

Understanding the Health Effects of Stationary Source Emissions

Dan Greenbaum, President
Health Effects Institute

Ozone Transport Commission
Boston
November 10, 2010



Understanding the Health Effects of Stationary Source Emissions



- What do we know about effects?
 - Criteria Pollutants
 - Hazardous Air pollutants (e.g. metals, gases)
- Quantifying Health and Economic Impacts
- Hope for the future
- Concluding thoughts



The Health Effects Institute

- 30 years of providing impartial, high-quality science on health effects of air pollution
- Joint core funding from
 - Government (U.S. EPA)
 - Industry (Worldwide Vehicle and Engine Manufacturers)
- Expanded partnerships with:
 - Oil, Chemical, other industries
 - DOE, FHWA, WHO, California, other agencies
 - USAID, ADB, Hewlett Foundation
- Science products responsive, widely credible to global leaders
 - *Targeted research*
 - Over 250 studies on ozone, carbon monoxide, particulate matter, diesel exhaust, benzene, butadiene, MTBE, others
 - *Re-Analysis*
 - e.g. Harvard Six Cities and American Cancer Society Studies on PM; 30 revised “time-series” PM studies
 - *Rapid Review*
 - Traffic Health Effects, MTBE, Diesel Exhaust Epidemiology, Air Toxics
 - Air pollution and Health in Asia



HEI Structure and Approach

- HEI structured to maintain credibility & transparency in often controversial regulatory debates
 - Balanced government and industry funding
 - Independent Board and Expert Science Committees
 - Not affiliated with sponsors – no perceived “point of view”
 - *Research Committee* selects all research competitively
 - Separate *Review Committee* intensively peer reviews all results
 - All results and data – both positive and negative – reported
- Does not take policy positions



The HEI Strategic Plan 2010 - 2015

Next Generation Multipollutant Approaches
For conventional air pollutants...
... and at the air quality – climate nexus

**Cross-cutting
Issues**

*Sensitive
Populations*
*(e.g. elderly, lower
SES)*

Innovation
*(e.g. data,
statistics, omics)*

Validation
*(e.g. statistics,
new toxicology)*

**1. Multi-
pollutant
Exposure,
Epidemiology
and
Toxicology
Research**

**2. Assessing
Health Effects
of
Emerging
Fuels
and
Technologies**

**3. Measuring
the Health
Outcomes of
Air Quality
Actions
(Accountability)**

**4. An
International
Perspective in
the Developed
and
Developing
World**

Better Informed Air Quality and Climate Decisions in the Developed and Developing World

HEI



Emissions of Potential Concern

(a subset...)

- **Particulate Matter and Gases**
 - **PM**
 - **NO_x (+VOCs) > Ozone**
- **The “Fellow Travelers” (HAPs)**
 - **Reactive transition metals** (e.g. Fe, Cd, Mn, Pb)
 - **Acid Gases** (e.g. HCl)
 - **Dioxins**
 - **Mercury**

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PM and Gases

- Major sources:
 - fossil fuel combustion (coal and oil)
- High levels of PM ($> 500 \mu\text{m}^3$) known to cause premature death
 - e.g. London 1952
- Studies in US, Europe, elsewhere have found association of PM with mortality at much lower levels ($<50 \mu\text{m}^3$)
 - no evidence of a “threshold” (safe level)



Effects of long-term PM_{2.5} Exposure

Extended Follow-Up of the American Cancer Society Study of PM and Mortality;
HEI Report #140, 2009

Tracking detailed effects in 600,000 people over 18 years

*Large effects, especially for heart disease
(18% - 24% increase in risk per 10 µg/m³)*

Commentary Table 3. Associations Between Various Causes of Death and Long-Term Exposure to PM_{2.5} in Two Time Periods from the Nationwide Analysis^a

Cause of Death	Standard Cox Model	Random Effects Cox Model ^b
HR per 10-µg/m³ Change in PM_{2.5} Exposure Level (Average for 1979–1983)		
All causes	1.03 (1.01–1.04)	1.04 (1.03–1.06)
Ischemic heart disease	1.12 (1.09–1.16)	1.18 (1.15–1.22)
Cardiopulmonary disease	1.06 (1.04–1.08)	1.09 (1.06–1.11)
Lung cancer	1.08 (1.03–1.14)	1.09 (1.03–1.15)
HR per 10-µg/m³ Change in PM_{2.5} Exposure Level (Average for 1999–2000)		
All causes	1.03 (1.01–1.05)	1.06 (1.04–1.08)
Ischemic heart disease	1.15 (1.11–1.20)	1.24 (1.19–1.29)
Cardiopulmonary disease	1.09 (1.06–1.12)	1.13 (1.10–1.16)
Lung cancer	1.11 (1.04–1.18)	1.14 (1.06–1.23)

Many Components in PM:

Sulfate is associated with premature mortality,
even in multi-pollutant analyses
(ACS Analyses in Lancet 2009)

	PM _{2.5} (1.0 µg/m ³)	Ozone (1.0 µg/m ³)	Sulphate (1.0 µg/m ³)	Elemental carbon (1.0 µg/m ³)
All-cause mortality (deaths=93 358)				
Single-pollutant	0.58 (0.22 to 0.95)	0.04 (-0.01 to 0.09)	1.11 (0.78 to 1.44)	5.51 (0.74 to 10.51)
Multiple-pollutant	..	0.01 (-0.06 to 0.07)	..	5.16 (-0.51 to 11.17)
Multiple-pollutant	..	0.02 (-0.01 to 0.06)	1.09 (0.76 to 1.43)	..
Multiple-pollutant	1.06 (0.73 to 1.40)	2.70 (-1.01 to 6.57)
Multiple-pollutant	..	0.01 (-0.04 to 0.06)	1.07 (0.73 to 1.40)	2.11 (-2.44 to 6.89)
Cardiopulmonary mortality (deaths=46 168)				
Single-pollutant	1.27 (0.76 to 1.79)	0.12 (0.03 to 0.21)	1.55 (1.03 to 2.08)	10.60 (2.92 to 18.86)
Multiple-pollutant	..	0.08 (-0.02 to 0.18)	..	6.55 (-2.05 to 15.91)
Multiple-pollutant	..	0.10 (0.04 to 0.16)	1.54 (1.05 to 2.03)	..
Multiple-pollutant	1.46 (0.94 to 1.97)	7.05 (1.11 to 13.35)
Multiple-pollutant	..	0.09 (0.01 to 0.17)	1.51 (1.01 to 2.01)	2.09 (-4.53 to 9.18)

Data from the American Cancer Society Cancer Prevention II cohort (n=352 242), with follow-up from 1982 to 2000. Spatial survival model included random effects at the 66 metropolitan statistical areas that had all pollutants recorded for the national cohort. Survival model is stratified by age (1 year), sex, and race. Pollution effects adjusted for 44 covariates measured at the individual level and seven covariates measured at the ecological level for the zip code area of residence and for the zip code area deviated from the metropolitan area average. Relative risks presented in the first row for each cause of death are from single-pollutant models, whereas those in subsequent rows indicate pollutants simultaneously included in survival models. See webappendix pp 26–27 for details. PM_{2.5}=particulate matter with aerodynamic diameter 2.5 µm or less.

Table 2: Percentage changes of relative risk based on µg/m³ range of pollutant concentration by selected causes of death for single-pollutant and multiple-pollutant models

Ozone

- Formed from Nitrogen Oxides (NO_x) and Volatile Organic Compounds (VOCs)
- Known to:
 - Cause inflammation in respiratory tract
 - Reduce ability to breathe (lung function) for some
 - Increase hospitalization for asthma, other lung diseases
- Recent multi-city evidence of effects on premature mortality
 - Effects have been demonstrated for short term exposure
 - Less evidence of mortality effects from long term exposure



Ozone reduces lung function (ability to breath) in sensitive individuals

Evidence growing at lower levels

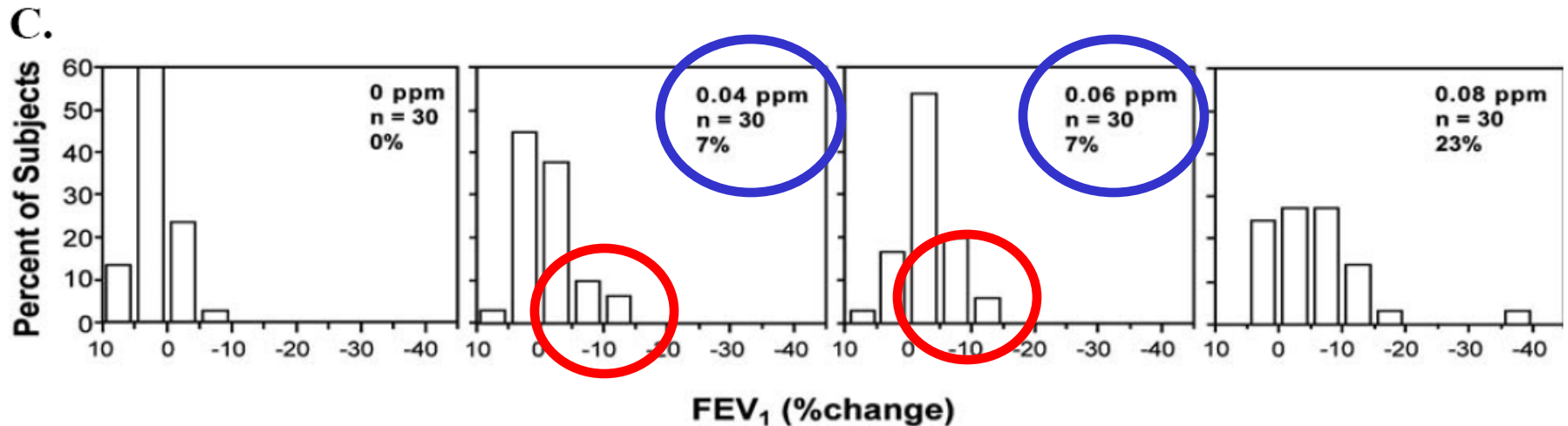
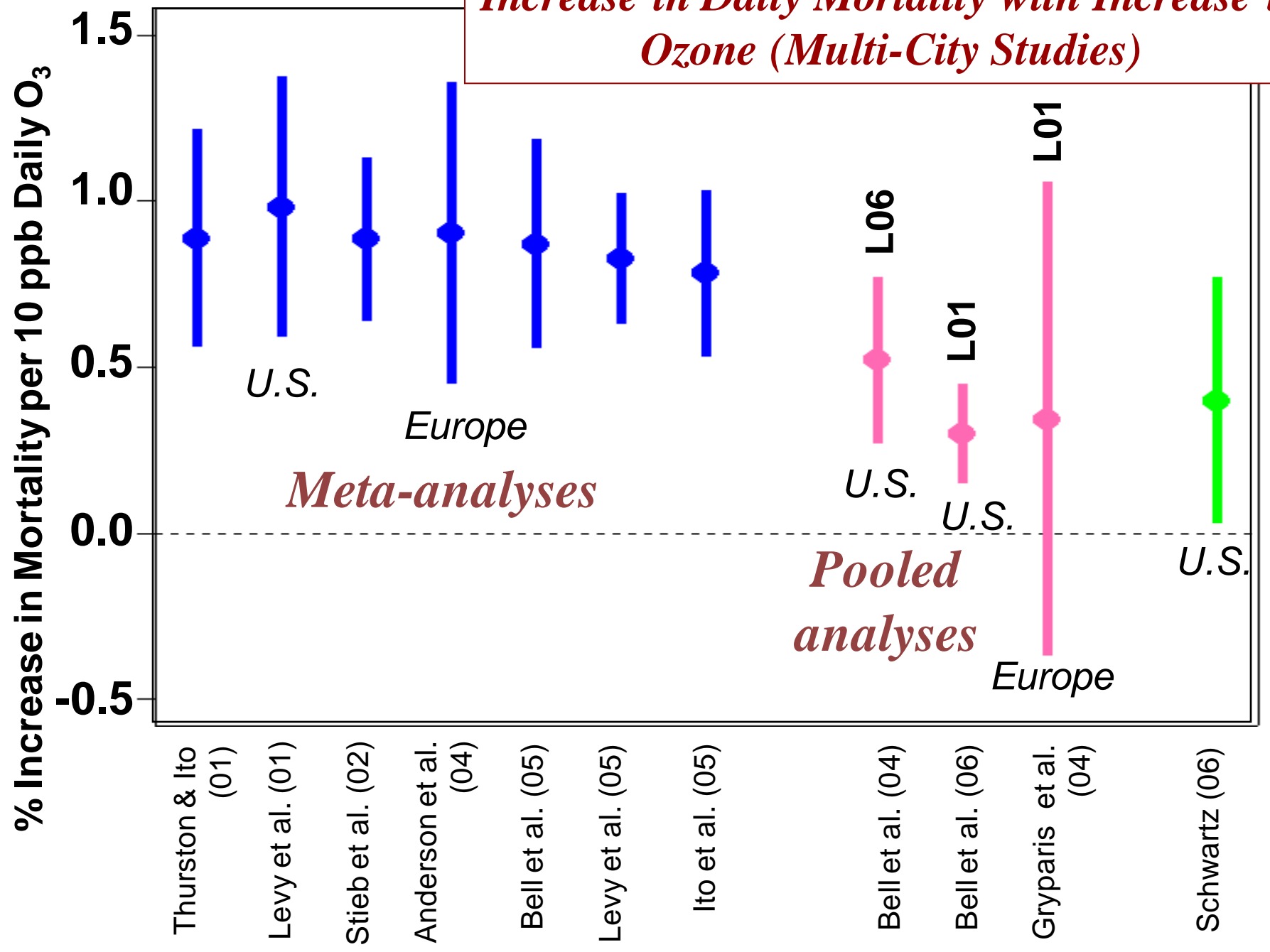


Figure 3-1C. Frequency distributions of FEV₁ changes following 6.6-h exposures to a constant concentration of O₃ or filtered air. The FEV₁ changes following O₃ exposures have been corrected for filtered air responses, i.e., they are O₃-induced FEV₁ changes. Note that the percentage in each panel indicates the distributions of % decrement.

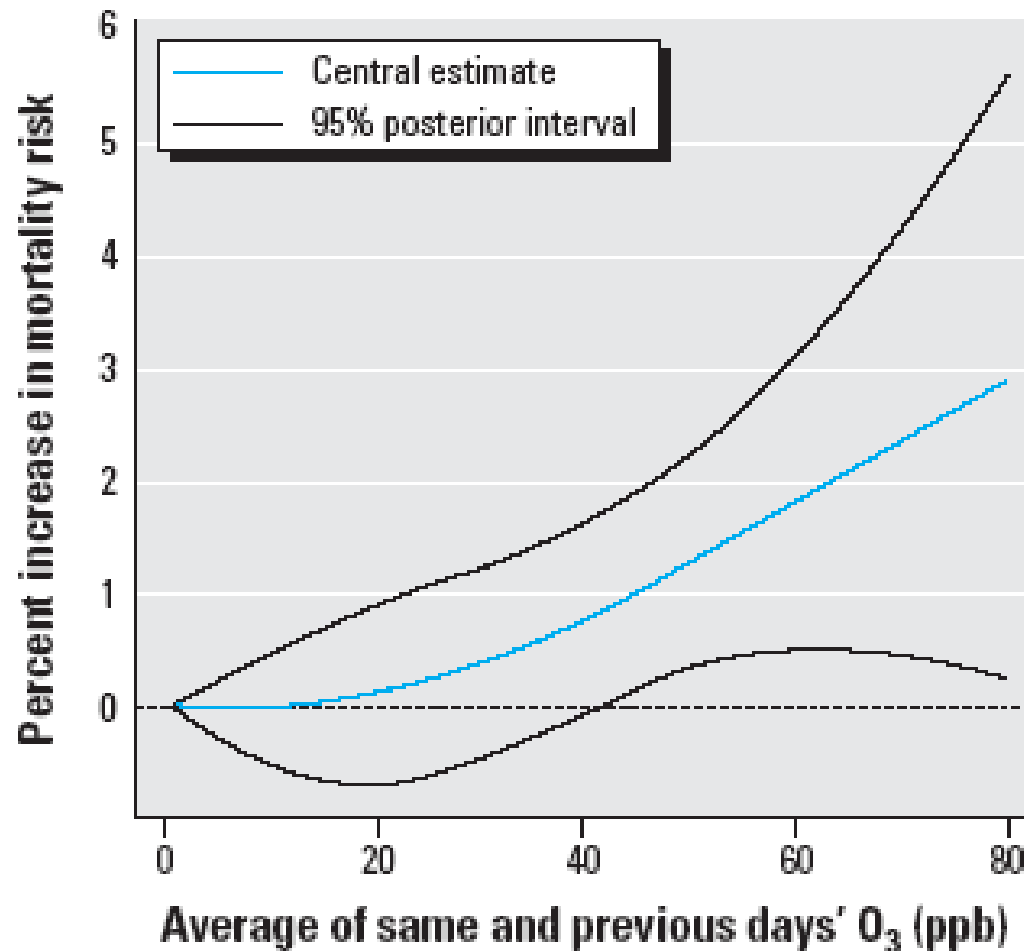
Source: Adams (2002, 2006), pre- and post- FEV₁ data for each subject provided by author.

Increase in Daily Mortality with Increase in Ozone (Multi-City Studies)



Ozone Effects on Daily Mortality 95 US Cities

Approximately 0.5% increase in mortality /10ppb
(Bell et al 2005)



ORIGINAL ARTICLE

Long-Term Ozone Exposure and Mortality

Michael Jerrett, Ph.D., Richard T. Burnett, Ph.D., C. Arden Pope III, Ph.D.,
Kazuhiko Ito, Ph.D., George Thurston, Sc.D., Daniel Krewski, Ph.D.,
Yuanli Shi, M.D., Eugenia Calle, Ph.D., and Michael Thun, M.D.

Table 3. Relative Risk of Death Attributable to a 10-ppb Change in the Ambient Ozone Concentration.*

Cause of Death	Single-Pollutant Model†			Two-Pollutant Model‡	
	Ozone (96 MSAs)	Ozone (86 MSAs)	PM _{2.5} (86 MSAs) <i>relative risk (95% CI)</i>	Ozone (86 MSAs)	PM _{2.5} (86 MSAs)
Any cause	1.001 (0.996–1.007)	1.001 (0.996–1.007)	1.048 (1.024–1.071)	0.989 (0.981–0.996)	1.080 (1.048–1.113)
Cardiopulmonary	1.014 (1.007–1.022)	1.016 (1.008–1.024)	1.129 (1.094–1.071)	0.992 (0.982–1.003)	1.153 (1.104–1.204)
Respiratory	1.029 (1.010–1.048)	1.027 (1.007–1.046)	1.031 (0.955–1.113)	1.040 (1.013–1.067)	0.927 (0.836–1.029)
Cardiovascular	1.011 (1.003–1.023)	1.014 (1.005–1.023)	1.150 (1.111–1.191)	0.983 (0.971–0.994)	1.206 (1.150–1.264)
Ischemic heart disease	1.015 (1.003–1.026)	1.017 (1.006–1.029)	1.211 (1.156–1.268)	0.973 (0.958–0.988)	1.306 (1.226–1.390)

Initial Evidence of Ozone Long Term Effects

Jerrett, et al March 2009



Some evidence of a threshold for long term effects (below 60 ppb)

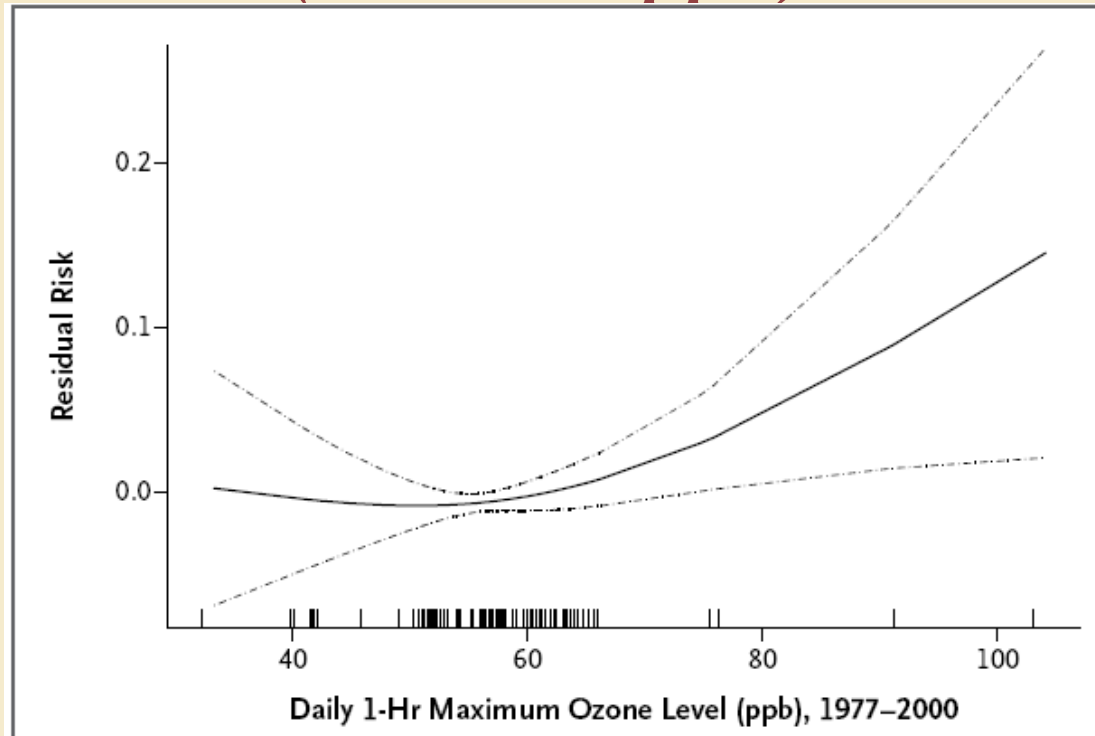


Figure 2. Exposure–Response Curve for the Relation between Exposure to Ozone and the Risk of Death from Respiratory Causes.

The curve is based on a natural spline with 2 df estimated from the residual relative risk of death within a metropolitan statistical area (MSA) according to a random-effects survival model. The dashed lines indicate the 95% confidence interval of fit, and the hash marks indicate the ozone levels of each of the 96 MSAs.



Ozone and PM Cardiovascular Effects?

- Extensive data on low-level lung effects of human exposure to ozone
 - Much less on cardiovascular effects, and multi-pollutant
- HEI RFA 10-1: Sought human controlled exposure experiments on cardiovascular effects
 - Older volunteers (e.g. 55)
 - Phase 1: Ozone exposures alone
 - Phase 2: Ozone exposures in an ambient setting (i.e. with other pollutants present)
- Three studies identified for Phase 1
 - Protocol development underway
- Revised Phase 2 RFA to issue in 2011



The “Fellow Travelers” (HAPs)

- **Reactive transition metals**
(e.g. Fe, Cd, Mn, Pb)
- **Acid Gases** (e.g. HCl)
- **Dioxins**
- **Mercury**



Metals

- Large number of **transition metals** in combustion emissions: Cd, Mn, Pb, Fe

- Various Sources:

- Coal
- Fuel Oil
- Vehicles

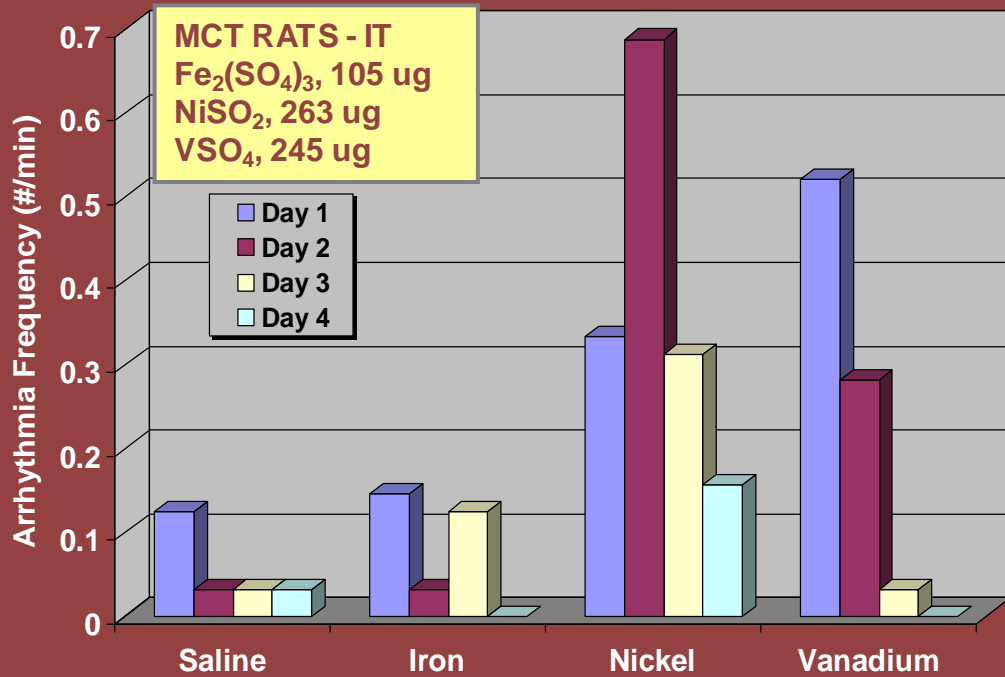
The periodic table is color-coded by groups. A legend in the upper right identifies the categories: Non Metals (white), Alkali Metals (blue), Alkaline Metals (cyan), Transition Metals (magenta), Rare Earth Elements (green), Noble Gases (black), Metalloids (yellow), Halogens (red), and Other Metals (grey). A black rectangular box highlights the transition metal region, which includes elements from Scandium (Sc) to Zinc (Zn) in period 4, and Yttrium (Y) to Cadmium (Cd) in period 5. The lanthanide and actinide series are shown at the bottom.

Group	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Period 1	1 H																	2 He
Period 2	3 Li	4 Be											5 B	6 C	7 N	8 O	9 F	10 Ne
Period 3	11 Na	12 Mg											13 Al	14 Si	15 P	16 S	17 Cl	18 Ar
Period 4	19 K	20 Ca	21 Sc	22 Ti	23 V	24 Cr	25 Mn	26 Fe	27 Co	28 Ni	29 Cu	30 Zn	31 Ga	32 Ge	33 As	34 Se	35 Br	36 Kr
Period 5	37 Rb	38 Sr	39 Y	40 Zr	41 Nb	42 Mo	43 Tc	44 Ru	45 Rh	46 Pd	47 Ag	48 Cd	49 In	50 Sn	51 Sb	52 Te	53 I	54 Xe
Period 6	55 Cs	56 Ba	57 La*	72 Hf	73 Ta	74 W	75 Re	76 Os	77 Ir	78 Pt	79 Au	80 Hg	81 Tl	82 Pb	83 Bi	84 Po	85 At	86 Rn
Period 7	87 Fr	88 Ra	89 Ac**	104 Rf	105 Db	106 Sg	107 Bh	108 Hs	109 Mt	110 Uu	111 Uu	112 Uu	113 Uu	114 Uuq	116 Uuh		118 Uuo	
*Lanthanides	58 Ce	59 Pr	60 Nd	61 Pm	62 Sm	63 Eu	64 Gd	65 Tb	66 Dy	67 Ho	68 Er	69 Tm	70 Yb	71 Lu				
**Actinides	90 Th	91 Pa	92 U	93 Np	94 Pu	95 Am	96 Cm	97 Bk	98 Cf	99 Es	100 Fm	101 Md	102 No	103 Lr				

- A variety of known effects, especially in occupational settings at high levels

- Neurotoxic effects
- Inflammation



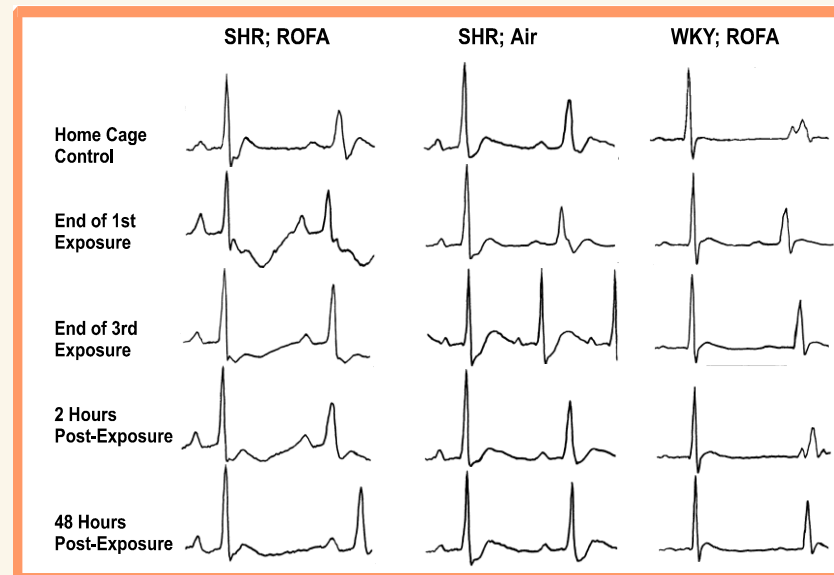


IT Metals Induce Arrhythmias

Campen et al., 2002

ROFA Inhalation Alters ECG Patterns in SH Rats

Kodavanti et al., 2000



Acid Gases

- Hydrogen Chloride, Hydrogen Fluoride, others
- Emitted from many sources
 - MACT in place or underway for cement kilns, boilers, incinerators and others (using HCl as metric)
- Health Effects
 - Corrosive to eyes, skin, mucous membrane
 - *Acute effects*: eye, nose, and respiratory irritation
 - *Chronic long term effects* (higher occupational exposures): gastritis, bronchitis, dermatitis
 - Generally not classified as to *cancer*

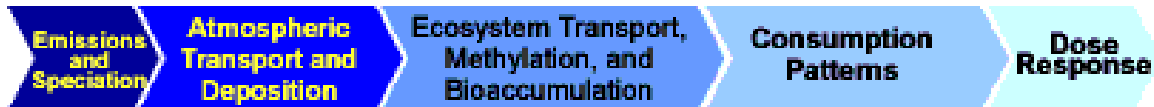
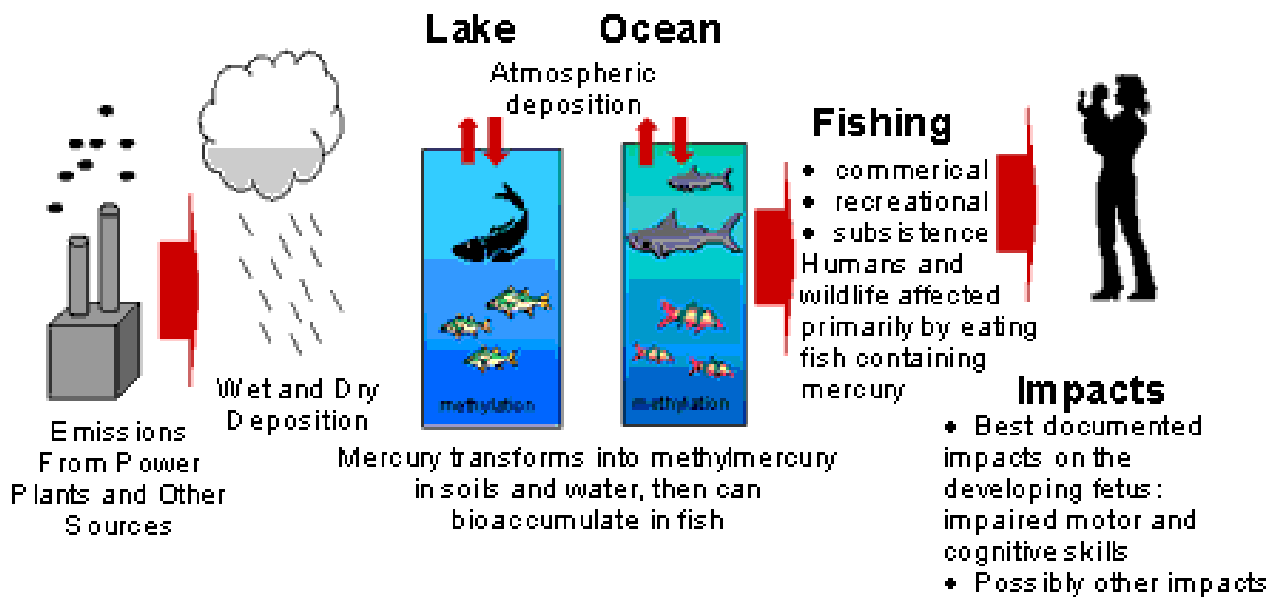


Dioxins

- Many sources:
 - Power plants estimated to contribute 5% of total (EPA)
- Cancer:
 - A range of compounds with differing toxicity
 - Most toxic (TCDD) seen as “likely to be a carcinogen” (NRC 2006)
 - Toxic Equivalency Factors (TEF) calculated to compare the range of compounds to the most potent
- Non-Cancer effects
 - Likely to cause toxicity to the immune system, though level at which that occurs needs better analysis (NRC)
 - Evidence of developmental and reproductive effects in animals, though not yet in humans



Mercury Routes of Exposure



Mercury Effects

- Known to cause significant neurological effects in high exposure “poisonings,” e.g.
 - Minimata, Japan
 - Iraq
- Evidence of learning, neurological effects at lower levels from diet studies in
 - Faroe Islands (positive)
 - New Zealand (positive)
 - Seychelles (no effects shown)
- US National Academy of Science (2000): Positive effects in 2 of 3 studies, EPA has basis for setting standards
- *Of special concern for effects in pregnant women and their children*



*How can we quantify the health
and economic impacts?*





BEST

Board on Environmental Studies and Toxicology

National Research Council Report, October 2009

Hidden Costs of Energy: Unpriced Consequences of Energy Production and Use

Presentation by

Jared Cohon
Carnegie Mellon University

Maureen Cropper
University of Maryland

Daniel Greenbaum
Health Effects Institute



Study Task and Approach

Task:

- Define and evaluate key external costs and benefits – related to health, environment, security, and infrastructure – that are associated with the production, distribution, and use of energy but not reflected in the market price of energy or fully addressed by current government policy.

Approach:

- The Full Lifecycle of Damages:
Emissions>>Ambient Concentration>>Exposure>>Effect>>
Monetized Damages
- Effects of air pollution on human health, grain crop and timber yields, building materials, recreation, and visibility of outdoor vistas.
- Health effects (mortality) provided larger estimated damages

Conclusions

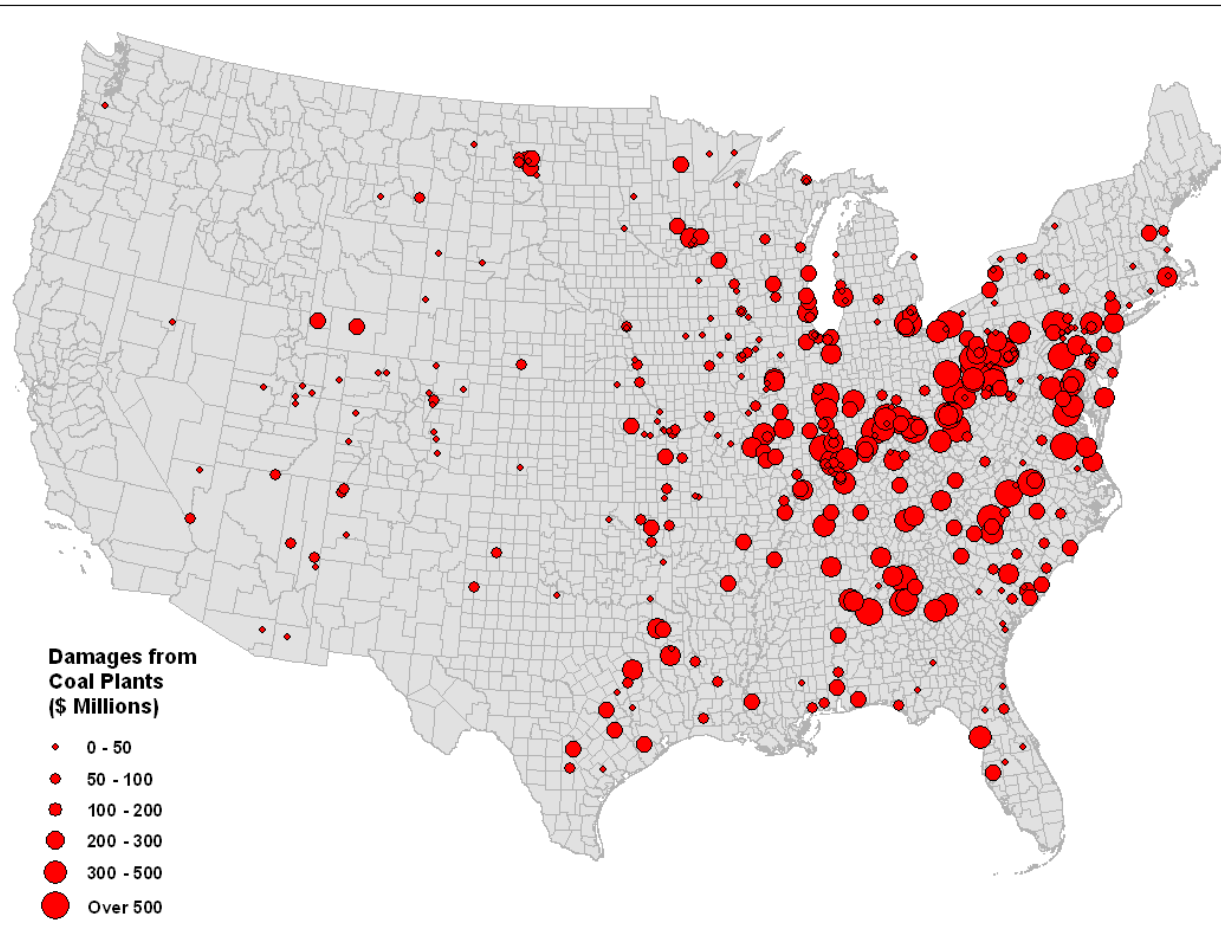
- Non-climate damages from electricity generation and transportation exceed \$120 billion for the year 2005. These damages are principally related to emissions of NO_x, SO₂, and PM.
- The above total is a substantial underestimate because it does not include damages related to climate change, health effects of hazardous pollutants, ecosystem effects, or infrastructure and security.
- How much a burden should be reduced depends on its magnitude *and* the cost of reducing it.
- Reducing emissions, improving energy efficiency, or shifting to cleaner methods of generating electricity could substantially reduce damages.



Electricity: Coal

\$62 Billion of Health and Other Non-Climate Damages in 2005

Damage Estimates based on SO₂, NO_x, and PM emissions



- Air Pollution Damages from Coal Generation for 406 plants, 2005
 - **3.2 cents/kWh**
- With control, damages lower in 2030
 - **1.7 cents/kWh**
- Damages related to climate-change effects are not included



Climate Change Damage Estimates (e.g. flooding, crops) may nearly double Health Effects Damages

Energy-Related Activity (fuel type)	Non-climate damage	Climate Damages (per ton CO ₂ -eq)		
		@ \$10	@ \$30	@ \$100
Electricity Generation (coal)	3.2 cts/kWh	1 cts/kWh	3 cts/kWh	10 cts/kWh
Electricity Generation (natural gas)	0.16 cts/kWh	0.5 cts/kWh	1.5 cts/kWh	5 cts/kWh
Transportation	1.1 to ~1.7 cts/VMT	0.15 to ~0.65 cts/VMT	0.45 to ~2 cts/VMT	1.5 to ~6 cts/VMT
Heat production (natural gas)	11 cts/MCF	70 cts/MCF	210 cts/MCF	700 cts/MCF



Hope for the Future

...based on the substantial progress we have already made





Advanced Collaborative Emissions Study (ACES)

**Emissions (Phases 1 and 2) and possible health effects (Phase 3)
of new advanced heavy duty engine and control systems and fuels
in the market 2007 – 2010.**



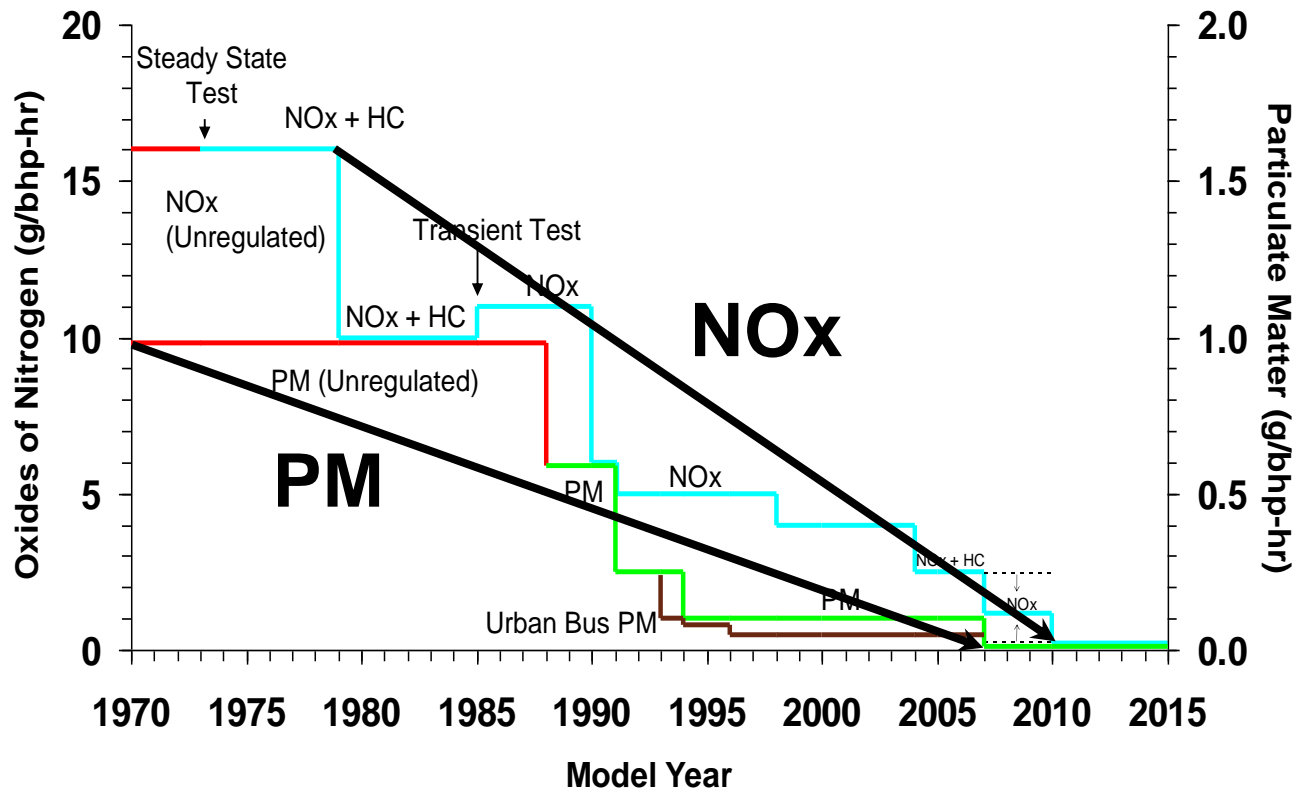
PROJECT SPONSORS

US Department of Energy (DOE) OVT and NETL
Engine Manufacturers Association (EMA)
US Environmental Protection Agency (EPA)
California Air Resources Board (ARB)
American Petroleum Institute (API)
Aftertreatment Manufacturers
Coordinating Research Council (CRC)



Improvements in PM and NOx Diesel Emission Standards

EPA Heavy-Duty Engine Emission Standards



ACES PHASE 1 Results:

Substantial Reductions below standards (except for NO_x which will be regulated in 2010)

Regulated Emissions Relative to EPA 2007 Standard Based on FTP Transient Cycle

	2007 EPA Standard (g/hp-hr)	Average ACES Engine Emissions (g/hp-hr)	ACES Emissions % Reduction Relative to the 2007 Certification Standard
CO	15.5	0.33	98
NMHC	0.14	0.0064	95
PM	0.01	0.0011	89
NO _x	1.2 ^a	1.075	10

^a Average value between 2007 and 2009, with full enforcement in 2010 at 0.20 g/hp-hr



Phase 1 Results: Unregulated Emissions

On a g/hr emission rate basis, the great majority of unregulated emission species were *substantially below the level observed with 2004 engine technology* used in CRC E55/59.

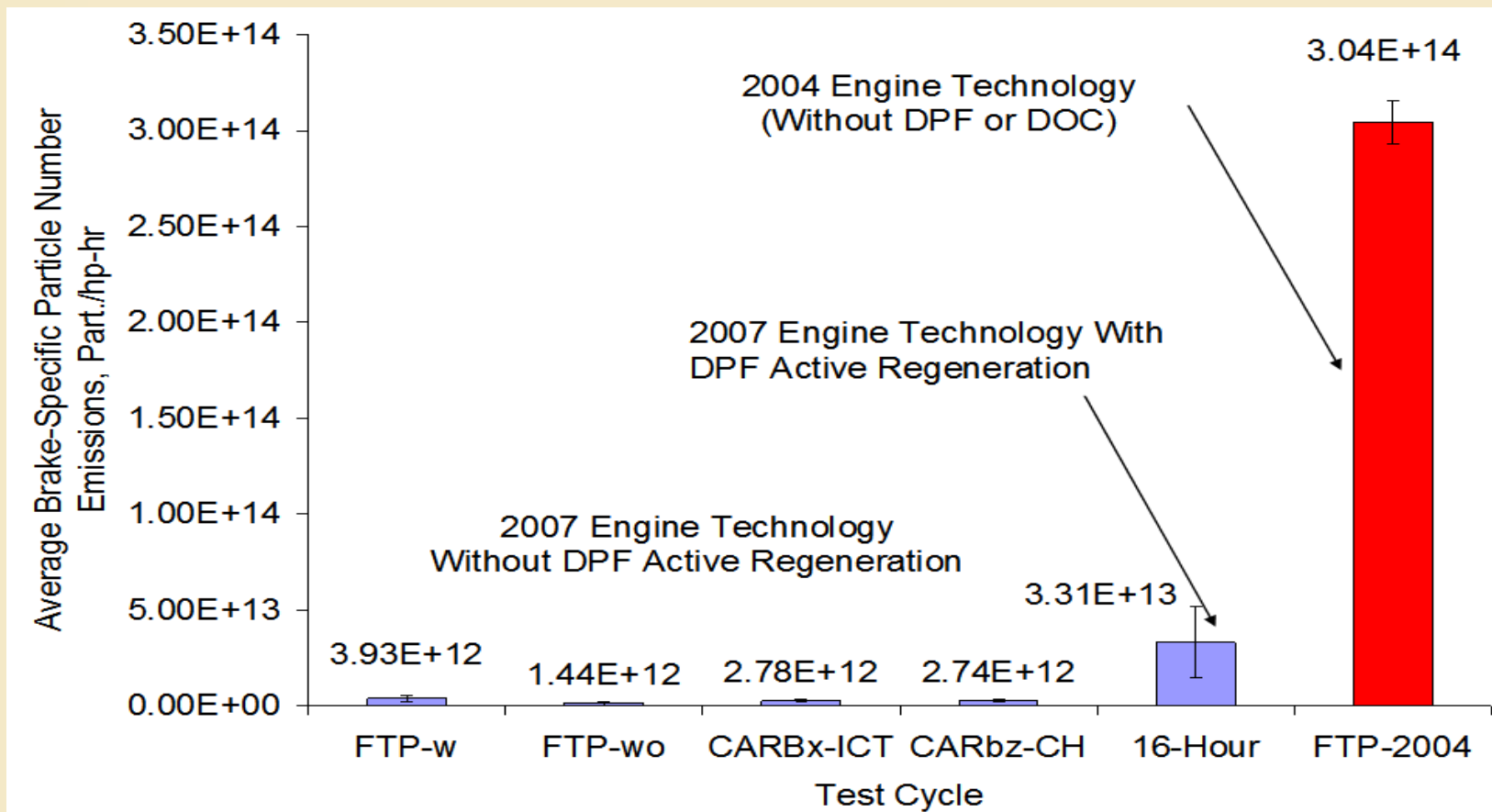
Compounds	% Lower Than 2004 Engine Technology	
	16-Hour Cycle	CARBx-ICT
Single Ring Aromatics	82%	69%
PAH	79%	26%
Nitro-PAH	81%	49%
Alkanes	85%	84%
Polar	81%	12%
Hopanes/Steranes	99%	99%
Carbonyls	98%	78%
Inorganic Ions	38%	100%
Metals and Elements	98%	90%
Organic Carbon	96%	78%
Elemental Carbon	99%	100%
Dioxins/Furans ^a	99%	N/A

^a Relative to 1998 Engine Technology

In general, the low exhaust temperature cycle CARBx-ICT showed less reduction for the hydrocarbon-based compounds, compared to the 16-Hour Cycle



Average Total Particle Number Emissions Reduced



- With regeneration, the particle number emissions average was approximately 90 percent lower than the level emitted by a 2004 engine technology, and without regeneration it was approximately 99 percent lower



***ACHIEVING CLEAN AIR AND PUBLIC HEALTH:
40 Years of Progress***

Dan Greenbaum, President
Health Effects Institute

The Clean Air Act at 40
September 14, 2010



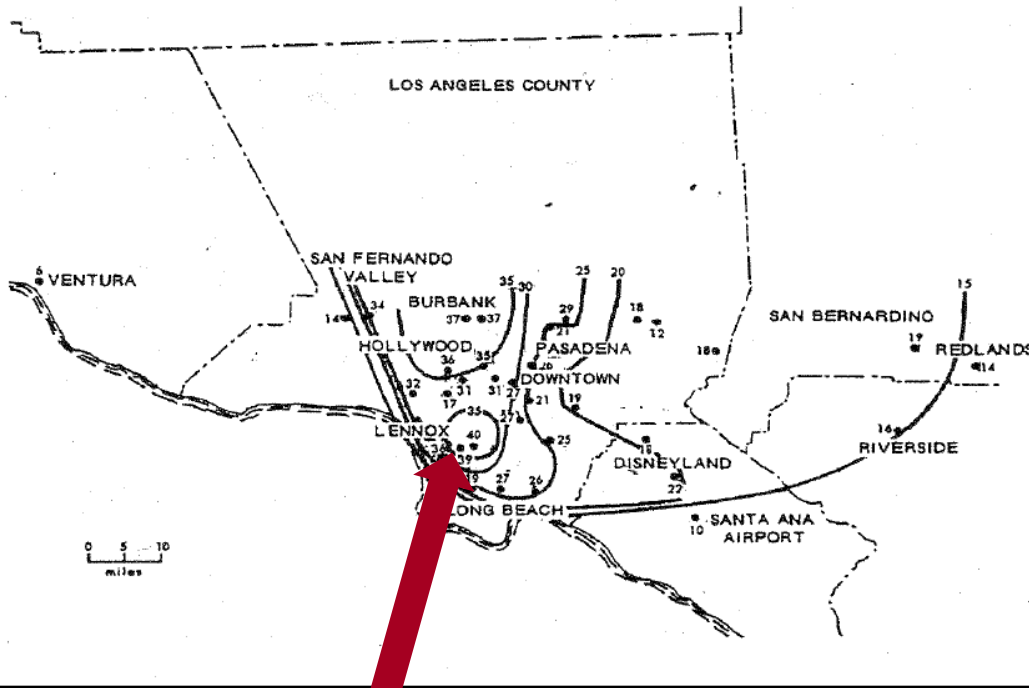
Clean Air Act Progress:

Carbon Monoxide
Levels in Los Angeles
1960s – 2000

Vehicles 

1960: 3.5 million
2008: 7.5 million

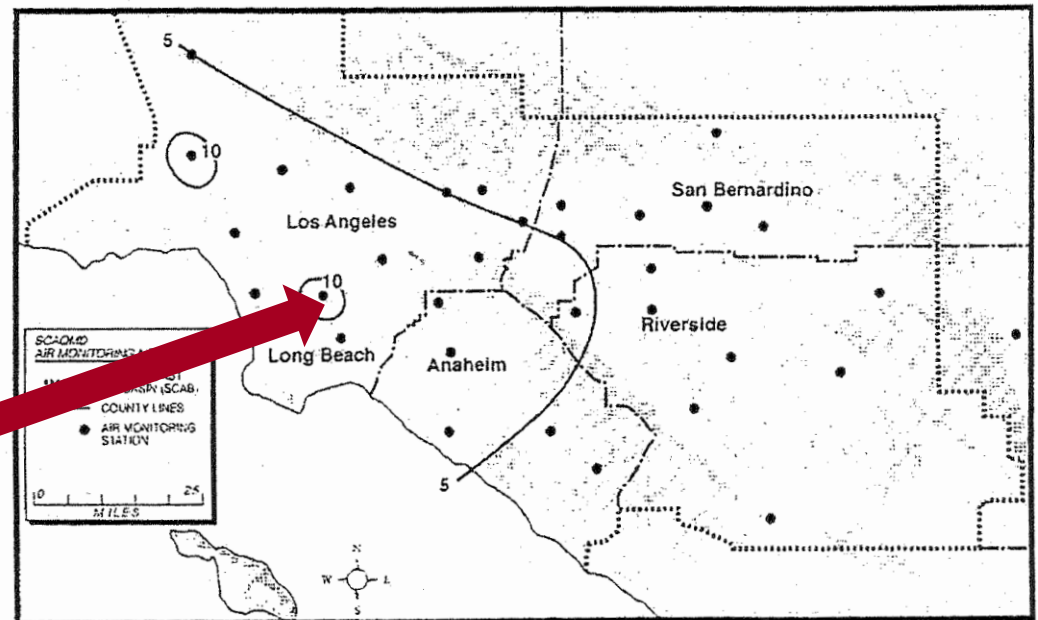
Source: NRC 2003



1956 – 1967
CO > 40 ppm

2000
CO < 10 ppm

CO NAAQS = 9 ppm



Measuring Progress:

Health Benefits 1990 – 2020

(August 2010 Draft)

Substantial Estimated Benefits:

- Mortality
- Bronchitis
- Heart Attack
- Emergency Room Visits
- Lost School and Work Days

Health Effect Reductions (PM2.5 & Ozone Only)	Year 2010	Year 2020
PM2.5 Adult Mortality	160,000	230,000
PM2.5 Infant Mortality	230	280
Ozone Mortality	4,300	7,100
Chronic Bronchitis	54,000	75,000
Acute Bronchitis	130,000	180,000
Acute Myocardial Infarction	130,000	200,000
Asthma Exacerbation	1,700,000	2,400,000
Hospital Admissions	86,000	135,000
Emergency Room Visits	86,000	120,000
Restricted Activity Days	84,000,000	110,000,000
School Loss Days	3,200,000	5,400,000
Lost Work Days	13,000,000	17,000,000

Exhibit 10. Differences in key health effects outcomes associated with fine particles (PM2.5) and ozone between the *With-CAAA90* and *Without-CAAA90* scenarios for the 2010 and 2020 study target years. (In number of cases avoided, rounded to 2 significant digits).

Measuring Progress:

Costs vs. Benefits

1990 – 2020

(August 2010 Draft)

Costs Substantially Exceeded by Benefits

- Largely estimated mortality benefits
- Without mortality benefits,
 - morbidity, visibility and other benefits also exceed costs
- Significant unquantifiable benefits

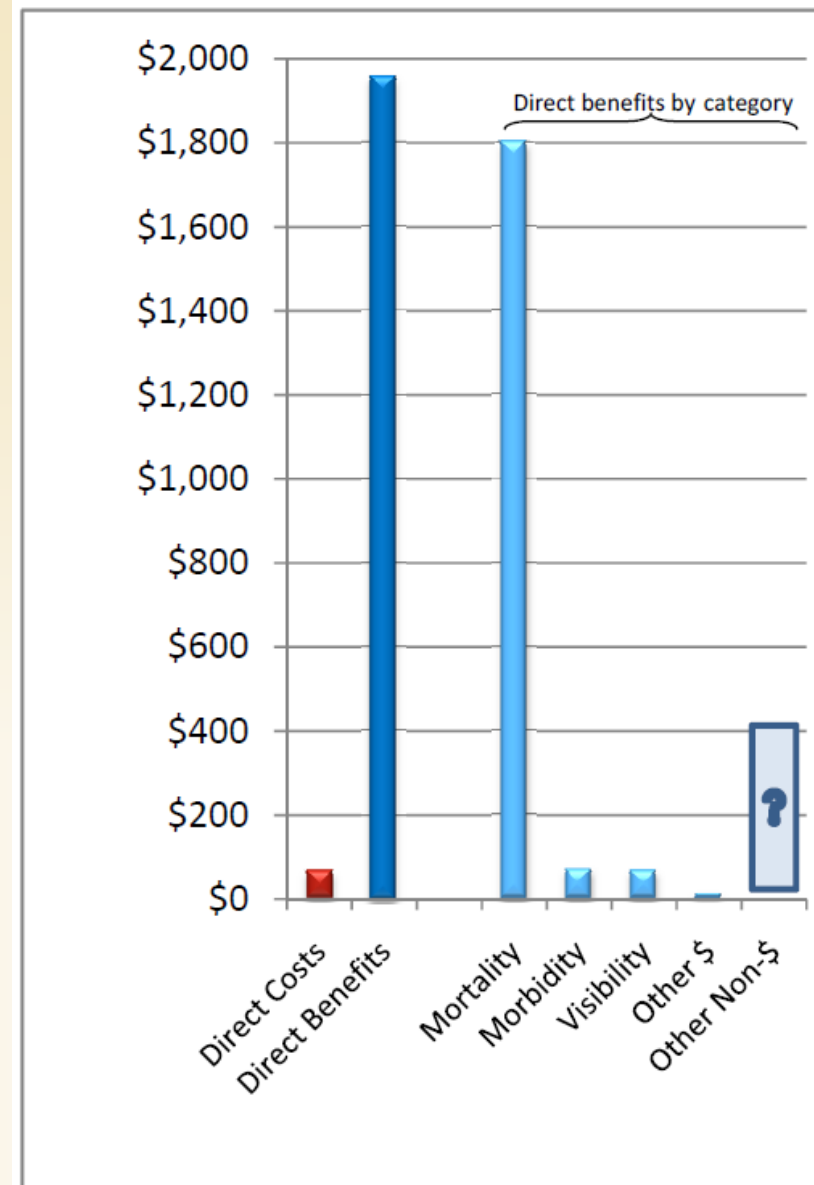


Exhibit 13. Year 2020 Primary Central Estimates of direct costs and direct benefits with breakdown of benefits by effect category. (In billions of year 2006 dollars).

Concluding Thoughts

- Significant effects:
 - *PM and gases*: mortality, other health effects
 - *Transition metals*: inflammation, neurotoxicity
 - *Acid Gases*: acute inflammation and irritation
 - *Dioxins*: likely carcinogens,
 - *Mercury*: developmental effects
- Substantial economic “damages” can be attributed to these emissions
- There is hope for the future!!!



Thank You

dgreenbaum@healtheffects.org

