Texas Commission on Environmental Quality

INTEROFFICE MEMORANDUM

To: Commissioners' Work Session **Date:** May 23, 2012

Thru: Bridget C. Bohac, Chief Clerk

From: Susana M. Hildebrand, P.E., Chief Engineer

Subject: EPA's Cost Benefit Analysis

At the June 1, 2012 Commissioners' Work Session, a discussion will be presented of the analysis of the methods and underlying assumptions the Environmental Protection Agency employs in its estimation of the costs and benefits of its regulations.

The presentation will incorporate the attached slides and will include information from the attached literature.

TCEQ

EPA's Benefit Cost Analysis

Susana Hildebrand, P.E., Chief Engineer
Michael Honeycutt, Ph.D.
Stephanie Shirley, Ph.D.

Background

- March 2011 – EPA published "Benefits and Costs of the Clean Air Act from 1990 to 2020 (Second Prospective Study)"
  - Benefits ($2T) outweigh costs ($65B) by 30 to 1
    - TCEQ staff examined this analysis, focusing on:
      - The studies used
      - The assumptions made
      - The methods employed

Regulatory Impact Analyses

- President requires RIAs (Regulatory Impact Analyses) from all agencies proposing major regulations

- RIA should help determine if the benefits of an action are likely and justify the costs or discover which of various possible alternatives would be the most cost-effective
  - (OMB circular A4, 09/2003)

- RIAs are NOT subject to peer or public review

Key legislation – Executive Orders

- EO12291 – Reagan, 1981
  - "Regulatory action shall not be undertaken unless the potential benefits to society for the regulation outweigh the potential costs to society…the alternative involving the least net cost to society shall be chosen"
- EO12866 – Clinton, 1993
  - Key change: benefits must justify the costs
- EO13563 – Obama, 2011
  - Benefits must justify the costs
  - New: equity, human dignity, fairness and distributive impacts are required to be considered
  - "Our regulatory system must protect public health, welfare, safety, and our environment while promoting economic growth, innovation, competitiveness, and job creation"

Use of $PM_{2.5}$ in RIAs

- EPA uses estimates of benefits from reducing $PM_{2.5}$ in its RIAs for rulemakings under the Clean Air Act
  - This is called "co-benefits" because a $PM_{2.5}$ reduction is expected from efforts to reduce other air pollutants
- Trend towards using $PM_{2.5}$ as primary source of benefits in most RIAs since 1997
  - Even when regulation is not intended to protect public health from exposures to ambient $PM_{2.5}$

Table 2. Summary of Degree of Reliance on $PM_{2.5}$-Related Co-Benefits in RIAs Since 1997 for Major Non-$PM_{2.5}$ Rulemakings under the CAA
(RIAs with no quantified benefits at all are not in this table. Where ranges of benefit and/or cost estimates are provided, percentages are based on upper bound of both the benefits and cost estimates. Estimates using the 7% discount rates are used in all cases.)

Year	RIAs for Rules NOT Based on Legal Authority to Regulate Ambient $PM_{2.5}$	$PM_{2.5}$ Co-Benefits Are >50% of Total	$PM_{2.5}$ Co-Benefits Are Only Benefits Quantified
1997	Ozone NAAQS (.12 1hr=>.08 8hr)	×	
1997	Pulp&Paper NESHAP		
1998	NOx SIP Call & Section 126 Petitions		
1999	Regional Haze Rule	×	
1999	Final Section 126 Petition Rule	×	
2004	Stationary Reciprocating Internal Combustion Engine	×	
2004	Industrial Boilers & Process Heaters NESHAP	×	×
2005	Clean Air Mercury Rule	×	
2005	Clean Air Visibility Rule/BART Guidelines	×	
2006	Stationary Compression Ignition Internal Combustion		
2007	Control of HAP from mobile sources	×	×
2008	Ozone NAAQS (.08 8hr =>.075 8hr)	×	
2008	Lead (Pb) NAAQS	×	
2009	New Marine Compress'n-Ign Engines >30 L per	×	
2010	Reciprocating Internal Combustion Engines NESHAP	×	×
2010	EPA/NHTSA Joint Light-Duty GHG & CAFES		
2010	SO2 NAAQS (1-hr, 75 ppb)	×	> 99.9%
2010	Existing Stationary Compression Ignition Engines	×	×
2011	Industrial, Comm, and Institutional Boilers NESHAP	×	×
2011	Indus'l, Comm'l, and Institutional Boilers & Process	×	×
2011	Comm'l & Indus'l Solid Waste Incin. Units NSPS &	×	×
2011	Control of GHG from Medium & Heavy-Duty		
2011	Ozone Reconsideration NAAQS	×	
2011	Utility Boiler MACT NESHAP (Final Rule's RIA)	×	≥ 99%
2011	Mercury Cell Chlor Alkali Plant Mercury Emissions	×	
2011	Sewage Sludge Incineration Units NSPS & Emission	×	×
2011	Ferroalloys Production NESHAP Amendments	×	×

2009
Change in Methodology

From Smith, 2012 testimony

Key Changes in $PM_{2.5}$ Methodology

- The Benefits and Costs of the Clean Air Act from 1990 to 2020 (March 2011)

  1. A no-threshold model for $PM_{2.5}$ that calculates incremental benefits down to the lowest modeled air quality levels

  2. Risks attributed to very low (background) levels of ambient $PM_{2.5}$

  3. Assumption of causal relationship between $PM_{2.5}$ and mortality

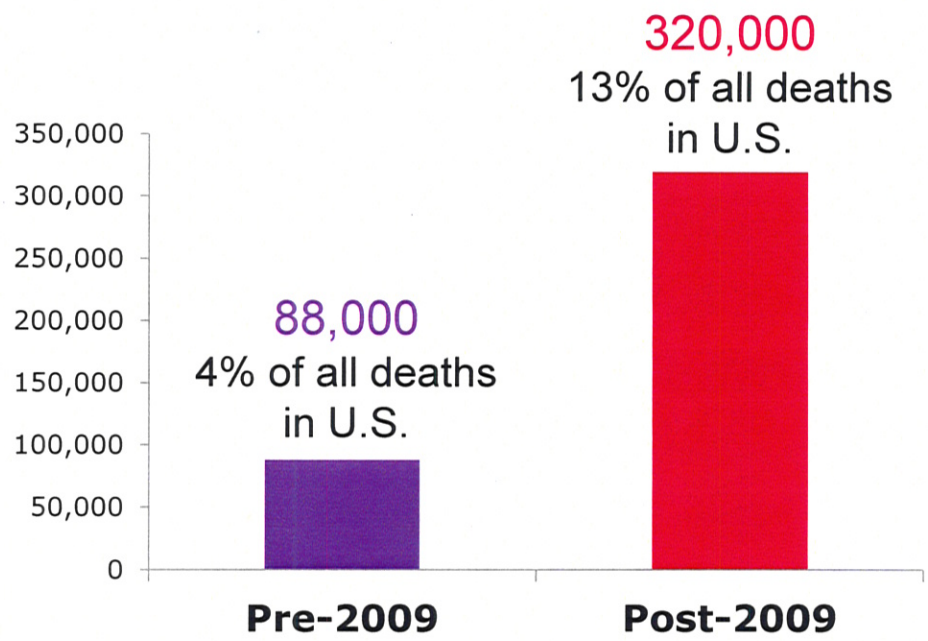
  4. A Value of Statistical Life (VSL)

Result of Key Changes in $PM_{2.5}$ Methodology

Change in deaths attributable to $PM_{2.5}$

Increased estimates of benefits

Number of Deaths due to $PM_{2.5}$ in 2005



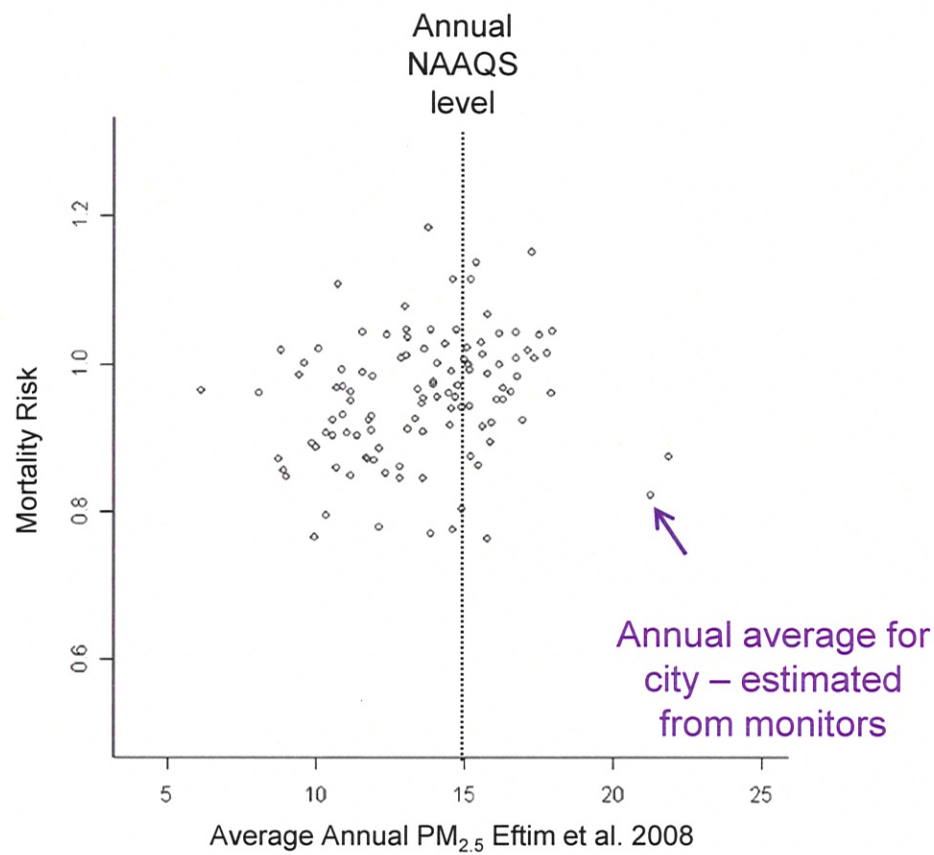
Source: EPA 2010 Quantitative Health Risk Assessment for $PM_{2.5}$ Table G-1

Despite improvement in air quality since the CAAA

1. No Threshold Model

- A no-threshold model for $PM_{2.5}$ that calculates incremental benefits down to the lowest modeled air quality levels

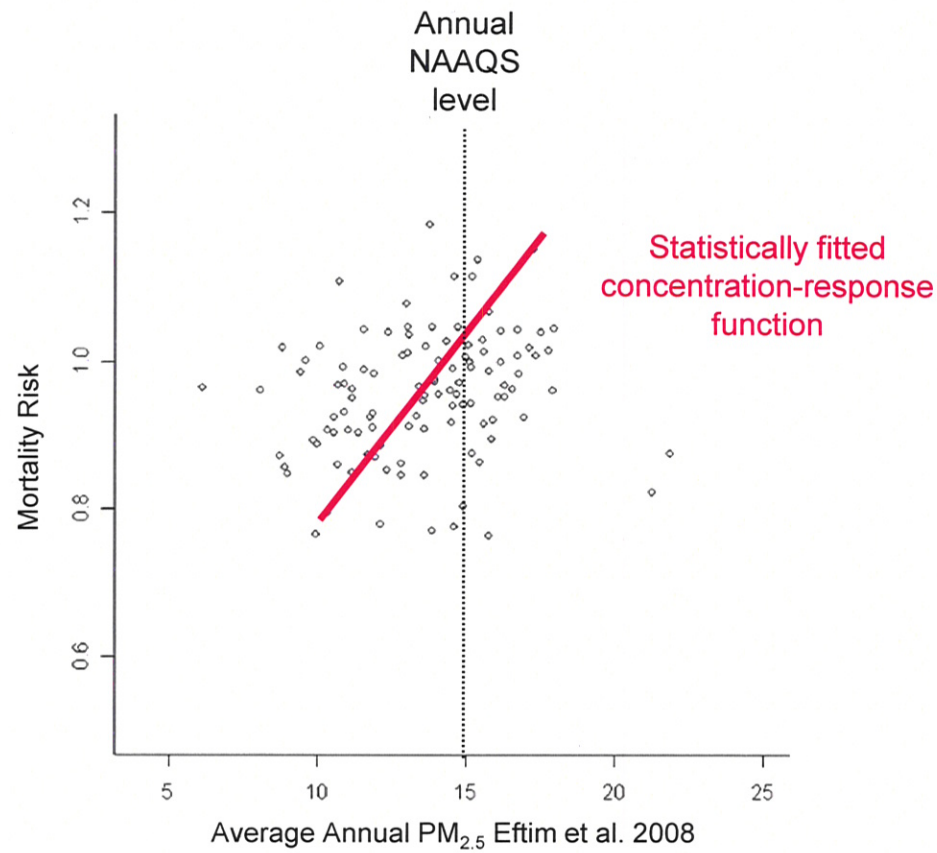
Based on death certificates collected in the city



Adapted from Smith 2011

1. No Threshold Model

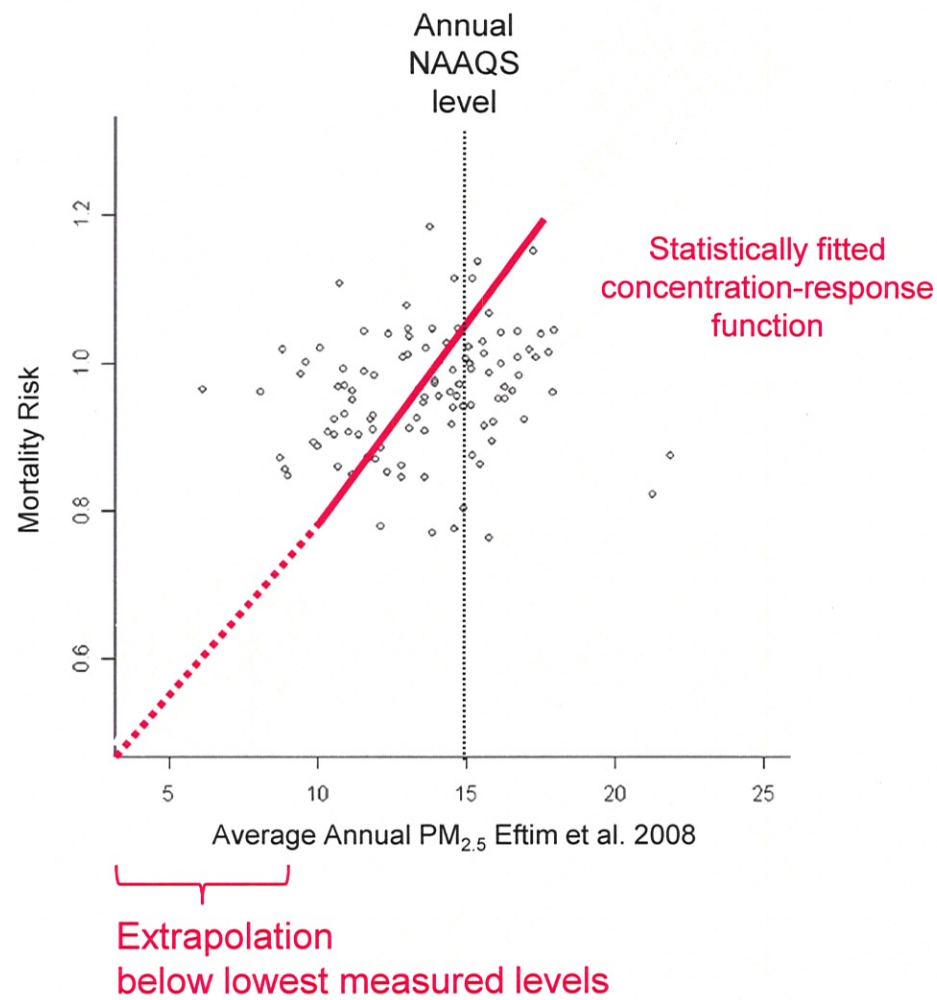
- A no-threshold model for $PM_{2.5}$ that calculates incremental benefits down to the lowest modeled air quality levels



Adapted from
Smith 2011

1. No Threshold Model

- A no-threshold model for $PM_{2.5}$ that calculates incremental benefits down to the lowest modeled air quality levels



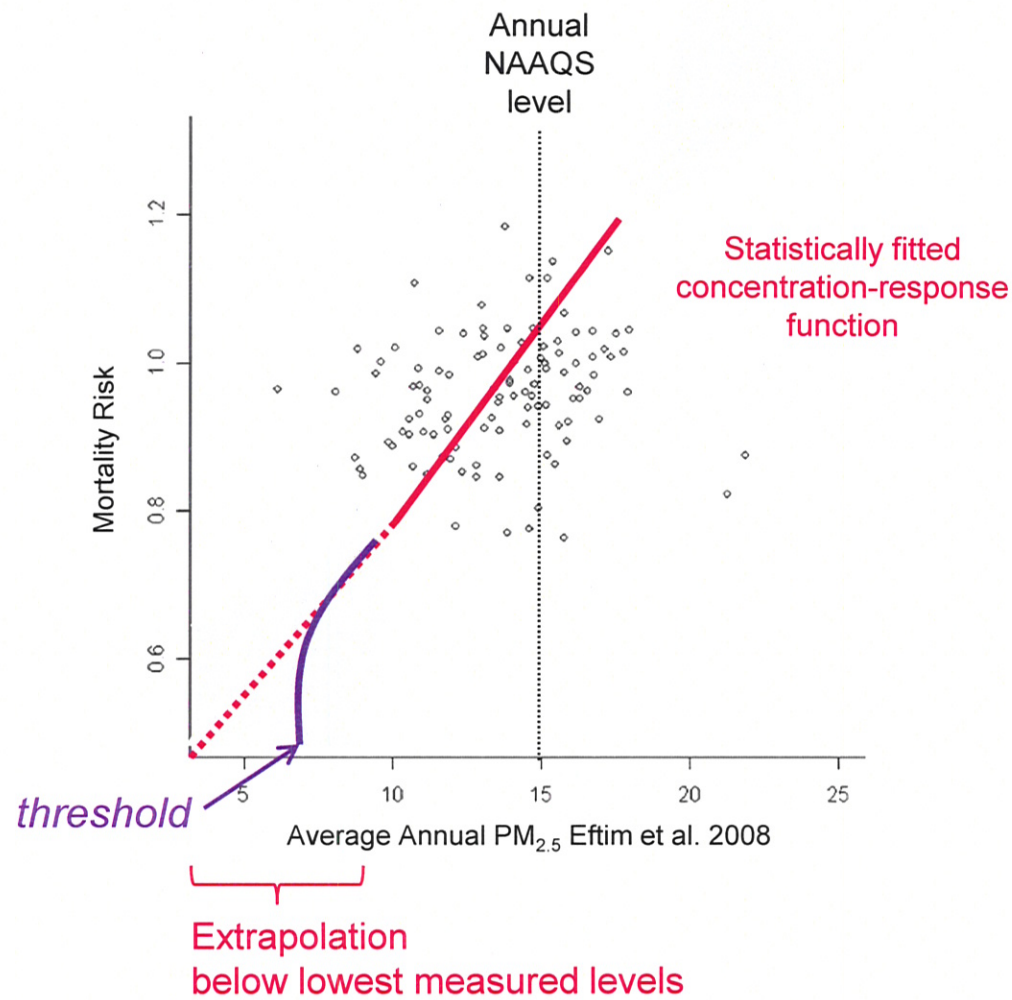
Adapted from
Smith 2011

1. No Threshold Model

- A no-threshold model for $PM_{2.5}$ that calculates incremental benefits down to the lowest modeled air quality levels

1. Question: what is the shape of the curve in the low-dose range?

2. Question: is there significant risk associated with ambient $PM_{2.5}$ levels?



Adapted from
Smith 2011

Clinical Exposure Studies Conducted by EPA

January 2010 – June 2011

41 Volunteers

Dose:35 – 750 ug/m^3

Results:

1 individual: elevated heart rate
1 individual: irregular heart beat*
39 individuals: no clinical effects

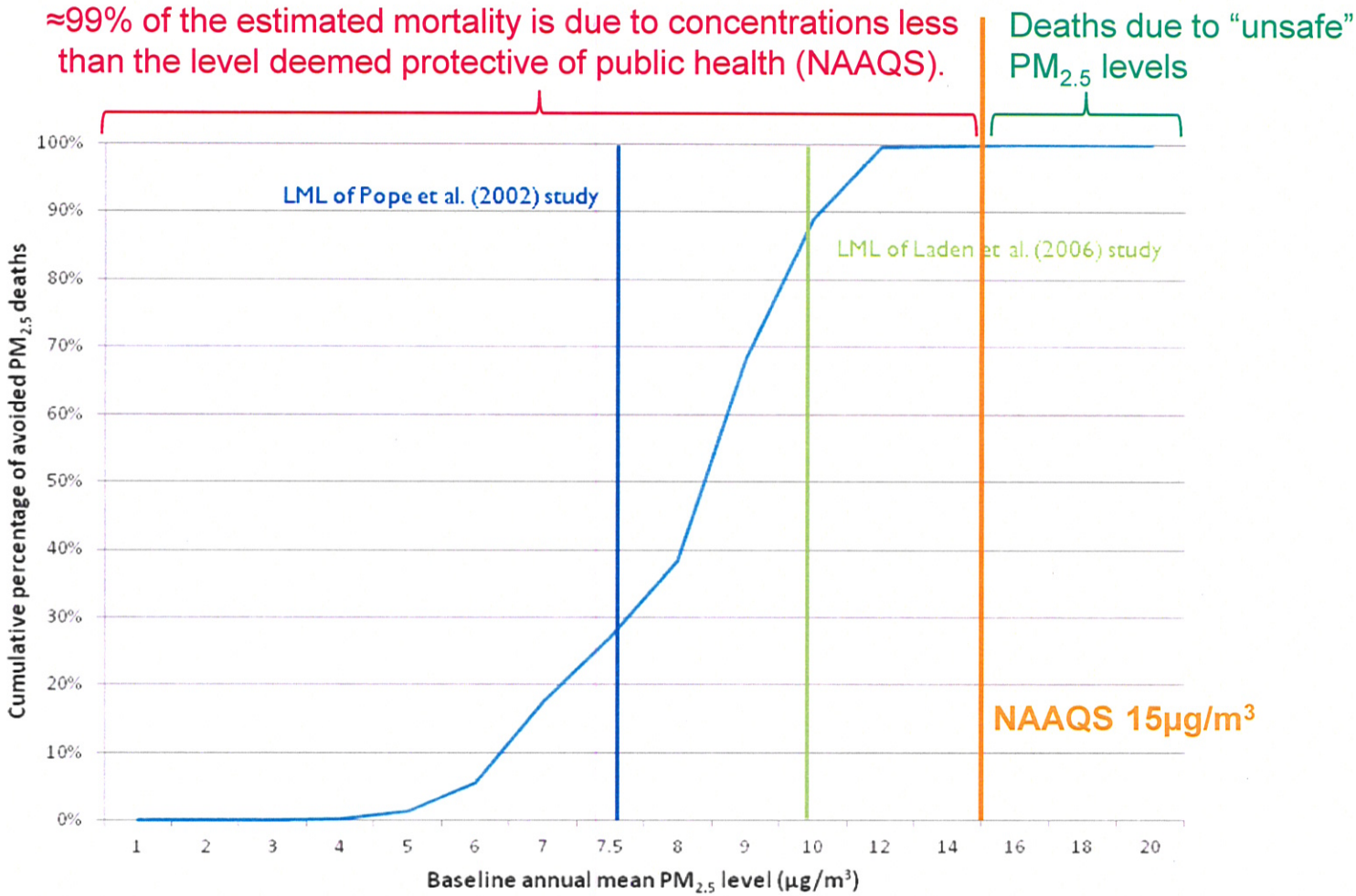
FOIA # HQ-FOI-02235-11

Exposure Date	SUBJECT	Entered Chamber	Exited Chamber	Filter Conc (ug/m3)	Clinical Effects*
1/5/2010	OMC019	11:02	13:02	205.27	No clinical effects requiring follow-up observed
1/6/2010	KCN112	9:34	11:34	153.58	No clinical effects requiring follow-up observed
2/9/2010	OMC021	10:52	12:52	442.49	No clinical effects requiring follow-up observed
3/9/2010	OMC023	10:45	11:08	750.83	No clinical effects requiring follow-up observed
3/23/2010	OMC024	10:49	12:49	147.42	No clinical effects requiring follow-up observed
4/13/2010	OMC025	10:43	12:43	431.06	No clinical effects requiring follow-up observed
4/20/2010	OMC026	11:19	13:19	336.56	No clinical effects requiring follow-up observed
4/27/2010	OMC027	11:00	13:00	257.18	No clinical effects requiring follow-up observed
4/28/2010	KCN111	9:13	11:13	154.36	No clinical effects requiring follow-up observed
5/4/2010	OMC028	10:54	12:54	326.78	No clinical effects requiring follow-up observed
5/5/2010	KCN113	9:26	11:26	578.95	No clinical effects requiring follow-up observed
5/11/2010	OMC022	10:51	12:51	247.77	No clinical effects requiring follow-up observed
6/8/2010	OMC030	10:48	12:48	257.12	No clinical effects requiring follow-up observed
6/15/2010	OMC031	11:28	13:28	468.96	No clinical effects requiring follow-up observed
6/29/2010	OMC033	11:04	13:04	321.36	No clinical effects requiring follow-up observed
7/13/2010	OMC034	10:49	12:49	177.02	No clinical effects requiring follow-up observed
7/15/2010	XCE224	11:10	13:10	137.19	No clinical effects requiring follow-up observed
8/10/2010	OMC035	11:00	13:00	411.98	No clinical effects requiring follow-up observed
8/12/2010	XCE225	10:59	12:59	157.63	No clinical effects requiring follow-up observed
8/25/2010	KCN114	9:55	11:55	232.91	No clinical effects requiring follow-up observed
9/9/2010	XCE226	10:55	12:55	87.36	No clinical effects requiring follow-up observed
9/23/2010	XCE228	11:05	13:05	174.61	No clinical effects requiring follow-up observed
10/6/2010	KCN115	9:31	11:31	131.50	No clinical effects requiring follow-up observed
10/7/2010	XCE227	11:21	12:10	111.68	Removed from chamber due to new onset of atrial fibrillation. Individual reverted to normal sinus rhythm approximately two hours later. Individual was admitted to the hospital overnight for observation and telemetry. Detailed in Ghio et al., 2011 Case Report, Environ Health Perspect doi:10.1289/ehp.1103877
11/18/2010	XCE229	11:14	13:14	59.09	No clinical effects requiring follow-up observed
12/2/2010	XCE231	10:55	12:55	35.60	No clinical effects requiring follow-up observed
1/6/2011	XCE233	11:05	13:05	43.65	No clinical effects requiring follow-up observed
1/24/2011	XCE232	10:47	12:47	150.63	No clinical effects requiring follow-up observed
1/31/2011	XCE234	11:03	13:03	90.95	No clinical effects requiring follow-up observed
2/3/2011	XCE236	11:12	13:12	57.91	No clinical effects requiring follow-up observed
2/10/2011	XCE235	11:12	11:35	66.26	Removed from chamber due to a short episode of an elevated heart rate during exposure. The individual denied any symptoms. This individual was provided with copies of the EKG and holter recording and referred to MD.
2/24/2011	XCE238	10:57	12:57	103.51	No clinical effects requiring follow-up observed
3/28/2011	XCE239	10:52	12:52	80.06	No clinical effects requiring follow-up observed
4/14/2011	XCE237	10:48	12:48	93.24	No clinical effects requiring follow-up observed
4/18/2011	XCE242	11:09	13:09	72.89	No clinical effects requiring follow-up observed
4/25/2011	XCE240	11:05	13:05	41.54	No clinical effects requiring follow-up observed
5/2/2011	XCE244	11:13	13:13	85.31	No clinical effects requiring follow-up observed
5/16/2011	XCE243	11:00	13:00	142.50	No clinical effects requiring follow-up observed
5/23/2011	XCE245	10:57	12:57	266.92	No clinical effects requiring follow-up observed
6/2/2011	XCE247	11:00	13:00	179.58	No clinical effects requiring follow-up observed
6/9/2011	XCE246	10:55	12:55	359.52	No clinical effects requiring follow-up observed

* Note : Clinical Effects is defined as requiring medical follow-up or referral to physician

** Case Report: Supraventricular Arrhythmia after Exposure to Concentrated Ambient Air Pollution Particles. Ghio et al. EHP. Feb. 2010. 120:275-277*

2. Risk Attributed to Ambient PM2.5

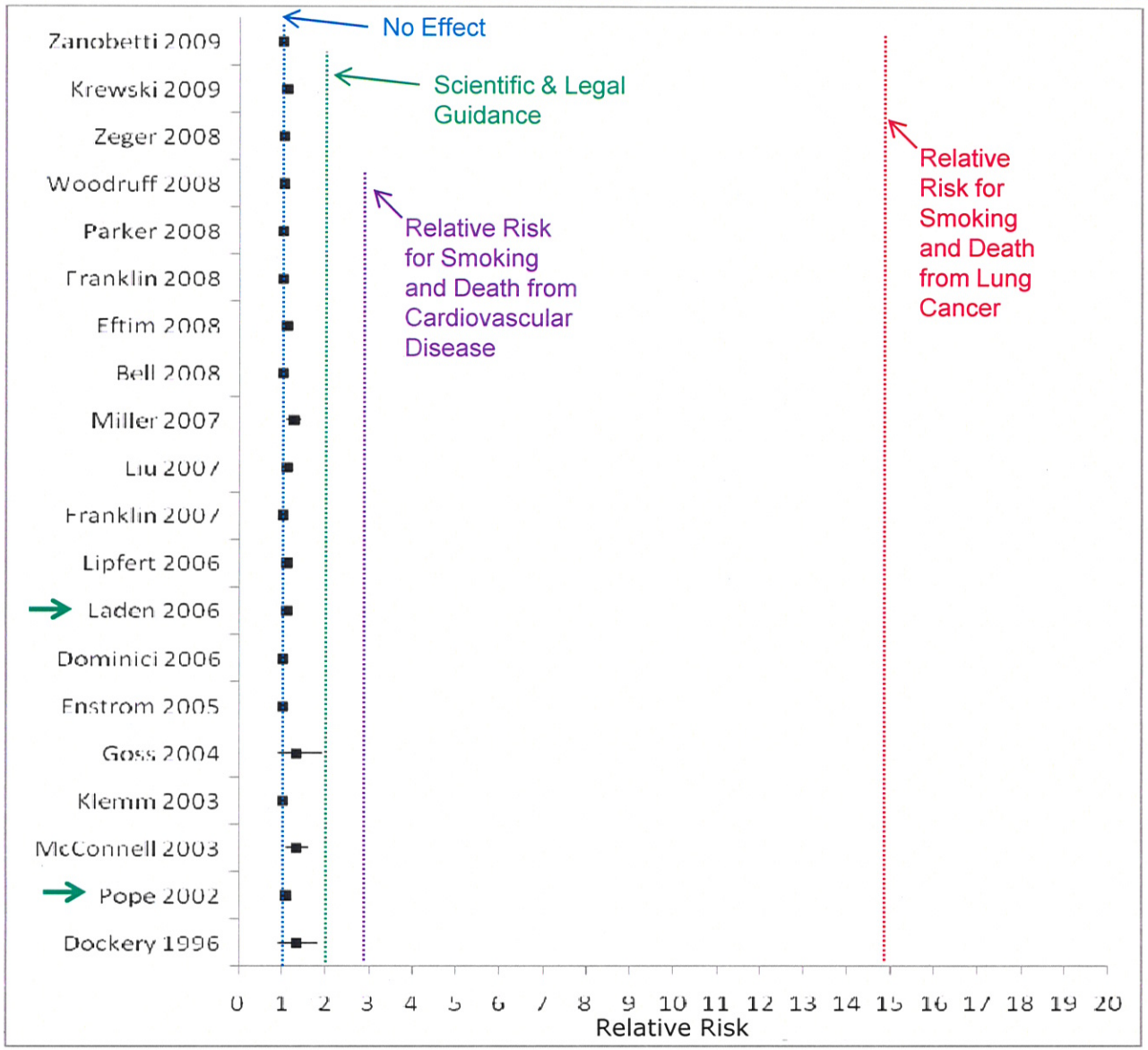


Of the total PM-related deaths avoided:

73% occur among population exposed to PM levels at or above the LML of the **Pope et al.** study.

11% occur among population exposed to PM levels at or above the LML of the **Laden et al.** study.

3. Assumption of Causality



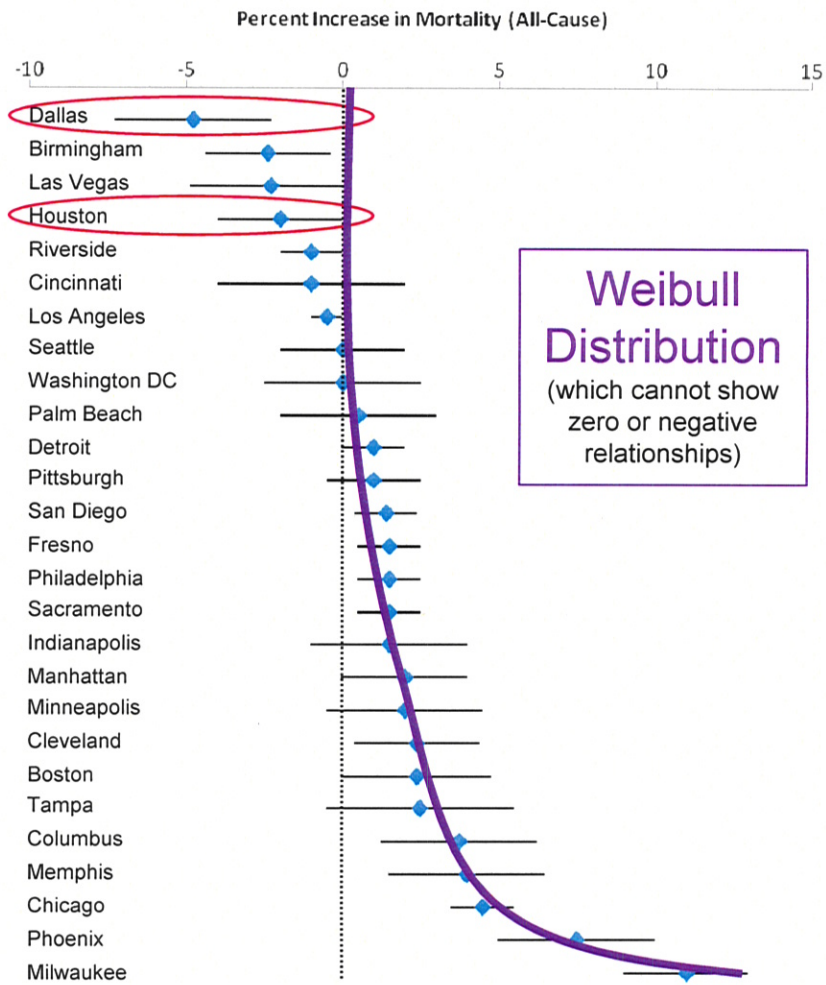
3. Assumption of Causality

- The epidemiology studies cannot show causality
- The analysis "assumes a causal relationship between $PM_{2.5}$ exposure and premature mortality…if the $PM_{2.5}$/mortality relationship is not causal, it would lead to a significant overestimation of net benefits"

-*EPA, The Benefits and Costs of the Clean Air Act from 1990 to 2020, March 2011*

$PM_{2.5}$ -Mortality Coefficient Estimates and 95% CI

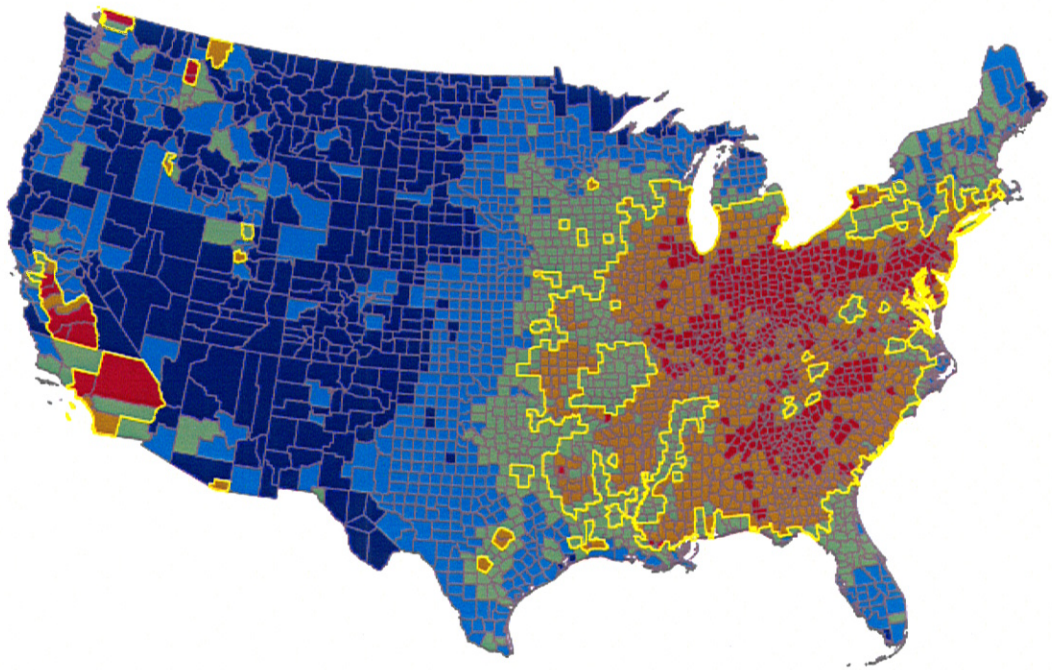
Adapted from Franklin et al. 2007



Estimates of the percent Increase in all-cause mortality with a 10 μg/m³ increase in previous day's concentration $PM_{2.5}$

Extrapolation of Mortality Estimates

Figure C-2. Distribution of $PM_{2.5}$ Mortality Risk in 2005



Percentage of total deaths due to PM2.5

- 0.85% to 2.6%
- 2.3% to 3.9%
- 4% to 5.1%
- 5.2% to 6.1%
- 6.1% to 9%
- Counties at or above the median risk level in 2005

From EPA – Regulatory Impact Analysis of the Proposed Toxics Rule: Final Report – March 2011

4. Value of Statistical Life
Definition

- A Value of Statistical Life (VSL) = value of risk reduction
  - A "statistical life" has traditionally referred to the aggregation of small risk reductions across many individuals until that aggregate reflects a total of one statistical life
  - The VSL has been a shorthand way of referring to the monetary value or tradeoff between income and mortality risk reduction, i.e. the willingness to pay for small risk reductions across large numbers of people
  - It has led to confusion because it has been interpreted as referring to the loss of identified lives

If risk was reduced
by 1 in 1,000,000
for 1 year
in a population of 200 million

savings of 200 **statistical** lives = value of risk reduction

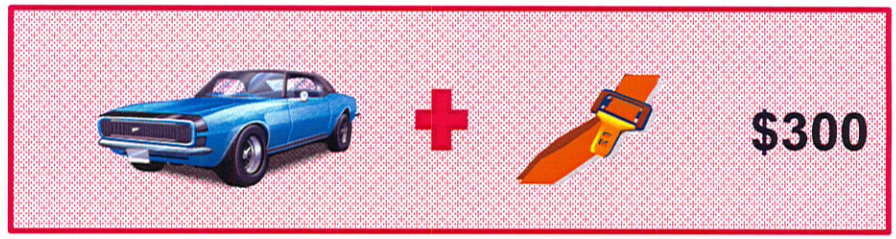
≠

savings of 200 **actual** lives

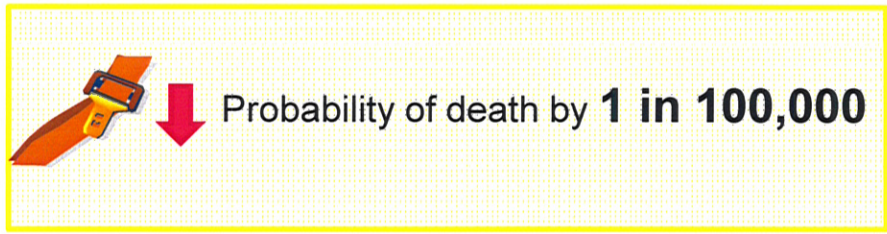
Deriving Value of Statistical Life

Willingness to Pay – Road Hazard Studies

- Example:
  - Cars with seatbelts cost $300 more than cars without seatbelts



  - Buying a car with that option reduces the probability of death by 1 in 100,000



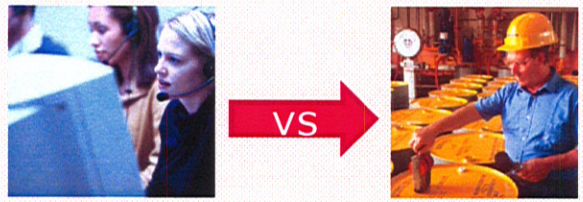
  - If people are willing to pay for this option, we can infer that the person is placing a valuation on his/her life of at least $300 x 100,000 = 30,000,000 ($30 million)

$300
x 100,000
= $30 million

Deriving Value of Statistical Life

Income vs. Risk – Occupational Studies

- Example:
  - A job carries a higher risk of injury, but pays $ 500 more per year



$ 500

  - The more dangerous job carries an increased risk of injury by 1 in 10,000

Probability of injury by **1 in 10,000**

  - If people are willing to pay for this option, we can infer that the individuals are placing a valuation on their lives of at least $500 x 10,000 = 5,000,000 ($5 million)

$ 500
x 10,000
= $5 million

Interpreting VSL in the Media

"When these new [EGU MACT] standards are finalized, they **will assist in preventing** 11,000 heart attacks, **17,000 premature deaths**, 120,000 cases of childhood asthma symptoms and approximately 11,000 fewer cases of acute bronchitis among children each year. Hospital visits will be reduced and nearly 850,000 fewer days of work will be missed due to illness."

- Lisa Jackson, EPA Administrator, 2011

This was interpreted as:

"EPA's proposed mercury and air toxics standards … **are projected to save as many as 17,000 American lives** …

- John D. Walke, Natural Resources Defense Council, 2011

"These new standards mark a huge step forward in clean air protections and **will be responsible for saving thousands of lives** each year."

- Albert A. Rizzo, MD, National Volunteer Chair of the American Lung Association

"The new EPA mercury standards **will save countless lives** and improve the quality of life for millions."

- New York Mayor Michael Bloomberg

Appropriate Use of Value of Statistical Life

The Benefits and Costs of the Clean Air Act from 1990 to 2020

TABLE 5-8. LIFE YEARS GAINED AND LIFE EXPECTANCY GAIN ESTIMATES FROM THE POPULATION SIMULATION MODEL

AGE COHORT		LIFE-YEARS GAINED IN SPECIFIC YEARS (ANNUAL)		CUMULATIVE LIFE YEARS GAINED THROUGH TARGET YEAR		LIFE EXPECTANCY GAINS (YEARS)		
START AGE	END AGE	2020	2040	2020	2040	2010	2020	2040
30	39	17,000	18,000	260,000	620,000	0.65	0.87	0.91
40	49	60,000	71,000	910,000	2,300,000	0.63	0.84	0.88
50	59	150,000	180,000	2,000,000	5,400,000	0.59	0.79	0.84
60	69	330,000	380,000	3,500,000	11,000,000	0.53	0.71	0.76
70	79	470,000	840,000	5,000,000	20,000,000	0.44	0.59	0.64
80	89	470,000	1,200,000	6,000,000	23,000,000	0.32	0.43	0.48
90	99	320,000	800,000	3,600,000	14,000,000	0.19	0.25	0.27
100+		60,000	200,000	490,000	3,100,000	0	0	0
	Total	1,900,000	3,800,000	22,000,000	80,000,000			

Note: Column entries to not add to totals due to rounding. Life expectancy results are incremental period conditional life expectancy gains at the start age of the cohort.

EPA VSL:$8,900,000

- Lives Saved vs. Life-Years Added
  - Deaths "prevented or avoided"
  - Gains in life expectancy

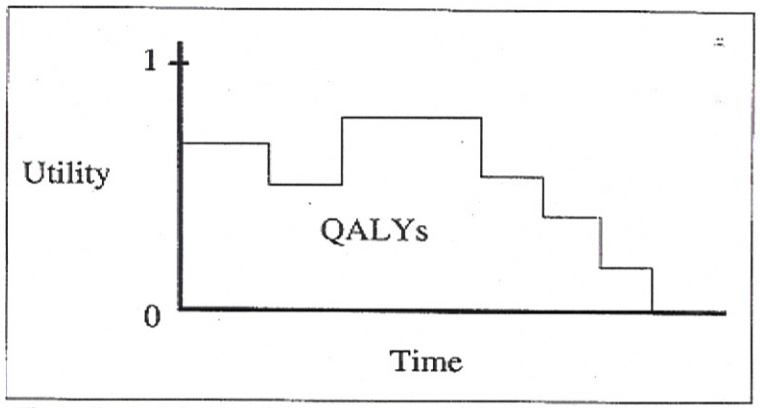


Figure: Determining Quality-Adjusted Survival—Length of life (time) is plotted against quality of life (utility). The area under the curve represents quality-adjusted survival measured in quality-adjusted life years (QALYs).

From Weeks 1995

- The median age of people who gain extra months of life from cleaner air is close to 80 years
- Adjustment of VSL for quality of life:
  - EPA VSL of $8,900,000 appropriate for healthy young adult (≈25)
  - 6:1 ratio for 25 vs. 80 year old

Clean Air Act - Benefits and Costs

reduced number of deaths in 2020 * value per statistical life saved
= 230,000 fewer deaths * $8,900,000 per life saved
≈ $2 trillion
Benefit/Cost = $2 trillion/$0.065 trillion* ≈ 30

life-years gained in 2020 * value per statistical life-year gained
=1,900,000 life-years gained * $150,000/life-year gained
≈ $0.3 trillion
Benefit/Cost = $0.3 trillion/$0.065 trillion* ≈ 5

Adjusted estimate of benefit:
$19 billion

Benefit/Cost = $0.019 trillion/$0.065 trillion* ≈ 0.3

Mercury & Air Toxics Standard

	Benefits from HAPs (billions)	"Co-Benefits" from non-HAPs (billions)
Mercury	$ 0.004-0.006	$ 1-2
Acid Gasses	$ 0	$ 32-87
Non-Hg Metals	$ 0	$ 1-2
Total	**≤$ 0.006**	**$ 33-90**

- MATS is estimated to prevent 0.00209 IQ point loss per child (starting immediately)
- Each child will gain 0.0956 school days over their lifetime
- 0.00209 IQ points x 244,468 children = 511 IQ points per year
- Assuming a net monetary loss per decrease in one IQ point of between ~$8,000 and ~$12,000 (in terms of foregone future earnings)
- Benefit = $4.2M to $6.2M

Table adapted from testimony by Anne E. Smith 2/2010 to Subcommittee on Energy and Power

Oil & Gas NSPS and NESHAPS

	Oil and Natural Gas NSPS (millions)	Oil and Natural Gas NESHAP Amendments (millions)
Benefits	NA	NA
Costs	- $15	$3.5
Non-monetized benefits	11,000 tons of HAP5 190,000 tons of VOC 1.0 million tons of methane Health effects of HAP exposure Health effects of $PM_{2.5}$ and ozone exposure Visibility impairment Vegetation effects Climate effects	670 tons of HAP 1,200 tons of VOC 420 tons of methane Health effects of HAP exposure Health effects of $PM_{2.5}$ and ozone exposure Visibility impairment Vegetation effects Climate effects

*"…**quantification of those benefits cannot be accomplished for this rule.** This is not to imply that there are no benefits of the rules; rather, it is a reflection of the difficulties in modeling the direct and indirect impacts of the reductions in emissions for this industrial sector with the data currently available."*

April 2012 RIA

PM Co-Benefits in RIAs

	$PM_{2.5}$ NAAQS	Utility Boiler MACT	Mercury Air Toxics Standard	Sewage Sludge Incineration Units	Ferroalloy NESHAP	Total Costs millions ($2006)
Estimated Statistical Deaths	15,000	11,900	2,650	25	14	
Cost	6,400	10,600	9,329	17	4	26,350

- Double counting benefits: same statistical lives counted in multiple rules
- Different costs: unique to each rule

Contact Information

Susana M. Hildebrand, P.E.
Chief Engineer
susana.hildebrand@tceq.texas.gov
(512) 239-4696

Michael Honeycutt, Ph.D.
Division Director, Toxicology
michael.honeycutt@tceq.texas.gov
(512) 239-1793

Health Effects of Poverty and Unemployment

- Poverty and unemployment have been recognized as risk factors for morbidity and mortality since the 1800's (Virchow, 1848)
  - As of March 2012, there are 4,850 publications on this topic

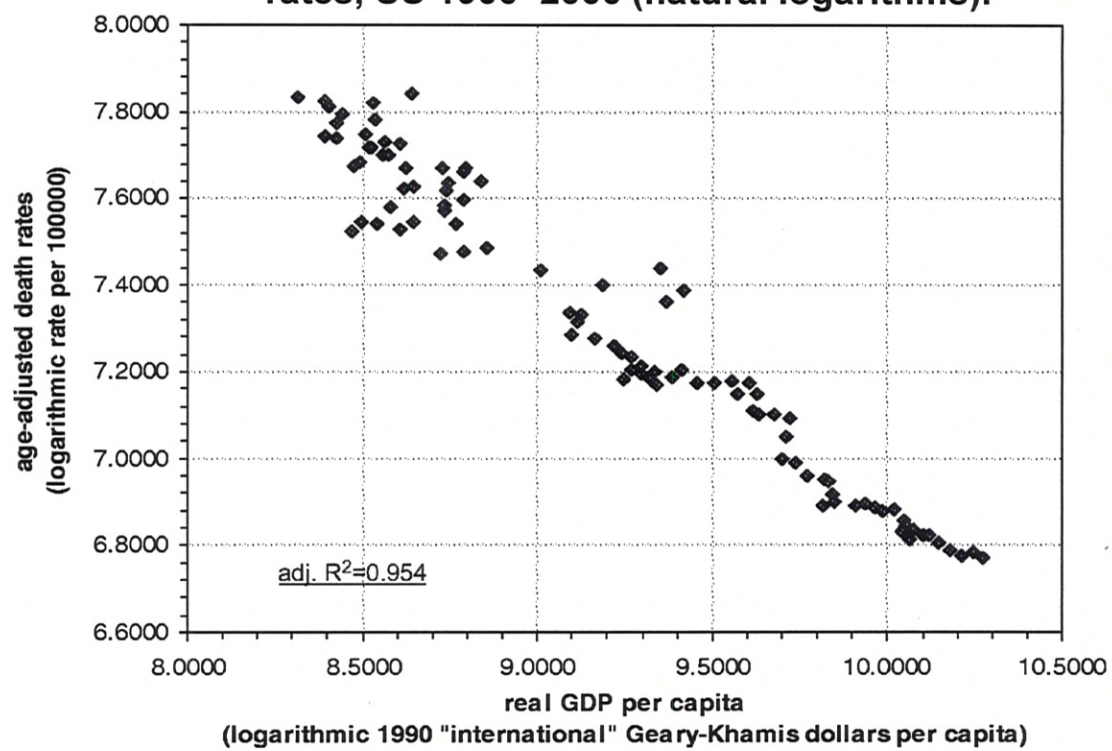
Unemployment and All-Cause Mortality

Meta-analyses stratified by gender and age [a]

Gender	Mean Age	HR (95% CI)
	Less than 40	1.73[b] (1.41, 2.11)
Women	40 to 49.9	1.34[b] (1.15, 1.56)
	50 to 65	0.94 (0.80, 1.11)
	Less than 40	1.95[b] (1.69, 2.26)
Men	40 to 49.9	1.86[b] (1.63, 2.12)
	50 to 65	1.17[c] (1.00, 1.36)

Roelfs et al. Soc Sci Med 2011; 72:840-54

Relation of real GDP per capita to age-adjusted death rates, US 1900–2000 (natural logarithms).

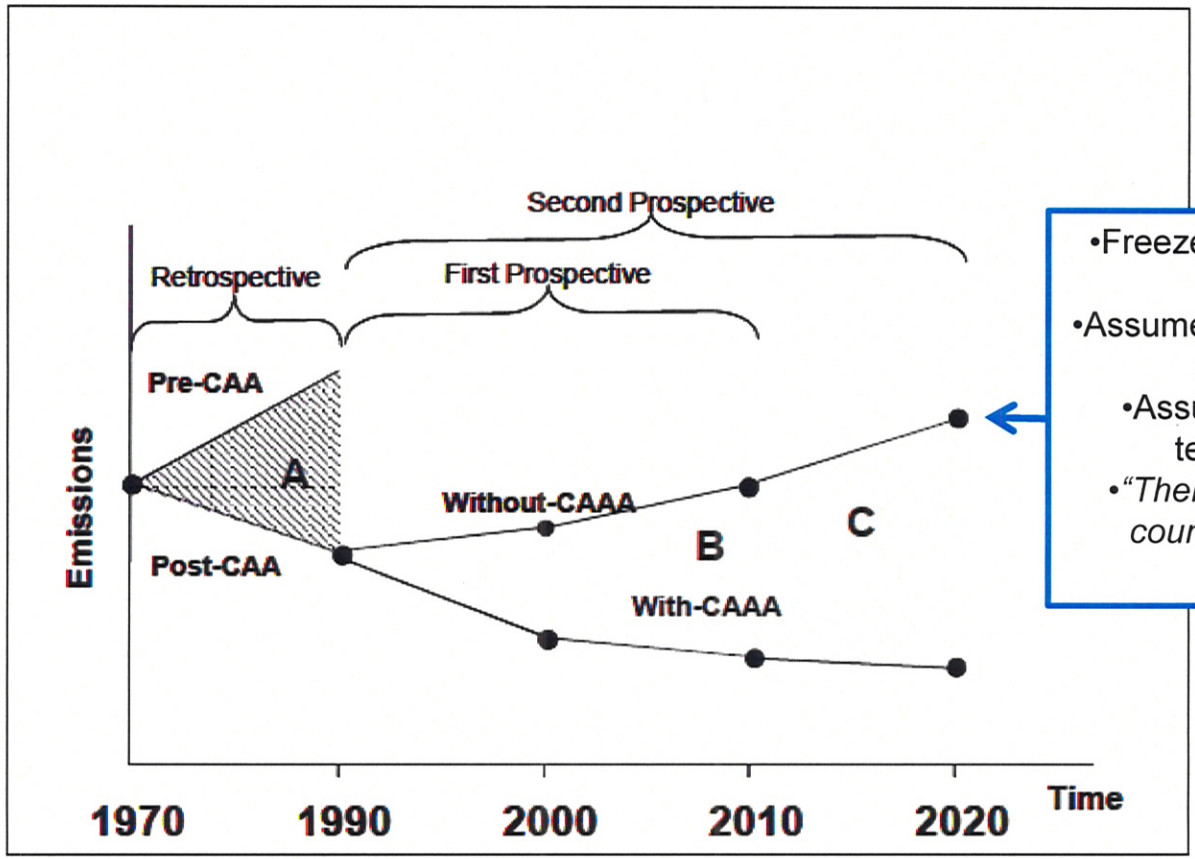


Brenner M H Int. J. Epidemiol. 2005;34:1214-1221

With CAAA vs. Without CAAA

The Benefits and Costs of the Clean Air Act from 1990 to 2020

FIGURE 1-1. CLEAN AIR ACT SECTION 812 SCENARIOS: CONCEPTUAL SCHEMATIC



- Freezes pollution controls at 1990 levels
- Assumes no additional state or local regulation after 1990
- Assumes no improvements in technology or efficiency
- *"There is no way to validate the counterfactual, without-CAAA scenario estimates"*

Oil & Gas NESHAPS

Table 4-7 Climate Methane Benefits Using 'GWP' Approach

SCC Value for 2015 emission reductions ($/ton CO_2 in 2008 dollars)[1]	Total Benefits based on 100 year GWP adjustment[2] (millions 2008$)	
	Final NSPS	Final NESHAP Amendments
$6 (mean 5% discount rate)	$100	$0.05
$25 (mean 3% discount rate)	$440	$0.20
$40 (mean 2.5% discount rate)	$700	$0.32
$76 (95th percentile at 3% discount rate)	$1,300	$0.60
Methane Emission Reductions[3] (MMT CO_2-e)	17.6	0.008

April 18, 2012 Press Conference

"Today's rules would yield significant reductions in methane, a potent greenhouse gas. EPA's Regulatory Impact Analysis for the rule estimates the value of the climate co-benefits that would result from this reduction at $440 million annually by 2015."
-Gina McCarthy

Reported monetized benefit: $0

Note: benefits calculated at 3%, but costs at 7%

Costs of the Clean Air Act and Amendments

Year	RIAs for Rules Not Targeting Ambient PM 2.5	PM Co-Benefits are >50% of Total	PM Co-Benefits Are Only Benefits Quantified	Cost ($ Billion)*
1997	Ozone NAAQS (.12 1hr=>.08 8hr)	x		9.60
1997	Pulp&Paper NESHAP			6.48
1998	NOx SIP Call & Section 126 Petitions			1.66
1999	Regional Haze Rule	x		1.74
1999	Final Section 126 Petition Rule	x		1.15
2004	Stationary Reciprocating Internal Combustion Engin NESHAP	x		0.25
2004	Industrial Boilers & Process Heaters NESHAP	x	x	0.86
2005	Clean Air Mercury Rule	x		0.90
2005	Clean Air Visibility Rule/BART Guidelines	x		1.50
2006	Stationary Compression Ignition Internal Combustion Engine NSPS			0.06
2007	Control of HAP from mobile sources	x	x	0.36
2008	Ozone NAAQS (.08 8hr =>.075 8hr)	x		8.20#
2008	Lead (Pb) NAAQS	x		3.20
2009	New Marine Compress'n-Ign Engines >30 L per Cylinder	x		1.90
2010	Reciprocating Internal Combustion Engines NESHAP - Comp. Ignit.	x	x	0.37
2010	EPA/NHTSA Joint Light-Duty GHG & CAFES			15.60
2010	SO2 NAAQS (1-hr, 75 ppb)	x	>99.9%	1.50
2010	Existing Stationary Compression Ignition Engines NESHAP	x	x	0.25
2011	Industrial, Comm, and Institutional Boilers NESHAP	x	x	0.49
2011	Indus'l, Comm'l, and Institutional Boilers & Process Heaters NESHAP	x	x	2.90
2011	Comm'l & Indus'l Solid Waste Incin. Units NSPS & Emission G'lines	x	x	0.28
2011	Control of GHG from Medium & Heavy-Duty Vehicles			2.00&
2011	Ozone Reconsideration NAAQS	x		8.20#
2011	Utility Boiler MACT NESHAP (Final Rule's RIA)	x	≥99%	9.60
2011	Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP	x		0.00
2011	Sewage Sludge Incineration Units NSPS & Emission Guidelines	x	x	0.02
2011	Ferroalloys Production NESHAP Ammendments	x	x	0.004
		Total:		60.67

+ MATS – 9.3

Partial Total: 69.97

- Cross State Air Pollution Rule
  - EPA estimated cost:$800 million annually
  - Independent analysis: $120 billion by 2015
- Boiler MACT
  - EPA estimated cost:$2.6 billion annually
  - Independent analysis:$14.5 billion

* ($2006)

Business Impact

The Benefits and Costs of the Clean Air Act from 1990 to 2020

FIGURE 8-6. PERCENT CHANGE IN INDUSTRY OUTPUT IN 2020: LABOR FORCE-ADJUSTED CASE

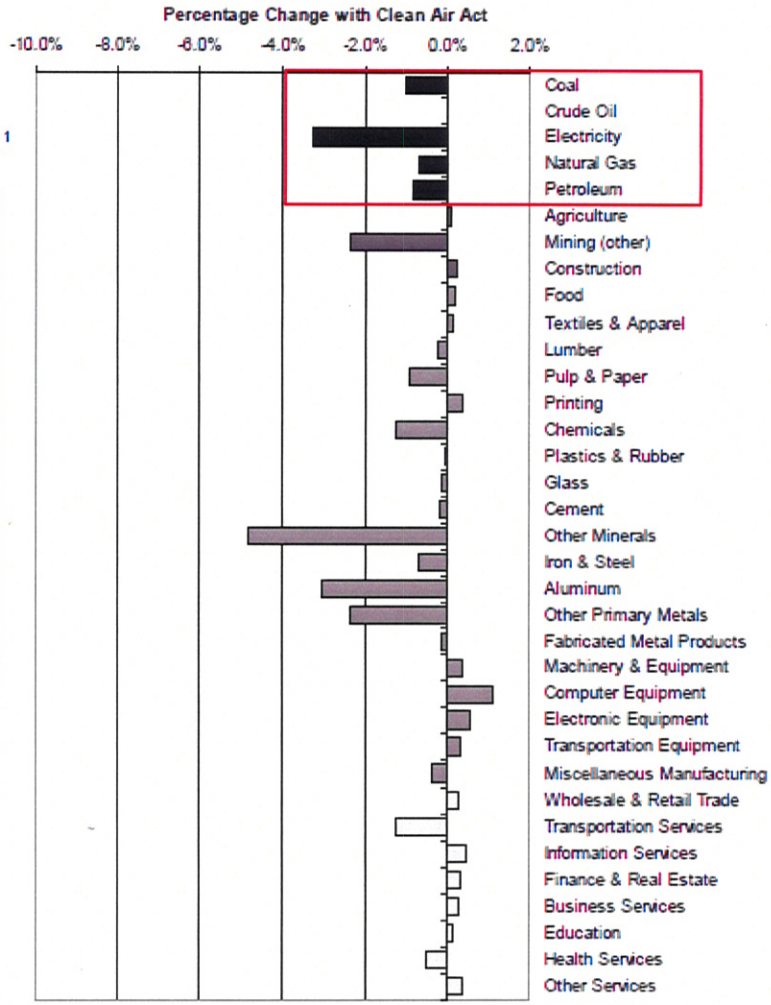


TABLE 8-8.

SUMMARY OF ANNUAL MACROECONOMIC IMPACTS: LABOR FORCE-ADJUSTED CASE[1]

VARIABLE	MODEL RUN	2010	2015	2020
GDP	With Clean Air Act ($ billion)	$15,027	$17,338	$20,202
	Without Clean Air Act ($ billion)	$15,059	$17,350	$20,197
	Change ($ billion)	−$32	-$12	$5
	% change	−0.21%	-0.07%	0.02%
Consumption	With Clean Air Act ($ billion)	$10,969	$12,699	$14,881
	Without Clean Air Act ($ billion)	$10,972	$12,696	$14,876
	Change ($ billion)	−$3	$3	$5
	% change	−0.03%	0.02%	0.03%
Hicksian EV (annual)	Change ($ billion)	$11	$22	$29
	% change	0.08%	0.13%	0.15%

Notes:

1. Results are expressed in year 2006 dollars.

Adjusted Benefits Estimate

Tony Cox, 2011:

($1.8 trillion initial estimate)

x (1/6 reduction factor for VSL if age or VSLY is considered)

x (0.5 probability that a true association exists)

x (0.5 probability that a true association is causal, given that one exists)

x (0.5 probability that ambient concentrations are above any thresholds or nadirs in the C-R function, given that a true causal C-R relation exists)

x (0.5 expected reduction factor in C-R coefficient by 2020 due to improved medication and prevention of disease-related mortalities)

= (1.8 trillion)*(1/6)*(0.5)*(0.5)*(0.5)*(0.5) = $19 billion

AN EVALUATION OF THE $PM_{2.5}$ HEALTH BENEFITS ESTIMATES IN REGULATORY IMPACT ANALYSES FOR RECENT AIR REGULATIONS

NERA
Economic Consulting

Final Report
Prepared for the Utility Air Regulatory Group

Anne E. Smith, Ph.D.
Senior Vice President
NERA Economic Consulting
1255 23rd Street NW
Washington, DC 20037

December, 2011

An Evaluation of the $PM_{2.5}$ Health Benefits Estimates in Regulatory Impact Analyses for Recent Air Regulations

Anne E. Smith, Ph.D.
NERA Economic Consulting[1]

December, 2011

Abstract.

When preparing its Regulatory Impact Analyses (RIAs) for regulations under the Clean Air Act (CAA) that are not intended to control ambient fine particulate matter ($PM_{2.5}$), the US Environmental Protection Agency (EPA) often predicts reductions of ambient $PM_{2.5}$ that may occur coincidentally, and attributes so-called "$PM_{2.5}$ co-benefits" to those coincidental reductions. This paper reviews and evaluates EPA's practice of including $PM_{2.5}$ co-benefits in its RIAs for non-PM rules. It is based on review of 57 individual CAA-related RIAs released since EPA promulgated its first $PM_{2.5}$ national ambient air quality standard (NAAQS) and finds that EPA has been relying on $PM_{2.5}$ co-benefits estimates to create an apparent benefit-cost justification for almost all of its non-PM CAA rules. This paper then evaluates that practice from multiple perspectives: theoretical, practical, scientific, and analytical. It concludes that co-benefits from separately-regulated pollutants, such as $PM_{2.5}$, should not be reported as part of the total benefits estimates in an RIA, nor should they be included in public announcements of the benefits of a new regulation. EPA should reform the manner in which it defines its baselines of emissions for each RIA, and provide more temporal information on benefits and costs to eliminate problems of double-counting. This paper also concludes that EPA should reform its current methods of calculating benefits from reductions in ambient $PM_{2.5}$ even in its PM-related rules, because it finds that as EPA's reliance on co-benefits has increased, EPA has shifted to less credible methods of estimating $PM_{2.5}$ benefits.

[1] The author acknowledges and thanks Suresh Moolgavkar, W. David Montgomery, and Mike King for their helpful and insightful comments and suggestions on drafts of this paper. The author also thanks Shirley Xiong for her careful and persevering assistance in reviewing and documenting the nearly 60 RIAs relied on in this study. Any remaining errors are solely my responsibility.

1. Introduction and Synopsis

Regulatory Impact Analyses (RIAs) are documents required by an Executive Order (EO) of the President to be submitted to the Office of Management and Budget (OMB) by all agencies proposing new major regulations. RIAs' quality and usefulness have been a substantial interest for the past thirty years. Policy analysts have written many papers and reports identifying the ways RIAs provide value to policy making and offering suggestions for improvement.[2] This paper reviews and evaluates the US Environmental Protection Agency's (EPA's) practice of estimating benefits from reducing ambient fine particulate matter ($PM_{2.5}$) in its RIAs for rulemakings under the Clean Air Act (CAA).

In recent years, EPA has relied on reductions of ambient $PM_{2.5}$ as the primary source of benefits in most of its RIAs for CAA-related regulations – even for regulations not specifically to protect the public health from exposures to ambient $PM_{2.5}$. When the regulation is not targeting $PM_{2.5}$, they are called "co-benefits" because they result from changes in ambient $PM_{2.5}$ levels projected to follow coincidentally from efforts to reduce other types of air pollutants. Questions and concerns have been raised by many in policy making and policy analysis communities about EPA's reliance on such co-benefits.

Based on review of CAA-related RIAs since 1997, this paper identifies the degree of EPA's reliance on $PM_{2.5}$ co-benefits. It then examines EPA's co-benefits practice from multiple perspectives: theoretical, practical, scientific, and analytical. It finds that the theoretical formulation of benefit-cost analysis (BCA) – a key underpinning of RIAs – does not support inclusion of co-benefits from pollutants subject to their own, separate regulation. Also, allowing such co-benefits to dominate RIAs detracts from RIAs' most valuable practical role, which is to help guide us toward regulations that provide cost-effective, minimally-complex management of societal resources. From a scientific perspective, this review finds EPA's estimates of the risks of $PM_{2.5}$ have become less and less credible as EPA has come to rely more and more heavily on them to justify regulation of other pollutants. It also finds that use of co-benefits in many RIAs being prepared simultaneously degrades the analytical rigor of benefits accounting across the body of RIAs as a group, with double-counting and related analytical maladies resulting.

Accordingly, this paper recommends changes in how RIA baselines are set in order to eliminate problems of double-counting and inappropriate benefit-cost comparisons. It also concludes that:

- Public announcements about the benefits of a new regulation should not include co-benefits of pollutants that are already directly regulated; nor should such co-benefits be included in the total benefits reported in RIA Executive Summaries.
- EPA should reform its practice of calculating benefits from reductions in ambient $PM_{2.5}$ by using more credible sets of risk analysis assumptions, and eliminating extrapolations.

---

[2] Examples include Morgenstern (1997), Hahn and Dudley (2007), and Harrington *et al.* (2009).

This paper is organized as follows. The next section provides background on the history and purpose of RIAs as the general requirement under which EPA is producing its CAA-related RIAs. It is followed by a summary of EPA's growing reliance on $PM_{2.5}$ co-benefits identified through a review of RIAs dating back to 1997, when EPA released its first regulation of ambient $PM_{2.5}$. The four sections after that examine EPA's co-benefits practice from four different perspectives: theoretical, practical, scientific, and analytical, and conclude the practice is problematic from each of these perspectives. The last section recaps findings and recommendations made throughout the paper and draws further conclusions.

2. History and Purpose of RIAs

The practice of using BCA for assessing the appropriateness of public policies dates back well before RIAs were required, but was relatively sporadic. This changed in 1981 when President Ronald Reagan issued EO 12291. EO 12291 required that a BCA be prepared and submitted to the OMB for each major regulation issued by the Federal government. That EO required that each new major rule be demonstrated to provide greater benefits than its costs,[3] using the term "regulatory impact analysis" for the document making this demonstration.[4] The requirement that benefits be greater than costs is certainly a prerequisite for passing any BCA test; nevertheless, the economically-proper definition of a benefit-cost optimum – the BCA basis for determining the appropriate stringency of a standard – is that the incremental or "marginal" cost of making a standard tighter is equal to the marginal benefits that such tightening would provide. This is because net benefits are at their maximum level when marginal costs equal marginal benefits. Subsequent guidance for conducting RIAs requires that several alternative standards be evaluated in the RIA. This is to help steer the selected alternative to the one that would offer the highest net benefits without requiring a precise optimization using marginal analysis.[5]

EO 12291's requirement that benefits exceed costs was at odds with the fact that many standards, including National Ambient Air Quality Standards (NAAQSs) for criteria pollutants, and National Emissions Standard for Hazardous Air Pollutants (NESHAPs) for air toxics, must be set without regard to costs. The CAA requires that each NAAQS

---

[3] Section 2 of EO 12291 specifically required, *inter alia*, that "(b) Regulatory action shall not be undertaken unless the potential benefits to society for the regulation outweigh the potential costs to society; (c) Regulatory objectives shall be chosen to maximize the net benefits to society; (d) Among alternative approaches to any given regulatory objective, the alternative involving the least net cost to society shall be chosen." These are overtly the requirements of standard BCA-based decision making.

[4] EO 12291, Section 3.

[5] Guidance has been provided over the years. The most current version of guidance, known as "Circular A-4," states that: "In general, both the benefits and costs associated with a regulation will increase with the level of stringency (although marginal costs generally increase with stringency, whereas marginal benefits may decrease). You should study alternative levels of stringency to understand more fully the relationship between stringency and the size and distribution of benefits and costs among different groups." (OMB, 2003, p.8).

be set at a level that protects the public health with "an adequate margin of safety,"[6] and has been interpreted to require that this level be set without regard for their costs. The CAA's requirements for NESHAP rules are more complex, but the most commonly required provision used under a NESHAP rule is maximum achievable control technology (MACT). The least stringent emissions level for a MACT (the "MACT floor") is determined based solely on the average performance achieved by the best 12% of existing technologies for a given production process, regardless of what cost achieving that MACT floor may impose on facilities that do not already have that emissions rate.[7] Thus the requirement of EO 12291 that all major regulations demonstrate they could pass the most basic requirement of BCA was instantly at odds with the legal framework for CAA rules. Resolution of this inconsistency was addressed by a provision in EO 12291 that the BCA provisions be applied "to the extent permitted by law."[8]

An executive order requirement for RIAs for all major regulations has remained in effect since 1981, but has evolved. In particular, President Bill Clinton issued EO 12866 in 1993 which revokes EO 12291, but replaces it with very similar requirements for assessment of regulatory impacts of major rulemakings.[9] Other than instituting some procedural changes, EO 12866 primarily moderates the emphasis of EO 12291 on meeting benefit-cost criteria. However, the change is one of degree only and the fundamental underpinnings of RIAs in BCA remains apparent. For example, EO 12866 states:

> *In deciding whether and how to regulate, agencies should assess all costs and benefits of available regulatory alternatives, including the alternative of not regulating. Costs and benefits shall be understood to include both quantifiable measures (to the fullest extent that these can be usefully estimated) and qualitative measures of costs and benefits that are difficult to quantify, but nevertheless essential to consider. Further, in choosing among alternative regulatory approaches, agencies should select those approaches that maximize net benefits (including potential economic, environmental, public health and safety, and other advantages; distributive impacts; and equity), unless a statute requires another regulatory approach.*[10]

---

[6] 42 USC §7409(b)(1).

[7] 42 USC §7412(d)(3)(A).

[8] EO 12291, Section 2.

[9] Interestingly, EO 12866 did not use the term "regulatory impact assessment" despite the similarity of its requirements. Apparently as a result, after the issuance of Clinton's EO 12866, and for the remainder of his years in office, the term RIA was largely replaced by "Economic Impact Analysis" (EIA) as the name of these EO-mandated documents. After about 2002, the term RIA came back into use, and remains the term used today. This temporary change in the common name for the regulatory assessment documents submitted to OMB can be confusing when performing reviews of RIAs and the methodological practices associated with them. This paper will refer to all of them as RIAs, although the titles of some of the documents cited do not use that term.

[10] EO 12866, Section 1(a).

As the excerpt above reveals, EO 12866 no longer requires that benefits be greater than costs. Also, it broadens the criteria to be considered to include more allowance for qualitative and non-quantifiable benefits. This is reinforced by the following language:

> *Each agency shall assess both the costs and the benefits of the intended regulation and, recognizing that some costs and benefits are difficult to quantify, propose or adopt a regulation only upon a reasoned determination that the benefits of the intended regulation justify its costs.*[11]

As a result, the purpose of RIAs has been more clearly defined as one of providing decision-relevant information in a structured, coherent and transparent format. Costs and benefits still play a central role, but are not treated as if they should be determinative, even in situations where the law allows consideration of costs. Most policy analysts, including most economists, have been supportive of this broader purpose of RIAs.

In 2011, President Barack Obama issued EO 13563 to supplement EO 12866: "to improve regulation and regulatory review." EO 13563 requires that "equity, human dignity, fairness, and distributive impacts" be considered, in addition to the requirements of EO 12866.[12] This last modification does not alter the fundamental view of the role and purpose of RIAs: EO 13563 states that the purpose of RIAs is that regulations will be adopted based "upon a reasoned determination that its benefits justify its costs" and also that each agency "tailor its regulations to impose the least burden on society" and "select, in choosing among regulatory approaches, those approaches that maximize net benefits."[13] It also, importantly, notes that agencies should strive to reduce regulatory requirements that are "redundant, inconsistent, or overlapping."[14]

Thus, RIAs have an original foundation in BCA, but they rarely have been used strictly as BCA-based decision documents. Their practical value in policy making is that they can provide a structured assessment of the merits of individual regulations, so that policymakers and the public can have a clear understanding of the overall burden and positive contributions of individual regulations.[15] This practical role is quite clearly stated in OMB's "Circular A-4," which is the present OMB guidance for conducting RIAs:

> *Regulatory analysis is a tool regulatory agencies use to anticipate and evaluate the likely consequences of rules. It provides a formal way of organizing the evidence on the key effects, good and bad, of the various alternatives that should be considered in developing regulations. The*

---

[11] EO 12866, Section 1(b)(6).

[12] EO 13563, Section 1(c)).

[13] EO 13563, Section 1(b).

[14] EO 13563, Section 3.

[15] See, for example, Arrow *et al.* (1996).

> *motivation is to (1) learn if the benefits of an action are likely to justify the costs or (2) discover which of various possible alternatives would be the most cost-effective.*[16]

Similarly, as RIAs are prepared for multiple different rules required by certain sections of law, such as a NESHAP under the CAA, the combined set of RIAs can help create an understanding of the overall merits of the way the controlling laws have been written. With time, good RIA practice should help identify laws that are poorly written, or regulatory approaches that implement laws in an ineffective manner. Political pressure to revise ineffective laws may result in better long-term legislation or regulatory practice. The fact that this role is recognized as valuable may be one reason why the requirement for RIAs has endured for thirty years and through Administrations with differing political perspectives. Whatever the merits of RIAs may be, they do face one problem with respect to serving their most valuable public policy objective. They are not subject to any formal public or peer review process other than review by OMB's Office of Information and Regulatory Affairs (OIRA).[17]

3. Evidence of the Predominance of $PM_{2.5}$ Co-Benefits in RIAs for Air Regulations

The research for this paper sought to identify and obtain all of the RIAs released for rulemakings under the CAA since 1997, which was the year in which EPA released its first NAAQS for $PM_{2.5}$. EPA's first estimates of mortality benefits from reducing ambient $PM_{2.5}$ were developed as part of that 1997 NAAQS policy decision, which makes 1997 an appropriate starting point for tracing the history of the use of $PM_{2.5}$ co-benefits in CAA-related RIAs. Creation of a list of RIAs to include in this study started with the identification of major regulations that the US EPA Air Office has submitted to OMB since 1997.[18] The list was then expanded to include RIAs for major air regulations still in the proposal stage.[19] This process identified 57 rules, 54 of which are final and 3 of which have been recently proposed and not yet been finalized.

---

[16] OMB (2003), pp. 1-2.

[17] The importance of extensive external review is highlighted in the sixth principle of the appropriate use of BCA in RIAs in Arrow *et al*. (1996), p. 221.

[18] This was done by going to http://www.reginfo.gov/public/do/eoAdvancedSearch# on the OMB website, and filtering for rules that met the following selection criteria: submitted by the EPA Air Office, concluded, final rule, major, and completed in any year since 1997. One rule that appeared on the resulting list was excluded (the renewable fuels standard known as "RFS2") because it is required under the Energy Independence and Security Act of 2007, not the CAA. One rule found on the OMB website fit the selection criteria, but did not appear when the filter was applied. This rule (the Petroleum Refineries NSPS rule concluded 12/10/08) was included.

[19] These were identified from RIAs posted on the EPA RIA website as of October 31, 2011 (at http://www.epa.gov/ttnecas1/ria.html). During initial research, the final RIA for the proposed air toxics rule for electricity generating units (EGUs) was all that was available. This rule, which is called the "EGU MACT rule" in this paper, was finalized in December 2011. This paper relies on results from the RIA for the final EGU MACT rule for its conclusions, but the paper quotes statements made by EPA and others that

A greater challenge was obtaining copies of the RIAs for all of the rules on this list. Neither OMB nor EPA maintains a complete, publicly-available record of RIAs. Also, an RIA is apparently not usually placed in its associated rulemaking docket. This situation may be a limitation in the completeness of dockets, or may be because RIAs are not required by the legal rulemaking procedures, but only by executive order.[20] Although *Federal Register* Notices of Rulemakings often summarize information from the RIAs, these sources were not always sufficient, because they may report total benefits estimated, but without the details needed to disaggregate those totals into direct benefits, $PM_{2.5}$ health-related co-benefits, and any other types of co-benefits.

At this point in time, this study has found either the final RIA itself, or sufficient information in other rulemaking documents to understand what EPA did to analyze the benefits and associated costs for 51 of the 57 rules identified, 48 of which are final rules and 3 of which are recently proposed rules. References for the source documents used are provided in Appendix A for all 57, with, where possible, page references for the relevant information on benefit and cost estimates found for each rule. Summary tables in the body of this paper are based on the references in Appendix A. Any RIAs that are quoted or described in greater detail in this paper are also listed in the References section of this paper.

This review of CAA RIAs since 1997 found that as EPA releases each of its proposed and final air quality rules, it typically emphasizes that the rule will generate health benefits that exceed its costs. However, close inspection of the associated RIAs reveals that a majority of those benefits – sometimes *all* of them – are from reductions in $PM_{2.5}$, even for air regulations that are targeting clean air objectives other than $PM_{2.5}$. For many of those regulations, the bulk of the benefits estimates in their RIAs are attributable to reductions in already-low concentrations of ambient $PM_{2.5}$ that EPA has predicted will occur *coincidentally* as a result of regulation of those non-PM pollutant(s).

Figure 1 provides a summary of the role of $PM_{2.5}$ co-benefits in all of the air regulation RIAs that were for rules <u>not</u> targeting $PM_{2.5}$-related health risks and which provided any quantified benefits estimates at all. The rulemakings are listed in chronological order (based on the date the rules were finalized), so that one can observe the trend over time since 1997 in how frequently EPA has relied on $PM_{2.5}$ co-benefits, and the general degree of importance those co-benefits played in lending EPA a benefit-cost case for each rule. Figure 1 shows that a trend towards almost complete reliance on $PM_{2.5}$-related health co-benefits has grown over time. The main exceptions in recent years have been rules addressing greenhouse gases (GHGs) under the CAA. (The GHG-related RIAs all report negative costs, so $PM_{2.5}$ co-benefits are not necessary to make a benefit-cost justification in any case.)

---

used numbers from the final RIA for the proposed EGU MACT rule. Care is taken to clarify where information in this paper refers to information released with the proposed EGU MACT rule.

[20] Although EO 12866 requires that all communications between OIRA and the agencies, White House and public be added to the docket, our experience has been that the RIAs themselves cannot been found in all the respective dockets.

Figure 1. Summary of Degree of Reliance on $PM_{2.5}$-Related Co-Benefits in RIAs Since 1997 for Major Non-PM Rulemakings under the CAA
(RIAs with no quantified benefits at all are not in this table. Where ranges of benefit and/or cost estimates are provided, percentages are based on upper bound of both the benefits and cost estimates. Estimates using the 7% discount rates are used in all cases.)

Year	RIAs for Rules Not Targeting Ambient PM 2.5	PM Co-Benefits Are >50% of Total	PM Co-Benefits Are Only Benefits Quantified
1997	Ozone NAAQS (.12 1hr=>.08 8hr)	×	
1997	Pulp&Paper NESHAP		
1998	NOx SIP Call & Section 126 Petitions		
1999	Regional Haze Rule	×	
1999	Final Section 126 Petition Rule	×	
2004	Stationary Reciprocating Internal Combustion Engine NESHAP	×	
2004	Industrial Boilers & Process Heaters NESHAP	×	×
2005	Clean Air Mercury Rule	×	
2005	Clean Air Visibility Rule/BART Guidelines	×	
2006	Stationary Compression Ignition Internal Combustion Engine NSPS		
2007	Control of HAP from mobile sources	×	×
2008	Ozone NAAQS (.08 8hr =>.075 8hr)	×	
2008	Lead (Pb) NAAQS	×	
2009	New Marine Compress'n-Ign Engines >30 L per Cylinder	×	
2010	Reciprocating Internal Combustion Engines NESHAP – Comp. Ignit.	×	×
2010	EPA/NHTSA Joint Light-Duty GHG & CAFES		
2010	SO2 NAAQS (1-hr, 75 ppb)	×	> 99.9%
2010	Existing Stationary Compression Ignition Engines NESHAP	×	×
2011	Industrial, Comm, and Institutional Boilers NESHAP	×	×
2011	Indus'l, Comm'l, and Institutional Boilers & Process Heaters NESHAP	×	×
2011	Comm'l & Indus'l Solid Waste Incin. Units NSPS & Emission G'lines	×	×
2011	Control of GHG from Medium & Heavy-Duty Vehicles		
2011	Ozone Reconsideration NAAQS	×	
2011	Utility Boiler MACT NESHAP *(Final Rule's RIA)*	×	≥ 99%
2011	Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP	×	
2011	Sewage Sludge Incineration Units NSPS & Emission Guidelines	×	×

The practice of including co-benefits in RIAs is not new. Examples where co-benefits of one sort or another played a significant role in the evaluation of an RIA can be found as early as 1984.[21] However, the current situation is dramatically different from past examples. First, the pollutant for which co-benefits are being claimed is already stringently regulated – in fact, the CAA language defining the required stringency implies stringency greater than the BCA optimum (this point is explained in the next section). Second, these $PM_{2.5}$ co-benefits not only dominate the majority of RIAs for EPA's non-PM rules, but in many cases they are the only benefit that is being quantified at all.

Section 6 will discuss a number of reasons why these $PM_{2.5}$ co-benefits are overstated. That discussion also implies that the direct benefits that EPA is estimating in its RIAs for rules that do directly address $PM_{2.5}$ risks are also overstated. However, the intervening two sections first address the reasons why reliance on $PM_{2.5}$ co-benefits in non-PM rulemakings is inappropriate theoretically, inconsistent with the stated objectives of requiring RIAs to be prepared, and promotes excessively complex, duplicative and cost-ineffective regulation for the nation.

4. EPA's Use of $PM_{2.5}$ Co-Benefits in RIAs Is Inconsistent with Theoretical Underpinnings of Benefit-Cost Analysis

Although RIAs are no longer intended to serve solely as BCAs, BCA is clearly a core tenet of the exercise of producing RIAs. For this reason, it is important to explore from a theoretical perspective what BCA implies about the use of "co-benefits." As this section will demonstrate, the theoretical underpinnings of BCA do not support EPA's practice of adding co-benefits of separately-regulated pollutants into the total benefits estimates in RIAs, even when there are significant interrelationships between the pollutants in terms of the technologies that control them, their atmospheric formation, or health or welfare responses. In fact, the analysis in this section demonstrates that EPA's co-benefits practice actually moves the resulting benefit-cost comparison *in the wrong* direction from what BCA would advise if the pollutants for which co-benefits are being calculated are criteria pollutants already regulated under a NAAQS.

The purpose of BCA when applied to a policy choice, such as determining the level of an emissions standard, is to identify the level that would provide the highest net benefits (and also to ensure that those net benefits would be positive). The optimization objective for choosing a required amount of reduction of a single pollutant is mathematically stated as:

$$\underset{\{P\}}{\text{Maximize}}\ B(P)-C(P) \qquad [i]$$

where "P" stands for the amount of reduction in the pollutant in question, B(P) represents the benefits achieved given the amount of emissions reduction P, and C(P) represents the

[21] See, for example, the case study on the RIA for the lead phase-out regulations by A. Nichols in Chapter 4 of Morgenstern (1997).

costs of achieving the emissions reduction P. The BCA-optimal degree of reduction is identified by mathematically differentiating equation (i) with respect to P and setting it equal to zero, as in equation (ii):

$$dB/dP - dC/dP = 0 \qquad \text{[ii]}$$

This is known as the "first-order condition" for the optimization. One can see from equation (ii) that in the single-pollutant situation the BCA-optimal reduction for P is where the marginal benefit of P equals the marginal cost of P.

Consider now the somewhat more complex situation of performing a BCA for multiple pollutants simultaneously. For example, if two pollutants are to be controlled so that the net benefits of the combined regulations are optimized, the optimization objective is written as:

$$\underset{\{P,\,H\}}{\text{Maximize}}\ \ B(P,H)-C(P,H) \qquad \text{[iii]}$$

where P stands for reduction in the first pollutant and H for reduction in the second pollutant.[22] Optimization of equation (iii) requires the simultaneous solution of two equations. The two equations are the first-order conditions for each pollutant separately, which requires differentiating the above objective function first with respect to P and second with respect to H and setting both equal to zero:

$$\partial B/\partial P - \partial C/\partial P = 0 \qquad \text{[iv-1]}$$

$$\partial B/\partial H - \partial C/\partial H = 0 \qquad \text{[iv-2]}$$

Equations (iv-1) and (iv-2) show that if the standards for multiple pollutants can be selected simultaneously, then the level at which to set each one is decided without any reference to the changes in benefits or costs that would come from any other of the simultaneously-regulated pollutants. In other words, there is no term in the joint optimality conditions that represents co-benefits from any of the other pollutants. The decision about levels for H must be made assuming that P will be set at its optimal level, and vice versa.

In reality, the CAA does not give EPA the ability to actually set regulations in this simultaneous manner. The optimality rules become more complex when one must choose the level of one pollutant given that levels of other, interrelated pollutants have been *separately* established. This more realistic BCA situation can be represented by building on the two-pollutant BCA optimization of equation [iii]. Assume now that the

---

[22] For a person who wishes to read through this section with a specific emissions control policy example in mind, think of P as representing $PM_{2.5}$, and think of H as representing an air toxic such as mercury. As $PM_{2.5}$ precursors are reduced, so too is mercury, and vice versa. Total benefits, B, are a function of the amount of reduction in both $PM_{2.5}$ and mercury. Similarly, changes in policy costs, C, are a function of the reduction in both $PM_{2.5}$ and mercury that must be achieved.

policymaker can only control one pollutant, say by choosing H, while accepting that P has already been regulated to the level $P=P^N$. Now, the optimization objective is stated as:

$$\underset{\{H\}}{\text{Maximize}} \;\; B(P^N,H)-C(P^N,H) \qquad [v]$$

This gives rise to a single first-order condition for H which replaces the first-order condition for H in equation (iv-2):

$$(\partial B/\partial H - \partial C/\partial H) = (\partial C/\partial P - \partial B/\partial P)*dP/dH, \textit{ evaluated at } P=P^N \qquad [vi]$$

Equation (vi) is similar to equation (iv-2), except that instead of choosing H make the left-hand side (LHS) equal to zero, the LHS now should be made equal to the complex term that appears on the right-hand side (RHS) of equation (vi). This RHS term includes the marginal cost and marginal benefit of P (evaluated at $P=P^N$), as well as a term dP/dH. These elements of the RHS term clearly relate to co-benefits from the other separately-regulated pollutant, P, but the RHS term is not equal to P's co-benefits, specifically.[23] Thus, equation (vi) reveals how interactions of P with H would properly be incorporated into a BCA for choosing the optimal level of H. One cannot conclude that there is a theoretical rationale for including co-benefits of separately-regulated pollutants in a BCA for setting a standard on another pollutant unless the term on the RHS of equation (vi) were equal to what EPA calculates as co-benefits (*i.e.*, $\partial B/\partial P*dP/dH$), which it clearly is not. In fact, as will be explained below, equation (vi) actually implies that adding a separately-regulated pollutant's co-benefits into a BCA-based evaluation of any other pollutant regulation can drive the resulting regulation in the opposite direction from what BCA would advise.

The first thing to note about the RHS term of equation (vi) is that if P^N were to be set at the BCA-optimal level for P, then the RHS would equal zero.[24] In that case, equation (vi) would be identical to equation (iv-2), thus indicating that H should be selected without any reference to any co-effects from P. In this case, co-benefits of a separately-regulated pollutant clearly should not play any role in the BCA for another pollutant.

Consider now the alternative case in which P^N is *not* set at the BCA-optimal level for P. Although the RHS of equation (vi) is non-zero in this case, it still indicates that EPA's method of including co-benefits from P in a BCA for H is inappropriate. The non-zero RHS term can be interpreted as a measure of the degree to which the non-optimized choice of P *diverges* from its own first-best optimality condition, multiplied by dP/dH. If

---

[23] For example, dP/dH, which stands for the change in the level of P when H is changed, reflects coincidental additional reduction in P beyond its existing reduction level for the given reduction requirement on H. In other words, dP/dH is the physical co-*reduction* in P when a standard is set on H. Thus, the co-*benefit* from coincidental changes in P that result from choosing a level for H, which is what EPA includes in its RIAs, would be equal to $\partial B/\partial P*dP/dH$.

[24] This is because the portion of the RHS term in parentheses is consistent with the LHS of equation (iv-1), and thus equals zero if P^N is set at its optimal level where its own marginal costs equal its own marginal benefits, as prescribed by equation (iv-1).

co-benefits are positive, and P^N is over-controlled with respect to its BCA-optimal level, then the RHS of equation (vi) will be positive, because over-control occurs when the marginal costs of P have exceeded its marginal benefits, which in turn means that $(\partial C/\partial H - \partial B/\partial H) > 0$.[25] Thus, if P has been *over*-controlled with respect of its BCA optimum, equation (vi) tells us that H should then be *under*-controlled with respect to *its* BCA optimum. That is, the LHS of equation (vi) will need to be positive too, which means that H should not be controlled beyond a point where its own marginal benefits become equal to or less than its own marginal costs.[26] Similarly, if P is under-controlled, then equation (vi) indicates that H should be controlled to a level beyond its own BCA-optimum.

The case of P being *over*-controlled is of particular interest and relevance to $PM_{2.5}$ co-benefits. This is because the legal framework that determines what the EPA Administrator must select as a standard for ambient $PM_{2.5}$ clearly drives towards over-control of $PM_{2.5}$ with respect to its own BCA optimum.[27] The key implication is that if $PM_{2.5}$ co-benefits should be considered at all in a BCA for another pollutant (such as air toxics or ozone), they should enter as a *negative* term on the benefits side of the ledger, and not as a positive one. Thus, estimates of the co-benefits from $PM_{2.5}$ *specifically* should not be incorporated into BCAs for any other pollutants being controlled under the CAA, and when they are included, this only serves to drive other policies *away* from what would be their appropriate level, based on BCA principles.

In fact, this conclusion can be stated more broadly, that co-benefits from any pollutant that is regulated as a criteria pollutant with a NAAQS that conforms with the requirements of CAA Section 109 should not be included in the BCA of any other pollutant. This is because Section 109 requires over-control of all criteria pollutants with respect to their own BCA optima, due to Section 109's requirement for an adequate margin of safety when setting a NAAQS.

---

[25] Because co-benefits are positive, dP/dH is positive (*i.e.,* if pollutant H is reduced, then pollutant P is also reduced), so the sign of the term on the RHS is equal to the sign of the term in parentheses.

[26] The degree of under-control of H is not easily inferred without more situation-specific technical information, but the term that determines how much to over- or under-control H is not determined by simply adding the monetary co-benefits of P to the BCA for H. One can determine however that the amount of adjustment is less than the monetary co-benefits of P: the adjustment should be equal to $(\partial C/\partial P - \partial B/\partial P) * dP/dH$, and this is less than $\partial B/\partial P * dP/dH$, which is the monetary measure of co-benefits of P.

[27] The requirement that a NAAQS be set at a level that protects the public health with a margin of safety is equivalent to saying that it should be set at a point where no further material public health improvements would be expected to be gained by tightening the standard any further. In fact, the "margin of safety" implies that the standard be set a notch beyond that point of no further expected gains. In terms of a benefits curve, this would be a point where the benefits curve (as a function of ambient pollutant reduction) becomes flat, or at least that there is a significant probability in the mind of the Administrator that it is flat by that point. When the benefits curve is flat, marginal benefit equals zero. However, since marginal costs will always be increasing for all levels of reduction up to 100% reduction, the point where the Administrator must set the NAAQS is a point where marginal costs are greater than marginal benefits. Thus the point where marginal cost *equals* marginal benefit, which defines the BCA-optimal degree of reduction, is at a lesser level of than what the CAA requires for a NAAQS determination.

The fact that the CAA results in over-control of criteria pollutants *with respect to their BCA-optima* can be no surprise to analysts familiar with BCA. However, it might be useful to bring this point into the open in the way RIAs are conducted for criteria pollutants (and any other pollutants that must be regulated without regard to costs). If a NAAQS has indeed been set to a point where it provides an adequate margin of safety, its RIA *should* show that it fails the marginal BCA test. If RIAs are to more effectively serve in the role of providing sound and policy-relevant information to policymakers and the public about the impacts of new regulations, they should strive to actually quantify the net cost that our society is paying for that margin of safety in its air pollution rules. The purpose of recrafting CAA-related RIAs in this manner would not be to demonstrate that NAAQS rules fail a benefit-cost test that the CAA does not allow in the first place; rather, it would be to inform ourselves about the *degree* to which we are accepting higher costs in order to have the extra degree of public health safety that is mandated by the CAA, and apparently desired by the public.

To summarize, the analysis in this section establishes that EPA's practice of adding co-benefits of pollutants that are separately subject to policymaker control into the total benefits reported in RIAs for other pollutants is not supported by BCA theory. This conclusion holds whether or not the other pollutants are being regulated to their own BCA-optimal levels. This analysis has also demonstrated that EPA's practice of including co-benefits of criteria pollutants in its RIAs actually intensifies the pressure towards over-control relative to what BCA would prescribe that already exists in the standard-setting requirements of the CAA.

5. EPA's Use of $PM_{2.5}$ Co-Benefits Subverts the Practical Purpose of RIAs as an Informational Device for Improving Policy Making

As noted in Section 2, RIAs are no longer viewed as having to justify policy decisions based solely on BCA principles. They are now viewed as assessments that should help inform the public and policymakers on a variety of impacts that can be anticipated to result from each new major regulation, all of which are viewed as having relevance to societal objectives. People who feel that this structured informational role of RIAs is more important than its BCA-related role *per se* may argue that there is merit in understanding each rule's co-benefits, even those that come from separately-regulated co-pollutants such as $PM_{2.5}$. Unfortunately, EPA's use of $PM_{2.5}$ benefits as the predominant (often the *only*) quantified benefit in RIAs undercuts the objective of providing the public with a transparent understanding of the relative merits of each type of regulation.

Quantification of $PM_{2.5}$ co-benefits creates particular problems when they are reported as part of the total benefits of a rule in the Executive Summary of an RIA. Including them in the summary of the RIA, which is where the benefits of a rule are compared to its costs, and then also including them in public announcements about net benefits and benefit-cost ratios of new rules creates confusion for the public and other audiences who have little time to study the details of the underlying analysis in each RIA. An example of the confusion that can be created is in the following quote from the EPA Administrator

regarding the benefits case for controlling air toxics from electricity generating units (EGUs), known as the "Proposed EGU MACT" rule:

> *When these new standards are finalized, they will assist in preventing 11,000 heart attacks, 17,000 premature deaths, 120,000 cases of childhood asthma symptoms and approximately 11,000 fewer cases of acute bronchitis among children each year. Hospital visits will be reduced and nearly 850,000 fewer days of work will be missed due to illness.*[28]

The fact is that every one of the benefits in the quote above comes from EPA's predicted $PM_{2.5}$ co-benefits, and not from any of the reductions in air toxics that are the purpose of that rule.[29] Anyone with the time and interest can read the RIA from which these numbers came, and may realize that these are $PM_{2.5}$-related benefits. However, not every such motivated reader would also realize that $PM_{2.5}$ is not an air toxic, and that those $PM_{2.5}$-related benefits would be more certain and more cost-effectively obtained through a different regulation altogether than an air toxics rule (*i.e.*, through the $PM_{2.5}$ NAAQS). By including those $PM_{2.5}$ co-benefits as part of the "total benefits" reported in the RIA for the Proposed EGU MACT rule, EPA encourages this misunderstanding.[30] The statements can become even more misleading in the hands of advocates outside of EPA. For example, the following statement in testimony by an advocate outside of EPA before the House Energy and Commerce Committee on the Proposed EGU MACT rule gives the distinct impression to readers that air toxics from power plants are killing and otherwise harming massive numbers of people:

> *EPA's proposed mercury and air toxics standards for power plants that burn coal and oil are projected to save as many as 17,000 American lives every year by 2015. These standards also will prevent up to 11,000 cases of heart attacks, 120,000 cases of asthma attacks, 11,000 cases of acute bronchitis among children, 12,000 emergency room and hospital visits and 850,000 lost work days every year.*[31]

---

[28] Quote from Administrator Jackson in EPA Air News Release (HQ), "EPA Extends Public Comment on Mercury and Air Toxics Standards," June 21, 2011. (In the RIA for the final EGU MACT rule the "17,000 premature deaths" has been reduced to 11,000, but this quote was made before the RIA for the final EGU MACT was released in December 2011.)

[29] This can be confirmed by reviewing the RIA for the Proposed EGU MACT rule, which is EPA (2011a).

[30] Any counter-argument that co-benefits are essential to add into an RIA's statement of a new rule's total benefits would require one to argue that RIAs have a BCA function. As has been demonstrated above, the principles of BCA, when scrutinized, prescribe that co-benefits of an already-regulated pollutant not be added in the comparison of benefits and costs of a regulation for a different pollutant.

[31] John D. Walke, Natural Resources Defense Council, Testimony at Hearing on "Recent EPA Rulemakings Relating To Boilers, Cement Manufacturing Plants, And Utilities," before the Subcommittee on Energy and Power, Committee on Energy and Commerce, U. S. House of Representatives, April 15, 2011.

EPA's reliance on co-benefits estimates thus undercuts the transparency that RIAs are supposed to bring to assessments of the impacts of new rules. Lack of transparency enables misleading advocacy.

Reliance on co-benefits creates another problem that undercuts the practical value of RIAs: it shields EPA from pressure to improve its ability to describe, characterize and even quantify the health and welfare benefits for the other pollutants that it is charged with regulating. The EGU MACT RIA is not the worst case in this regard. As Figure 1 shows, during the period 2009-2011, EPA released 13 CAA-related RIAs that provided quantitative estimates of benefits and in which $PM_{2.5}$ was a co-benefit rather than a direct benefit. $PM_{2.5}$ co-benefits accounted from more than half of the total benefits in all but 2 of the 13.[32] EPA did not even attempt to quantify the direct benefits in 6 of those RIAs: $PM_{2.5}$ co-benefits accounted for 100% of the total benefits identified in those RIAs. In two more of those 13 RIAs, although some direct benefits estimates were provided, $PM_{2.5}$ co-benefits accounted for more than 99% of the total reported benefits. In essence, EPA has been abdicating its responsibility to make a clear direct benefits case for its air rules, particularly those for air toxics. Furthermore, although EPA has quantified direct benefits for the new standards it has set for other criteria pollutants, it is relying on its $PM_{2.5}$ co-benefits estimates to create its case that those other NAAQS revisions will produce benefits greater than their costs, when in fact their direct benefits are often miniscule compared to their costs.

Clearly, EPA's $PM_{2.5}$ co-benefits habit is allowing EPA to avoid grappling with the important task of making a case that all of these other pollutants really require tighter controls. It may be possible for that case to be made for some of those pollutants, but a high degree of complacency and analytical laziness has instead taken root as EPA has found it can more easily rely on simplistically-derived estimates of co-benefits from a pollutant that it has every authority it needs, and indeed the legal *requirement*, to directly regulate to levels that are safe for the public health. This situation is completely at odds with the purpose of RIAs, which is to provide a consistent, credible and thoughtful evaluation of the societal value gained with the increased regulatory burden that new rulemakings create. It also stymies scientific progress in risk assessment techniques and associated knowledge.

In summary, $PM_{2.5}$ co-benefits have become a device for keeping some regulations of dubious public policy value from transparent scrutiny. Although many of those regulations may be mandated by law, the degree of stringency imposed requires judgment by the Administrator, and co-benefits may be masking judgments that would not otherwise pass scrutiny. On a longer-term basis, this practice is also preventing RIAs from playing their most meaningful practical role, which is to help the policymaking

---

[32] The two RIAs out of the 13 rulemakings during 2009-2011 in which $PM_{2.5}$ co-benefits were not the predominant form of quantified benefits were rules to reduce mobile source greenhouse gases. EPA estimated negative costs for both of those greenhouse gas rules, so their benefits would have exceeded their costs even with zero direct benefits. EPA has developed a method for quantifying benefits from reductions in greenhouse gases, so while it does also report $PM_{2.5}$ co-benefits is in those two RIAs, the emphasis on them as the benefit-cost justification for the rule is less pronounced.

community identify those frameworks for regulation that may not be providing the value that they were originally expected to provide when enacted or otherwise established. Including $PM_{2.5}$ co-benefits in all air-related RIAs thus is preventing any meaningful identification of ways to reduce regulatory burdens while still meeting our national air quality objectives.

6. $PM_{2.5}$ Risk Estimates Have Become Increasingly Less Credible as EPA's Use of Them as Co-Benefits in Non-PM RIAs Has Grown

This paper has demonstrated that BCA theory does not support the role that EPA gives to $PM_{2.5}$ co-benefits, and that excessive reliance on them in its regulatory justifications is undercutting the more general purposes of RIAs for guiding policy making. This review of EPA's RIAs has also found that as EPA has used $PM_{2.5}$ co-benefits to justify more and more of its non-$PM_{2.5}$ rules, it has also moved to less and less scientifically-credible methods for estimating those co-benefits. These changes in methodology and assumptions have inflated the $PM_{2.5}$ co-benefits estimates dramatically (and also the direct $PM_{2.5}$ benefits estimates in rulemakings targeting $PM_{2.5}$).

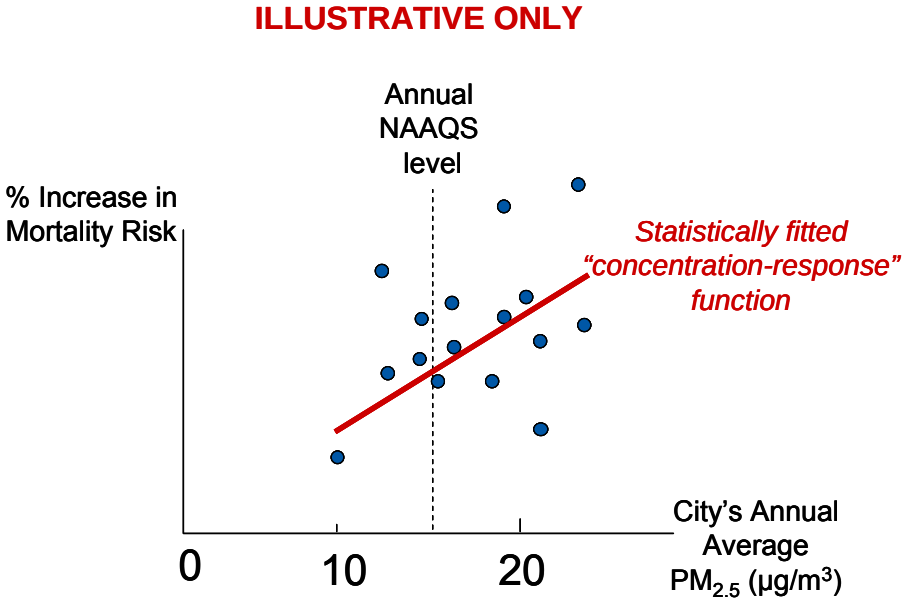
To explain the methodological issues, it is necessary that the reader first understand the general elements of the scientific basis for EPA's $PM_{2.5}$ risk calculations. These calculations are all based on the presumption that statistical correlations between health effects levels and ambient air quality are causal in nature. The illustrative example of how these studies are performed and then used to calculate risk changes from hypothetical changes in ambient $PM_{2.5}$ levels will be based on the "chronic exposure" studies that are the starting point for EPA's estimates of mortality risks from $PM_{2.5}$. The focus of this discussion will be on mortality because $PM_{2.5}$ mortality benefits estimates account for over 90% (and as much as 97%) of the total $PM_{2.5}$-health benefits estimates in EPA's RIAs. All of the categories of $PM_{2.5}$ morbidity benefits account for as little as 3% of the estimated $PM_{2.5}$ co-benefits estimates, particularly when the upper-bound mortality estimates are reported.[33]

For the chronic exposure mortality-risk studies, a database is created of many individuals living in multiple cities across the US. Researchers then track the survival outcomes of those individuals over time to build up estimates of the relative mortality risk at each age level in each of the different cities where these individuals, or "cohorts" live. After enough deaths have been observed (which can require as much as a decade), the researchers assess whether a statistical correlation exists between the estimated relative mortality risk in each city and the cities' average ambient $PM_{2.5}$ concentrations. This statistical analysis also attempts to control for all the other major factors that contribute to mortality risk, which is a daunting and perhaps elusive goal, given limitations in the availability of the relevant data.

[33] A single morbidity category, chronic bronchitis, accounts for about half of all the morbidity benefits value. EPA's estimates of chronic bronchitis risks are also based on a "chronic exposure" type of study. The rest of the morbidity benefits (as little as 1.5% of the total $PM_{2.5}$ co-benefits) are based on "acute exposure" types of studies that differ in a number of ways from the illustrative example that this section provides.

Figure 2 provides a simplified illustration of the way that population risk information from the chronic exposure studies produces an equation that EPA uses to calculate mortality risks from current and changed levels of $PM_{2.5}$. Each dot in the figure represents the percent increase in mortality risk for an entire city,[34] plotted against each city's respective annual average monitored ambient $PM_{2.5}$ concentration. The heights of the dots on the vertical axis should be viewed as the percent differences in mortality risks that remain across the cities after first controlling for and removing other risk factors for which data can be obtained (*e.g.*, age, income level, smoking status, weight, local climate, *etc*.). The placement of each dot on the horizontal axis reflects that city's average concentration of ambient $PM_{2.5}$ as measured at central monitoring stations. The statistical analysis then estimates the line through these data points that provides the most likely explanation of their scatter.[35] The most important attribute of this line is its slope, *i.e.*, the percent risk increase per additional $\mu g/m^3$ of ambient $PM_{2.5}$. The estimated slope is a single constant percent per $\mu g/m^3$ from the city with the lowest measured $PM_{2.5}$ concentration to that with the highest measured concentration. This statistically-fitted curve is called the "concentration-response" function, because it associates risk with city-wide concentrations of ambient $PM_{2.5}$ measured at monitoring stations. Monitored concentrations serve as rough proxies for individuals' exposures to $PM_{2.5}$, which certainly vary among the individuals within a city, but are not known.

Figure 2. Illustration of Basis of Concentration-Response Functions in Cross-City Comparisons of Relative Mortality Risks



[34] The absolute risk varies by age, but the statistical estimation method used in the $PM_{2.5}$ chronic exposure mortality studies (the "Cox Proportional Hazards model") assumes that any increase in risk due to a city's average ambient $PM_{2.5}$ level increases risk by the same proportion for all age groups. Thus, relative risks can be summarized as a single dot for each city in the study.

[35] The formula for the fitted relationship is in a "log-linear" form. It appears as a line in the illustrative figure because the y-axis units are the *percentage* increase in risk per increment of $PM_{2.5}$ concentration, which is stated in absolute units.

The ambient concentrations observed in each study fall within a range. For example, in the illustrative figure, the range is roughly from 10 $\mu g/m^3$ to 25 $\mu g/m^3$. There is no factual evidence to indicate the shape of the concentration-response function below or above this range, which is why the red line in Figure 2 does not extend beyond the range of the observed data. A term that will figure prominently in the discussion below is the *lowest measured level* (LML) of ambient average $PM_{2.5}$. This term refers the average city-wide $PM_{2.5}$ concentration of the "cleanest" city in the dataset from which a concentration-response function has been estimated. In the illustrative figure, the LML is about 10 $\mu g/m^3$.

Each chronic exposure study has a different number of cities, or data points. One widely-cited database is called the American Cancer Society (ACS) cohort. This database includes people (the "cohort") residing in over a hundred cities. Statistical estimates of a $PM_{2.5}$-mortality slope based on the ACS cohort usually include from 50 to 160 cities, or data points as shown in Figure 2. A commonly-cited slope estimate based on the ACS cohort is from Pope *et al.* (2002), which is often used by EPA to estimate its lower bound $PM_{2.5}$ mortality benefits estimates. Another widely-cited database is the Harvard "Six-Cities" cohort. As its name implies, it offers a slope estimated from only six data points. One slope estimate based on the Six Cities cohort is reported in Laden *et al.* (2006), which is notable here as the study on which EPA presently bases its upper bound $PM_{2.5}$ mortality benefits estimates.

Most (but not all) $PM_{2.5}$ researchers studying the ACS, Six-Cities, and several other cohort datasets have reported that the estimated slope of the concentration-response curve is positive, and statistically significant. However, this fact does not eliminate uncertainties about the size of the risk, nor about whether the association is causal. A discussion of the many uncertainties that remain is provided before turning to how the estimated relationship is being used by EPA to predict benefits from regulations that would reduce ambient concentrations of $PM_{2.5}$ in certain areas of the US.

First, there are usually relatively few data points near the upper and lower ends of the range of ambient $PM_{2.5}$ data. This causes the confidence interval on the slope estimate to widen progressively as one moves from concentrations near the average among the cities studied out to the extremes of the observed data. The confidence interval on the relative risk associated with cities with concentrations at the LML may be very wide. This means that the slope may be lower or higher than that which has been estimated over the full range of data. One may not even be able to statistically assert that the $PM_{2.5}$-risk relationship is non-zero for concentrations at or near the LML, even when the average slope estimated over the full range of $PM_{2.5}$ levels is statistically significant. Thus, there is much greater uncertainty about the size of the $PM_{2.5}$ effect at lower ambient concentrations, such as at the LML, than is usually acknowledged.

Second, data point "scatter" lies beneath the average relationship that the fitted line summarizes. This implies that the estimated concentration-response curve will be a poorer predictor of the change in risk that will be experienced in any specific city than it may be for predicting average risks over many different cities. It also follows that

uncertainties in predicted responses to reductions in $PM_{2.5}$ levels in cities that were not in the original dataset will be even greater than the statistical confidence intervals imply.

Third, there is great uncertainty on the true shape of the concentration-response relationship. Researchers report they have not been able to identify any shape that is statistically superior to the log-linear form, but this does not mean that the actual relationship is log-linear. There are numerous problems in the quality of the data being used that can undercut the ability to detect shape. Thus, even within the observed data range, uncertainty remains about the shape of the estimated concentration-response function. However, there is no ability at all to determine statistically whether or not the slope of the curve continues unchanged below the LML, as ambient $PM_{2.5}$ concentrations approach near-zero levels, because there are no observations in that range at all.[36] In situations such as this, researchers usually attempt to use mechanistic understanding of the phenomenon being estimated to guide shape assumptions. However, EPA has not been able to provide any mechanistic explanation of how current ambient levels of $PM_{2.5}$ may increase risk of death, and so there can be no help from mechanistic reasoning. Any extrapolation of the concentration-response relationship below the LML is therefore subject to much greater uncertainty than the statistical confidence intervals might suggest.

Fourth, there is substantial uncertainty in defining the appropriate concentration to serve as the best proxy for levels of $PM_{2.5}$ to which the individuals in the study cohort have been exposed.[37] EPA now states that the LML for the ACS cohort is 7.5 $\mu g/m^3$, and 10 $\mu g/m^3$ for the Six-Cities cohort. However, the LML for the ACS cohort averaged about 10 $\mu g/m^3$ during 1979-1983, which spans the time that cohort was recruited (in 1982). The LML for the Six-Cities cohort averaged about 11 $\mu g/m^3$ during 1979-1985, although that cohort was recruited earlier still, in 1974-1977. But even relying on these earlier, higher concentration levels as estimates of the levels that might account for observed differences in mortality risk levels is open to question. Recall that the estimates of differences in mortality risk across cities are built up by following the survival outcomes of the people in each city over many years. This means that the observations of their mortality risks at each age, if attributable to air pollution at all, could be a result of exposures they experienced many years in the past, or that they accumulated over a long period of time.

Take the ACS cohort as an example. The ACS cohort was first established in 1982. At the time that the individuals were recruited for the ACS study, they had to be at least 30 years old and their average age in 1982 was 56 years. Thus all of the individuals in the ACS database had been exposed to US pollution levels since at least 1952 (*i.e.*, 30 years before 1982), and the average individual in the database experienced US pollution levels

---

[36] Efforts to explore shape near the LML have produced inconsistent results. Some researchers report finding upward curvature and others report finding downward curvature. At present, no consensus or weight of evidence can be said to exist on this matter.

[37] Recall also that the term "concentration-response function" is used to remind us that none of these studies actually measure what *exposures* the individuals tracked were receiving. An assumption is made that the average exposure across individuals in each city can be approximated by the readings at that city's ambient monitors. All individuals in a city are assumed to be exposed to the same concentrations.

dating back to 1926. As researchers using the ACS database have stated "In the 1950s, levels of air pollution in most North American and European cities were 10 to 50 times higher than those found today."[38] Since the mortality risk estimated for each city is based on many years of tracking these people, recent average $PM_{2.5}$ concentrations such as those in 2000 cannot be viewed as indicative of the $PM_{2.5}$ exposure level that most affected their observed survival outcomes. Those individuals who had not already died by 2000 would have already lived at least 44 years of their lives while being exposed to earlier, higher $PM_{2.5}$ levels. To say that the estimated mortality-risk relationship has been observed down to the level of the lowest $PM_{2.5}$ concentration most recently measured in any of these cities is close to assuming that recent lower levels of $PM_{2.5}$ accounted for the health outcomes of people who died as much as several decades ago. The same issues are present with the Six-Cities and all other cohorts being used in $PM_{2.5}$ epidemiological studies of risks due to chronic exposures to $PM_{2.5}$.

Fifth, none of the $PM_{2.5}$-risk estimates that EPA relies on for a concentration-response function slope assumption has been estimated while also accounting for the relative levels of pollutants other than $PM_{2.5}$. The presumption is being made that $PM_{2.5}$ is the sole air pollutant contributing to observations of an increased average mortality risk associated with higher average ambient pollution. Nevertheless, some studies have controlled for other pollutants. For example, the ACS cohort's slope with respect to $PM_{2.5}$ was found in 2000 to be much smaller and statistically insignificant when another pollutant (SO_2) was included in the analysis.[39] Since 2000, not a single study based on that ACS cohort has reported an estimate of the $PM_{2.5}$ slope that came from a model that also accounted for SO_2.

Sixth, unlike other pollutants, the chemical and physical composition of $PM_{2.5}$ varies over space and time,[40] but none of these statistical studies have sufficient data yet to try to determine the degree to which some $PM_{2.5}$ constituents account for more of the observed associations than other constituents. The concentration-response functions that EPA constructs from these types of epidemiological studies all assume that every one of the multiple types of $PM_{2.5}$ is equally potent. This assumption is not realistic when one considers the wide variety in the chemical properties of the many major components of ambient $PM_{2.5}$ concentrations. This fact creates substantial unquantified uncertainty in estimates of benefits from future decreases in $PM_{2.5}$ concentrations except in the unlikely case there all the individual constituents would be reduced by the same percentage. This source of uncertainty in benefits estimates becomes particularly extreme for regulations would only reduce one type of $PM_{2.5}$ constituent. Reduction of that single type of $PM_{2.5}$

---

[38] Krewski *et al*. (2000), p. 33.

[39] Krewski *et al*. (2000).

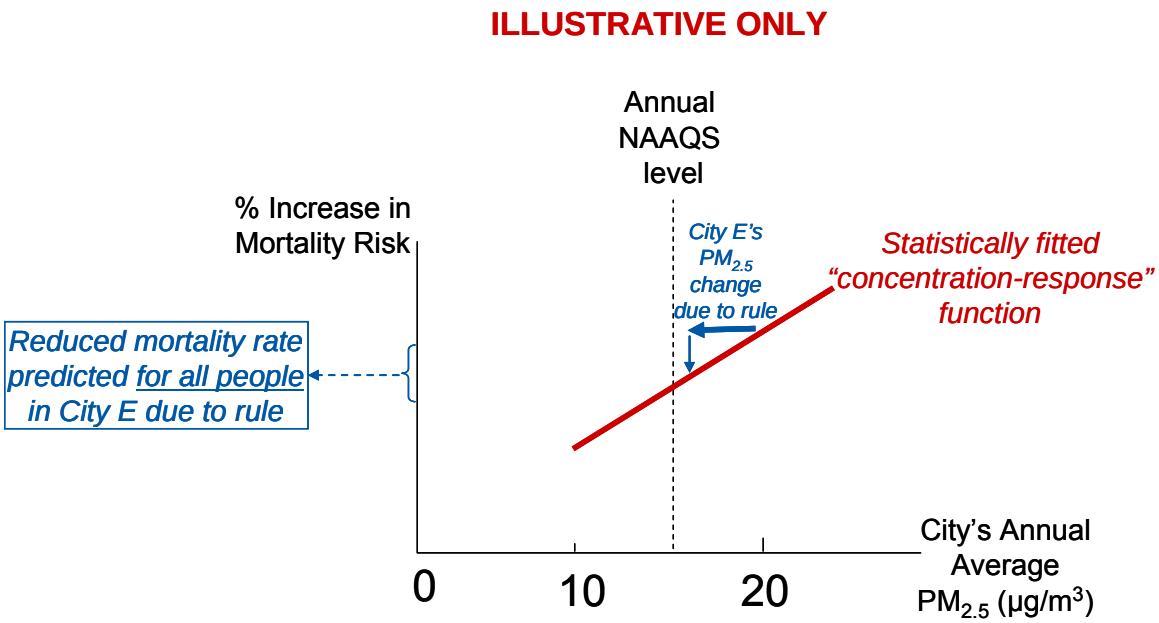
[40] $PM_{2.5}$ comprises all compounds in the ambient air that are not in the form of a gas; it includes compounds that are as physically different as solid particles and very fine liquid droplets. Chemically, the constituents that may be found in the ambient mass that counts as $PM_{2.5}$ include a diversity of compounds including dust, soot (elemental carbon), sulfates, nitrates, and secondary organic compounds. Some are soluble and some are insoluble, and each has its own distinct physiological impact when inhaled. Not only is $PM_{2.5}$ a hodge-podge of compounds, but the mix differs dramatically from location to location and temporally at any given location.

could have no effect at all on health, or it could have even greater effect than EPA predicts using its equal-toxicity assumption. EPA has never attempted to quantify this uncertainty in any of its benefits estimates. This is a particular concern for co-benefits estimates in RIAs for non-PM rules, because co-benefits are often based on changes in a single constituent that happens to be linked with reductions of the non-PM pollutant. For example, almost all of the 11,000 deaths attributable to $PM_{2.5}$ co-benefits in the Final EGU MACT rule RIA are due to reductions in sulfates alone.

These six uncertainties represent just a few of the uncertainties that exist for the "concentration-response" function's ability to predict how much mortality will be reduced if national ambient $PM_{2.5}$ is decreased. At present, the only statement of statistical uncertainty that EPA provides for an estimate based on any individual epidemiological study reflects only the statistical confidence of the overall slope of the estimate. As explained above, this is not an appropriate measure of the uncertainty of predictions of risk at concentrations at the extreme ends of the observed dataset, such as those at and just above the LML. But these six types of technical problems imply larger uncertainties than even the expanded confidence bounds would imply, and undercut confidence in interpreting the statistical association as causal in nature. Nevertheless, EPA uses the slope estimates from these studies to predict risk from changes in $PM_{2.5}$ that will occur in the future, and in many locations that were not even studied, as explained next.

The next figure, Figure 3, illustrates how EPA uses the slope that is estimated from cohort studies to project $PM_{2.5}$ deaths due to changes in baseline $PM_{2.5}$ levels. *First, and foremost, EPA starts by presuming that the statistically-estimated concentration-response slope represents a causal relationship with $PM_{2.5}$ and that pollutant alone.* As the figure shows, EPA just takes the average slope from one of the studies, and then determines how much the mortality rate in a given city (not necessarily one in the original study) will be reduced if its ambient $PM_{2.5}$ concentrations are reduced. Consider, for example, "City E" in the illustrative figure, which has a baseline annual average $PM_{2.5}$ concentration of 20 $\mu g/m^3$ that is projected to decline to about 16 $\mu g/m^3$ under an hypothetical regulation. EPA's risk assessment calculation for that regulation would assume that every person residing in City E will experience a drop in mortality risk equal to the vertical drop along the concentration-response function, as indicated by the blue arrows in Figure 3.

Figure 3. Illustration of Calculation of Reduced Premature Deaths Due to Changes in Predicted Ambient Average $PM_{2.5}$ Levels in a City ("City E")



In its national benefits analyses, EPA performs the same computation as shown for illustrative "City E" for the changes in $PM_{2.5}$ concentrations that EPA projects for every county in the US. All those changes – assumed to benefit every resident of any county or city in which a change in $PM_{2.5}$ is projected to occur – are added up to produce EPA's estimate of the national reduction in deaths due to $PM_{2.5}$ from a regulation. Very small changes in $PM_{2.5}$ (and therefore in $PM_{2.5}$-related mortality risk) thus can produce very large changes in estimated premature deaths, if spread over a population of about 300 million people. For example, the 11,000 deaths that EPA attributes as co-benefits in the RIA for the Final EGU MACT rule involve median changes in $PM_{2.5}$ concentrations of 0.36 $\mu g/m^3$ at simulated monitors.[41] Further, (as will be shown later in this paper) almost all of those small changes in ambient concentrations occur at very low levels of baseline $PM_{2.5}$ concentrations – levels for which no observed concentration-response function exists.

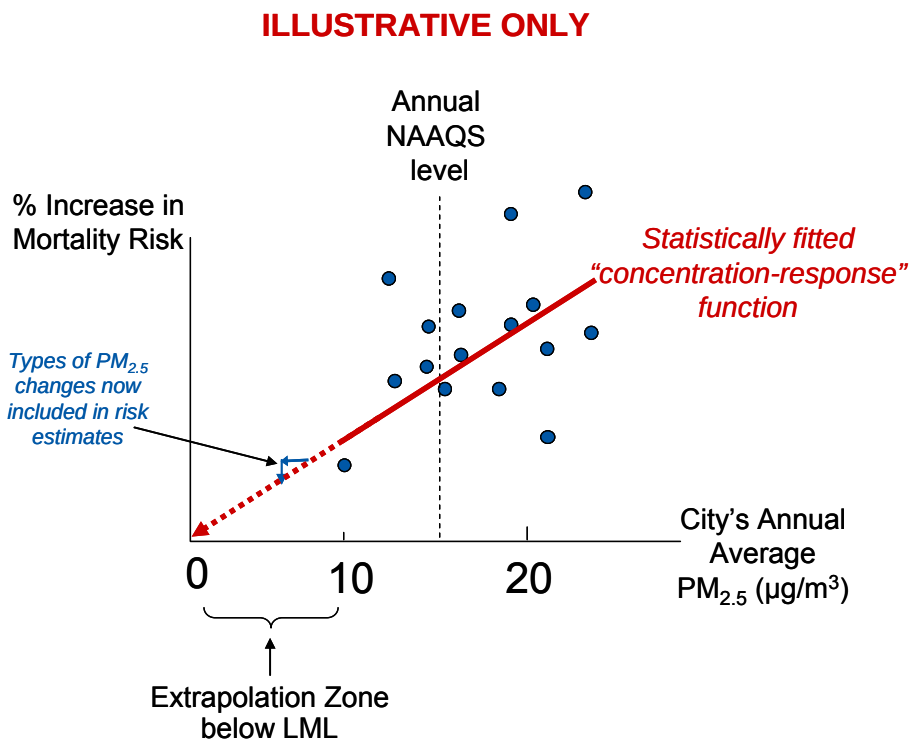
It should be apparent from the discussion above that EPA's estimates of the benefits from regulations that will reduce concentrations of $PM_{2.5}$ in certain locations are fraught with uncertainties, even for changes in $PM_{2.5}$ concentrations that occur above the most recently measured LML. However, in 2009, EPA modified its $PM_{2.5}$-mortality risk formula in a way that greatly increased its benefits estimates. In the illustrative examples above, risks were not computed for changes below the LML in the underlying epidemiological studies. That is, if ambient $PM_{2.5}$ in a location was already below the end of the curve (*e.g.*, at 10 $\mu g/m^3$ in the figures), then prior to 2009, EPA did not assume

[41] See EPA (2011c), p. 5B-4.

there was any further potential for $PM_{2.5}$-related mortality risk reduction in that location. However, starting in 2009, EPA decided that it would calculate risks to the lowest level projected by its air quality models, even though no observed or empirical evidence exists for what the slope of the concentration-response may be in that low-concentration zone.

Figure 4 shows this methodological change, building on the graphical illustrations of Figure 2 and Figure 3. Instead of calculating risks only in areas with $PM_{2.5}$ down to the LML of the study – the point at which all scientific evidence of a statistical association ends – EPA now assumes risks continue *at the same rates* to levels well below the range in which there is any scientific evidence to support those calculations. "Extrapolation" is the use of quantitative relationships outside of the range of evidence on which it was based.[42]

Figure 4. Illustration of the Extrapolation EPA Is Now Using to Calculate Risks in Areas with Ambient $PM_{2.5}$ Concentrations below the Lowest Measured Level in the Original Statistical Study



[42] Most elementary lectures on statistical methods such as regression warn the students that extrapolation of any statistically-derived relationships is a highly dubious exercise. For example, a summary of statistical regression methods carries the following warning: "After computing the regression line, you must not use it to predict values of the response for values of the explanatory variable outside the range of the data used to compute the line in the first place. This practice, called extrapolation, is dangerous because the original data can only produce a formula that describes the association for values found in the original data" (see http://emp.byui.edu/BrownD/Stats-intro/dscrptv/dscrptv_2_qunt_vars/smpl_lnr_rgrsn_ref.htm, accessed November 22, 2011, emphasis in original.)

In other words, in 2009, EPA suddenly started including an entirely new set of presumed risks in its RIAs, based entirely on an extrapolation that has little to no scientific support and without assessing the statistical confidence for predictions of risk changes even at the LMLs of the studies that EPA started from. This created a major change in the level of national mortality estimated to be due to $PM_{2.5}$ that EPA is assuming, because the majority of the US population resides in locations where ambient $PM_{2.5}$ concentrations are below 10 μg/m^3. Where EPA previously did not presume any risk for those people, EPA is now attributing as much health benefit per person in those areas from very small changes in $PM_{2.5}$ (*e.g.*, 0.36 μg/m^3) as it attributes per person for the same size change in areas that have ambient levels above the LML, and even in areas with $PM_{2.5}$ exceeding the "safe" $PM_{2.5}$ NAAQS level of 15 μg/m^3.

EPA's change in its risk analysis assumptions also dramatically inflated its estimates of baseline mortality due to $PM_{2.5}$ in areas with $PM_{2.5}$ above the LML. Prior to 2009, EPA assumed their risk was elevated only in proportion to the degree that their location's ambient $PM_{2.5}$ concentration exceeded the LML, but after 2009, EPA started to assume their risk was elevated in proportion to the degree that their location's ambient $PM_{2.5}$ exceeded background levels (which EPA assumes is about 1 μg/m^3).[43] For example, in an area with $PM_{2.5}$ equal to 16 μg/m^3, EPA used to calculate risks for an excess exposure of 6 μg/m^3 (*i.e.*, 16 minus an LML of about 10). Now, for that same population, EPA is assuming an excess exposure of 15 μg/m^3 (*i.e.*, 16 minus a background of about 1). This decision to calculate risks below the LML increased the estimated mortality risk in that illustrative type of location (*i.e.,* one that is above the LML) by 250%.

Overall, the decision in 2009 to extrapolate risks below the LML caused EPA's estimates of total US deaths due to $PM_{2.5}$ to nearly quadruple. Prior to 2009, EPA was calculating (for its upper bound RIA benefits estimates) that $PM_{2.5}$ caused up to 88,000 deaths nationwide in the relatively clean year of 2005; then, overnight in 2009, EPA changed that number to 320,000 deaths – an increase of a factor of 3.6.[44]

The fact that EPA's methodological change would increase EPA's estimates of deaths due to $PM_{2.5}$ in the year 2005 by a factor of 3.6 (or by about 232,000 more deaths) was never reported or peer reviewed. Although EPA points to concurrence from a committee

---

[43] "Background" level is supposed to represent the ambient concentration that would still remain if all manmade sources of emissions were to be eliminated. EPA formally uses an estimate it calls "policy-relevant background," which is supposed to represent US ambient concentrations if all US, Canadian and Mexican manmade emissions were to be eliminated, but does allow for contributions to US ambient concentrations from emissions in other locations such as Asia, Europe and South America.

[44] The fact that this inflation from 88,000 to over 320,000 was due solely to the decision to extrapolate below the LML is directly observable in EPA's *Quantitative Health Risk Assessment for $PM_{2.5}$* (EPA, 2010b). Appendix G of that document shows the 2005 national risk estimates based on the epidemiological study by Laden *et al.* when calculated just down to the LML, and also when calculated down to zero concentrations (EPA, 2010b, Table G-1, pp. G-6 to G-7). Although a risk estimate that extrapolates below the LML appears in this appendix, none of the CASAC-approved risk estimates in the main body of EPA (2010b) includes risks below the LML. The fact that EPA is actually using an estimate of 320,000 deaths due to $PM_{2.5}$ in 2005 is explicitly stated in EPA's RIA for the Cross-State Air Pollution Rule (EPA, 2011b, pp. 2-3).

of its Science Advisory Board (SAB) that it could extrapolate risks below the LML in its RIAs, there was no public discussion about that decision's implications for a huge inflation in the estimated number of lives that could still be saved through yet-more reductions of $PM_{2.5}$. There is also no evidence that the SAB committee that was asked to opine on this decision was offered any information on its quantitative implications. (Notably, that SAB committee was *not* CASAC. As a result, EPA is now using these inconsistent estimates of baseline $PM_{2.5}$-related deaths simultaneously in different contexts – EPA is using the smaller number of baseline deaths in its CASAC-reviewed risk analyses for the $PM_{2.5}$ NAAQS review, and it is using the larger number of baseline deaths in its RIAs that are generating the large co-benefits for non-$PM_{2.5}$ regulations, such as for air toxics regulations and for non-PM NAAQS, such as ozone.)

The quantitative inflation in $PM_{2.5}$-related mortality benefits through the non-scientific process of extrapolation below the LML is dramatic in its own right, but its lack of credibility becomes more clear when one considers what it means about the fraction of all deaths in the US that are due to $PM_{2.5}$. EPA's presumption that 320,000 deaths in 2005 in the US were "due to $PM_{2.5}$" means that over 13% of all deaths in the US *on average* were due to $PM_{2.5}$.[45] The estimate of 13% of all deaths may seem implausible, but the fractions at the regional level are what gives one pause. These can be seen in Figure 5, which is found in EPA's final RIA for the Proposed EGU MACT rule.[46] Its legend has been adapted here to be consistent with the upper bound $PM_{2.5}$ mortality co-benefits estimates in the Final EGU MACT and other post-2009 RIAs. In other words, the scale shown in red font on Figure 5 is the scale that is consistent with 320,000 deaths due to $PM_{2.5}$ in 2005. It shows that EPA is assuming as a starting point for its benefits calculations that 16% to 22% of all deaths in 2005 were due to $PM_{2.5}$ in large expanses of the Eastern US (*i.e.*, in all of the red-colored counties on the map).

Another inference can be made from EPA's post-2009 method of extrapolating $PM_{2.5}$-related mortality risks below the LML. It implies that about 25% of all deaths *nationwide* were due to $PM_{2.5}$ as recently as 1980.[47] These assumptions, which underpin EPA's co-benefits calculations, stretch the bounds of credibility, and thus undercut the credibility of all of EPA's $PM_{2.5}$-related mortality benefits estimates.

EPA's post-2009 baseline risks are so large because EPA now assumes that there is no tapering off of relative risk as $PM_{2.5}$ exposure approaches zero. For years there has been a debate about whether the concentration-response relationship can truly be linear down

---

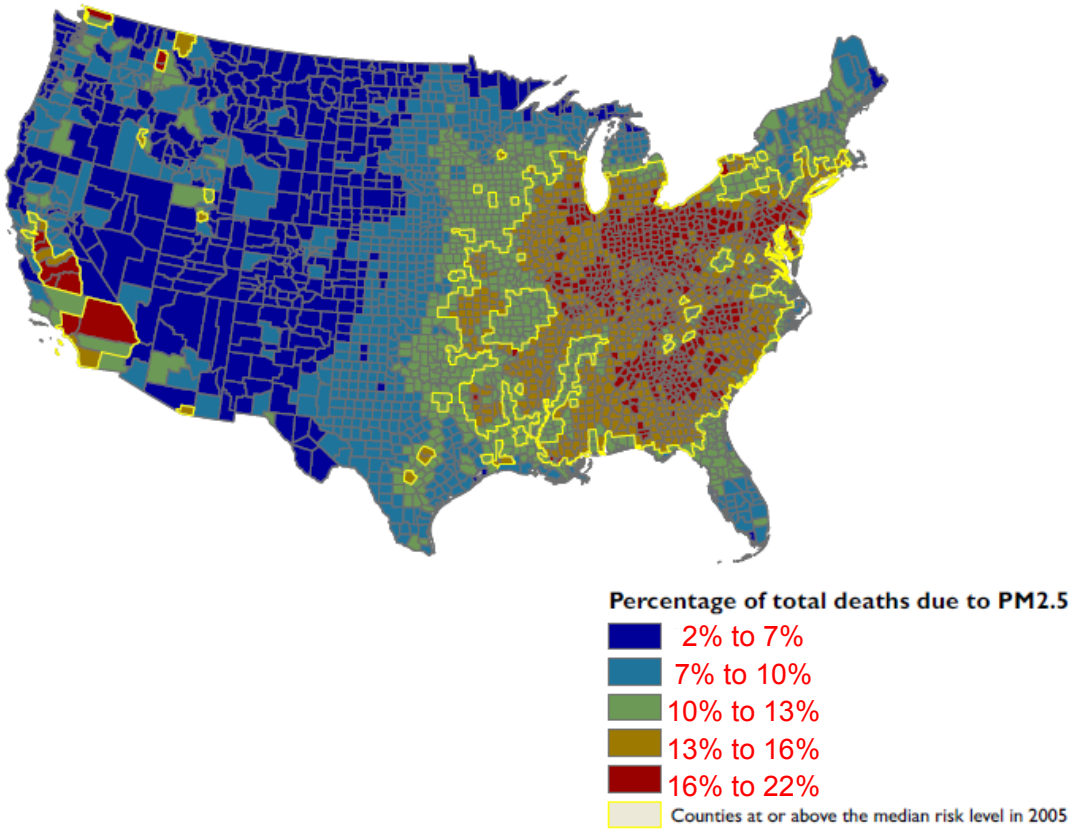
[45] In contrast, the estimate of 88,000 deaths is 4% of all US deaths. Although this starts to seem like a small number in comparison to the 13% that EPA now endorses, it is most likely also an overstatement of the true risks, for reasons discussed above, such as the difficulty in identifying the correct concentration to which elevated mortality risks should be attributed, the uncertainty in the appropriate LML to apply, and the presumption of causality itself in these risk calculations.

[46] Although this figure comes from the final RIA for the Proposed EGU MACT rule (EPA, 2011a), it is still applicable to the Final EGU MACT rule because it reports EPA's estimates of historical (*i.e.*, 2005) levels of mortality risk, which have not been affected by any of the changes in baselines or MACT-related co-reductions of ambient $PM_{2.5}$ that occurred between the proposed and final EGU MACT rule.

[47] See Smith (2011), pp. 14-16 for how this calculation is done.

to zero, but this debate has been focused on questions of statistical power and on basic principles of toxicology. The implication of the linear-to-zero/no-threshold assumption has never been debated in terms of its implication that an implausible proportion of total deaths in the US would be due to $PM_{2.5}$ – but perhaps now it should be debated that way too.

Figure 5. EPA-Produced Map Showing Percentage of Total Deaths due to $PM_{2.5}$ in the Year 2005, with Legend Adjusted by Author to Represent the $PM_{2.5}$ Risk Slope that EPA Uses for its Upper Bound $PM_{2.5}$ Risk Calculations. [48]



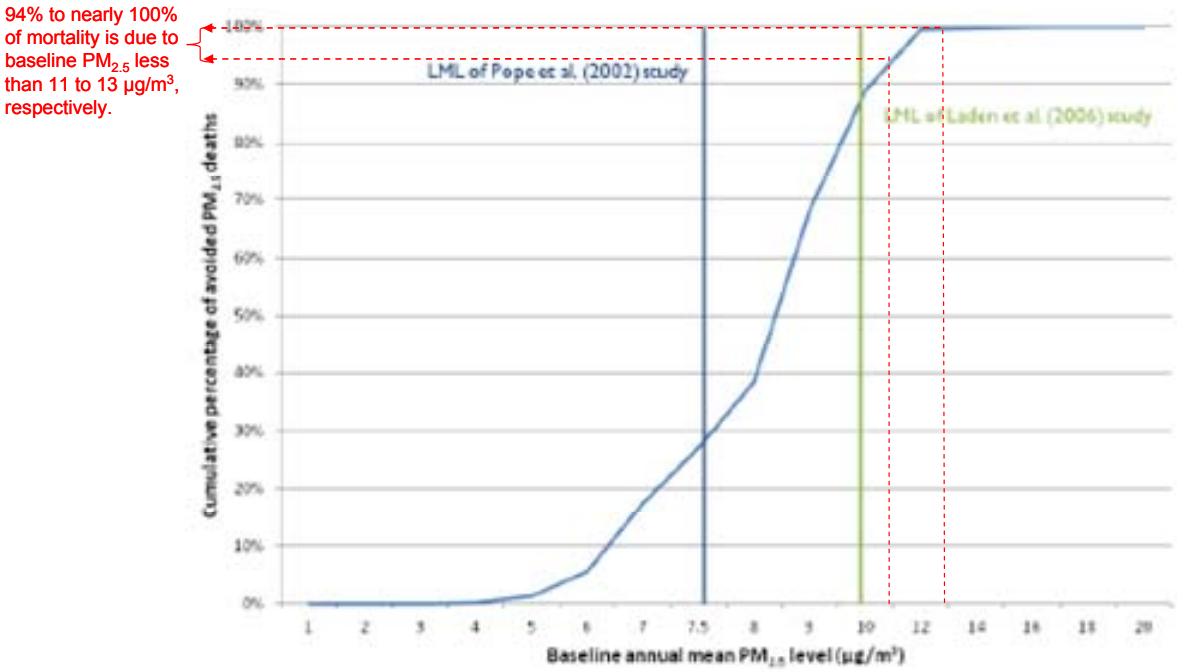
EPA's 2009 inflation in the number of estimated "deaths due to $PM_{2.5}$" has its greatest impact on risks calculated for very low $PM_{2.5}$ levels. Thus, its primary impact has been to increase co-benefits estimates for regulations that are not related to attaining the $PM_{2.5}$ NAAQS, such as the EGU MACT standard. That is, where EPA previously estimated

[48] Figure copied from EPA (2011a), Figure C-2. However, the figure in the RIA is presented for a $PM_{2.5}$ concentration-response slope that is not the one EPA uses to calculate its upper bound estimate of lives saved from the EGU MACT due to $PM_{2.5}$ co-benefits. That is, the text in EPA (2011a) explaining the derivation of the figure indicates that it is based on a $PM_{2.5}$ concentration-response slope from Krewski *et al*. (2009). EPA's current upper bound estimates of lives saved from $PM_{2.5}$ is based a concentration-response slope from Laden *et al.* (2006). Since the 2005 $PM_{2.5}$ levels in each county in the map would not change (they are historical data), the risk range for the scale can readily be recalculated for the Laden *et al*. slope, as done in this paper. Smith (2011) explains how this adjustment is made.

zero co-benefits from $PM_{2.5}$ reductions in areas already below the LML, EPA has created a reservoir of perhaps over 100,000 deaths that it can tap into as co-benefits from new non-PM regulations.

The extent to which this inflationary extrapolation enhances co-benefits estimates can be seen in Figure 6, which is taken from the RIA for the Final EGU MACT RIA.[49] This figure reports (on the vertical axis) the percentage of EPA's estimate of the EGU MACT's total $PM_{2.5}$ mortality co-benefits (*i.e.*, the 11,000 lives saved) that is attributable to ambient $PM_{2.5}$ concentrations at or below the level reported on the x-axis. It shows that nearly all of those 11,000 deaths are in populations that are in areas that are already in attainment with the current $PM_{2.5}$ annual NAAQS of 15 µg/m^3.[50] Under current EPA policy, all of those estimated deaths would be deaths of people living in areas that are protected with an "adequate margin of safety" from $PM_{2.5}$ risks.

Figure 6. Copy of Figure 5-15 from EPA's RIA for the Final EGU MACT Rule Showing that 94% to Nearly 100% of the $PM_{2.5}$ Co-Benefits in that RIA Are Due to Changes in Exposures to Annual Average Ambient $PM_{2.5}$ that Will Still Be Deemed Safe by EPA after Revising the $PM_{2.5}$ NAAQS.



[49] EPA (2011c), Figure 5-15.

[50] This fact can be inferred from the figure in the following way. The blue S-shaped curve in Figure 6 indicates on the vertical axis the percent of the RIA's $PM_{2.5}$ co-benefits estimate that is attributable to baseline $PM_{2.5}$ exposures at or below the $PM_{2.5}$ concentration on the horizontal axis. This is known as a "cumulative distribution." The point on the horizontal axis where the S-shaped curve just reaches 100% indicates the level of baseline $PM_{2.5}$ at or below which *all* (*i.e.*, "100%") of the estimated $PM_{2.5}$ co-benefits occur. As one can see, the vertical reading on the blue S-shaped curve is about 100% at 15 µg/m^3, which means that about 100% of EPA's estimated $PM_{2.5}$ co-benefits from the EGU MACT would be based on reductions in annual average $PM_{2.5}$ exposures that are already below the health-protective level of the current $PM_{2.5}$ standard.

Figure 6 also shows that if EPA had not extrapolated below the LML, about 89% of the estimated upper bound co-benefits of the EGU MACT would have been estimated as zero.[51] This is confirmed in the RIA, which reports that of the 11,000 estimated avoided premature deaths, only 1,200 are in areas where to baseline $PM_{2.5}$ concentrations are above the LML.[52]

The 15 μg/m^3 annual $PM_{2.5}$ NAAQS is under review now, and EPA staff (with CASAC's concurrence) has stated that it will consider revising the annual $PM_{2.5}$ NAAQS to somewhere in the range of 11 to 13 μg/m^3.[53] EPA's reluctance to set the annual $PM_{2.5}$ NAAQS anywhere below 11 to 13 μg/m^3 would appear to reveal the extent to which EPA does not itself feel that risk estimates below that range are credible; if it did view them as credible estimates, surely EPA and CASAC would be compelled to propose a lower $PM_{2.5}$ NAAQS.

Dotted red lines have been added to Figure 6 to show that between 94% and nearly 100% of the 11,000 $PM_{2.5}$ mortality benefits that EPA has estimated from the Final EGU MACT are attributed to estimated $PM_{2.5}$ concentrations below levels that will be deemed protective of the public health with an adequate margin of safety even if EPA revises the annual $PM_{2.5}$ NAAQS to a level within its recommended range of 11 μg/m^3 to 13 μg/m^3.[**] If those concentrations are safe, then it is not appropriate for EPA to be calculating them as co-benefits justifying non-PM regulations such as the EGU MACT rule. Thus those estimates are non-credible from a scientific standpoint.

Further, the remaining <1% to 6% of estimated mortality reductions (*i.e.,* ~0 to ~660 avoided premature deaths out of EPA's estimated 11,000) that are attributable to baseline concentrations between whatever the new $PM_{2.5}$ NAAQS level may be and the upper end of the x-axis (*i.e.*, at about 15 μg/m^3) should, if anything, be counted as direct benefits of the revised $PM_{2.5}$ NAAQS. They are overstated due to issues discussed above concerning the use in chronic exposure studies of recent ambient data rather than average ambient concentrations experienced over the cohort's lifetime, and due to EPA's presumption that there is no uncertainty in the causality of the statistical associations. However, even a more appropriately calculated lower estimate should not be considered a co-benefit for the EGU MACT or other non-PM regulation; it should be counted as a benefit of the $PM_{2.5}$ NAAQS. Placing them in the co-benefits category is tantamount to double-counting them, will be explained in Section 7.

---

[51] The LML for the upper bound is at the green vertical line in the figure.

[52] EPA (2011c), Table 5-20, p. 5-101.

[53] EPA, (2010a), p. 2-106.

[**] Note: A previous version of this report erroneously stated that the lower bound of the range was 84%.

7. EPA's Baselines and Reporting of Benefit and Costs Estimates for a Single Year Cause Double-Counting

This paper has already shown that:

- Co-benefits have no support in BCA theory,
- Use of $PM_{2.5}$ co-benefits in RIAs is undercutting the more general, practical objectives of RIAs,
- EPA's current calculations of $PM_{2.5}$ risks are unsupported by data or scientific principles, and
- The resulting magnitude of EPA's risk estimates are *prima facie* non-credible.

These points should give any thoughtful person reason to call for a stop to the practice of using co-benefits as the primary benefit justification for new rules, and to call for a stop to the estimation of $PM_{2.5}$ risks below the range of observed associations. However, there is yet another significant concern that merits discussion in this paper: EPA may be double-counting many of the $PM_{2.5}$ benefits as it moves from one RIA to the next.

EPA has argued that it does not double-count the $PM_{2.5}$ benefits because it includes all existing regulations in the baseline of emissions for each of its RIAs for another rule. If EPA were doing so thoroughly and consistently, double-counting would not be a concern. However, this is not the actual case.

First, many RIAs are being prepared simultaneously. In 2010, 6 final major CAA-related RIAs and at least 7 proposed RIAs were released for CAA-related rulemakings. In 2011, 7 final and at least 4 proposed CAA-related RIAs were released. This creates a constant source of confusion and potential for double-counting. For example, the RIA for the Proposed EGU MACT rule applied the Proposed CATR rule in *its* baseline, while the RIA for the Final EGU MACT rule applied the Final CSAPR rule in *its* baseline (simply because the CSAPR rule was finalized in the interim between the proposal and finalization of the EGU MACT rule). This change of baseline appears to be the primary reason why the EGU MACT rule's estimated $PM_{2.5}$-related co-benefits for mortality fell from 17,000 to 11,000 when the RIA for the final rule was released. This reveals the extent to which double-counting can occur due to seemingly small differences in what specific rules are included in an RIA's baseline. Moreover, *neither* of the EGU MACT RIAs' baselines included compliance with other existing regulations that have yet to be fully implemented, such as the new 1-hour SO_2 NAAQS, the new NO_2 NAAQS or even the 2006 daily or annual $PM_{2.5}$ NAAQS themselves. In fact, there is a very small amount of co-benefit in the RIA for the Final EGU MACT that is due to reduction of baseline $PM_{2.5}$ exceeding the 15 $\mu g/m^3$ annual NAAQS level.[54] Small as that amount is, it is direct evidence that double-counting can and does occur across all CAA RIAs as a group.

[54] See EPA (2011c), Figure 5-14, p. 5-100.

Clearly the development of an RIA's baseline plays a critical role in the estimates of benefits (and costs) of a rule. The significance of the baseline in determining the benefit and cost comparisons of an RIA has been pointed out by other reviewers of RIAs.[55] However, only one of the previous RIA critiques reviewed in this study has addressed the question of how a baseline affects $PM_{2.5}$ co-benefits. In a review of proposed and final RIAs for the first mercury rule, which was under development during the period 2001-2005, O'Neill takes issue with the fact that coincidental $PM_{2.5}$ reductions from mercury regulation did not appear in the RIA for the mercury rule proposal released in 2004 (*i.e.*, the proposal for what became the Clean Air Mercury Rule, CAMR, which was finalized in 2005). The reason the co-benefits did not appear in the proposed CAMR's RIA was because by the time that rule had been proposed, another new rule that was designed specifically to reduce those same $PM_{2.5}$ levels also was in its final stages (*i.e.*, the Clean Air Interstate Rule, CAIR). As a result, the CAIR rule was incorporated into the baseline for the final CAMR rule's RIA, and what might have once appeared to be co-benefits of CAMR were actually recognized as direct benefits of CAIR. O'Neill also criticizes the fact that EPA chose the single year 2020 to assess the incremental benefits of CAMR, which was the point in time at which the CAIR rule would have been fully implemented. The choice of year as well as the choice of placing CAIR in the baseline had the effect of attributing all of CAIR's benefits to CAIR, rather than allowing any of them to appear as co-benefits from CAMR.

One reasonable response to O'Neill's criticism is that since CAIR was a rule specifically designed to control $PM_{2.5}$, any $PM_{2.5}$-related benefits that might be derived from CAIR should rightfully appear as direct benefits in the RIA for CAIR. In fact, one could contend that they never should have been viewed as co-benefits of the CAMR (or any other possible non-PM rule) as long as the $PM_{2.5}$ NAAQS was in place, because throughout the period when the first mercury rule was being crafted, one could fully anticipate that the $PM_{2.5}$ NAAQS would require implementation. Even if CAIR had not been in development stages at that same time, any non-PM RIA, such as a mercury RIA, should have assumed full implementation of the $PM_{2.5}$ NAAQS anyway. That would have had the same effect as the simple and appropriate act of moving the $PM_{2.5}$-related benefits estimates off of the mercury rule's co-benefits ledger and onto the direct benefits ledger of a $PM_{2.5}$ rulemaking.

The point raised by O'Neill does highlight how the baseline can alter whether $PM_{2.5}$ changes will be counted as co-benefits to justify a non-PM rule or not. However, it does not address double-counting, which is another concern that arises from EPA's choices of RIA baselines. It is nearly impossible to keep the baselines straight when multiple regulations are in the proposal stage at the same time. However, a simple prescription can be applied to EPA's current practice that would help minimize the problem. If any RIA will be accounting for co-benefits from a pollutant that it does not directly address, such as those from $PM_{2.5}$ in a NESHAP rulemaking, then the baseline for that RIA should include "existing" rules, even if not fully implemented yet. It should also explicitly

[55] See, for example, Morgenstern in Chapter 3 of Morgenstern (1997) and O'Neill in Chapter 6 of Harrington *et al.* (2009).

incorporate any reasonably anticipated future standards and/or rulemakings that will deal with that pollutant before allowing any co-benefits from that pollutant to be counted in some unrelated RIA. This may be an uncertain task, but it can certainly be handled by at least considering two baselines:

> Baseline A: Include only the present level of current standards, but ensure that all of them are simulated as attained at their respective attainment deadlines.
>
> Baseline B: Incorporate reasoned assumptions regarding levels of new regulations that are known to be on the verge of modification, even if not yet promulgated or even proposed, and accounting for their future attainment deadlines. (For example, Baseline B would incorporate a reasoned estimate of the most stringent potential level of a tightened $PM_{2.5}$ NAAQS level that may be implemented within the next decade.)

This recommendation conforms with OMB guidance for performing RIAs, which states "When more than one baseline is reasonable and the choice of baseline will significantly affect estimated benefits and costs, you should consider measuring benefits and costs against alternative baselines."[56] Morgenstern also has highlighted the value of considering multiple baselines to highlight the role it plays in RIA findings.[57]

In neither of the alternative baselines should $PM_{2.5}$ co-benefits be calculated based on extrapolation of the concentration-response relationship below the data range over which its slope has been estimated. Indeed, the limit for extrapolation should not be based on the *most recent* LML among the cities in the database; the calculation of risks should be curtailed at a level reflective of the concentrations that the individuals in the cohort experienced on average across their lives.

Another change that is required in order to mitigate double-counting is that EPA stop reporting its benefits and cost estimates for a single year. Regulatory compliance costs and benefits should be considered on a present value basis. EPA's practice of reporting the costs and benefits for a single year can be misleading, especially if the baseline of emissions is declining after the single year selected. For example, $PM_{2.5}$, SO_2, and NO_2 can all be expected to keep declining after 2016 even in the absence of an EGU MACT rule because there are specific standards for each off those pollutants that will take effect between now and 2020. However, in the RIA for the EGU MACT, EPA reports its $PM_{2.5}$ co-benefits only for 2016, at a point in time where $PM_{2.5}$ levels should be on a steady decline through 2019 (which is the latest attainment date for the 2006 $PM_{2.5}$ NAAQS). Thus, there must be a declining trend in baseline risks, and hence the EGU MACT's $PM_{2.5}$ co-benefits soon will be much smaller than EPA reports in the RIA for the single year, 2016. In contrast, the annual costs that EPA reports for that rule will not be declining. Choosing 2016 as the single year for reporting the benefits and costs from the EGU MACT gives an overstated impression of the size of the benefits relative to their

---

[56] OMB (2003), p. 15.

[57] Morgenstern in Chapter 3 of Morgenstern (1997), p. 35.

costs. In fact, it is tantamount to double-counting of co-benefits, because the reported "annual co-benefits" in 2016 includes mortality and morbidity risks that will be gained (and attributed to) the $PM_{2.5}$ and other existing rules just a couple of years later. If benefits and costs are reported for only a single year, that year should be selected as one in which all other regulations in the baseline will be fully implemented.

8. Summary and Conclusions

The key findings of this paper, which is based on review of the benefit and cost evidence in RIAs for major CAA-based rules dating back to 1997, are:

- EPA is relying heavily on coincidental "co-benefits" from $PM_{2.5}$ reductions to create the impression of a benefit-cost justification for many air regulations that are not intended to address $PM_{2.5}$.
- Consideration of co-benefits for a separately-regulated pollutant is not supported by benefit-cost analysis (BCA) theory, and EPA's excessive reliance on them undercuts the broader practical value of RIAs, which is to provide structured and transparent information to help avoid and reduce redundant and ineffective regulations.
- In 2009, EPA vastly increased the levels of mortality risks that it attributes to $PM_{2.5}$ (and hence inflated its estimates of $PM_{2.5}$ benefits and co-benefits) simply by starting to assign risks down to background levels of $PM_{2.5,}$ below the most recent of the lowest measured levels (LMLs) in the epidemiological studies. This created non-credible estimates of risks from ambient exposures that are well within the safe range established by the $PM_{2.5}$ NAAQS.
- Identifying an appropriate lower bound below which risk estimates are not scientifically supported is not as simple as identifying the most recently-observed LML among cities in a chronic exposure epidemiological study. The exposure level to attribute to the observed mortality differences could be much earlier in time, given that such studies track mortality outcomes dating several decades back, based on people whose lifetime exposures date back to well before 1950.
- The decision to inflate the $PM_{2.5}$ risk estimates by presuming risks continue at an unchanged rate down to background has its greatest impact on co-benefits estimates because – for rules that do not address $PM_{2.5}$ directly – a much greater share of their incremental reduction of $PM_{2.5}$ will occur in areas that are already in attainment with the $PM_{2.5}$ NAAQS (and thus have $PM_{2.5}$ levels that EPA has deemed safe).
- Poor choices of baselines and EPA's practice of reporting benefits and costs for only a single year leads to double-counting of the $PM_{2.5}$-related benefits and co-benefits.

Based on the above observations, several recommendations and conclusions follow for aligning RIA methods with BCA principles, and for improving the quality and usefulness of RIAs that EPA produces. These include:

- Baselines in RIAs should incorporate implementation of all reasonably anticipated standards, even if formal rules to implement them are not yet in place. Estimates of benefits from $PM_{2.5}$ reductions will thus remain the direct benefits of $PM_{2.5}$-specific rules, and double-counting will be avoided. Any temporary benefits from early introduction of $PM_{2.5}$ reductions via a non-$PM_{2.5}$ rule should be identified as temporary only, and not reported as the co-benefits in a single, "snapshot" year, which implies those benefits would be permanent.
- Co-benefits from a pollutant that EPA already regulates under separate rulemakings should not be allowed to serve as a component of the total benefits reported in the Executive Summary of RIAs for rules that target different public health or welfare concerns. The current practice of doing so subverts the practical values of preparing RIAs, leads to unnecessary regulatory complexity, and incentivizes use of less credible methods of risk estimation. Co-benefits should not be reported as part of the total benefits estimates in an RIA, nor should they be included in public announcements of the benefits of a new regulation.
- EPA should stop using its scientifically non-credible method of extrapolating $PM_{2.5}$ risks below the LML. If EPA does persist in producing estimates of benefits or co-benefits from changes in concentrations below the LML, those estimates should be kept clearly separated from all other $PM_{2.5}$-related mortality benefits estimates, not be added to any other PM-related benefits estimates, and should be accompanied by a clear statement that there is no scientific evidence about the shape or existence of any concentration-response function in that range of ambient $PM_{2.5}$ concentrations.
- For benefits estimates based on $PM_{2.5}$ concentrations above the LML, EPA should be offering quantitative estimates of the uncertainties associated with its risk estimates, *taking account of the expanded confidence interval for estimates nearing the LML*. Confidence ranges based only on the statistical error of the slope estimate are not an appropriate measure of the statistical confidence of its predicted changes in health risk for reductions in $PM_{2.5}$, especially those well below the average $PM_{2.5}$ in the underlying epidemiological study.

In all, EPA's use of co-benefits in its RIAs should end for several reasons. It scares the public into believing that large numbers of people die prematurely were it not for implementation of new rules on pollutants for which EPA has not actually identified any current public health risk. EPA's use of co-benefits also gives EPA a shield to justify building a complex web of rules when EPA could (and is already obligated to) provide almost all of those purported health-protective benefits with just a single rule, if warranted: the $PM_{2.5}$ NAAQS. If large effects below the level of the $PM_{2.5}$ NAAQS were deemed credible, the appropriate policy remedy would be to tighten the $PM_{2.5}$ standard. The fact that EPA does not take this simple, streamlined approach hints at the degree to which the Agency realizes that its co-benefits calculations do not reflect true public health risks. But finally, promoting the goal of further $PM_{2.5}$ risk reductions by way of rules for totally different categories of emissions is just bad policy. This cannot possibly result in a cost-effective path to addressing a nation's clean air needs.

References

Arrow K. D., M. L. Cropper, G. C. Eads, R. W. Hahn, L. B. Lave, R. G. No11, P. R. Portney, M. Russell, R. Schmalensee, V. K, Smith, and R. N. Stavins. 1996. "Is There a Role for Benefit-Cost Analysis in Environmental, Health and Safety Regulation?" *Science*. Vol. 272, pp. 221-222.

EPA. 2011a. *Regulatory Impact Analysis of the Proposed Toxics Rule: Final Report*. March. Available at http://www.epa.gov/ttnecas1/regdata/RIAs/ToxicsRuleRIA.pdf.

EPA. 2011b. *Regulatory Impact Analysis of the Final Transport Rule.* June. Available at http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf.

EPA. 2011c. *Regulatory Impact Analysis for the Final Mercury and Air Toxics Standards*. EPA-452/R-11-011. December. Available at http://www.epa.gov/ttnecas1/regdata/RIAs/matsriafinal.pdf.

EPA. 2010a. *Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards*. EPA-452/R-11-003. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. April. Available at: http://www.epa.gov/ttnnaaqs/standards/pm/data/20110419pmpafinal.pdf.

EPA. 2010b. *Quantitative Health Risk Assessment for Particulate Matter*. EPA-452/R-10-005. Office of Air Quality Planning and Standards, Research Triangle Park, N.C. June. Available at: http://www.epa.gov/ttn/naaqs/standards/pm/data/PM_RA_FINAL_June_2010.pdf.

Executive Order 12291. 1981. "Federal Regulation." 46 *Fed. Reg.* 13193, February 17. Available at: http://www.archives.gov/federal-register/codification/executive-order/12291.html.

Executive Order 12866. 1993. "Regulatory Planning and Review." 58 *Fed. Reg.* 51735, October 4. Available at: www.whitehouse.gov/omb/inforeg/eo12866.pdf.

Executive Order 13563. 2011. "Improving Regulation and Regulatory Review." 76 *Fed. Reg.* 3821, January 18. Available at: http://www.regulations.gov/exchange/sites/default/files/doc_files/President%27s%20Executive%20Order%2013563_0.pdf.

Hahn R. W. and P. M. Dudley. 2007. "How Well Does the US Government Do Benefit-Cost Analysis?" *Review of Environmental Economics and Policy*. Vol. 1(2), pp. 192-211.

Harrington, W., L. Heinzerling, and R. D. Morgenstern (eds.) 2009. *Reforming Regulatory Impact Analysis*. Resources for the Future, Washington DC. April.

Krewski D., R. T. Burnett, M. Goldberg, K. Hoover, J. Siemiatycki, M. Jerrett, M. Abrahamowicz, and W. H. White. 2000. *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality.* Special Report. Health Effects Institute, Cambridge, Massachusetts. July.

Krewski D., M. Jerrett, R. T. Burnett, R. Ma, E. Hughes, Y. Shi, Y, *et al*. 2009. *Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality*. HEI Research Report Number 140. Health Effects Institute, Boston, MA. May.

Laden, F., J. Schwartz, F. E. Speizer, and D. W. Dockery. 2006. "Reduction in Fine Particulate Air Pollution and Mortality." *American Journal of Respiratory and Critical Care Medicine*. Vol 173, pp. 667-672.

Morgenstern, R. D. (ed.) 1997. *Economic Analyses at EPA: Assessing Regulatory Impact*. Resources for the Future, Washington, DC.

OMB. 2003. *Circular A-4, Regulatory Analysis*. Available at: http:/www.whitehouse.gov/sites/default/files/omb/assets/regulatory_matters_pdf/a-4.pdf.

Pope, C. A., III, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito, G. D. Thurston. 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to fine Particulate Air Pollution." *JAMA*. Vol. 287(9), pp. 11332-1141.

Smith, A. E. 2011. *Technical Comments on the Regulatory Impact Analysis Supporting EPA's Proposed Rule for Utility MACT and Revised NSPS (76 FR 24976)*. Prepared for and submitted to EPA EGU MACT Docket by Utility Air Regulatory Group (Attachment 13 in Docket Reference EPA-HQ-OAR-2009-0234-17775). August 3. Available at: http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OAR-2009-0234-17775.

Appendix A.

Sources of Cost and Benefit Information on CAA RIAs since 1997 that Were Reviewed in this Study

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
7/12/1997	1997	Ozone NAAQS *(To revise the 0.12 1hr standard to a 0.08 8hr standard)*	Ozone	Final RIA. See pp.13-2 for cost, pp. 12-64 for other co-benefits, pp. 12-1 for total benefits. Full attainment (F/A) numbers were estimated by scaling partial attainment (P/A) numbers for target benefits, PM co-benefits and PM mortality rates.	http://www.epa.gov/ttn/oarpg/naaqsfin/ria.html
7/16/1997	1997	$PM_{2.5}$ NAAQS *(To implement the first $PM_{2.5}$ standard at 15/65 annual/daily averages, supplementing the PM_{10} standard)*	$PM_{2.5}$	Final RIA. F/A numbers provided. Target benefit estimate comes from: low- 19.8-4.3 = 15.5; high- 109.7-8.1 = 101.6. Cost: pp.13-2. Other co-benefits: pp.12-64. Total benefits: pp.13-2. Mortality rates: see pp.12-43 for P/A numbers. F/A estimated by 15.5/14.5 * 3300 = 3528 for low; 101.6/96.1 * 15,600 = 16493 for high.	http://www.epa.gov/ttn/oarpg/naaqsfin/ria.html
8/14/1997	1997	Hospital/ Medical/ Infectious Waste Incinerators NSPS and EG	Many pollutants	Final rule FR. See pp. 29 for cost and benefits. All quantified benefits are PM.	http://www.gpo.gov/fdsys/pkg/FR-1997-09-15/pdf/97-23835.pdf#page=1
9/22/1997	1997	Highway Heavy-Duty Engines and Diesel Engines	NO_x, HC	Final RIA. See pp. 97 for cost.	http://www.regulations.gov/#!searchResults;rpp=10;po=0;s=EPA-HQ-OAR-2003-0012-0949
10/27/1997	1997	Pulp & Paper NESHAP	HAPs, VOC, TRS	Final rule FR. See pp. 120 for cost, pp. 126 for benefits. Included air related numbers only. Identified negative co-benefits (due to SO_2, CO, PM, NO_x increases).	http://www.gpo.gov/fdsys/pkg/FR-1998-04-15/pdf/98-9613.pdf#page=1
12/16/1997	1997	Locomotive Emission Standards	NO_x, PM	1998 Regulatory support document. See pp. 120 for NPV (7% discount rate) of the total cost, no annualized figure provided.	http://www.regulations.gov/#!searchResults;rpp=10;po=0;s=EPA-R03-OAR-2009-0956-0038

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
8/14/1998	1998	National VOC Standards for Architectural Coatings	VOC	Proposed rule and notice of public hearing FR. See pp. 6 for cost.	http://www.gpo.gov/fdsys/pkg/FR-1996-06-25/pdf/96-16009.pdf#page=1
8/27/1998	1998	Non-Road Diesel Engines	NO_x, HC	Final RIA. See pp. 72 for cost.	http://www.epa.gov/nonroaddiesel/frm1998/nr-ria.pdf
9/2/1998	1998	Revised NO_x Std: New Fossil-Fuel-Fired Steam Generating Units	NO_x	Insufficient information.	http://www.gpo.gov/fdsys/pkg/FR-1997-07-09/pdf/97-17950.pdf#page=1
9/23/1998	1998	NO_x SIP Call & Section 126 Petitions	NO_x	RIA. Ranges quoted are for low to high assumption sets. See pp. ES-3 for cost, pp. ES-6 for total benefits, pp. 4-50 for a breakdown of benefits, pp. 4-23 for mortality rates. All ozone related benefits are target benefits.	http://www.epa.gov/ttn/oarpg/otag/sipriav2.zip
3/1/1999	1999	Phase II Emission Stds for New Nonroad Spark-Ignition Non-Handheld Engines <19 kW	HC, NO_x	Final RIA. See pp. 7-15 for fuel savings (0.2 billion per year), pp. 7-13 for cost (0.132 billion) for a net cost savings of 0.0907.	http://www.epa.gov/otaq/equip-ld.htm
4/22/1999	1999	Regional Haze Rule	Visibility	RIA. See pp. 9-48 and 9-51 for benefits, pp. 10-20 for cost, pp. 9-55 and 9-61 for mortality rates. Reported 1.0 dv/10years levels, ranges quoted for benefits and mortality rates are the low and high ends across Case A and Case B. Total benefit is calculated accordingly.	http://www.epa.gov/ttnecas1/regdata/RIAs/rhria.zip
4/30/1999	1999	Final Section 126 Petition Rule	NO_x	RIA. See pp. ES-3 for cost, pp. ES-11 for benefits, pp. ES-7 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/126fn0.zip
12/21/1999	1999	Gasoline Sulfur Control Requirements	NO_x, PM	Final FR. See pp. 88 for benefits and mortality rate, pp. 86 for cost.	http://www.gpo.gov/fdsys/pkg/FR-2000-02-10/pdf/00-19.pdf#page=1

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
3/1/2000	2000	Phase 2 Emission Stds for New Nonroad Small Spark Ignition Handheld Engines <19 kW	HC, NO_x	Final RIA. See pp. 110 for annualized cost (.234-.284) and fuel savings (0.094).	http://www.epa.gov/ otaq/equip-ld.htm
7/28/2000	2000	Control of Emissions from 2004 and Later Model Year Highway Heavy-Duty Engines	Many pollutants	RIA. See pp. 89 and 106 for cost.	http://www.regulations.gov/#!searchResults;rpp=10;po=0;s=EPA-HQ-OAR-2003-0012-0950
10/19/2000	2000	Protection of Stratospheric Ozone Reductions	Stratospheric O_3	RIA. See pp. 4 for cost.	http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OAR-2008-0009-0012
12/21/2000	2000	Heavy-Duty Engine & Diesel Fuel Sulfur Control Reqm 2007	NO_x, PM	Final RIA. See pp. xvi for cost and benefits, pp. xvii for mortality rates.	http://www.epa.gov/oms/highway-diesel/regs/exec-sum.pdf
9/13/2002	2002	Emissions from Nonroad & Recreational Spark-Ignition Engines	NO_x, VOC, CO, PM	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2001-10-05/pdf/01-23591.pdf#page=1
8/27/2003	2003	PSD & NSR: Routine Maintenance and Repair	all	Insufficient Information.	http://www.epa.gov/fedrgstr/EPA-AIR/2003/October/Day-27/a26320.htm
2/26/2004	2004	Stationary Reciprocating Internal Combustion Engine NESHAP	HAPs	RIA. See pp. ES-5 for cost, pp.8-40 for mortality rates (50% NO_x emission reduction), pp. 8-45 for benefits. Assuming the social cost of 0.255 on pp. ES-7 is a typo. Mortality rates are prorated assuming 25% NO_x emission reduction. Ozone and PM_{10} benefits are regarded as "other benefits".	http://www.epa.gov/ttnecas1/regdata/RIAs/RICERIA-finalrule.pdf
2/26/2004	2004	Plywood & Composite Wood Products NESHAP	organic HAPs	RIA. See pp. ES-1 for cost.	http://www.epa.gov/ttnecas1/regdata/RIAs/pcwp-finalruleRIA.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
2/26/2004	2004	Automobile & Light-Duty Vehicle Manufacturing NESHAP	HAPs	RIA. See pp. ES-5 for cost. Did mention ozone and PM co-benefits from VOC reduction, but did not quantify.	http://www.epa.gov/ttnecas1/regdata/RIAs/autolightdutyRIAfinaltotal.pdf
2/26/2004	2004	Industrial Boilers & Process Heaters NESHAP	HAPs, HCl, metals	RIA. See pp.ES-1 for cost, pp. 10-45 for mortality rates and benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/indboilprocheatfinalruleRIA.pdf
5/7/2004	2004	Non-Road Diesel Rule	PM, NO_x, HAPs	RIA. See pp.9-42 to 43 for mortality rates and benefits, pp. 9-52 for costs. Other co-benefits include 2.5-3.4 (pp. 9-27) reductions in unpleasant odors, and 2.15 PM welfare benefits.	http://www.epa.gov/nonroad-diesel/2004fr.htm#ria
3/10/2005	2005	Clean Air Interstate Rule	NO_x,SO_2 *(SO_2 as precursor of amb. $PM_{2.5}$)*	RIA. See pp. 1-2 for benefits and cost, pp. 1-4 for mortality rates.	http://www.epa.gov/cair/pdfs/finaltech08.pdf
3/15/2005	2005	Clean Air Mercury Rule	Hg	RIA. See pp. 11-14 for target benefits, pp.7-13 for cost, pp.12-8 for $PM_{2.5}$ co-benefits and mortality rates.	http://www.epa.gov/ttn/ecas/regdata/RIAs/mercury_ria_final.pdf
6/15/2005	2005	Clean Air Visibility Rule/BART Guidelines	Visibility	RIA. See pp. 1-3 for cost and benefits, pp. 1-5 for mortality rates.	http://www.epa.gov/oar/visibility/pdfs/bart_ria_2005_6_15.pdf
3/15/2006	2006	Inclusion of Delaware and New Jersey in CAIR	NO_x,SO_2 *(SO_2 as precursor of amb. $PM_{2.5}$)*	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2005-05-12/pdf/05-5520.pdf#page=1
3/15/2006	2006	Sec. 126 from NC to Reduce Interstate Transport of PM & O3; FIPs to Reduce Interstate Transport of PM & O3; Revisions to CAIR; Revisions to Acid Rain Program	NO_x,SO_2 *(SO_2 as precursor of amb. $PM_{2.5}$)*	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2005-08-24/pdf/05-15529.pdf#page=1

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
6/28/2006	2006	Stationary Compression Ignition Internal Combustion Engine NSPS	many	RIA. See pp. 1-2 for cost, pp. 6-4 for benefits. Direct PM benefit is recorded as target benefit. Benefits from NO_x and SO_2 are recorded as PM co-benefits. Premature mortality prevention accounts for 90% of the total benefit, but cannot be quantified (pp.6-5). Benefits are quoted at 3% discount rate (pp.6-4).	http://www.epa.gov/ttnecas1/regdata/RIAs/ci_nsps_ria_reportfinal06.pdf
9/21/2006	2006	$PM_{2.5}$ NAAQS *(To revise from 15/65 annual/daily averages, to 15/35)*	$PM_{2.5}$	RIA. See pp. ES-9 to 10 for costs and benefits, pp. 5-100 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/Executive%20Summary.pdf http://www.epa.gov/ttnecas1/regdata/RIAs/Chapter%205--Benefits.pdf
2/8/2007	2007	Control of HAP from mobile sources	HAPs	Final RIA. See pp. 12-20 for mortality rate (Pope et al. only), pp. ES-10 for PM benefits, pp. ES-11 for cost.	http://www.epa.gov/otaq/regs/toxics/fr-ria-sections.htm
3/28/2007	2007	Clean Air Fine Particle Implementation Rule	$PM_{2.5}$	Insufficient Information.	http://www.gpo.gov/fdsys/pkg/FR-2005-11-01/pdf/05-20455.pdf#page=1
2/14/2008	2008	Control of Emissions form New Locomotives & Marine Diesel Engines <30 L per Cylinder	PM, NO_x	RIA. See pp. 6-52 for cost and benefits, pp. 6-44 for PM mortality, pp. 6-46 for a breakdown of benefits.	http://www.epa.gov/oms/regs/nonroad/420r08001a.pdf
3/12/2008	2008	Ozone NAAQS *(To revise the 0.08 8hr standard to 0.075 8hr standard)*	Ozone	RIA. See pp.ES-3 for cost and visibility benefits, pp. 6-62 for target benefits, pp. 6-48 for mortality rates, pp. 6-64 for PM co-benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/452_R_08_003.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
8/18/2008	2008	Control of Emissions form Non-road Spark-Ignition Engines & Equipment	VOC, NO_x, PM,CO	Final rule FR. See pp. 122 for mortality rates, pp. 124 for a breakdown of benefits, pp. 128 for cost and total benefits. Target benefit is all PM; other benefits include visibility and ozone benefit.	http://www.gpo.gov/fdsys/pkg/FR-2008-10-08/pdf/E8-21093.pdf#page=1
10/15/2008	2008	Lead (Pb) NAAQS	Pb	RIA. See pp. ES-11 for cost and benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/finalpbria.pdf
12/10/2008	2008	Petroleum Refineries NSPS	SO_2, NO_x, PM, VOC	RIA. See pp. 7-6 for benefits, pp. 7-12 for cost. Direct benefit is PM benefit, PM 2.5 co-benefits include benefits from reductions in PM precursors.	http://www.epa.gov/ttnecas1/regdata/RIAs/finalpetroleumrefineriesnspsria43008.pdf
9/16/2009	2009	GHG Mandatory Reporting Rule	GHGs	Final rule FR. See pp. 105 for cost.	http://www.epa.gov/climatechange/emissions/notices.html
12/17/2009	2009	Control of Emissions from New Marine Compress'n-Ign Engines >30 L per Cylinder	NO_x	Final rule FR. See pp. 69 for benefits and cost, pp. 67 for PM and Ozone ("other") benefit, pp. 65 for mortality rates.	http://edocket.access.gpo.gov/2010/pdf/2010-2534.pdf
2/17/2010	2010	Reciprocating Internal Combustion Engines NESHAP - Compression Ignition	Organic, metal HAPs	RIA. See pp.1-1 for cost, pp.7-1 for $PM_{2.5}$ co-benefits, and pp.7-8 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/CIRICENESHAPRIA2-17-10cleanpublication.pdf
3/31/2010	2010	EPA/NHTSA Joint Light-Duty GHG Emission Stds & CAFES	GHGs	Final FR. See pp. 213 for benefits and cost, pp. 206 for PM benefits, pp. 205 for mortality rates,	http://www.gpo.gov/fdsys/pkg/FR-2010-05-07/pdf/2010-8159.pdf#page=1
5/12/2010	2010	Greenhouse Gases PSD and Tailoring Rule	GHGs	RIA. See pp.18 for cost. No benefits quantified.	http://www.epa.gov/ttnecas1/regdata/RIAs/riatailoring.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
6/2/2010	2010	SO_2 NAAQS *(To set a 75 ppb 1-hr standard)*	SO_2	See pp. ES-9 for cost and benefits, pp. 5-30 for mortality rates. Negative cost indicates regulatory relief benefit. Numbers quoted are for Step 1, 25000 tpy.	http://www.epa.gov/ttnecas1/regdata/RIAs/fso2ria100602full.pdf
8/6/2010	2010	Portland Cement Manufacturing Industry NSPS & NESHAP Amendment	HC, HAPs, PM	RIA. See pp. 1-2 for cost, pp. 6-1 for $PM_{2.5}$ co-benefits, pp. 6-15 for mortality rates. All benefits of the rule are from NESHAP portion. NESHAP does not target $PM_{2.5}$ or PM_{10} mass, but all the benefits of the NESHAP (and of the NESHAP + NSPS) are $PM_{2.5}$ ambient concentration. Therefore, all are co-benefits.	http://www.epa.gov/ttnecas1/regdata/RIAs/portlandcementfinalria.pdf
8/10/2010	2010	Existing Stationary Compression Ignition Engines NESHAP	HAPs	Final RIA. See pp. 7-10 for mortality rates, pp. 7-9 for benefits, pp. 1-1 for costs.	http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OAR-2008-0708-0571
2/21/2011	2011	Industrial, Comm'l, and Institutional Boilers NESHAP	HAPs	Final FR. See pp. 27 for benefits and mortality rates, pp.29 for cost.	http://www.gpo.gov/fdsys/pkg/FR-2011-03-21/pdf/2011-4493.pdf#page=1
2/21/2011	2010	Indus'l, Comm'l, and Institutional Boilers & Process Heaters NESHAP	HAPs	RIA. See pp.1-1 for cost, pp.6-1 for $PM_{2.5}$ co-benefits pp. 6-8 for mortality rates.	http://www.epa.gov/airquality/combustion/docs/boilerria20100429.pdf
2/21/2011	2011	Comm'l & Indus'l Solid Waste Incineration Units NSPS and Emission Guidelines	CO, Pb, HAPs	RIA. See pp. 1-1 for cost, pp. 1-2 for benefits, pp. 5-10 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/CISWIRIAfinal110221_psg2.pdf
7/1/2011	2011	Cross State Air Pollution Rule (CSAPR)	NO_x,SO_2 *(SO_2 as precursor of amb. $PM_{2.5}$)*	RIA. See pp.1 for mortality rates, pp.2 for cost, table 1-3 on pp. 6 to 7 for benefits. Other co-benefits include visibility (4.1) + social cost of carbon (0.6) = 4.7. Sum up all the remaining items to get target benefits.	http://www.epa.gov/airtransport/pdfs/FinalRIA.pdf

Concluded Date	Year of Document Used	RIA Name	Target Pollutant	Document Type / Page References	URL
8/8/2011	2011	Control of GHG from Medium & Heavy-Duty Vehicles	GHGs	Final RIA. See pp. 9-45 for benefits and cost, net cost = 24.7 (technology cost) - 166.5 (fuel savings), pp. 8-86 for PM benefit and pp.8-81 for mortality rates. Benefit range is derived from different assumptions for social cost of carbon in 2012.	http://www.epa.gov/otaq/climate/documents/420r11901.pdf
9/6/2011	2010	Reconsideration of Ozone NAAQS	Ozone	RIA. F/A (75 ppb) numbers quoted. See pp. S1-4 for cost, pp.3-11 for mortality rates and benefits, pp.3-6 for visibility. Target benefit refers to ozone benefit here.	http://www.epa.gov/ttnecas1/regdata/RIAs/s1-supplemental_analysis_full.pdf
12/16/2011	2011	EGU MACT Rule	Hg, HAPs	RIA. See p. ES-2 for cost; pp. ES-6/7 for direct benefits, $PM_{2.5}$ co-benefits, & social cost of carbon. SCC is quoted at 3% discount rate. See p. ES-5 for mortality counts.	http://www.epa.gov/ttnecas1/regdata/RIAs/matsriafinal.pdf
N/A	2011	Mercury Cell Chlor Alkali Plant Mercury Emissions NESHAP	Hg	RIA. See pp.1-2 for cost and total benefit, pp. 5-1 for $PM_{2.5}$ co-benefits, pp. 5-11 for mortality rates, and pp. 5-16 for the social cost of carbon (other co-benefits).	http://www.epa.gov/ttnecas1/regdata/RIAs/mercurycell.pdf
N/A	2011	Oil and Natural Gas Industry NSPS & NESHAP Amendment	VOC, SO_2, HAPs, Methane	RIA. See pp.1-4 and pp. 1-6 for costs.	http://www.epa.gov/ttnecas1/regdata/RIAs/oilnaturalgasfinalria.pdf
N/A	2011	Sewage Sludge Incineration Units NSPS & Emission Guidelines	Hg	RIA. See pp. 3 for cost and benefit, pp. 5-11 for mortality rates.	http://www.epa.gov/ttnecas1/regdata/RIAs/ssiria110201.pdf

www.researchsolutions.com
1.866.WE.FIND.IT

Regular

Order ID: 1454351
Group ID: 144182
Group #: 8 / 8
Ordered: 03.27.2012

Client Reference

None Given

Citation

[Risk Anal], 2011 Nov

Reassessing the Human Health Benefits from Cleaner Air

Cox LA Jr

0272-4332

Notes

English Only

We're in the office: 6am-6pm (PT) | 7am-7pm (MT) | 8am-8pm (CT) | 9am-9pm (ET)

NOTICE: This material MAY BE PROTECTED by title 17 of the U.S. Copyright Code. For additional information please visit the Copyright Clearance Center (www.copyright.com) and Research Solutions (www.researchsolutions.com/copyright-policy.php).

 DOI: 10.1111/j.1539-6924.2011.01698.x

Reassessing the Human Health Benefits from Cleaner Air

Louis Anthony (Tony) Cox, Jr.*

Recent proposals to further reduce permitted levels of air pollution emissions are supported by high projected values of resulting public health benefits. For example, the Environmental Protection Agency recently estimated that the 1990 Clean Air Act Amendment (CAAA) will produce human health benefits in 2020, from reduced mortality rates, valued at nearly $2 trillion per year, compared to compliance costs of $65 billion ($0.065 trillion). However, while compliance costs can be measured, health benefits are unproved: they depend on a series of uncertain assumptions. Among these are that additional life expectancy gained by a beneficiary (with median age of about 80 years) should be valued at about $80,000 per month; that there is a 100% probability that a positive, linear, no-threshold, causal relation exists between $PM_{2.5}$ concentration and mortality risk; and that progress in medicine and disease prevention will not greatly diminish this relationship. We present an alternative uncertainty analysis that assigns a positive probability of error to each assumption. This discrete uncertainty analysis suggests (with probability >90% under plausible alternative assumptions) that the costs of CAAA exceed its benefits. Thus, instead of suggesting to policymakers that CAAA benefits are almost certainly far larger than its costs, we believe that accuracy requires acknowledging that the costs purchase a relatively uncertain, possibly much smaller, benefit. The difference between these contrasting conclusions is driven by different approaches to uncertainty analysis, that is, excluding or including discrete uncertainties about the main assumptions required for nonzero health benefits to exist at all.

KEY WORDS: Air pollution health effects; Clean Air Act Amendment 1990; risk-cost-benefit analysis; uncertainty analysis; Weibull distribution

1. INTRODUCTION

Media, regulatory, and advocacy reports and recommendations frequently suggest that particulate matter in outdoor air ($PM_{2.5}$) kills people and causes serious health problems. For example, an Environmental Protection Agency (EPA) website warns that "[n]umerous scientific studies have linked particle pollution exposure to a variety of problems, including irregular heartbeat; nonfatal heart attacks; and premature death in people with heart or lung disease" (www.epa.gov/pm/health.html). Conversely, regulatory proposals to further decrease currently permitted levels of pollutants are increasingly supported by reference to large estimated or predicted health benefits from doing so. For example, in early 2011, the EPA released the results of its cost-benefit analysis of the 1990 Clean Air Act Amendments (CAAA). The assessment made two striking claims:[1] (1) as of 2020, the CAAA would produce estimated health benefits valued at approximately $2 trillion (i.e., two thousand billion) per year, compared to estimated compliance costs of only about $65 billion per year; and (2) the uncertainties in the cost-benefit analysis are small enough so that: "***The extent to which estimated benefits exceed estimated costs*** and an in-depth analysis of uncertainties ***indicate that it is extremely unlikely the costs of 1990 Clean Air Act Amendment programs would exceed***

*Address correspondence to Louis Anthony (Tony) Cox, Jr., Cox Associates and University of Colordo, 503 Franklin St., Denver, CO 80218, USA; tcoxdenver@aol.com.

 0272-4332/11/0100-0001$22.00/1 © 2011 Society for Risk Analysis

their benefits under any reasonable combination of alternative assumptions or methods identified during this study" (emphasis in original).

This article reexamines the assumptions, methods, and numbers used in reaching these encouraging conclusions. We come to the very different conclusion that EPA's evaluation of health benefits is unrealistically high, by a factor that could well exceed 1,000, and that it is therefore very likely that the costs of the 1990 CAAA exceed its benefits, plausibly by more than 50-fold. Our reasoning involves reexamining specific uncertainties (including model uncertainty, toxicological uncertainty, confounder uncertainty, and uncertainty about what actually affects the timing of death in people) that were acknowledged qualitatively, but whose discrete contributions to uncertainty in health benefits were not quantified in EPA's cost-benefit analysis.

Of greater methodological interest than the difference in conclusions is the difference in uncertainty analyses. If EPA's assessment of uncertainties were correct, then it should be impossible, or at least very improbable, for any plausible variations in assumptions to reverse the main conclusion that CAAA benefits greatly exceed its costs. Yet, this is precisely what we will show: plausible variations in assumptions easily reverse the sign of the estimated net benefits. The principal methodological challenge that we seek to address in this article is, therefore, to diagnose the features of the uncertainty analysis that led to such a confident—but, we think, erroneous—conclusion, and to understand how to avoid such overconfidence in future risk-cost-benefit analyses.

2. THE MAIN LOGIC OF EPA'S BENEFIT ASSESSMENT

Most of the benefit that EPA projects for 2020 is based on the estimated number and dollar value of mortalities that it expects will be prevented in that year by having cleaner air. The main calculation is simple:

$$\begin{aligned} \textit{Benefit} = {} & \textit{reduced number of deaths in } 2020 \\ & \times \textit{value per statistical life saved} \\ & \approx (230{,}000 \text{ fewer deaths}) \\ & \times (\$8{,}900{,}000 \text{ per life saved}) \approx \$2 \text{ trillion.} \end{aligned} \tag{1}$$

The rationales for these two input values are provided in detail in EPA's report.[2] The 230,000 number for fewer deaths is given in Table 5-6, and is the estimated reduction in mortalities for all adults over 30 from reduced fine particulate matter ($PM_{2.5}$) in air. The value per statistical life saved (VSL) in 2020, of \$8,900,000, is given in Table 5-4. The actual calculation is more refined, as it includes discounting and additional sources of benefits (e.g., from reduced morbidities and improved visibility, as well as reduced mortalities from lower ozone levels), but these make little difference to the final answer, which is still close to \$2 trillion worth of benefits for 2020, and even more in later years. As EPA states: "These avoided deaths are valued at 1.8 trillion (\$2006), with primary low and primary high bounds on this estimate of \$170 billion to \$5.5 trillion."[2] The reduction to \$1.8 trillion reflects the passage of time before the estimated benefit is received. We will therefore focus on this simple calculation, and on the uncertainty analysis that led to these stated lower and upper bounds, as these calculations drive both the main quantitative results and the important qualitative conclusion that the value of health benefits almost certainly greatly exceeds the costs of compliance, which EPA estimates (Table 7-4 of Ref. 2) as \$65 billion for 2020.

3. EPA'S INPUT ESTIMATES AND WEIBULL UNCERTAINTY ANALYSIS

The upper and lower bounds for the \$2 trillion main estimate of benefits reflect uncertainties about the number of mortalities that will not occur in 2020 because of cleaner air, as well as uncertainty about the correct VSL value to use. The reduction in mortalities is estimated by multiplying estimated reductions in $PM_{2.5}$ concentrations by an estimated concentration-response potency factor, called the C-R coefficient. As EPA explains (p. 5-10): "We based the primary C-R coefficient estimate of the Second Prospective study on a Weibull distribution with a mean of 1.06 percent decrease in annual all-cause mortality per one $\mu g/m^3$. This mean is roughly equidistant between the results of the two most well-studied PM cohorts, the ACS cohort (0.58, as derived from Pope *et al.*, 2002) and the Six Cities cohort (1.5, as derived from Laden *et al.*, 2006), both of whose results have been robust to continued follow-up and extensive reanalysis." Similarly, for the VSL number, EPA again used a Weibull uncertainty distribution, noting (Table 5-4) that: "[The] Mean Value of Statistical Life (VSL) [is] based on 26 wage-risk and contingent valuation studies. A Weibull distribution, with a mean of \$7.4 million

(in 2006$), provided the best fit to the 26 estimates." The $7.4 million value is shown for 1990 income levels, and is updated to $8.9 million for 2020 income levels. These two Weibull distributions, for the C-R coefficient and VSL, together with estimated reductions in $PM_{2.5}$ concentration levels due to CAAA, drive the $2 trillion benefit estimate and the upper and lower bounds around it.

4. QUESTIONING THE LOGIC: DOES THE MAIN CALCULATION MAKE SENSE?

EPA's main analysis, as just summarized, has the commendable virtues of simple, transparent logic and well-documented input values and reasoning. It invites open inspection by the scientific community, so that any important deficiencies can be noted and, if necessary, repaired. To such deficiencies, we now turn.

The logic of Equation (1) requires estimating the reduction in mortalities in 2020 due to cleaner air (reduced $PM_{2.5}$ concentrations) resulting from the CAAA. The total number of mortalities in 2020 *with* the CAAA is (or should be) calculated as follows: from the number of mortalities that would have occurred in 2020 without the CAAA (as estimated from population sizes and mortality rates), *subtract* the number of mortalities that are postponed until 2021 or later because of reduced mortality rates attributed to the CAAA, and *add* the number of mortalities that are postponed until 2020 from 2019 or earlier because of reduced mortality rates attributed to the CAAA. The difference between this number (i.e., the mortalities that occur in 2020 *with* the CAAA) and the original number (i.e., the mortalities that occur in 2020 *without* the CAAA) is the total change in mortalities in 2020 attributable to the CAAA. In other words:

deaths in 2020 with CAAA
= *number that would have occured without CAAA*
− *deaths postponed because of CAAA until after* 2020 + *deaths postponed because of CAAA until 2020 from earlier.* (2a)

Similarly:

change in deaths in 2020 attributable to CAAA
= − *deaths postponed because of CAAA until after 2020* + *deaths postponed because of CAAA until 2020 from before 2020.* (2b)

Unfortunately, EPA's calculation includes only the first term in the change (that is, the reduction –*deaths postponed because of CAAA until after 2020*), but neglects the second term (that is, the increase *deaths postponed because of CAAA until 2020 from before 2020*). EPA estimates that the average gain in life expectancy is less than a year (Table 5-8), so if mortality rates and population sizes change relatively slowly between 2019 and 2021, the deaths postponed from 2020 to 2021 or later will be approximately equal to the deaths postponed until 2020 from 2019 or earlier. Hence, *the net change in deaths in 2020 will be approximately zero,* instead of the 230,000 reduction in mortality estimated by EPA.

More generally, calculating "deaths avoided" in any year is not the correct risk analysis tool to use to estimate the health benefits from a regulation or other intervention. A correct analysis (e.g., using age-specific hazard functions and life table analysis) shows that even a regulation that successfully decreases age-specific mortality rates, thereby increasing life expectancy, may not reduce the annual number of deaths in the population.

4.1. Example: Accounting for Deaths

The following simplified numerical example illustrates why a regulation that extends lives does not necessarily reduce deaths per year. For simplicity, suppose that, before the regulation, each individual lived to age 80, and then had a 50-50 chance of either dying at age 80, or of surviving for a further decade and dying at age 90. Suppose that the regulation prevents early deaths, so that all those who would have died at age 80 now die at age 90. (Those who would have died at age 90 still do so.) Now, how many people die each year before and after the regulation? (Assume a constant birth rate of N people per year, which is unaffected by the regulation.) Before the regulation, N people die each year (half of the cohort of N people born 80 years ago + half of those born 90 years ago, for a total of $N/2 + N/2 = N$). After the regulation has fully taken effect, N people still die each year (all of the cohort born 90 years ago). Although half of the population lives longer, the number of deaths per year is unchanged.

Of course, even if the net deaths prevented in 2020 are zero (as will necessarily be true under steady-state conditions, and as will be approximately true if the population size is changing relatively slowly on a time scale of months to years), this would not imply that increasing life expectancy by several

months has no value. But it does suggest that Equation (1)'s emphasis on reduced mortalities in 2020 is the wrong way to capture this value. The problem is that the main projected health benefits are not (or should not be) deaths "prevented" or "avoided," or lives "saved," but rather life-years gained. EPA's own report[2] explains this well: "Avoided premature mortality is one of the more commonly cited results of benefits analyses for air pollution control. However, as noted in the valuation section of this chapter, *a more accurate description of the benefit of clean air is a reduction in the risk of mortality* for the exposed population over many years, which results in the extension of lives (sometimes referred to as 'lives saved'). *Other useful metrics of the benefit of cleaner air are the number of life years that are gained through the reduction of mortal risks*, and the number of years of life expectancy gained on average throughout the population" (emphases added).

Accordingly, we can redo the calculation of benefits in Equation (1), switching from deaths "prevented" or avoided (which, in reality, do not occur, since reducing $PM_{2.5}$ does not confer immortality) to life-years gained. EPA estimates (Table 5-8 of Ref. 2) that, in 2020, almost 2 million (1,900,000) life-years will be gained because of cleaner air caused by CAAA. No value of a statistical life-year ($VSLY) gained is explicitly identified in EPA's report, and values in the literature vary widely, depending on the elicitation techniques used and the ages of the recipients. We tentatively use a $VSLY value of $150,000 per life-year, which has previously been used for life-years gained by cancer patients through the year 2020.[3] (For comparison, if we assumed, simplistically, that the $8,900,000 VSL number used in Equation (1) consists of at least 50 equally valued life-years, then the corresponding $VSLY value would be at most $8,900,000/50 = $178,000. This ignores the important complexities of age-specific $VSLY values and discounting, but suggests that the estimated $VSLY value of $150,000/year is not grossly inconsistent with EPA's $VSL number.) Equation (1) can now be replaced with the following estimated value of health benefits based on estimated life-years gained:

$$\begin{aligned} \textit{Benefit} &= (\textit{life-years gained in } 2020) \\ &\quad \times (\$\textit{VSLY per life-year gained}) \\ &= (1{,}900{,}000\,\textit{life-years gained}) \\ &\quad \times (\$\textit{150,000/ life-year gained}) \\ &= 0.3\,\textit{trillion}. \end{aligned} \tag{3}$$

Thus, simply replacing lives saved with life-years gained, and hence the $VSL value in Equation (1) with the $VSLY value in Equation (3), reduces the benefit estimate from about 2 trillion to about 0.3 trillion.

As it happens, a similar numerical conclusion results even if the logic of Equation (1) is retained, but the ages at which projected benefits occur are taken into account. EPA estimates the median age of people whose lives are "saved" (i.e., who gain extra months of life) from cleaner air to be close to 80 years (Table 5-8 of Ref. 2). The $VSL of $8,900,000 is appropriate for a healthy young adult of age about 25 (Fig. 1 in Ref. 4). The $VSL for people aged close to 80 has been estimated to be approximately one-sixth of the $VSL for people aged about 25 (Fig. 1 in Ref. 5). Thus, using $VSL numbers that condition on the ages at which benefits are received would also reduce the estimated value of health benefits substantially, with a sixfold reduction reducing EPA's current estimate of $1.8 trillion to a revised estimate of $0.3 trillion. Despite this numerical coincidence, however, we believe that Equation (3) is logically (and perhaps ethically) preferable to Equation (1), since, as EPA notes, the true benefit from reduced mortality rates is gain in expected life-years.

Two other ways of framing the simple analysis in this section may help to clarify the crucial role that conditioning on age should play in health effects benefits assessments. The first is that, in very round numbers, about 2 million life-years gained in 2020 can create about $2 trillion worth of benefit only if each life-year gained is valued at about a million dollars (or, equivalently, at about $80,000 per additional month of life). This is a much higher value than many octogenarians place on their own gains in life-years.[4,5] Second, applying a $VSL value appropriate for a 25-year old to people who are mostly over 75 (and a significant fraction of whom are over 100 years old, according to EPA's[2] Table 5-8, since this group contributes as much to estimated life-years gained as the 40–49-year olds in 2020) suggests that cleaner air is being evaluated, perhaps inadvertently, as a sort of fountain of youth, restoring the full value of youth (e.g., with at least another 50 years of healthy life expectancy) to people who, in fact, have a median age close to 80 years.[2] This appears to be overly optimistic.

Health economics studies of the costs and benefits of medical interventions routinely quantify health benefits in terms of the monetized value of

quality-adjusted life-years (QALYs), per the recommendation of the U.S. Panel on Cost Effectiveness.[6] QALY values of between $50k and $100k per year are common, and values up to $200k can be found, but these values are substantially less than the $1 million that EPA implicitly places on a life-year (without adjusting for quality). Thus, whether one uses monetized values of "lives saved," or life-years added, or quality-adjusted life-years added, EPA's valuation appears to be higher than others for similarly aged (and perhaps similarly infirm) people by approximately one order of magnitude.

5. QUESTIONING THE MAJOR PREMISE: WILL FURTHER REDUCING $PM_{2.5}$ CONCENTRATIONS REALLY REDUCE MORTALITY RATES FURTHER?

Even more important than uncertainty about the correct value of $VSL (or of $VSLY) is uncertainty about the correct value of the C-R coefficient that converts reductions in $PM_{2.5}$ concentrations to corresponding projected reductions in mortality rates. If the C-R coefficient is zero, then the health benefit from further reducing $PM_{2.5}$ concentrations is also zero. The assumption that the C-R coefficient is positive might be wrong. EPA's qualitative discussion of key uncertainties (Table 5-11 of Ref. 2) explicitly identifies this crucial assumption, noting that the "[a]nalysis assumes a causal relationship between PM exposure and premature mortality based on strong epidemiological evidence of a PM/mortality association. However, epidemiological evidence alone cannot establish this causal link." The possible impact of this assumption is identified as being a "potentially major" overestimate of benefits. However, EPA rates its confidence in its causal hypothesis as "High," arguing that: "The assumption of causality is suggested by the epidemiologic and toxicological evidence and is consistent with current practice in the development of a best estimate of air pollution-related health benefits. At this time, we can identify no basis to support a conclusion that such an assumption results in a known or suspected overestimation bias." EPA's quantitative uncertainty analysis goes further, implicitly expressing 100% confidence that this causal hypothesis is correct. The Weibull uncertainty distribution for the C-R coefficient implicitly assigns a probability of 100% to the C-R coefficient being positive, and hence a 0% probability to it being zero (or negative). Thus, the qualitative assessment of "high" confidence is implicitly mapped to a quantitative assessment of 100% certainty. Such complete certainty is unwarranted by available data and knowledge, as discussed next.

5.1. Is There Really a Significant Positive Association Between $PM_{2.5}$ and Mortality Rates?

Being unable to identify a basis for a suspicion of an overestimation bias does not, of course, guarantee that there is no such bias. Nor, perhaps, should a (subjectively perceived) suggestion of the possibility of a causal relation in epidemiological and toxicological data, or consistency with current practices in developing best estimates, warrant a very confident conclusion—let alone 100% confidence—that a causal relation necessarily exists.

For toxicological data, both theory and empirical evidence indicate that the inflammation-mediated adverse health effects of fine particulates probably have thresholds below which the C-R coefficient is zero.[7,8] For example, low levels of exposure induce increased antioxidant (AOX) production in the lung, but higher levels induce levels of reactive oxygen species (ROS) that overwhelm the very limited capacity of the lung to increase AOX. Increased disease risks occur only when there is an imbalance that leads to a sustained high-ROS lung environment and other changes that disrupt homeostasis, which may not occur at or near currently permitted exposure levels.[7]

For epidemiological data, the National Academy of Sciences and several expert statisticians have long questioned whether reported findings of significant positive C-R coefficients may be misinterpreting ambiguous data as showing evidence of a significant association where none actually exists.[9–11] This concern is based largely on awareness that multiple testing biases (which inflate the rate of false positives) can arise when pollution health effects investigators make numerous modeling choices (e.g., of which pollutant summaries, confounders, covariates, and interactions to include in the model; what logarithmic or other transformations to apply to them; lags in variables; model form specifications; treatment of exposure uncertainties, etc.) in arriving at the reported significant positive associations. Attempts to increase the objectivity of conclusions about PM-mortality rate associations, by considering all of the effect estimates from many (e.g., thousands of) computer-generated models that fit the available data approximately well, without applying human judgment to select or defend any particular model or conclusion,

have found that the hypothesized associations may not exist. For example, some Bayesian model-averaging (BMA) analyses (averaging effects over multiple models, weighted by their likelihoods in light of the data) of time series studies have reported, contrary to findings based on subjective selection of modeling assumptions, that available time series data sets give no overall indication of a positive relation between $PM_{2.5}$ and mortality rates.[9,10] The probability that current or lagged $PM_{2.5}$ values help to predict mortality rates (compared to predictions based solely on nonpollutant variables such as temperature and pressure) ranges from about 2% to 7% in some BMA studies,[10] and associations between pollutants (including particulate matter and ozone) and mortality rates have been reported as not significantly different from zero in some Canadian and U.S. investigations.[9–11] However, the BMA analyses were conducted for time series data, and thus are not directly comparable to the long-term cohort studies used by EPA. We do not know whether the conclusion that only a small fraction of models show any association of PM with mortality rates would hold in the two main studies (the Harvard Six Cities and American Cancer Society cohort study data) used in EPA's assessment.[2]

Against such reports of apparently nonexistent associations, some investigators whose methods and models have informed EPA's benefits assessment have argued that "Bayesian approaches in general and BMA in particular are not panaceas for model selection." (This is certainly true, as they are not model selection methods at all.) They further note that Bayesian approaches may be mistakenly applied, and therefore urge that "model selection should be informed by substantive knowledge about the environmental health processes influencing the outcome."[12] This response appears to reject the BMA principle that model selection should not be used at all (since even the "best" single model is almost certain to be wrong), but to advocate instead the continued use of judgment, "informed by substantive knowledge," as a basis for selecting models (which, in turn, may drive conclusions that are more consonant with the modelers' judgments about what should be found). This reopens the possibility that confirmation bias (selecting modeling assumptions that are favored by prior expectations or expert opinions) and multiple selection bias might explain some reported positive associations. For example, imposing an *a priori* modeling assumption that mortality rates increase approximately in direct proportion to pollutant levels at low concentrations (e.g., in a Cox proportional hazards model), or that the C-R function is linear at low concentrations (and piecewise linear overall), makes it possible to conclude that the C-R curve "is linear, clearly continuing below the current U.S. standard of 15 $\mu g/m^3$."[13] In this way, substantive knowledge or *a priori* assumptions and model selection constraints can still be used to produce low-dose linear results and a positive C-R coefficient. EPA (2011, Table 5-11, p. 5-42) identifies the "[a]ssumption of a linear, no-threshold model for PM and ozone mortality" as a "[k]ey uncertainty," but assesses as "High" its subjective confidence that this choice of model is correct, and as "Probably minor" the potential impact on overestimation of benefits of considering other model forms. This contrasts with time series studies that conclude that considering other models removes all significant positive associations between $PM_{2.5}$ and mortality rates.[9–11] A possible explanation is that the one cohort study that EPA's table cites in support of these crucial conclusions (an EPA—(Health Effects Institute)-funded study of the ACS data)[14] focused on models (random effects Cox proportional hazards and Poisson regression models) that imply low-dose linearity, rather than considering other (e.g., J-shaped or threshold) models that could have revealed absence of a positive C-R relationship at low exposure levels.

Even without further discussing, or seeking to resolve, how best to use computationally intensive statistical methods such as BMA to more fully characterize uncertainty about the C-R coefficient, it seems clear that there is significant uncertainty about whether a true association exists between ambient $PM_{2.5}$ concentrations and mortality rates; it is still being investigated. This crucial uncertainty should not be lightly dismissed. EPA's qualitative caveat deserves emphasis, that the "[a]nalysis assumes a causal relationship between PM exposure and premature mortality based on strong epidemiological evidence of a PM/mortality association, [but] epidemiological evidence alone cannot establish this causal link." Even this caveat should perhaps be tempered further, by acknowledging that calling the epidemiological evidence favoring a PM/mortality rate association "strong" may largely reflect unvalidated modeling assumptions (e.g., of a linear, no-threshold C-R relation at ambient levels). Frequent minority reports of statistically significant *negative* C-R coefficients, in models that allow this possibility, raise further questions about the coherence and reality of claimed

statistical associations. For example, a recent review of associations between $PM_{2.5}$ and all-cause and cause-specific mortality in 27 U.S. communities indicated *reduced* all-cause mortality rates at increased levels of $PM_{2.5}$ for one-third of the communities, including Birmingham, Dallas, Houston, Las Vegas, and Riverside.[12]

To understand the sensitivities of EPA's cost-benefit assessment to plausible alternative assumptions, it suffices to note that the question of whether a real (data-driven, rather than model selection-driven) association exists between $PM_{2.5}$ concentrations and mortality rates remains open, apparently on fairly substantive grounds, with at least some BMA studies indicating rather strongly (e.g., with greater than 95% probability) that there is no such association.[10] Lack of consistency in effects across studies, and large, unexplained heterogeneity in apparent C-R functions (including some with negative slopes) across locations, add qualitative reasons to question whether a stable, coherent association exists.[9–11,15] This uncertainty should be explicitly addressed and, if possible, quantified, as BMA analyses seek to do, rather than being tacitly ignored, for example, by using only a single average value across all studies[15] (perhaps with subjective confidence intervals) without explaining or resolving the observed negative relations.

5.2. Are Reported Statistical Associations Causal?

Even if a real (model-independent) statistical association exists, it might not be causal. Many investigators have recognized that covariates and confounders (such as proportion of high-risk minorities in a city, or correlates of wealth or education or poverty, or residual confounding by weather variables, including high temperatures or barometric pressures, or changes and trends in these variables) might create significant statistical associations between pollution and mortality rates, even if the former does not necessarily cause the latter. Similarly, regressing some trend variables (e.g., declining cause-specific and overall mortality rates in the wake of innovations such as angioplasty, anti-smoking campaigns, and aspirin therapy) against others (such as declining ambient pollution levels in recent decades) can show strong, statistically significant associations between them, with reductions in exposure concentrations routinely being followed (as well as preceded) by reductions in mortality rates, even if there is no causal relation between them.

Such threats to valid causal interpretation of statistical associations are well recognized by many investigators in pollution health effects research, but approaches for trying to address them have varied widely. To meet such challenges and threats to valid causal inference more decisively, econometric and statistical tests for potential causation between variables have been extensively developed, using concepts such as conditional independence and Granger causality for multiple time series.[16] However, these methods have generally not yet been applied to $PM_{2.5}$ and mortality data. Thus, the question of whether any statistical associations might be causal has not been settled using such relatively formal, objective tests. As previously noted, inconsistencies across cities and studies, and large, unexplained heterogeneity in C-R coefficients estimated in different studies, including some significant negative ones, undermine the coherence of proposed causal interpretations. Thus, the question of whether there is any positive *causal* relation between ambient levels of ozone and $PM_{2.5}$ and mortality rates is a second important, discrete, uncertainty that should be explicitly addressed in uncertainty analysis. It cannot be answered by arguing that there is a positive *statistical* association between them (at least under some model selection assumptions), since statistical and causal relations have no necessary connection (e.g., omitted confounders can make statistical relations positive even if causal relations are zero or negative). Nor can it be settled by concluding, based on expertise, that the data are *consistent* with such a relationship,[2] if the data are at least equally consistent with the opposite conclusion of no relationship.[9–11] We conclude that the question remains unsettled, and that it is appropriate for inclusion in uncertainty analysis.

EPA's benefit assessment[2] (pp. 5-7 to 5-10) recognized and addressed the possibility of confounding in its uncertainty analysis, as follows:

> Several other sources of uncertainty exist in the relationship between ambient pollution and the health outcomes, including model uncertainty, potential confounding by factors that are both correlated with the health outcome and each other, and potential misclassification of the study population exposures. . . . EPA recently conducted an expert elicitation (EE) study, which is the formal elicitation of subjective judgments, in order to more fully characterize the uncertainty surrounding the $PM_{2.5}$/mortality C-R function. This study allowed experts to consider and integrate several sources of uncertainty in the form of a probability distribution for the C-R function . . . [W]e rely exclusively on longterm studies to quantify PM mortality effects. This is because cohort studies are able to discern changes in mortality

> rates due to long-term exposure to elevated air pollution concentrations. ... Based on consultations with the Council's Health Effects Subcommittee (HES), the 812 Project Team developed a distribution of C-R function coefficients (i.e., the percent change in annual all-cause mortality per one $\mu g/m^3$ change in annual average $PM_{2.5}$) for use in the PM-mortality C-R function for the Second Prospective study. This distribution is rooted in the epidemiological studies that most inform our understanding of the PM mortality C-R function, but reflects the broader findings of the EE study. We based the primary C-R coefficient estimate of the Second Prospective study on a Weibull distribution with a mean of 1.06 percent decrease in annual all-cause mortality per one $\mu g/m^3$. This mean is roughly equidistant between the results of the two most well-studied PM cohorts, the ACS cohort (0.58, as derived from Pope *et al.*, 2002) and the Six Cities cohort (1.5, as derived from Laden *et al.*, 2006), both of whose results have been robust to continued follow-up and extensive re-analysis.

Thus, EPA explicitly recognized the possibility of confounding. It dealt with uncertainty about whether confounding, model uncertainty, or other sources of noncausal associations (such as exposure measurement error or misclassification) might have created an overall false-positive association between $PM_{2.5}$ concentrations (C) and health responses (R) in past studies, by (1) relying on two studies that reported significant positive associations; (2) discarding, or leaving unexplained, studies that reported significant negative or nonsignificant C-R associations; and (3) applying subjective expert judgments, encoded as a Weibull distribution (which cannot show zero or negative relationships), to conclude that a positive causal relation exists with 100% subjective confidence probability. (The Weibull distribution implicitly assigns a subjective probability of zero to the discrete possibility that fully controlling for all relevant confounders and trends would yield a negative or zero causal estimate of the percent decrease in annual all-cause mortality per $\mu g/m^3$ of $PM_{2.5}$.)

Whether or not its conclusion is correct, this *process*—considering only data and probability distributions that support or logically require, *a priori*, the conclusion of a positive relation between exposure and health effects—is surely not adequate for characterizing uncertainty about whether the underlying assumption that the true (unconfounded) C-R relation is positive is correct. The results of expert elicitation, expressed in the form of a distribution for a (causally interpreted) C-R coefficient, are also of uncertain value as a guide to objective truth, insofar as (1) the elicited distribution does not match, and hence does not explain, the empirically observed distribution of C-R coefficients in meta-analyses of dozens of reputable studies, about one-third of which are negative;[15] and (2) no one currently knows how or whether further reducing ambient concentrations of $PM_{2.5}$ *per se*—as opposed to reducing other activities, conditions (e.g., temperature extremes), and pollutant constituents (e.g., various oxides and sulfates) that are strongly correlated with $PM_{2.5}$—would cause any incremental reductions in mortality risks.

Experts, like other people, may have opinions on these matters. But one cannot elicit from experts knowledge that they do not have. In this case, correct "substantive knowledge" of causal relations showing how reducing current and future ambient $PM_{2.5}$ concentrations would affect human health, or how and whether other confounders and their interactions affect the estimated C-R relations for $PM_{2.5}$, is not yet available. We do not yet even know which components of $PM_{2.5}$ (if any), possibly interacting with which other pollutants, cause adverse health effects under present ambient exposure conditions. This does not necessarily imply that no new action should be taken. But it does imply that, if $PM_{2.5}$ *per se* is not involved in causing increased mortality rates, then further reductions in ambient $PM_{2.5}$ levels may not produce any health benefits. This possibility should be acknowledged in uncertainty analysis of human health benefits by including a substantial discrete (nonzero) uncertainty about causation.

5.3. Do C-R Relations Have Thresholds or J-Shapes?

Even if historical data showed a clear, unambiguous, causal relation between concentration and response, with past declining levels of $PM_{2.5}$ (or C) consistently causing proportionally declining levels of mortality rates (or R), it would be important to establish whether further reductions in C would produce further reductions in R. At some point, C might reach (or might already have decreased below) a threshold below which no further reductions in R are gained. Uncertainty about that threshold, and about whether current standards are already below it, should then be quantified. Or, conceivably, continuing reductions in C might eventually pass a point after which further reductions in C actually increase R, as suggested by a few of the U-shaped or J-shaped empirical C-R model curves fit to PM_{10} data for the 20 largest U.S. cities.[17] (This might also explain the previously noted disconcerting finding of negative C-R coefficients in approximately one-third of cities

examined.)[15] The probability of this contingency, and the conditional probability that we have already approached or passed the bottom of such a J-shape if it exists (at least in some cities), should limit the certainty with which additional reductions in C can be projected to cause additional reductions in R.

Existing discussions in the air pollution health effects literature of the possibility of a threshold for the C-R function largely recapitulate the technical issues in discussions of whether a nonzero C-R coefficient exists at all. Those who favor a linear, no-threshold model argue that model selection criteria such as the Akaike information criterion (AIC) can be used to support it,[13] insofar as other models tested typically do not give significantly better AIC scores. Those who are more agnostic about the linear no-threshold model point out that the AIC is not appropriate for this use: in simulation experiments (for which the correct model is known) it only identified the correct model about half the time, and assuming a linear no-threshold model can greatly under- or overestimate true mortality risks if nonlinearities are present but are mistakenly assumed away based on AIC.[18] Defenders of the linear no-threshold model further counter that population data exhibit no thresholds, but appear to be linear down to the lowest levels measured.[17] Skeptics could respond that this pattern is precisely what one might expect if there really is a population threshold, but individual exposure estimates contain unmodeled errors, as in past studies; thus, the observed population C-R function is irrelevant to determining whether there is a true C-R threshold.[7] That errors in past exposure estimates cast doubt on inferences about health effects is starting to be more widely acknowledged,[19] but no one has yet advanced a model that clearly fits most of the data, reconciles past inconsistencies, accounts for past measurement errors, and resolves the debate with an obviously correct C-R relation.

As in the case of uncertainty about whether a positive C-R coefficient exists, the role of uncertainty analysis is not to resolve whether threshold or J-shaped C-R relations exist (at least at some locations), but only to make sure that any significant uncertainties about these possibilities are identified and explicitly addressed in the presentation and evaluation of uncertain health benefits projected from reductions in exposure concentrations. However, EPA's quantitative uncertainty analysis implicitly assumes that there are *no* uncertainties about the correct form (linear, no-threshold with probability 100%) or sign (positive with 100% probability, zero or negative with 0% probability) of the C-R relation. Admitting that there are such uncertainties would substantially change both the uncertainty analysis and the evaluation of projected health benefits, as explained in Section 6.

5.4. How Much Smaller Will Future C-R Coefficients Be Than Past Ones?

Finally, suppose that a true, causal, linear C-R coefficient were to be confidently identified in historical data. Even this would not warrant a conclusion that *future* reductions in pollutant levels would achieve proportionate reductions in mortality rates and gains in life-years. Progress in medicine, including preventive medicine, is steadily shrinking the pool of highest-risk individuals whose lives might, under the above assumptions, be most prolonged by reducing pollutant levels. Long-term trends such as improved diet and exercise among adults, greater use of low-dose aspirin regimens and other medications to successfully prevent cardiovascular disease events, smoking reduction and cessation programs, and new treatment and therapeutic options (such as angioplasty), have led to a long-term trend of declining mortality risks from key diseases identified by EPA as being reduced by lower pollutant levels.

Some experts interpret epidemiological data as showing that "[s]hort-term and long-term studies clearly indicate that relatively modest exposures to particulate matter in the ambient air are associated with increased morbidity and mortality due to coronary heart disease."[20] Death from coronary heart disease (CHD), especially among the elderly and patients with preexisting cardiovascular diseases, has been proposed as a specific mortality risk caused by high levels of particulate pollution.[20,21] The future relation between $PM_{2.5}$ and mortality risks, therefore, may be affected by trends in CHD risk. These trends show very significant declines in CHD mortality risks over the past four decades, with further declines expected. For example, a recent review[22] that quantified temporal trends in cardiovascular mortality risks in Minneapolis–St. Paul, MN found that mortality rates for nonhospitalized men fell by about 2/3 between 1985 and 2008 (from about 150/100,000 in 1985 to about 50/100,000 in 2008). Similarly, for the United States as a whole, a recent World Health Organization (WHO) study[23] found that "[s]ince the late 1970s, age-adjusted CHD mortality rates have been halved in most industrialized countries,

including the United States. . . . Approximately 44% of the substantial CHD mortality decline in the United States between 1980 and 2000 was attributable to changes in major risk factors [smoking, systolic blood pressure, total blood cholesterol, body mass index], and 47% to specific cardiological treatments. These findings resembled those from other industrialized countries. . . . In conclusion, implementing evidence-based policies to better control tobacco use and achieve a healthier diet across the population could potentially halve future CHD deaths in the United States." Although future reductions in risk will depend on future trends in diet, exercise, obesity, aging of the population, and medical interventions and treatments, there appear to be continued large opportunities for continued risk reductions in the United States.[23] As such trends continue, the incremental benefits from additional pollution control might also be expected to shrink, as fewer people remain at high risk of mortality from pollution-associated diseases such as CHD. Thus, analysis of projected future benefits should include uncertainty about the size of future C-R coefficients, rather than assuming that C-R coefficients estimated from historical data will remain unchanged by current and future trends.

6. DISCRETE VERSUS CONTINUOUS UNCERTAINTY ANALYSIS

The major uncertainties about the health effects of reducing pollutant concentrations in air can be summarized as the following series of questions:

- *Is there a true C-R association?* What is the probability that there is a true (independent of model selection assumptions) statistical association between lower pollutant concentrations (C) and lower mortality rates (R)?
- *Is it causal?* Assuming that there is a true C-R association, is it causal? That is, will future reductions in $PM_{2.5}$ and ozone cause corresponding reductions in future mortality rates? Or, to the contrary, is the association noncausal, for example, reflecting past downward trends in both variables that do not represent a causal relation, and that do not support manipulating future mortality rates by changing future ambient pollutant concentrations? What is the probability that the C-R association (if there is one) is causal?
- *Is it linear nonthreshold (or are present and future concentrations above any applicable threshold, or on the ascending part of any J-shaped C-R function)?* Assuming that a causal C-R relation exists in past data, for concentration levels prevalent then, how likely is it that a similar C-R coefficient will continue to hold in future, as ambient concentrations continue to fall? What is the probability that we are *not* yet at or below a nadir, threshold, or threshold-like nonlinearity in the C-R function, which would cause future reductions in C to produce no further reductions in R, and hence no incremental health benefits? Colloquially, we might wonder: How clean is clean enough to achieve the possible health benefits, and have we already reached a point past which no additional health benefits will accrue to additional reductions in concentrations? What is the probability that this is not the case?
- *Is it stable over time?* What is the probability that C-R coefficients will remain approximately the same, despite improvements in prevention and medical treatments for the diseases that are associated with pollutants? If the C-R coefficient does decline in future, as the at-risk population declines, then what is its new expected value?

A notable aspect of these uncertainties is that they are discrete: one can imagine answering yes or no to each one, if perfect information were available, and can therefore envision assigning a discrete probability (a number between 0 and 1) to each of these possibilities now, based on the imperfect information available now.

For purposes of plausible numerical illustration and sensitivity analysis, suppose that the quantitative probabilities for answers to these questions are as follows:

- *Probability that there is a true association* $\leq$ 50%. As discussed previously, the data on associations are conflicting, with both positive and negative significant C-R associations being reported, and with results of individual studies appearing to be sensitive to choices of statistical methods and modeling assumptions.[9] BMA analyses that permit the possibility of no association between $PM_{2.5}$ and mortality rates generally find little support for the hypothesis that such an association exists at ambient concentrations; the probability that it does

exist was estimated as between about 2% and 7% in a recent BMA analysis.[10] Against this conclusion, many experts are convinced that the combined findings from multiple epidemiological studies "clearly indicate that relatively modest exposures to particulate matter in the ambient air are associated with increased morbidity and mortality due to coronary heart disease,"[20] and that the main remaining scientific challenge is to figure out why and how this occurs. It is not necessary to choose between these conclusions for purposes of illustrating how such uncertainties affect final benefits estimates. Instead, we will use a 50% probability of a true association as a point of departure for quantitative analysis, and then use sensitivity analysis to understand how changes in this probability affect conclusions.

- *Probability that a true association, if it exists, is causal* (and not explained away by trends, confounding, or the distinction between statistical and causal associations) ≤ 0.5. The inconsistencies and large unexplained differences in estimated C-R functions, signs, and magnitudes across studies—even for the same chemical, such as ozone—and failure to find clear evidence of a coherent causal relation at ambient levels in clinical, laboratory, or experimental studies despite decades of evidence might suggest a "more likely than not" conclusion that there is not a causal relation. A probability interval of ≤ 0.5 captures this uncertain probability. This is strengthened by some past reviews and conclusions, for example, that "[b]ecause the mortality risk estimates from important observational epidemiologic studies are extremely weak, derived from studies unable to control for relevant confounding causes, and inconsistent by location, toxicologic and clinical information is necessary to judge the likelihood and degree to which such findings are causal. Toxicologic data on typical forms of pollution-derived PM strongly suggest that current ambient concentrations in the United States are too small to cause significant disease or death. . . . The expectation that lives will be saved by reducing ambient $PM_{2.5}$ in the United States is not supported by the weight of scientific evidence, although other bases for regulating PM may be justifiable."[24]
- *Probability that currently permitted concentrations are above any threshold* or nadir in the C-R function (if any; or probability that there is no such threshold or nadir) ≤ 0.5. Repeated finding of negative as well as positive C-R coefficients might suggest the possibility of a J-shaped function, and other reasons for expecting a threshold have previously been discussed.[7] The inconsistencies and difficulties in identifying any clear positive C-R association[24] also suggest that we could now be below the clearly rising portion of a C-R function.
- *Expected reduction factor in C-R coefficient by 2020* ≤ 0.5, assuming that a linear no-threshold causal C-R coefficient exists. Improved medical diagnosis, intervention (e.g., with aspirin therapy, smoking cessation programs, or cholesterol-reducing measures, including changes in diet and exercise), and treatment, which are increasingly being used, can potentially reduce premature CVD mortalities by 80–90% or more, but an aging or more obese population over the next decade could modestly increase the C-R slope.[23] Based on these opposed trends, and in light of the past four decades of declining CVD risks, an overall reduction factor of 0.5 might not be implausible as a starting point for the reduction in any real C-R coefficient, although higher or lower values might be developed with additional modeling. We will use 0.5 as a point of departure for understanding the sensitivity of estimated benefits to this factor.

Each of these factors is conditioned on the outcomes of all of its predecessors being such that a positive linear no-threshold causal C-R coefficient exists.

With these rough estimates of factors (using the upper end of each uncertainty interval, e.g., 0.5 for the interval ≤ 0.5), the health benefits estimated by EPA would be adjusted as follows:

> (1.8 trillion initial estimate) × (1/6 reduction factor for \$VSL if age or \$VSLY is considered) × (0.5 probability that a true association exists) × (0.5 probability that a true association is causal, given that one exists) × (0.5 probability that ambient concentrations are above any thresholds or nadirs in the C-R function, given that a true causal C-R relation exists) × (0.5 expected reduction factor in C-R coefficient by 2020 due to improved medication and prevention of disease-related mortalities) = (1/6)*0.5*0.5*0.5*0.5 = \$19 billion.

In round numbers, the combination of assumptions shown here reduces the estimated value of health benefits in 2020 100-fold, from \$2 trillion to \$20 billion. Instead of being much greater than the

estimated compliance costs of $65 billion, the estimated value of health benefits is substantially less. This conclusion is robust to changes in any of the above factors, in that replacing any of them with 1 (no reduction) would not change the conclusion that benefits are less than costs. Sensitivity analysis of this product is trivial: changing any factor by k-fold changes the entire product by k-fold, for any $k > 0$.

The point of departure values for this calculation were deliberately chosen realizing that they might be conservative (erring in the direction of overstating C-R relations and health benefits), with all factors ≤ 0.5 being rounded up to 0.5 for purposes of this initial calculation. Two main changes that might increase realism would significantly reduce estimated benefits. One would be to accept at face value the BMA results indicating that a true positive association between present ambient levels of $PM_{2.5}$ and mortality rates is unlikely.[10] This could reduce the point of departure for this probability by about a further order of magnitude, from 0.5 to about 0.05, and hence reduce the plausible high estimate for monetized value of annual health benefits from $19 billion to $1.9 billion. (A strong conviction that causality is very unlikely would have a similar effect.) The other main change would be to replace the 1/6 reduction factor for $VSL with a reduction factor of 1/10 for $VSLY (or value of a QALY), as discussed earlier. This would reduce annual benefits estimates by a further factor of 0.6 (e.g., from $1.9 billion to approximately $1 billion). Such numbers are well below the $65 billion in annual compliance costs estimated by EPA. If these two alternative values were used as points of departure, then the conclusion that expected benefits are less than expected costs would be robust to changes that increased any or all of the remaining factors to 100%.

These calculations are all based on expected values (albeit for upper bounds, if the points of departure are indeed conservative). For a risk-averse individual or society, however, the economic value of an uncertain benefit is *less* than its expected value, by an amount that depends on the risk premium for uncertainty. A further reduction in the evaluation of the uncertain benefits (by perhaps a further factor of 2, depending on the risk premium) would be needed to take into account risk aversion for uncertain gains.

Such further adjustments and refinements, while possibly producing a more realistic answer, might also invite inessential controversy. Our main conclusion, that the 1990 CAAA benefits plausibly amount to only a small fraction of their direct compliance costs (the only costs considered in the $65 billion estimate), does not depend on choosing very small probabilities as points of departure, nor on greatly revising EPA's value judgments about $VSL or $VSLY, nor on incorporating risk aversion for uncertain benefits. Rather, it is a robust result of the combination, in series, of multiple substantial uncertainties. The result is robust in the sense that no particular one of these uncertainties (and no small subset, if the suggested smaller points of departure are adopted) is essential for reaching this conclusion.

However, the conclusion that the 1990 CAAA human health benefits are very probably substantially smaller than compliance costs offers a sharp contrast to EPA's more reassuring conclusion that: "***The extent to which estimated benefits exceed estimated costs*** *and an in-depth analysis of uncertainties* ***indicate that it is extremely unlikely the costs of 1990 Clean Air Act Amendment programs would exceed their benefits under any reasonable combination of alternative assumptions or methods identified during this study***"[1] What explains the difference? The key is that *EPA's uncertainty analysis ignores discrete uncertainties,* such as those we have enumerated, in favor of a continuous, nonnegative probability distribution—the Weibull distribution—that puts zero probability density on zero or negative numbers as possible values. Applied to the crucial C-R coefficient, the Weibull uncertainty distribution implies 100% confidence that the coefficient is positive, notwithstanding the numerous studies that have reported zero or negative coefficients. This framing of the uncertainty analysis only allows one to ask (and experts to answer) *how large* are the assumed benefits, but not to question (or provide opinions on) whether they exist. Thus, the major discrete uncertainty about whether positive benefits exist at all—the main focus of our analysis—is simply assumed away in EPA's Weibull uncertainty distribution.

In this context, EPA's conclusion that "it is extremely unlikely the costs of 1990 Clean Air Act Amendment programs would exceed their benefits under any reasonable combination of alternative assumptions or methods identified" is no surprise. It is little more than an assertion of a tautology, similar to: "Assuming that I am right, it is extremely unlikely that any reasonable combination of alternative assumptions would show that I am wrong." But this style of uncertainty analysis fails to cast any light on uncertainty about the major premise. The not-implausible numbers offered above suggest that the

probability of zero human health benefits might well exceed 1 – (0.5*0.5*0.5) = 87.5% based solely on uncertainties about whether there is a positive, causal (not due to confounding or coincidence of trends), nonthreshold C-R relation for current and future ambient pollutant concentrations (assuming, conservatively, that there is a 50% probability for each of these three uncertainties—if smaller points of departure are used, then the probability of zero human health benefits exceeds 99%). Thus, assuming that the probability of zero (or negative) human health benefits is zero substantially understates the true uncertainty, and hence substantially overstates the uncertain benefits of the CAAA.

The use of a Weibull uncertainty distribution also conceals other discrete uncertainties, arising from blinkered analysis and failure of imagination. For example, suppose that the CAAA creates more clear days and bright skies. How, if at all, would this affect peak temperatures during the summer, and mortality rates among people without air conditioners? How, if at all, would it affect the future burden of skin cancer? The cost-benefit analysis does not say, and the Weibull uncertainty analysis implicitly disregards all such uncertainties due to drivers of possible costs and benefits not already identified in the analysis. Or suppose that, contrary to our expectations, the CAAA does prolong the last year of life for a substantial fraction of elderly patients suffering from heart, lung, and circulatory diseases. How would this affect health care costs and living costs, and the benefits of longer life, as evaluated by patients and their families? These costs and uncertainties are similarly implicitly assumed away in EPA's analysis, but considering them might further increase the probability that the net benefits of the CAAA are negative, and further increase the estimated ratio of costs to benefits, beyond the roughly 65-to-1 value (or more) suggested by some of our calculations.

7. CONCLUSIONS

EPA's cost-benefit assessment indicates that the CAAA offers the United States an admirable return on investment: an expenditure of only $65 billion per year secures health benefits valued at nearly $2 trillion per year. If this is credible, any policymaker would be foolish to refuse it. EPA's uncertainty analysis suggests that the large excess of estimated costs over estimated benefits is not only credible, but virtually certain: it is deemed to be "extremely unlikely" that any reasonable changes in assumptions would reverse the sign of net benefits.

However, these promising conclusions depend essentially on the use of a Weibull distribution in the uncertainty analysis, which tacitly assumes away any possibility of serious, discrete errors or uncertainties in the key assumptions—especially about whether future reductions in $PM_{2.5}$ and ozone will necessarily cause future increases in life expectancy. Yet, this is exactly the uncertainty that a large, data-driven literature questions. Numerous reported findings of unexplained negative and highly heterogeneous positive C-R coefficients call into question the assumption that any such single, positive, causal coefficient exists.

Taking into account such discrete uncertainties suggests that the probability of achieving positive benefits could well be small (e.g., less than 12.5%, or less than 1%, if some BMA results[10] are used) so that EPA's uncertainty analysis assuming that this probability is 100% may be more misleading than informative. A policymaker to whom the CAAA costs and benefits are presented as: "Pay $65 billion for a chance of probably much less than 20% to achieve benefits of about $300 billion," as our numbers suggest, might well decide that the cost is too great for the uncertain benefits. Even if all of our suggested specific numbers are rejected and replaced by more carefully developed ones, it seems that considering discrete uncertainties has important effects on the analysis, and that such uncertainties should be presented to policymakers if they are to make well-informed decisions to serve the public interest.

We do not believe that the calculations presented here prove beyond doubt that the CAAA costs far more than it produces in benefits. We do think that they make this possibility very likely, and hence well worth including, instead of assuming away, in any uncertainty analysis of CAAA cost-benefit comparisons.

ACKNOWLEDGMENTS

I thank my friend Adam Finkel for drawing my attention to EPA's assessment of the monetized value of human health benefits from the 1990 CAAA (and its favorable reception by some leading economists), and for suggesting that I comment on it. This research was supported in part by the Center for Media and Public Affairs (CMPA) at George Mason University. I am grateful to the Area

Editor of *Risk Analysis*, Suresh Moolgavkar, and to several anonymous referees, as well as the reviewers at CMPA, for questions, comments, and suggestions for greater clarity that improved the final exposition. The research questions addressed, methods and models applied, and conclusions reached are solely mine.

REFERENCES

1. EPA. The Benefits and Costs of the Clean Air Act from 1990 to 2020: Summary Report. EPA. March, 2011.
2. EPA. The Benefits and Costs of the Clean Air Act from 1990 to 2020. 2011.
3. Yabroff KR, Bradley CJ, Mariotto AB, Brown ML, Feuer EJ. Estimates and projections of value of life lost from cancer deaths in the United States. Journal of the National Cancer Institute, 2008; 100(24):1755–1762.
4. Aldy JE, Viscusi WK. Age differences in the value of statistical life: Revealed preference evidence. Review of Environmental Economics and Policy, 2007; 1(2):241–260.
5. Murphy K, Topel R. The economic value of medical research. Pp. 9–40 in Murphy K, Topel R (eds). Measuring the Gains from Economic Research: An Economic Approach. Chicago, IL: University of Chicago Press, 2003.
6. Gold M, Siegel JE, Russell LB, Weinstein MC (eds). Cost-Effectiveness in Health and Medicine. New York: Oxford University Press, 1996.
7. Cox LA Jr. An exposure-response threshold for lung diseases and lung cancer caused by crystalline silica. Risk Analysis, 2011; 31(10).
8. Stoeger T, Reinhard C, Takenaka S, Schroeppel A, Karg E, Ritter B, Heyder J, Schulz H. Instillation of six different ultrafine carbon particles indicates a surface area threshold dose for acute lung inflammation in mice. Environmental Health Perspectives, 2006; 114(3):328–333.
9. Clyde M . Model uncertainty and health effect studies for particulate matter. Environmetrics, 2000; 11(6):745–763.
10. Koop G, Tole L. Measuring the health effects of air pollution: To what extent can we really say that people are dying from bad air? Journal of Environmental Economics and Management, 2004; 47:30–54. doi: 10.1.1.164.6048. Available at: http://citeseerx.ist.psu.edu/viewdoc/summary? Accessed October 12, 2011.
11. Koop G, Tole L. An investigation of thresholds in air pollution–mortality effects. Environmental Modelling & Software, 2006; 21(12):1662–1673.
12. Thomas DC, Jerrett M, Kuenzli N, Louis TA, Dominici F, Zeger S, Schwartz J, Burnett RT, Krewski D, Bates D. Bayesian model averaging in time-series studies of air pollution and mortality. Journal of Toxicology and Environmental Health Part A, 2007; 70(3-4):311–315.
13. Schwartz J, Coull B, Laden F, Ryan L. The effect of dose and timing of dose on the association between airborne particles and survival. Environmental Health Perspectives, 2008; 116(1):64–69.
14. Krewski, D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, et al. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Research Report (Health Effects Institute). 2009; 140:5–114.
15. Franklin M, Zeka A, Schwartz J. Association between PM2.5 and all-cause and specific-cause mortality in 27 US communities. Journal of Exposure Science and Environmental Epidemiology, 2007; 17(3):279–287.
16. Faes L, Nollo G, Chon KH. Assessment of Granger causality by nonlinear model identification: Application to short-term cardiovascular variability. Annals of Biomedical Engineering, 2008; 36(3):381–395.
17. Daniels MJ, Dominici F, Samet JM, Zeger SL. Estimating particulate matter-mortality dose-response curves and threshold levels: An analysis of daily time-series for the 20 largest US cities. American Journal of Epidemiology, 2000; 152(5): 397–406.
18. Roberts S, Martin MA. The question of nonlinearity in the dose-response relation between particulate matter air pollution and mortality: Can Akaike's information criterion be trusted to take the right turn? American Journal of Epidemiology, 2006; 164(12):1242–1250.
19. Sheppard L, Burnett RT, Szpiro A, Kim SY, Jerrett M, Pope CA, Brunekreef B. Confounding and exposure measurement error in air pollution. Air Quality, Atmosphere & Health, 2011, doi:10.1007/s11869-011-0140-9.
20. Simkhovich BZ, Kleinman MT, KlonerRA. Particulate air pollution and coronary heart disease. Current Opinion in Cardiology, 2009; 24(6):604–609.
21. Simkhovich BZ, Kleinman MT, Kloner RA. Air pollution and cardiovascular injury epidemiology, toxicology, and mechanisms. Journal of the American College of Cardiology, 2008; 52(9):719–726.
22. Adabag AS, Luepker RV, Roger VL, Gersh BJ. Sudden cardiac death: Epidemiology and risk factors. Nature Reviews Cardiology, 2010; 7:216–225.
23. Capewell S, Ford ES, Croft JB, Critchley JA, Greenlund KJ, Labarthe DR. Cardiovascular risk factor trends and potential for reducing coronary heart disease mortality in the United States of America. Bulletin of the World Health Organization, 2010; 88(2):81–160.
24. Green LC, Armstrong SR. Particulate matter in ambient air and mortality: Toxicologic perspectives. Regulatory Toxicology and Pharmacology, 2003; 38(3):326–335.