolic, Mortality, Other Endpoints

  Appendix 8-9: Welfare Effects- Ecological, Climate

  Appendix 10: Process

EPA United States Environmental Protection Agency

Summary
Causality Determinations - Health

Health Effects		
Short-term Exposure		
	2013 Ozone ISA	Current Ozone ISA
Respiratory Effects	Causal	Causal
Metabolic Effects	No Causality Determination	Likely to be Causal*
Cardiovascular Effects	Likely to be Causal	Suggestive of, but not sufficient to infer
Nervous System Effects	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Mortality	Likely to be Causal	Suggestive of, but not sufficient to infer
Long-term Exposure		
Respiratory Effects	Likely to be Causal	Likely to be Causal
Metabolic Effects	No Causality Determination	Likely to be Causal*
Cardiovascular Effects	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Nervous System Effects	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Reproductive Effects – Fertility and Reproduction	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer
Reproductive Effects – Pregnancy and Birth Outcomes		Suggestive of, but not sufficient to infer
Cancer	Inadequate	Inadequate
Mortality	Suggestive of, but not sufficient to infer	Suggestive of, but not sufficient to infer

Red text = new determination or change in causality determination from 2013 Ozone ISA
* New Causality Determination

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Appendix 3: Respiratory Effects and Short-term Ozone Exposure

Recent evidence supports and extends the conclusions of the 2013 Ozone ISA that there is a causal relationship between short-term ozone exposure and respiratory effects.

- Evidence spanning decades from Controlled Human Exposure, Epidemiologic and Animal Toxicological studies
  - Controlled Human Exposure Studies: Well-established endpoints showing ozone-induced effects at 60-70 ppb and higher (e.g., lung function decrements, respiratory symptoms, inflammation)
  - Epidemiologic Studies: Panel studies and emergency department visit/hospital admission studies at ambient ozone concentrations
  - Animal Toxicological Studies: Large body of evidence demonstrates ozone-induced changes in lung function measures, inflammation, increased airway responsiveness, and impaired lung host defense

Appendix 3: Respiratory Effects and Short-term Ozone Exposure (Cont.)

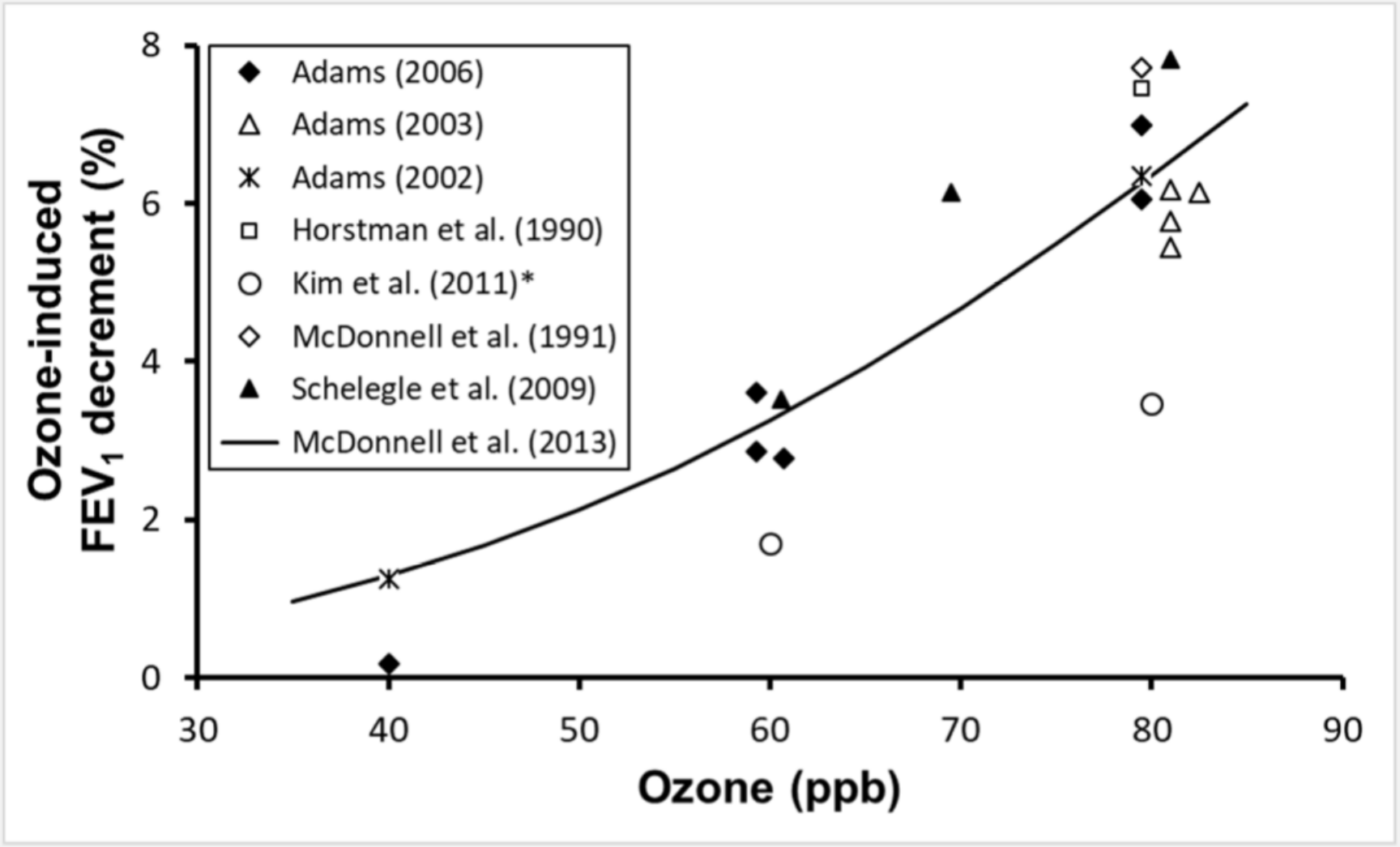

Fig IS.4-1 **Cross-study comparisons of mean ozone-induced forced expiratory volume in one second (FEV_1) decrements in young healthy adults following 6.6 hours of exposure to ozone.**

Appendix 6: Mortality and Short-term Ozone Exposure

Recent evidence changes the causality determination from a likely to be causal relationship (2013 Ozone ISA) to a suggestive of, but not sufficient to infer, a causal relationship between short-term ozone exposure and mortality.

- Limited evidence for a biologically plausible mechanism by which ozone exposure could lead to mortality given the limited evidence for cardiovascular morbidity
- Limited coherence with controlled human exposure and epidemiologic studies of subclinical cardiovascular effects and cardiovascular morbidity
- Consistent, positive associations between short-term ozone exposure and total mortality reported in U.S. and Canadian epidemiologic studies

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Summary
Causality Determinations - Welfare

Ecological Effects		
	2013 Ozone ISA	Current Ozone ISA
Visible Foliar Injury	Causal	Causal
Reduced Vegetation Growth	Causal	Causal
Reduced Plant Reproduction	No separate causality determination; included with plant growth	Causal
Increased Tree Mortality	No Causality Determination	Likely to be Causal
Reduced Crop Yield	Causal	Causal
Altered Herbivore Growth and Reproduction	No Causality Determination	Likely to be Causal
Altered Plant-Insect Signaling	No Causality Determination	Likely to be Causal
Reduced Carbon Sequestration	Likely to be Causal	Likely to be Causal
Reduced Productivity	Causal	Causal
Alterations of Below-ground Biogeochemistry	Causal	Causal
Alteration of Terrestrial Community Composition	Likely to be Causal	Causal
Alteration of Ecosystem Water Cycling	Likely to be Causal	Likely to be Causal
Effects on Climate		
	2013 Ozone ISA	Current Ozone ISA
Radiative Forcing	Causal	Causal
Temperature, Precipitation and Climate-related Variables*	Likely to be Causal	Likely to be Causal

Red text = new determination or change in causality determination from 2013 Ozone ISA
*Referred to as "Climate Change" in the 2013 Ozone ISA

OZONE PA REVIEW

EPA
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Policy Assessment for the Review of the Ozone National Ambient Air Quality Standard

External Review Draft

Staff from the
Office of Air Quality Planning and Standards
U.S. Environmental Protection Agency

December 5-6, 2019
Clean Air Scientific Advisory Committee

Primary Standard: Overview of Health Effects Evidence

- The health effects evidence continues to be strongest for respiratory effects
  - Causal relationship between short-term O_3 exposure and respiratory effects, likely causal relationship* for such effects with longer-term exposure
    - Strongest evidence comes from controlled human exposure studies, with epidemiologic studies also reporting associations between short-term O_3 and respiratory hospital admissions and emergency department visits (and other respiratory health outcomes)
  - Key effects in controlled human exposure studies of healthy adults, exposed during exercise, are lung function decrements and respiratory symptoms
    - Statistically significant findings for both endpoints for 6.6-hour exposures (5 hours of exercise) at and above 70 ppb, and statistically significant decrements at 60 ppb
    - Studies of 6.6-hour exposures at/above 80 ppb document greater lung function decrements and respiratory symptom scores, and also other respiratory response indicators
  - At-risk populations include people with asthma, children, as well as outdoor workers
    - ~8% of U.S. population has asthma, with much higher rates in some population groups
  - Uncertainties still remain from the last review regarding the population groups that may be at greatest risk and the extent of effects at low concentrations

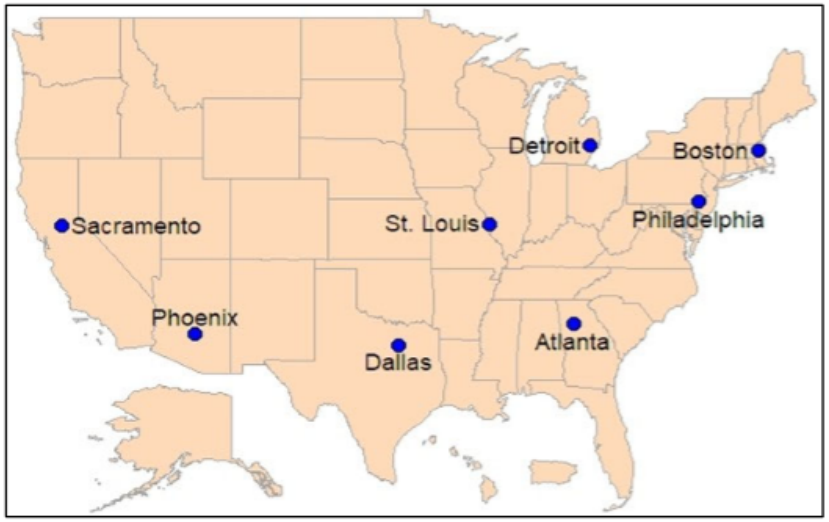
*The draft ISA also concludes there to be likely causal relationships for short- and long-term O_3 with metabolic effects.

Primary Standard: Exposure and Risk Analysis - Features of Study Areas

- Study Area Selection Criteria (PA, section 3D.2.1)
  - Have at least 10 ambient air O_3 monitors for the 2015-2017 period;
  - Combined statistical area (CSA)/metropolitan statistical area (MSA) ambient air monitor design values between 60-80 ppb
  - CSA/MSA population between 2 to 10 million;
  - Anticipated reasonable air quality model performance; and
  - Reasonable geographic distribution across continental U.S.

Modified from Draft PA, Appendix 3D, Table 3D-1. Study area features.

Study Area	U.S. Climate Region	CSA/MSA Population (millions)	Ambient Air Monitors (n)	Design Values (ppb)	
				2017	2008, 2010
Atlanta	Southeast	6.6	11	75	95, 80
Boston	Northeast	8.3	22	73	82, 76
Dallas	South	8.0	20	79	91, 86
Detroit	Upper Midwest	5.4	11	73	82, 75
Philadelphia	Northeast	7.2	19	80	92, 83
Phoenix	Southwest	4.9	28	76	81, 77
Sacramento	West	2.6	18	86	99, 99
St. Louis	Ohio Valley	2.9	12	72	82, 77

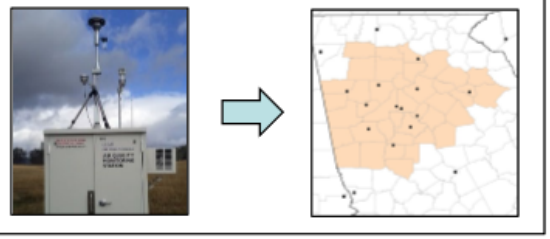


Draft PA, Appendix 3D, Figure 3D-1. Location of eight study areas.

Primary Standard: Exposure and Risk Analysis - Ambient Air Concentrations

- Objectives
  - Address fine-scale temporal and spatial variability in ambient air O_3 concentrations
  - Reflect specific air quality scenarios
- Approach to estimating concentrations (e.g., for scenario just meeting current standard)
  - Ambient air monitoring data (PA, section 3C.3)
    - O_3 measurements provide fine-scale temporal (hourly) and broad spatial variability
  - Air quality modeling (PA, section 3C.4 and 3C.5)
    - Hourly concentrations observed at monitor sites adjusted with spatially/temporally varying model-based factors such that highest study area DV met air quality scenario target
  - Spatial Interpolation (PA, section 3C.6)
    - Inverse distance weighting using nearest neighbor monitors to estimate O_3 concentrations for fine-scale (census tract) spatial variability

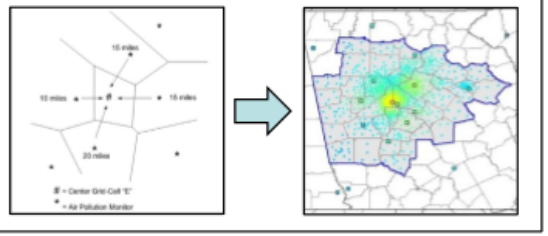
Ambient Air Monitor Data [1]



Air Quality Modeling [2]

Urban Area	75 ppb	70 ppb	65 ppb
Atlanta	0%	25%	44%
Boston	+7%	14%	40%
Dallas	15%	32%	45%
Detroit	+18%	21%	47%
Philadelphia	23%	43%	53%
Phoenix	14%	49%	68%
Sacramento	45%	58%	72%
Saint Louis	+11%	13%	38%

Spatial Interpolation [3]



[1] Draft PA, Appendix 3C, Figure 3C-3. Map of the Atlanta study area monitoring sites. (as an example)
[2] Draft PA, Appendix 3C, Table 3C-19. Percent NOx emissions changes used for each urban area to just meet each of the air quality scenarios evaluated.
[3] Draft PA, Appendix 3C, Figure 3C-91. Annual 4th highest MDA8 O_3 based on HDDM adjustments in Atlanta. (70 ppb as an example)

Primary Standard: Exposure and Risk Analysis - Estimating Exposure

- Approach uses Air Pollution Exposure Model (APEX) (PA, section 3D.2)
  - Population-based human inhalation exposure and risk model that links fine spatial and temporal scale ambient air O_3 concentrations with study area population demographics, human activity data, and physiological attributes of study populations
  - Estimates the complete time-series of O_3 exposures and simultaneously occurring breathing rates for simulated individuals as they perform activities within the microenvironment they visit
    - This is key to both the exposure and risk estimation because the adverse health effect depends on the exposed individuals having an elevated ventilation rate
- Outputs (PA, section 3D.2.7)
  - Counts of simulated people experiencing O_3 exposures at selected levels and at particular exertion rates of interest
  - Complete time-series of O_3 exposures (and ventilation rates) for simulated individuals (minute-by-minute, hourly, daily etc.)

Primary Standard: Exposure & Risk Analysis – Risk Estimates

- Comparison to Benchmarks - current standard (PA, section 3D.3.2.1)
  - % of children with asthma experiencing a day with 7-hour exposure at/above benchmark, while at elevated exertion
    - 80 ppb benchmark: At most, 0.1% in any year in any study area (zero children estimated to experience more than one day)
    - 70 ppb benchmark: At most, 1% in any year in any study area (0.1% estimated to experience more than one day)
    - 60 ppb benchmark: Less than 9%, on average across years and study areas (less than 5% estimated to experience more than one day)
- Lung Function Risk – current standard, via E-R function approach (PA, section 3D.3.3)
  - % of children with asthma experiencing a day with a FEV_1 reduction of at least:
    - 20% Decrement: At most, 0.4% in any year in any study area (0.2% estimated to experience more than a day)
    - 15% Decrement: At most, 1% in any year in any study area (0.6% estimated to experience more than a day)
    - 10% Decrement: At most 3.3%, on average across years and study areas (<3% estimated to experience more than a day)
  - Higher estimates using the MSS model, with increased uncertainty

Primary Standard: Preliminary Conclusions

- Health effects evidence newly available in this review is generally consistent with evidence base in last review.
- Exposure and risk estimates for air quality conditions just meeting the current standard generally reflect the ranges of estimated exposures and risks from the last review.
- Preliminary PA conclusion is that the available evidence and quantitative information, including uncertainties, do not call into question the adequacy of protection provided by the current standard, and thus, support consideration of retaining the current standard, without revision.
- Accordingly, the draft PA does not identify alternative standards for further evaluation.

Secondary Standard: Preliminary Conclusions

- Welfare effects evidence is generally consistent with evidence base in last review.
  - Growth-related effects: Exposure estimates for air quality conditions meeting the current standard virtually all at/below 19 ppm-hrs (the W126 index associated with 6% RBL for median species).
    - Focus on RBL as surrogate for other vegetation-related effects continues to be supported by the current information as approach for judging adequacy of protection provided by the current standard
  - Visible foliar injury: Current evidence does not indicate the occurrence of elevated severity or extensive leaf damage in areas that meet current standard
  - Climate effects: Evidence does not support climate risk estimation for O_3 concentrations that meet current standard.
- Preliminary conclusion is that the available evidence and quantitative information, including uncertainties, do not call into question the adequacy of protection provided by the current standard, and thus, support consideration of retaining the current standard, without revision.
  - Accordingly, the draft PA does not identify alternative standards for further evaluation.

ADDITIONAL INFORMATION

https://yosemite.epa.gov/sab/sabpeople.nsf/webcommittees/CASAC

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EPA Clean Air Scientific Advisory Committee (CASAC)

The Clean Air Scientific Advisory Committee (CASAC) provides independent advice to the EPA Administrator on the technical bases for EPA's National Ambient Air Quality Standards.

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Recent additions and CASAC Recent Happenings *More about RSS news feeds (USA.gov)*

Current Activities

- Oxides of Nitrogen, Oxides of Sulfur, and Particulate Matter Integrated Science Assessment - Ecological Criteria (Second External Review Draft)
- Oxides of Nitrogen, Oxides of Sulfur, and Particulate Matter Risk and Exposure Assessment Planning Document for Secondary (Welfare-based) National Ambient Air Quality Standards (NAAQS)
- Ozone Integrated Science Assessment (2019)
- Ozone Policy Assessment (2019)
- Particulate Matter Policy Assessment for the National Ambient Air Quality Standards (NAAQS)

Upcoming and Recent Meetings

- 12/03/2019 - 12/06/2019 Public Meeting of the Chartered Clean Air Scientific Advisory Committee (CASAC) on Particulate Matter and Ozone

More Meetings

Meeting

Public Meeting of the Chartered Clean Air Scientific Advisory Committee (CASAC) on Particulate Matter and Ozone

12/03/2019 to 12/06/2019

Federal Register Notice Announcing the Meeting

Type of Meeting	Advisory
Location	Embassy Suites by Hilton Raleigh Durham Research Triangle, 201 Harrison Oaks Boulevard, Cary, North Carolina, 27513
Expert Committee or Panel	CASAC
Contacts	Aaron Yeow 202-564-2050 yeow.aaron@epa.gov

Agenda

To discuss CASAC's Draft Report on EPA's Policy Assessment for Particulate Matter, to peer review EPA's Integrated Science Assessment for Ozone and Related Photochemical Oxidants (External Review Draft – September 2019), and to peer review EPA's Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards (External Review Draft – October 2019)

Agenda. (PDF, 4 pp., 199,064 bytes)

Advisory Activity (or Activities) Discussed

Particulate Matter Policy Assessment for the National Ambient Air Quality Standards (NAAQS)

Ozone Integrated Science Assessment (2019)

Meeting Materials

Disclaimer Although not required to do so, EPA generally posts public comments submitted to the SAB, CASAC or Council and their subcommittees on the internet to make them easily available to the public. Posting of public comments is not an Agency endorsement of, or agreement with, any information or viewpoints presented in the public comment, nor is it an Agency endorsement of the quality or correctness of such information and viewpoints. In addition, mention of any trade names or commercial products in posted meeting material does not constitute a recommendation by EPA or the SAB for use.

Category	Meeting Material
Agency Briefing Material	EPA Presentation - Policy Assessment for the Review of the Ozone National Ambient Air Quality Standard. (PDF, 33 pp., 700,988 bytes)
Agency Briefing Material	EPA Presentation - Review of the Integrated Science Assessment for Ozone. (PDF, 56 pp., 5,591,887 bytes)
Committee Members' Comments	11-27-19 Preliminary CASAC Member Comments on the Ozone ISA. (PDF, 100 pp., 1,135,242 bytes)
Committee Members' Comments	11-27-19 Preliminary CASAC Member Comments on the Ozone PA. (PDF, 51 pp., 1,177,036 bytes)
Committee Members' Comments	12-2-19 Preliminary Ozone ISA Comments from Dr. James Boylan. (PDF, 5 pp., 338,545 bytes)
Committee Members' Comments	12-4-19 Preliminary Ozone PA Comments from Dr. James Boylan. (PDF, 4 pp., 189,454 bytes)
Committee Members' Questions for Non-member Consultants	Ozone ISA Questions for Consultants from Dr. Corey Masuca. (PDF, 2 pp., 118,360 bytes)
Committee Members' Questions for Non-member Consultants	Ozone ISA Questions for Consultants from Dr. James Boylan. (PDF, 1 pp., 110,816 bytes)
Committee Members' Questions for Non-member Consultants	Ozone ISA Questions for Consultants from Dr. Mark Frampton. (PDF, 2 pp., 117,511 bytes)
Committee Members' Questions for Non-member Consultants	Ozone ISA Questions for Consultants from Dr. Sabine Lange. (PDF, 2 pp., 153,874 bytes)
Committee Members' Questions for Non-member Consultants	Ozone ISA Questions for Consultants from Dr. Steven Packham. (PDF, 8 pp., 443,168 bytes)
Committee Members' Questions for Non-member Consultants	Ozone ISA Questions for Consultants from Dr. Tony Cox. (PDF, 10 pp., 273,661 bytes)
Committee Members' Questions for Non-member Consultants	Ozone PA Questions for Consultants from Dr. Corey Masuca. (PDF, 2 pp., 136,025 bytes)
Committee Members' Questions for Non-member Consultants	Ozone PA Questions for Consultants from Dr. James Boylan. (PDF, 1 pp., 118,497 bytes)
Committee Members' Questions for Non-member Consultants	Ozone PA Questions for Consultants from Dr. Sabine Lange. (PDF, 2 pp., 225,571 bytes)
List of public speakers	List of Registered Public Speakers. (PDF, 2 pp., 113,358 bytes)
Non-member Consultants' Responses to Committee Members' Questions	Responses to CASAC Questions on the Ozone ISA from Dr. Dan Jaffe. (PDF, 5 pp., 140,203 bytes)
Non-member Consultants' Responses to Committee Members' Questions	Responses to CASAC Questions on the Ozone ISA from Dr. David Parrish. (PDF, 17 pp., 1,570,096 bytes)
Non-member Consultants' Responses	Responses to CASAC Questions on the Ozone ISA from Dr. Duncan Thomas

OZONE PUBLIC SPEAKERS

Public Comment Period - December 4, 2019, 8:30 am
on the Ozone ISA

#	Speaker's Name	Organizational Affiliation(s)
1	Gretchen Goldman	Union of Concerned Scientists
2	Julie Goodman	Gradient
3	Chris Frey	North Carolina State University
4	David G. Hill*	American Lung Association
5	Gary Ewart*	American Thoracic Society
6	Jennifer Richmond-Bryant	North Carolina State University
7	Randy Mandel*	Ramboll
8	Rashid Shaikh*	Health Effects Institute
9	John Dale Dunn*	Heartland Institute of Chicago
10	Bob Paine*	AECOM
11	Stewart Holm*	American Forest & Paper Association

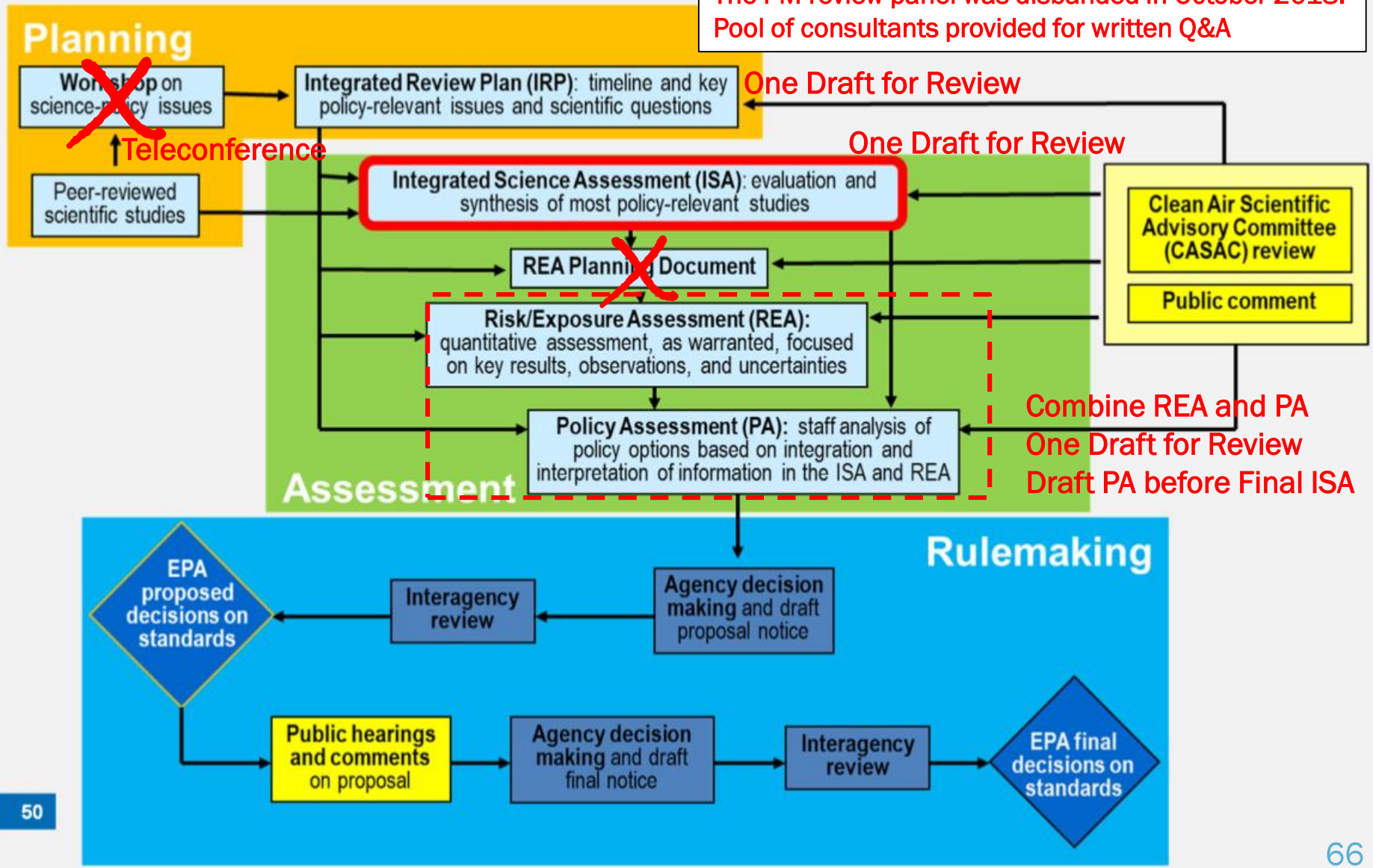
Public Comment Period - December 5, 2019, 1:00 pm
on the Ozone PA

#	Speaker's Name	Organizational Affiliation(s)
1	Gretchen Goldman	Union of Concerned Scientists
2	Julie Goodman*	Gradient
3	Chris Frey	North Carolina State University
4	Albert Rizzo*	American Lung Association
5	James Enstrom*	UCLA (retired) and Scientific Integrity Institute
6	Anne Smith*	NERA Economic Consulting
7	Gary Ewart*	American Thoracic Society
8	Chad Whiteman*	U.S. Chamber of Commerce
9	John Bachmann	None
10	Courtney Taylor*	Ramboll
11	John Dale Dunn*	Heartland Institute of Chicago
12	David Heinold*	AECOM
13	Daren Bakst*	The Heritage Foundation

Streamline NAAQS Review Process

Overview of the NAAQS Review Process

No ozone review panel was formed.
The PM review panel was disbanded in October 2018.
Pool of consultants provided for written Q&A



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