Black Carbon and Health:
What we know about exposure and effects

Dan Greenbaum, President
Health Effects Institute

Black Carbon Science Symposium
US EPA Regions 2 and 9
San Francisco, California
November 14, 2012

The Air Pollution/Climate Nexus

- The scope and sources of exposure
- The health evidence from:
  - Toxicology
  - Epidemiology
- Estimating Public Health Impact
  - The new Global Burden of Disease
  - The UNEP Assessment of BC and CH4

The Health Effects Institute

Trusted Science ➡ Cleaner Air ➡ Better Health

- An independent non-profit institute providing trusted science on the health effects of air pollution for 30 years
- Joint core support from
  - Government (US EPA) and Industry (Worldwide Motor Vehicle)
  - Partnerships with WHO, ADB, CAI-Asia, EU, US DOE, industries, Hewlett foundation, others
- Independent Board and Expert Science Committees
  - Oversee and intensively peer review all science
  - International experts (e.g. China (Tsinghua, Fudan), India, Thailand)
- Over 270 studies, scientific reviews, reanalysis conducted around the world, including
  - Public Health and Air Pollution in Asia (PAPA) program
- ***Understanding local impacts in a global context***

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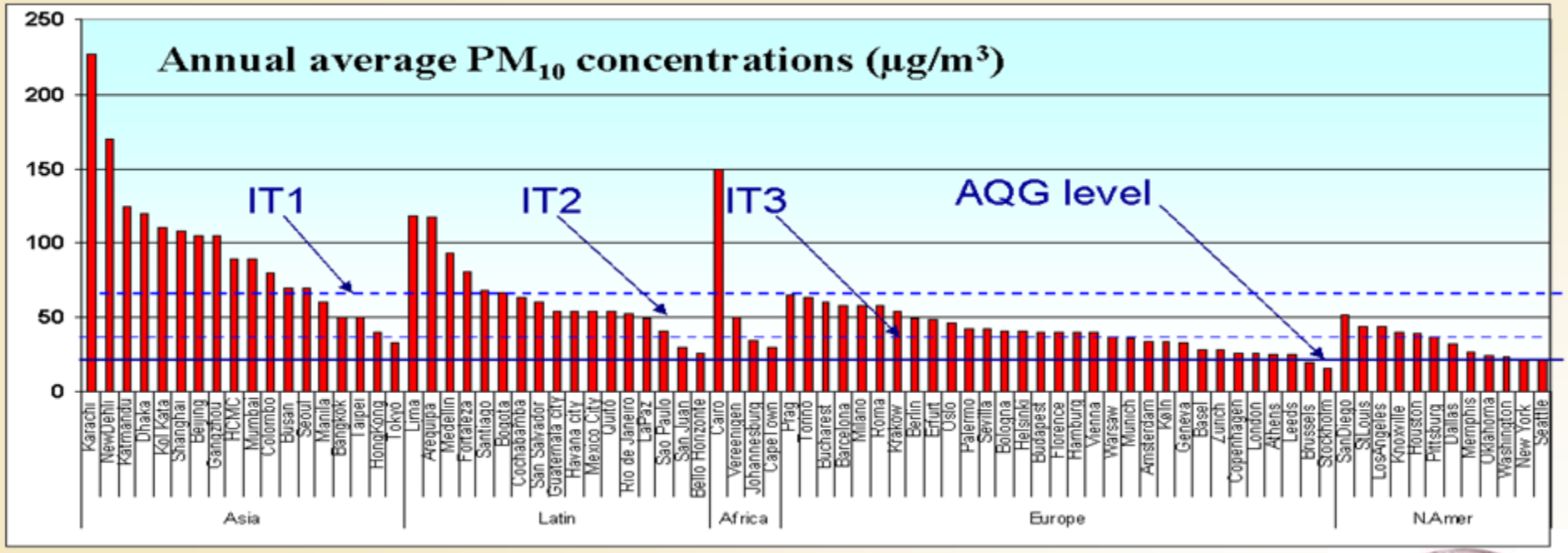
PM Air Pollution: A Problem Worldwide

Ambient Levels of Particulate Matter Exceed Current WHO Air Quality Guidelines

Especially in Some Asian Countries



World Health Organization 2006

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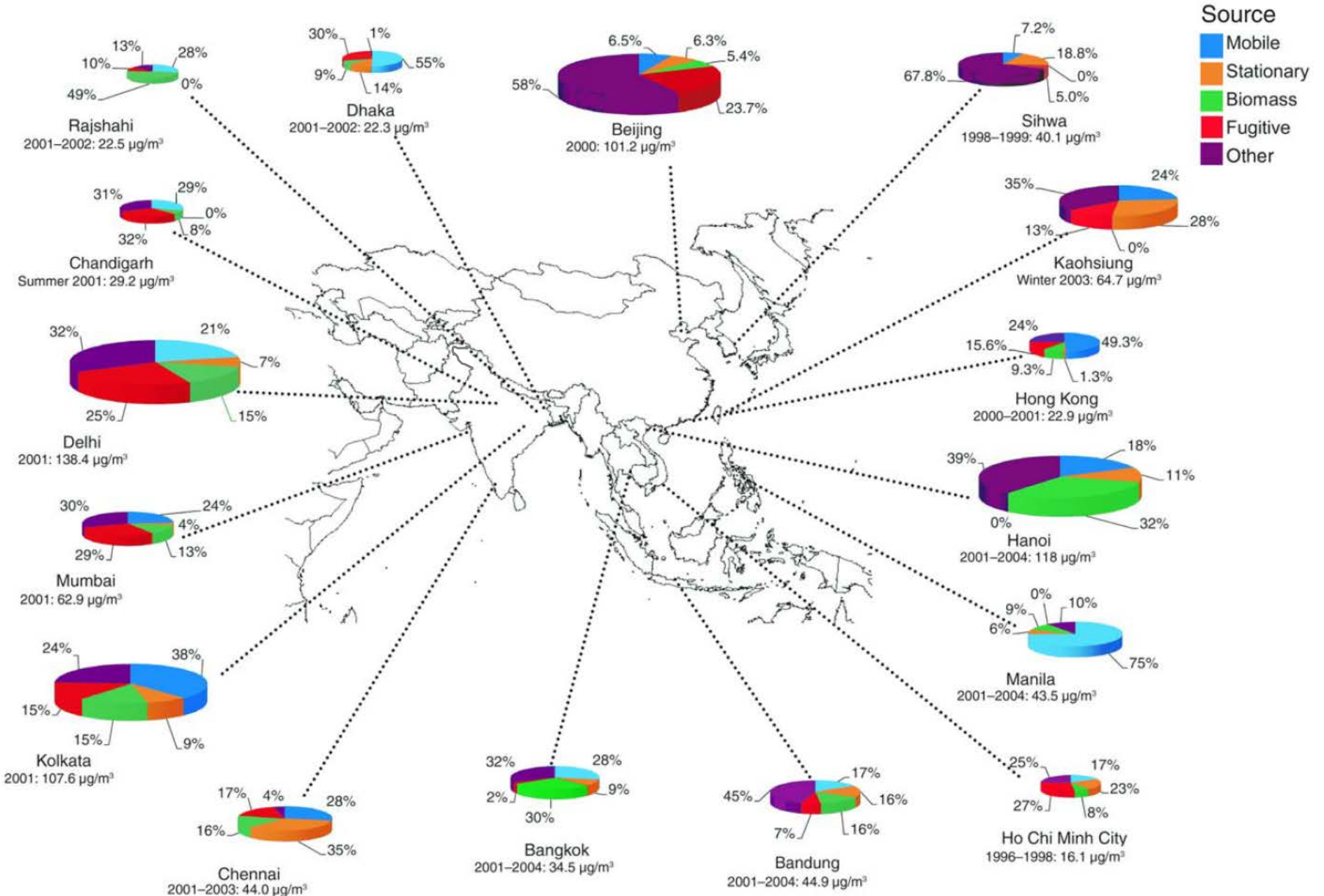
Asia PM 2.5 Source Apportionment: Vehicles ~20% - 35%

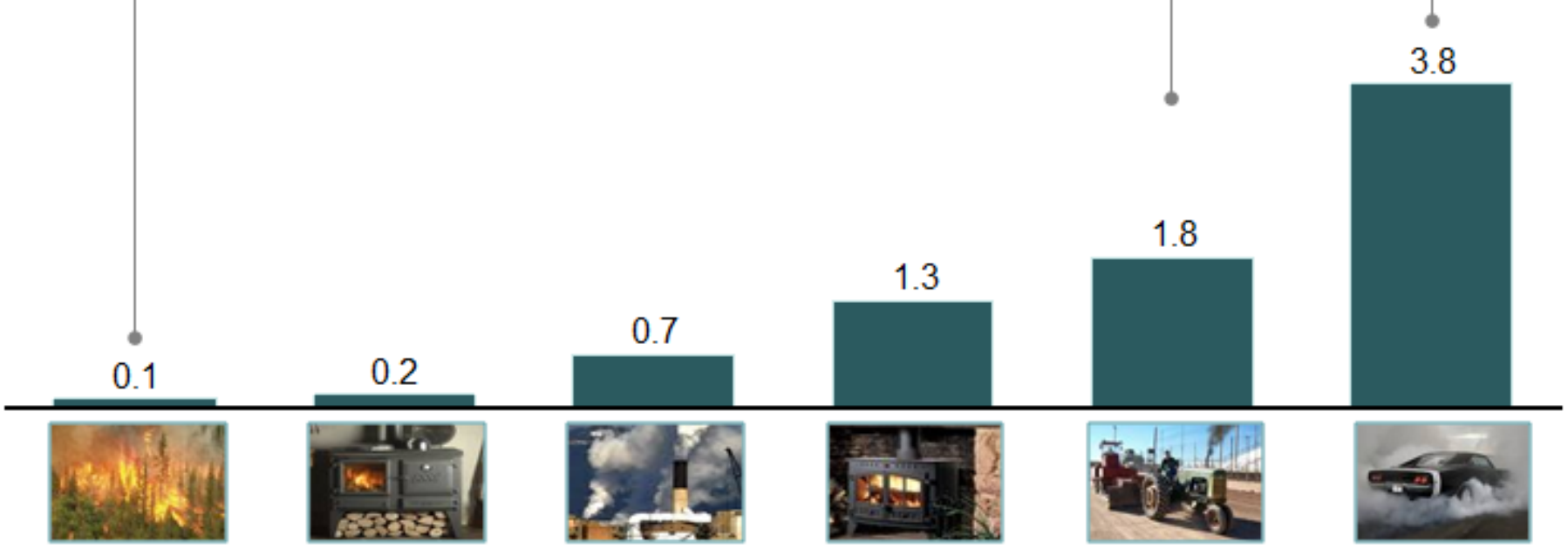


Source: Chowdhury, HEI

Ratio of black carbon to organic carbon

Smoke from open burning of biomass or wildfires appears gray because of the high ratio of OC which scatters sunlight and therefore appears light colored

Soot from diesel combustion appears black because of its high content of black carbon, the light-absorbing component of aerosols



Note: All sources emit significant quantities of other pollutants that may warm or cool the climate, including CO_2 (warming), NO_x (ozone and N_2O warming, nitrate cooling), and SO_2 (sulfate cooling)

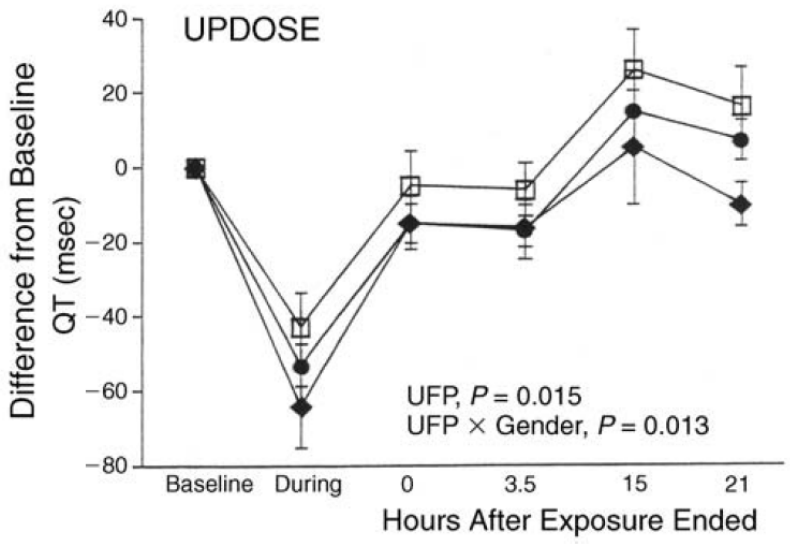
SOURCE: Non-CO2 Climate Forcers Report (2010), Bond (2007), GAINS

Toxicology Evidence

- Relative few studies directly of black carbon (as understood by atmospheric chemists)
- Two examples:
  - Ultrafine carbon particles
  - Effects of diesel emissions with a particle filter
    - i.e. the effects of the absence of carbon

HEI Human Study of Ultrafine Carbon Particles (Frampton et al)

- Carefully control exposures of healthy young adults at rest and during exercise
- Showed effects on heart rate variability, other CVD markers



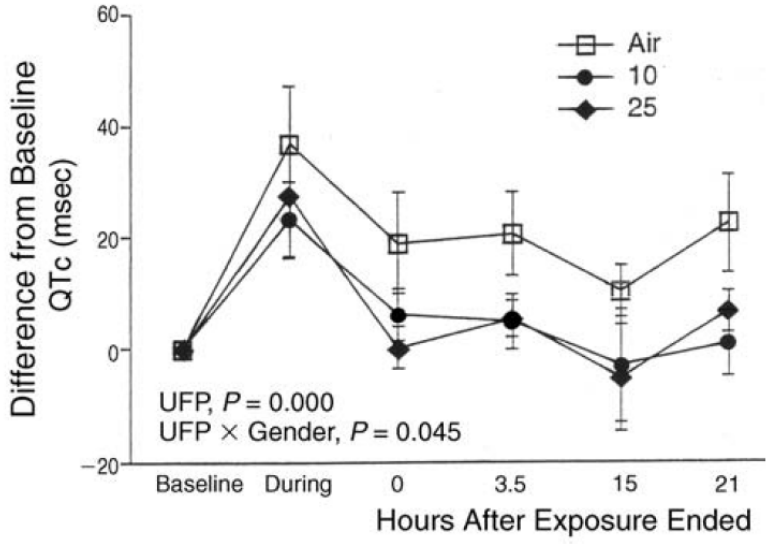


Figure 28. Cardiac repolarization, UPDOSE. Differences from baseline. QT interval and QT corrected with the Bazett formula. Data represent mean ± SE.

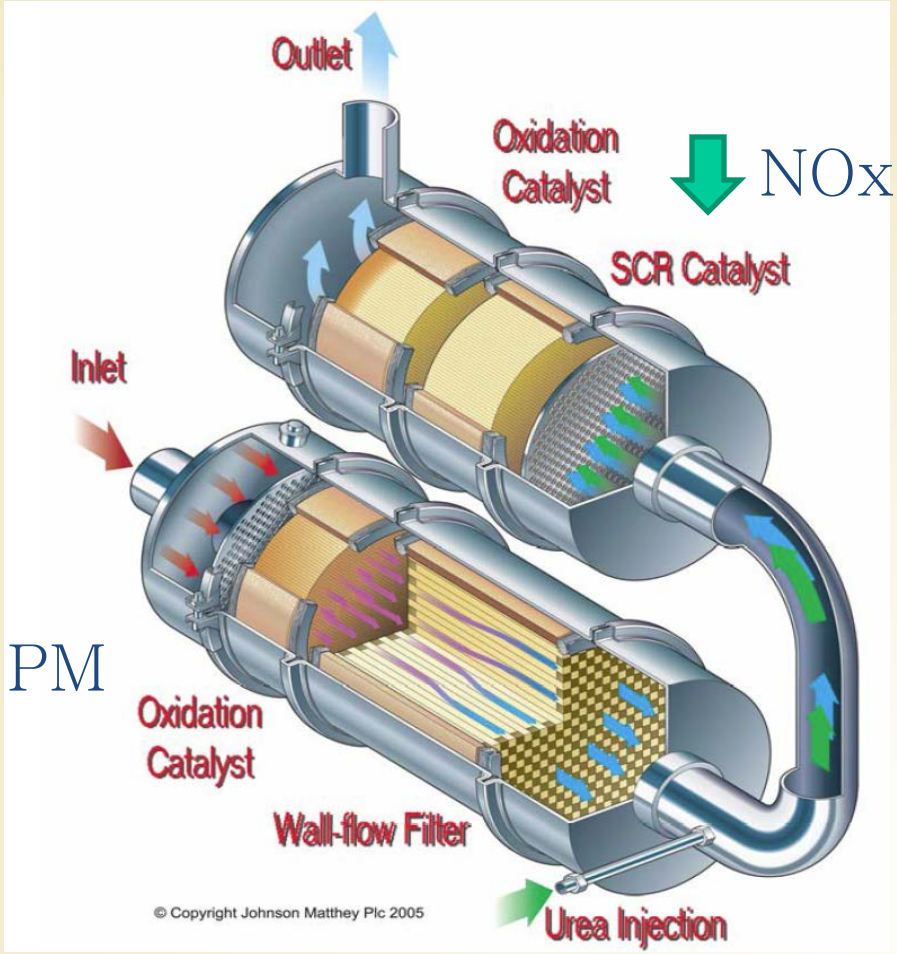
Testing what happens when Technologies and Fuels Change

"New Technology" Diesel

- Heavy Duty Truck and Bus Engines
  - 2007 EPA Requirements
    - 15 ppm sulfur diesel fuel
    - PM control (filter and catalyst)
    - Some NOx control (primarily exhaust gas recirculation)
  - 2010 EPA Requirements
    - Above plus:
      - *Advanced NOx control (selective catalytic reduction)*
    - Similar changes in light duty, off road vehicles

Key Need: Exhaust Treatment Systems

—Particle Removal and NOx Elimination



DOC+ DPF+ SCR

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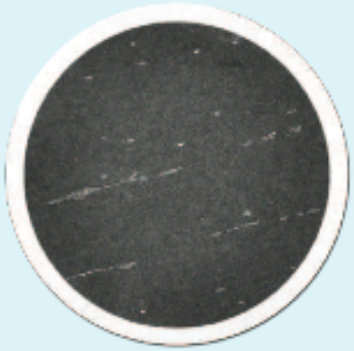
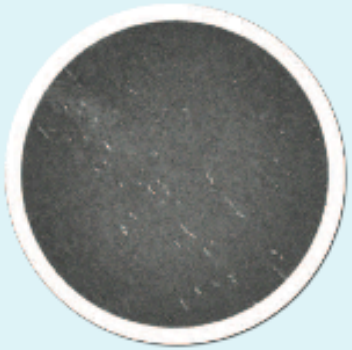
Diesel particulate filters can achieve dramatic emission reductions

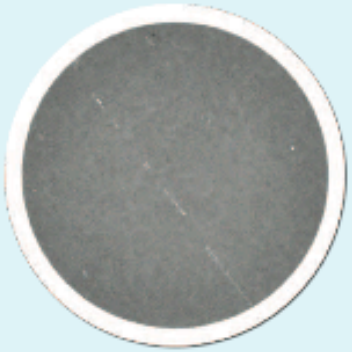


No Retrofit System
Uncontrolled Diesel Exhaust



Retrofitted with
Diesel Oxidation Catalyst (DOC)
(Level 1)

Old technology
Little black carbon removal
Little utrafine PM removal
Does not remove lube oil ash



Retrofitted with
Partial Filter
(Level 2)

Little black carbon removal
Little utrafine PM removal
Does not remove lube oil ash

Retrofitted with
Diesel Particulate Filter (DPF)
(Level 3)

New technology
Used on all new trucks since 2007
>85% black carbon removal
>85% ultrafine removal
>85% lube oil ash removal

Overview: The exhibits above are actual PM collection samples from an engine testing laboratory used to collect and measure diesel particulate matter (PM) emissions. Test conditions are:

- Test Cycle: UDDS (Urban Dynamometer Driving Schedule)
- Test Distance: 5.5 miles over 17 minutes
- Fuel Consumed During Test: 1.1 gallons
- Test Vehicle: Heavy-duty truck with a 370 hp Cummins engine (1999 model year)
- PM material on collection samples is 1/1,800th of actual

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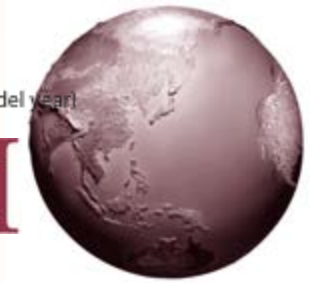


The HEI Advanced Collaborative Emissions Study (ACES)

Evaluating Emissions of Advanced Technology Diesels

- New 2007/2010 engine/control systems and fuels designed to result in substantially reduced emissions.
- Substantial public health benefits are expected from these reductions.
- *But, w*ith any new technology it is prudent to ensure there are no adverse impacts to public health and welfare.

ACES is moving to *answer these important questions*:

Phase 1: 2007 Engine Emissions Characterization Completed

Dramatic reductions

Phase 2: 2010 Engine Emissions Characterization

Testing Underway; report in 2013

Phase 3: 2007/2010 Engine Emissions Health Effects Testing

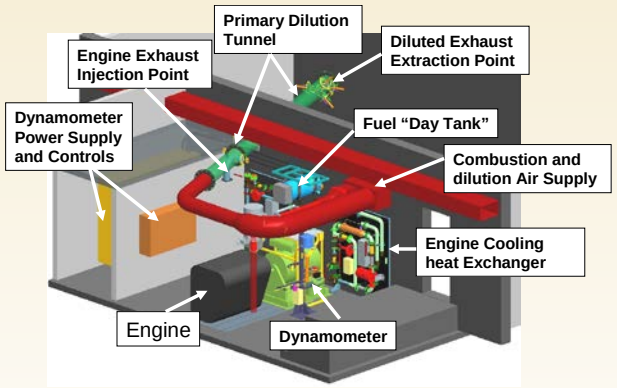
-Short term health biological screening complete

-Few to no health effects observed

-Longer term (cancer) testing well underway

-Early results promising

Final report expected 2014



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Characteristics of New vs. Old Diesel PM

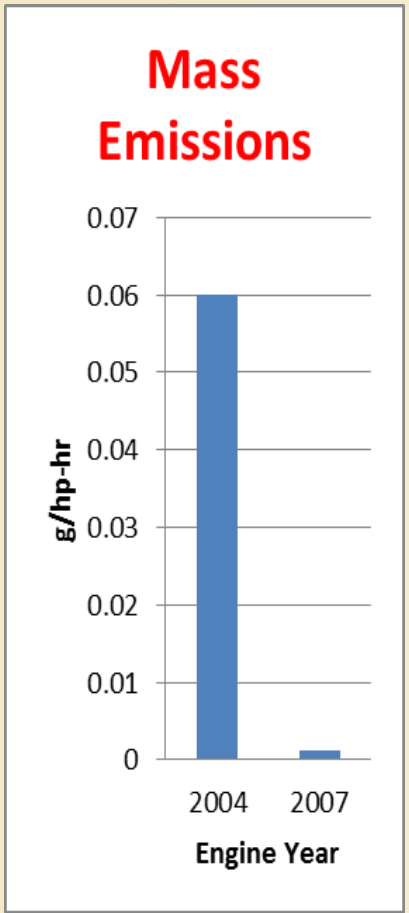
HEI/CRC ACES Results Compared to earlier Testing:

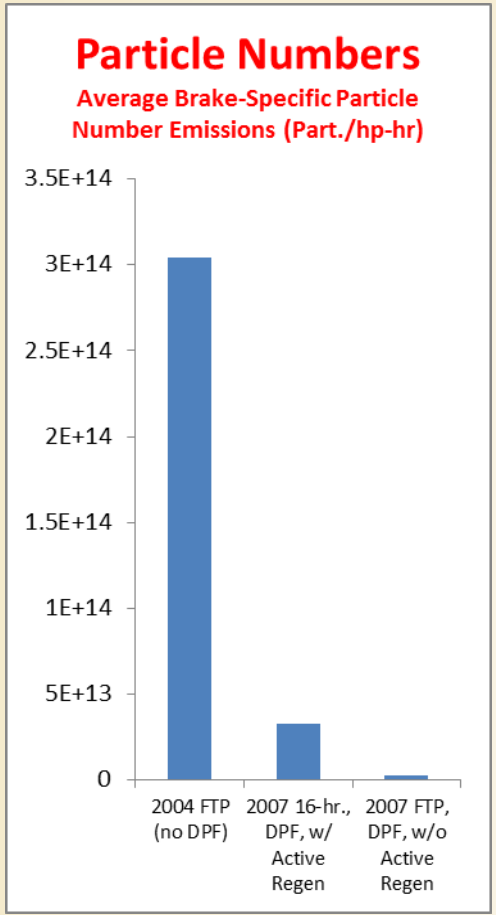
Dramatic Reductions

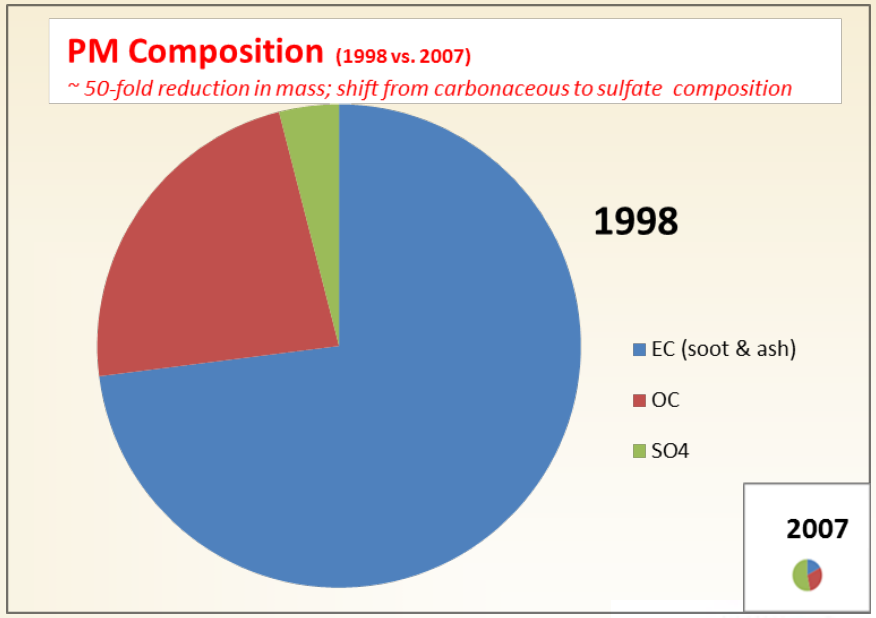
98% reduction in mass

90% - 99% reduction in Ultrafine Particles

Substantial reduction in carbon particles







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Greater than 90% reduction in PAHs (including known carcinogens)

Many PAHs now below detection limits (Khalek et al 2011)

- Polycyclic Aromatic Hydrocarbons (PAHs) have been of major concern in diesel exhaust
- Many known to cause cancer
- Some of the most toxic are so low they can no longer be measured

Table 8. PAH and nitroPAH average emissions for all 12 repeats of the 16-hr cycles for all four 2007 ACES engines and for a 2000-technology engine running over the FTP transient cycle.[16]

PAH and NitroPAH Compounds	2007 Engines[a] (mg/bhp-hr)	2000-Technology Engine[a, b] (mg/bhp-hr)	Percent Reduction
Naphthalene	0.0982000 ± 0.0423000	0.4829	80
Acenaphthylene	0.0005000 ± 0.0005000	0.0524	98
Acenaphthene	0.0004000 ± 0.0001000	0.0215	98
Fluorene	0.0015000 ± 0.0009000	0.0425	96
Phenanthrene	0.0077000 ± 0.0025000	0.0500	85
Anthracene	0.0003000 ± 0.0001000	0.0121	97
Fluoranthene	0.0006000 ± 0.0006000	0.0041	85
Pyrene	0.0005000 ± 0.000400	0.0101	95
Benzo(*a*)anthracene	<0.0000001	0.0004	>99
Chrysene	<0.0000001	0.0004	>99
Benzo(*b*)fluoranthene	<0.0000001	<0.0003	>99
Benzo(*k*)fluoranthene	<0.0000001	<0.0003	>99
Benzo(*e*)pyrene	<0.0000001	<0.0003	>99
Benzo(*a*)pyrene	<0.0000001	<0.0003	>99
Perylene	<0.0000001	<0.0003	>99
Indeno(123-*cd*)pyrene	<0.0000001	<0.0003	>99
Dibenz(*ah*)anthracene	<0.0000001	<0.0003	>99
Benzo(*ghi*)perylene	<0.0000001	<0.0003	>99
2-Nitrofluorene	0.00000360 ± 0.00000410	0.0000650	94
9-Nitroanthracene	0.0000148 ± 0.0000213	0.0007817	98
2-Nitroanthracene	0.00000040 ± 0.00000090	0.0000067	94
9-Nitrophenanthrene	0.00002110 ± 0.00002090	0.0001945	89
4-Nitropyrene	<0.00000001	0.0000216	>99
1-Nitropyrene[c]	0.00001970 ± 0.00002430	0.0006318	97
7-Nitrobenz(*a*)anthracene	0.00000020 ± 0.00000020	0.0000152	99
6-Nitrochrysene	<0.00000001	0.0000023	>99
6-Nitrobenzo(*a*)pyrene	<0.00000001	0.0000038	>99

Notes: [a]The significant figures signify the detection limit in mg/bhp-hr; [b]SD data were not provided by ref 15. [c]Previous work showed artifact formation during filter collection of the compounds highlighted in bold.

ACES PHASE 3B – HEALTH RESULTS

Key Findings at LRRI

The majority of the analyses showed no difference between diesel exhaust exposure and clean air control.

Histopathology analysis revealed mild/minimal exposure-related hyperplasia in the rats after 3 months of exposure, but not in mice. The hyperplasia increased at 12 months, but was still considered mild/minimal severity.

Statistically significant findings were noted for several indicators of oxidative stress and inflammation in rats and mice at 3 months (fewer findings in mice).

Pulmonary function assessments in rats showed slight differences in exposed rats compared with control after 3 and 12 months of exposure.

Note: When designing the study, it was expected that at the high concentration (at 4.2 ppm NO_2) some NO_2-related effects may be observed. Results so far are not inconsistent with that.

Other analyses of reduced carbon diesel: Vascular inflammation with and without a filter (Lucking et al 2011)

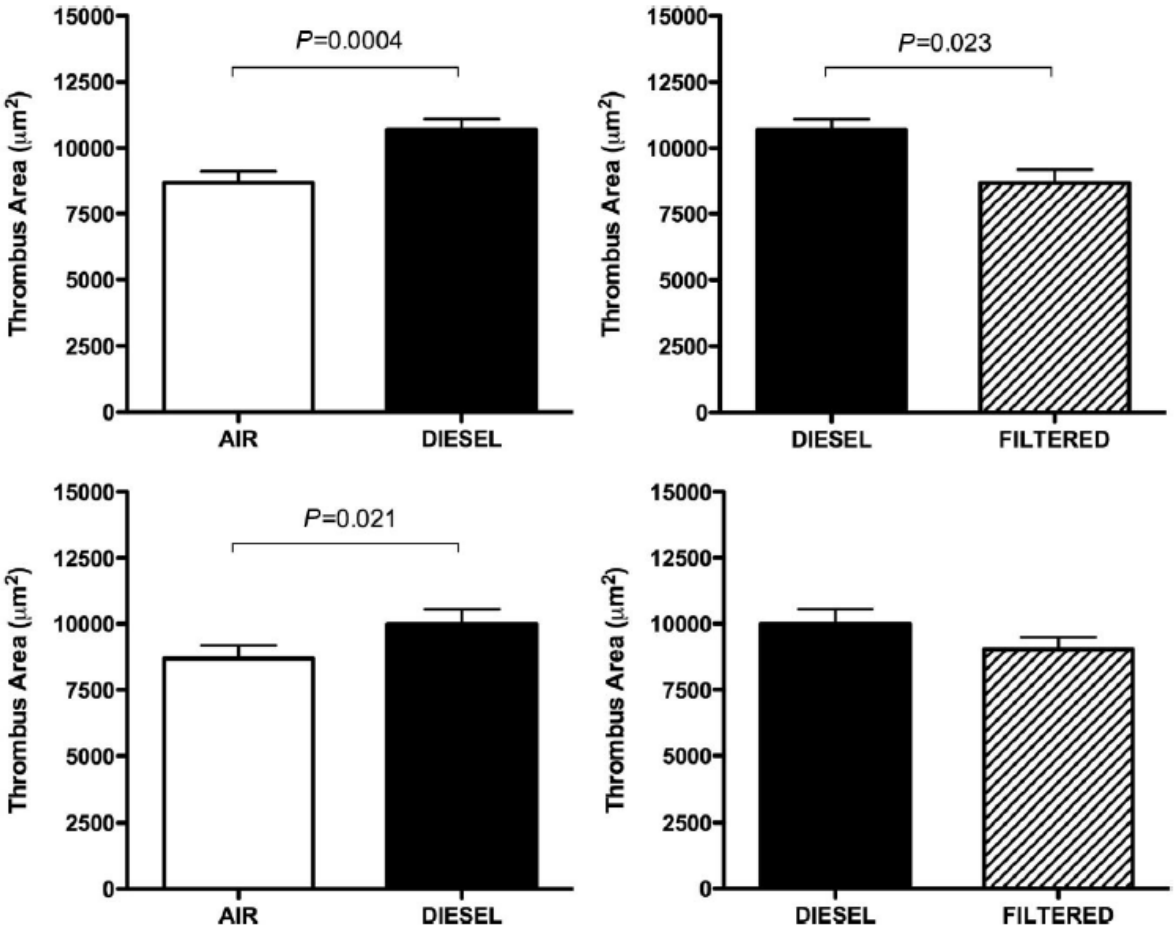


Figure 4. Ex vivo thrombus formation assessed with the Badimon chamber at 2 hours after exposure. Data from the low-shear chamber are shown in the top panels. Data from the high-shear chamber are shown in the bottom panels. Filtered air exposure is shown in white, diesel engine exhaust exposure in black, and filtered diesel exposure in hatched bars.

Epidemiology

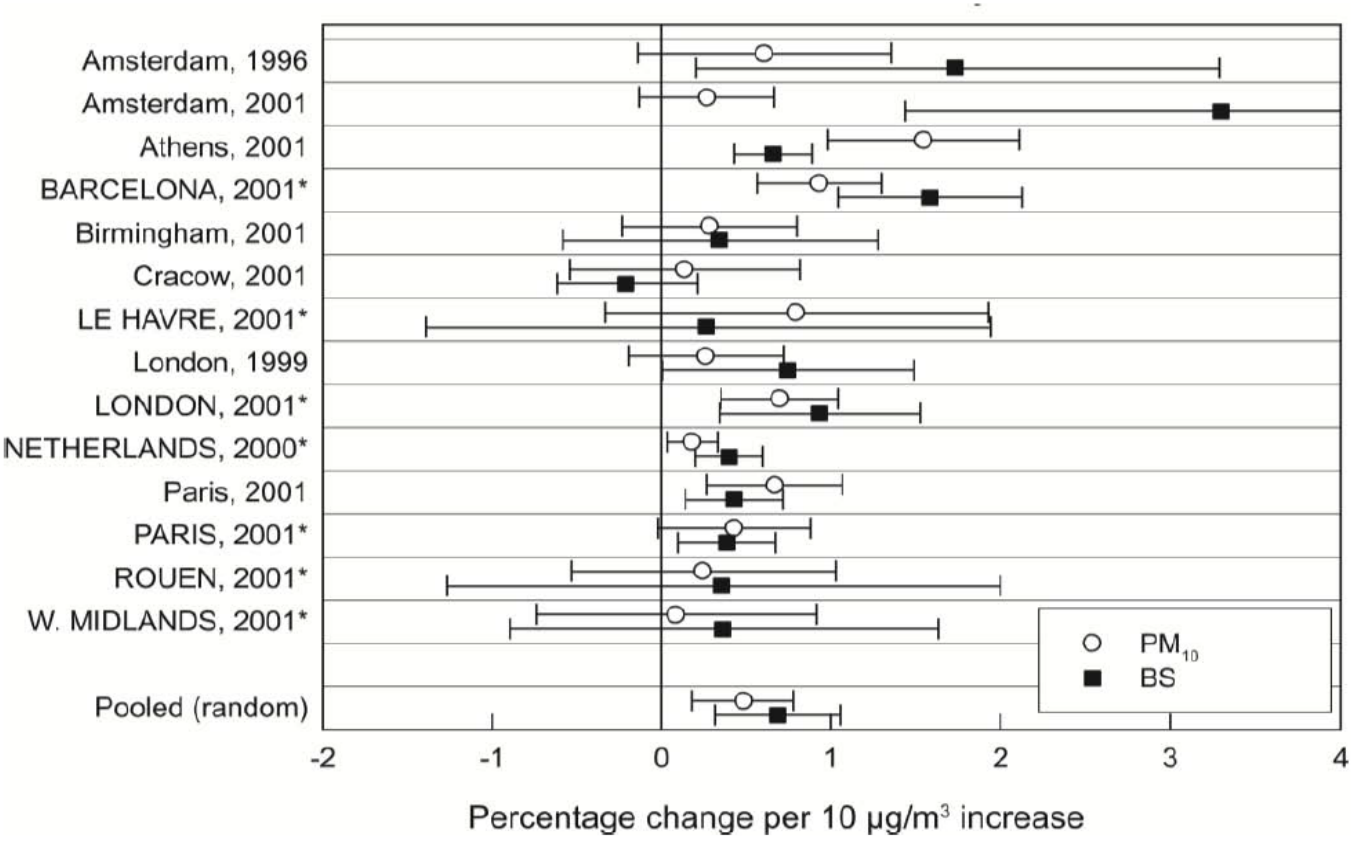
- A large number of studies of short term PM exposure and effects (e.g. time series studies)
- A smaller but growing number of long term (cohort) studies

A Challenge: Measuring Black Carbon for Epidemiology Studies

- Many studies historically in Europe/UK using Black Smoke, an optical measure.
- Works well as an indicator of health risk
- But relation to mass varies by location, season, and over time
- Elemental carbon of growing interest and now measured widely in US ambient monitoring networks
- Not a fully specific method – based on a thermo-optical procedure that is not as yet standardized
- At similar size distributions, however, EC seems to link better to the measures of black carbon mass used by the climate community

European Daily Time-series studies PM2.5 and Black Smoke

Fig. 1. Single-city, single-pollutant estimates for PM_{10} and BS and all-cause mortality



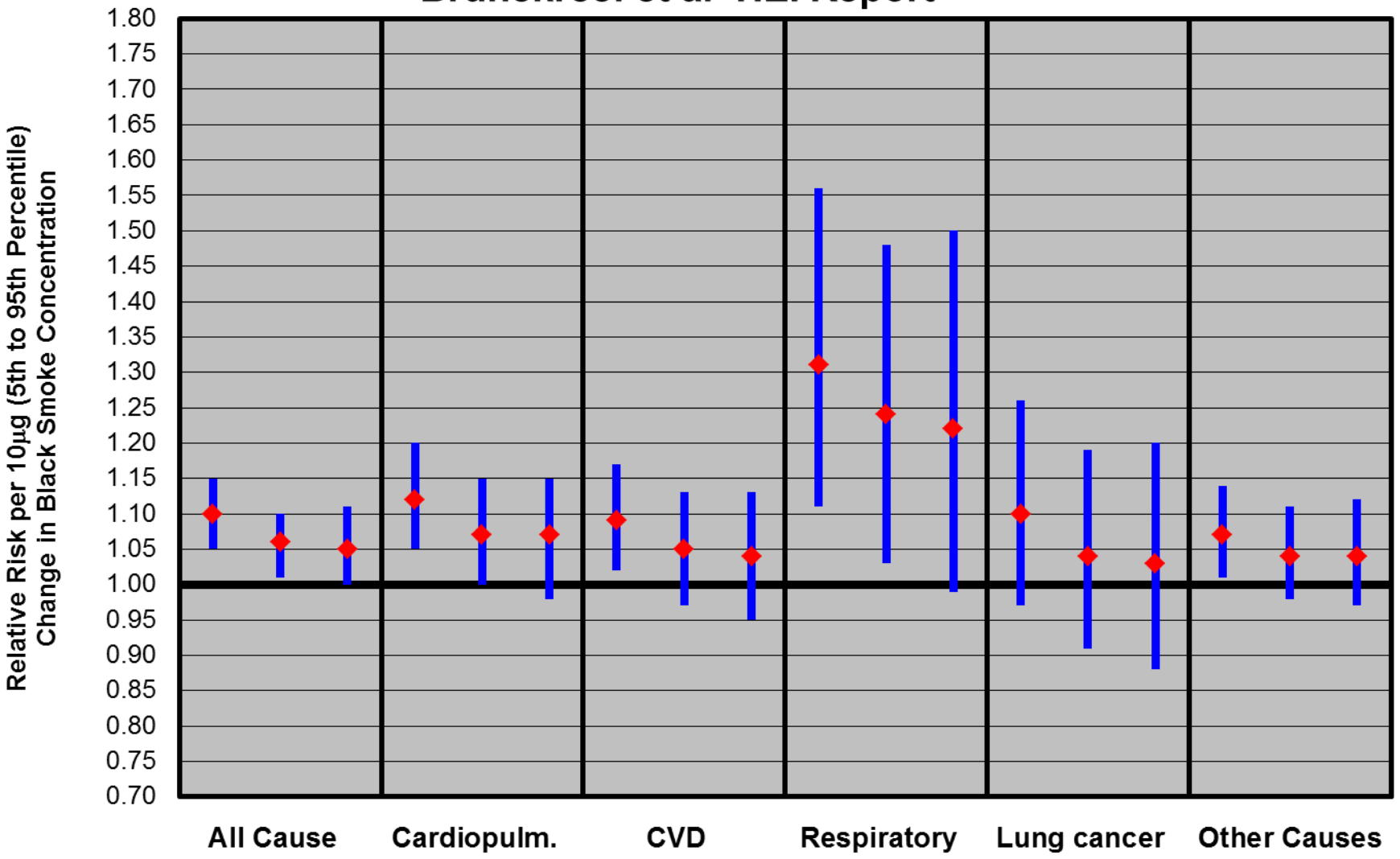
*Cities or areas included in the pooled estimate (year indicates year of publication).

Source: WHO 2012

HEI Study of effects in a Dutch Cohort (Brunekreef et al)

- Large population in a diet and nutrition study
- Measures of Black Smoke, other pollutants, and indicators of traffic

Relative Risk of Mortality for Full Cohort by Cause
Black Smoke Exposure
Brunekreef et al HEI Report*



*confounding adjustments: left bar: age, gender; center: + active smoking; right: +active smoking, SES

THE LANCET 374: 2091–2103, 2009 (Dec).

Smith KR, Jerrett M, Anderson H, Burnett R, Stone V, Derwent R, Atkinson R, Cohen A, Shonkoff S, Krewski D, Pope III CA, Thun M, Thurston G

Health and Climate Change 5

Part of a series of 6 papers on health and climate co-benefits

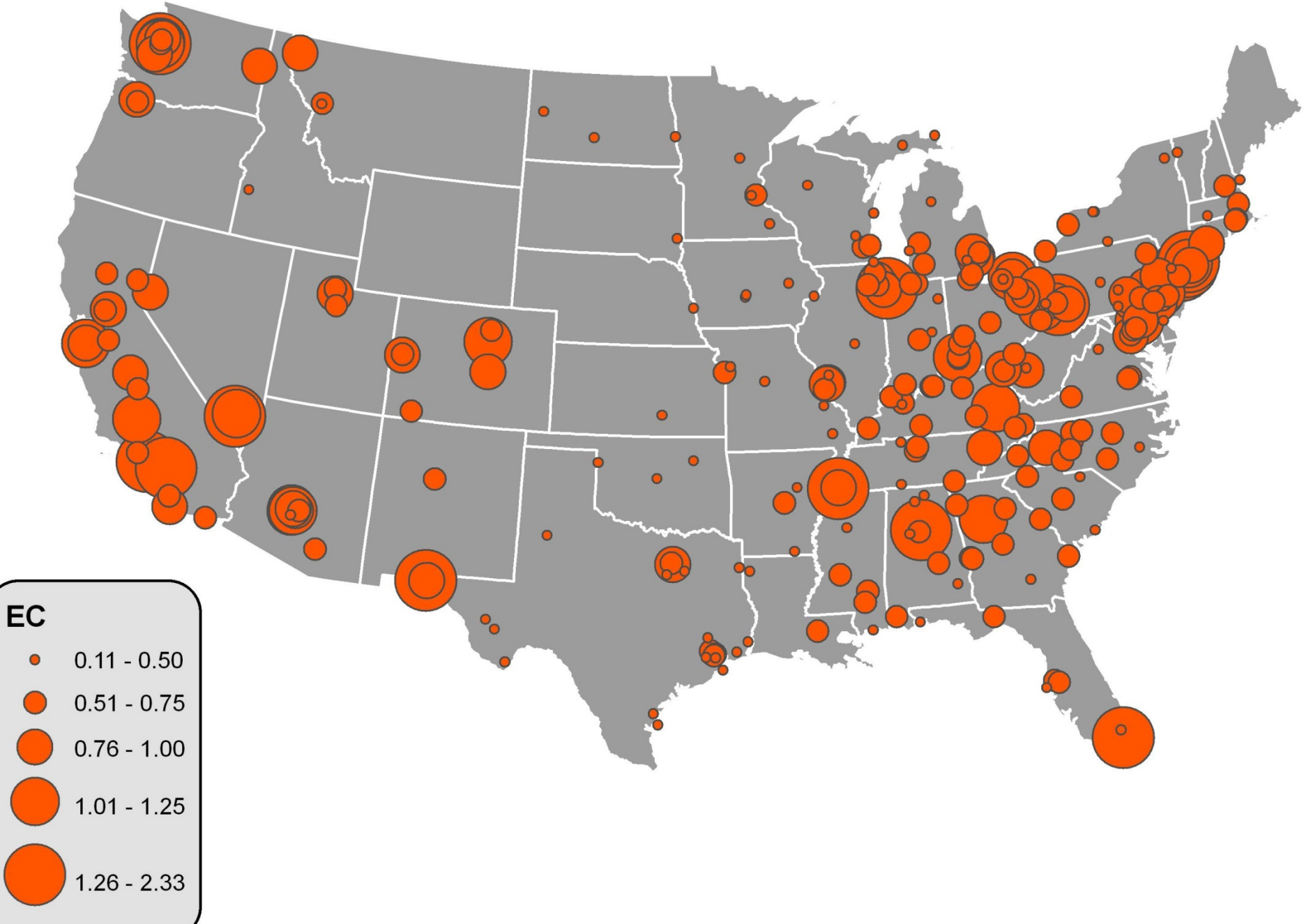
Public health benefits of strategies to reduce greenhouse-gas emissions: health implications of short-lived greenhouse pollutants

Kirk R Smith, Michael Jerrett, H Ross Anderson, Richard T Burnett, Vicki Stone, Richard Derwent, Richard W Atkinson, Aaron Cohen, Seth B Shonkoff, Daniel Krewski, C Arden Pope III, Michael J Thun, George Thurston

In this report we review the health effects of three short-lived greenhouse pollutants—black carbon, ozone, and sulphates. We undertook new meta-analyses of existing time-series studies and an analysis of a cohort of 352 000 people in 66 US cities during 18 years of follow-up. This cohort study provides estimates of mortality effects from long-term exposure to elemental carbon, an indicator of black carbon mass, and evidence that ozone exerts an independent risk of mortality. Associations among these pollutants make drawing conclusions about their individual health effects difficult at present, but sulphate seems to have the most robust effects in multiple-pollutant models. Generally, the toxicology of the pure compounds and their epidemiology diverge because atmospheric black carbon, ozone, and sulphate are associated and could interact with related toxic species. Although sulphate is a cooling agent, black carbon and ozone could together exert nearly half as much global warming as carbon dioxide. The complexity of these health and climate effects needs to be recognised in mitigation policies.

New Results Comparing Effects of Ozone, Sulfate, and EC with $PM_{2.5}$

- 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 in the 66 metropolitan statistical areas that had all pollutants recorded for the national cohort.
- Survival model stratified by age, 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.


EC
0.11 - 0.50
0.51 - 0.75
0.76 - 1.00
1.01 - 1.25
1.26 - 2.33

	$PM_{2.5}$ (1·0 μg/m³)	Ozone (1·0 μg/m³)	Sulphate (1·0 μg/m³)	Elemental carbon (1·0 μg/m³)
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)

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.

Smith, et al., Lancet, 2009

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Relatively Consistent Long-term results

EC risks consistently higher than PM

(WHO 2012)

Table 10. RR for mortality related to long-term exposure to $PM_{2.5}$ and EC per 1 µg/m^3

Reference	Cohort	R PM-BCP	Cause	RR $PM_{2.5}$	RR EC
Smith et al., 2009	500 000 adults, aged 20–87 years, United States	NA	All causes	1.006 (1.002–1.010)	1.06 (1.01–1.11)
			Cardiopulmonary	1.012 (1.008–1.018)	1.11 (1.03–1.19)
Lipfert et al., 2006	70 000 male veterans, United States	0.54	All causes	1.006 (0.993–1.020)	1.18 (1.05–1.33)
Beelen et al., 2008[a]	120 852 adults; aged 55–69 years, Netherlands	>0.8[b]	Natural causes	1.006 (0.997–1.015)	1.05 (1.00–1.10)
			Respiratory	1.007 (0.972–1.043)	1.20 (0.99–1.45)
			Cardiovascular	1.004 (0.990–1.019)	1.04 (0.95–1.12)
			Lung cancer	1.006 (0.980–1.033)	1.03 (0.89–1.18)
			Other	1.008 (0.996–1.021)	1.04 (0.97–1.11)
Filleul et al., 2005[a,c]	14 284 adults, aged 25–59 years, France	0.87[d]	Natural causes	1.010 (1.004–1.016)	1.06 (1.03–1.09)
			Cardiopulmonary	1.012 (1.002–1.023)	1.05 (0.98–1.11)
			Lung cancer	1.000 (0.983–1.019)	1.03 (0.93–1.14)
Pooled effect (fixed)[e]			All causes	1.007 (1.004–1.009)	1.06 (1.04–1.09)

Air Pollution and Public Health

- Several global efforts to estimate public health impact of air pollution
  - New Global Burden of Disease (2012)
  - UNEP Assessment (2011)
- Estimate impact for ***all*** pollution from all sources
  - i.e. PM2.5, Ozone
- To date, have not estimated specific effects of black carbon
  - Which is one, though not the largest, component
  - Key challenge: characterizing what among the indoor and outdoor carbon emissions ***are*** black carbon

The Global Burden of Disease (GBD)

- GBD is a systematic worldwide effort to estimate risks from ***all*** major risk factors
  - E.g. smoking, diet, road traffic, outdoor and indoor air pollution
  - Disability Adjusted Life Years (***DALYs***) and ***mortality***
- Last completed for Year 2000 (Published 2002)
  - Most recent update by WHO: 1.34 million deaths worldwide
- Newest version to be published in *The Lancet* in December
  - HEI led outdoor air pollution analysis, contributed to household pollution analysis

Global Burden of Disease 2010

- Extensive new analysis across multiple "risk factors"
  - 800 global scientists from Universities, WHO, Governments
- Major Changes for outdoor air pollution
  - Substantially enhanced exposure estimates
  - New refined estimate of the "concentration-response" relationship

New, More Extensive Global Estimates Based on Measurements, Remote Sensing

(Brauer, et al, 2011)

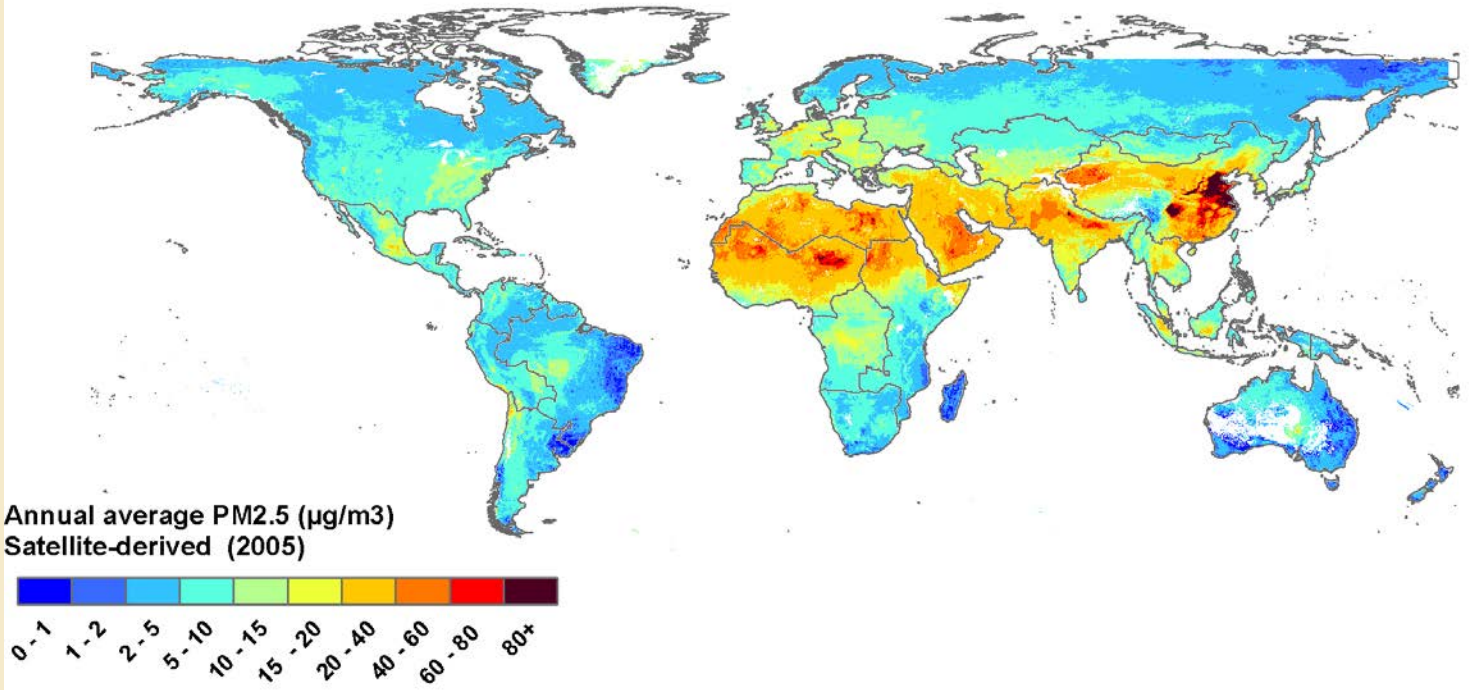
ENVIRONMENTAL Science & Technology

Article

pubs.acs.org/est

Exposure Assessment for Estimation of the Global Burden of Disease Attributable to Outdoor Air Pollution

Michael Brauer,*,† Markus Amann,‡ Rick T. Burnett,§ Aaron Cohen,∥ Frank Dentener,⊥ Majid Ezzati,# Sarah B. Henderson,▽ Michal Krzyzanowski,○ Randall V. Martin,◆,¶ Rita Van Dingenen,⊥ Aaron van Donkelaar,◆ and George D. Thurston+



A New "Concentration – Response" Graph for PM 2.5, and active and passive smoking

Big Question: Smoking exposures much higher, but "relative risks" are lower

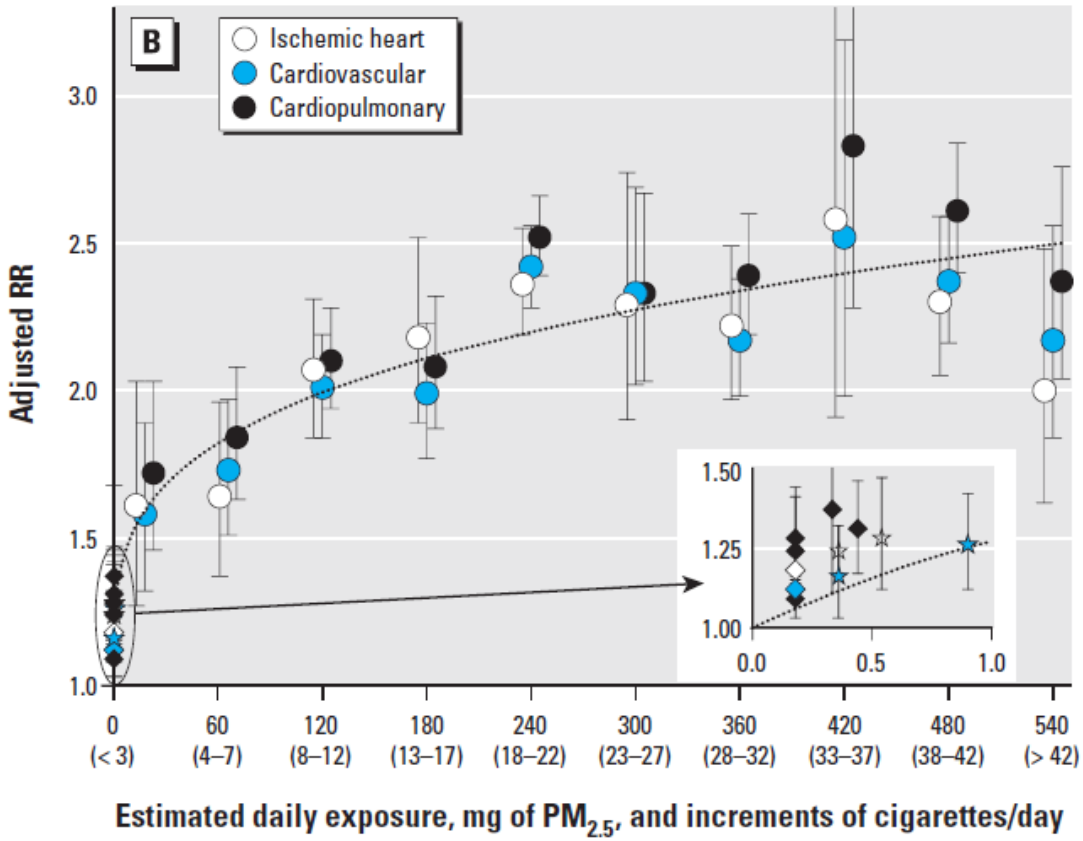
Key new finding: *Risk goes up with $PM_{2.5}$ inhaled dose regardless of source*

- Change in risk is steepest at lowest levels – from PM and Second-hand smoke

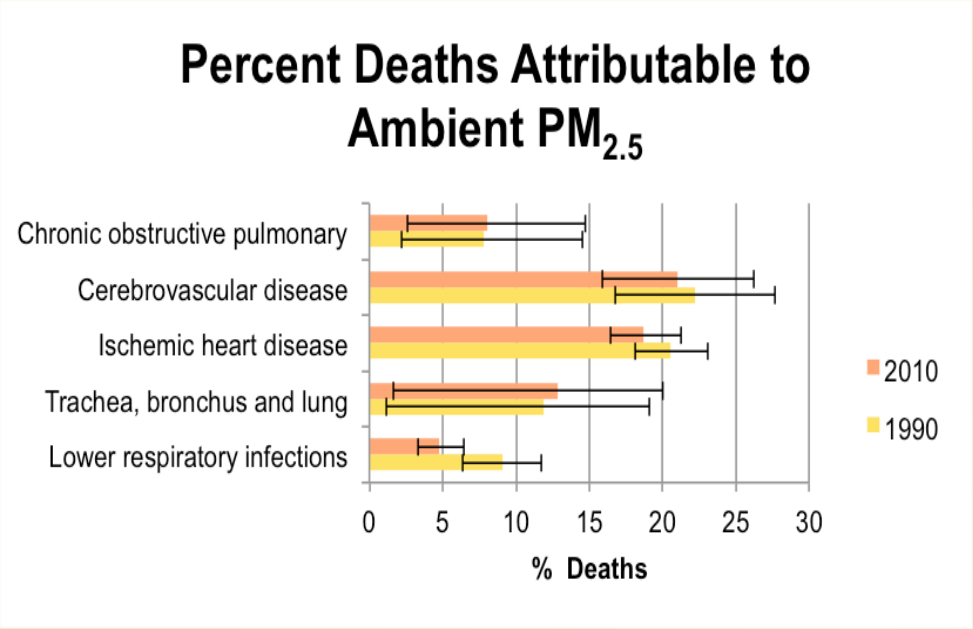
Intervention to reduce exposure at at lower levels will gain greatest health improvement

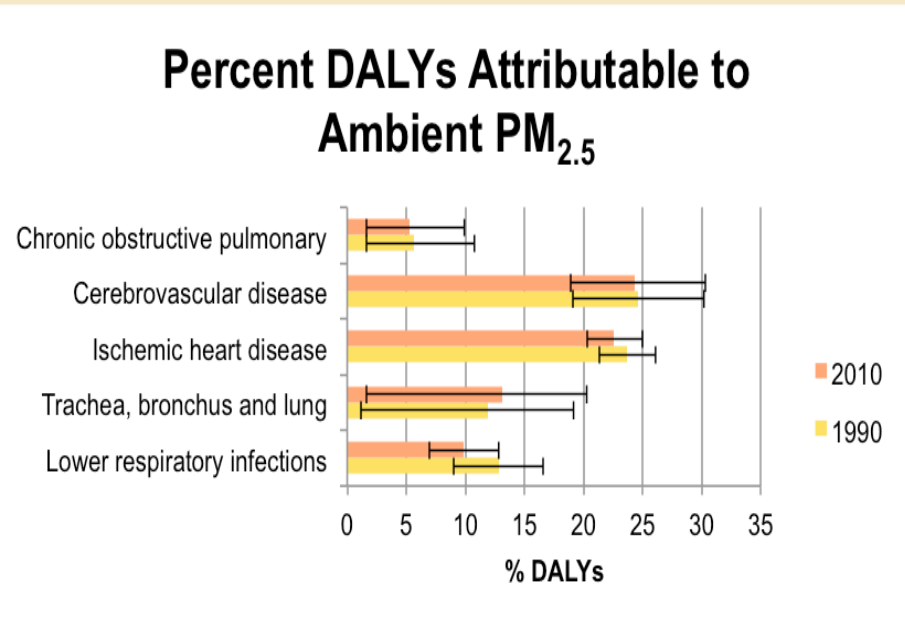
Circle: Active Smoking
Diamond: Passive Smoking
Square: PM2.5

Pope, et al 2011



Preliminary estimates of deaths, DALYs attributable to $PM_{2.5}$





- There is expected be a substantial increase in estimated premature deaths related to air pollution (compared to 2000)
  - **Largely attributable to cardiovascular disease – IHD and stroke**
- Air Pollution is expected to rise among other risk factors
- Also, a significant increase in Disability Adjusted Life Years (DALYs)
  - Key new evidence on Acute Lower Respiratory Disease in Children
    - The top cause of child mortality worldwide

HEI

UNEP Assessment 2011

- Substantial warming benefits from series of CH4 and BC Measures
- ~2.4 million premature deaths avoided in 2030 from PM2.5 reductions resulting from the measures (i.e. BC *plus* other PM reductions)
- Also, significant crop benefits

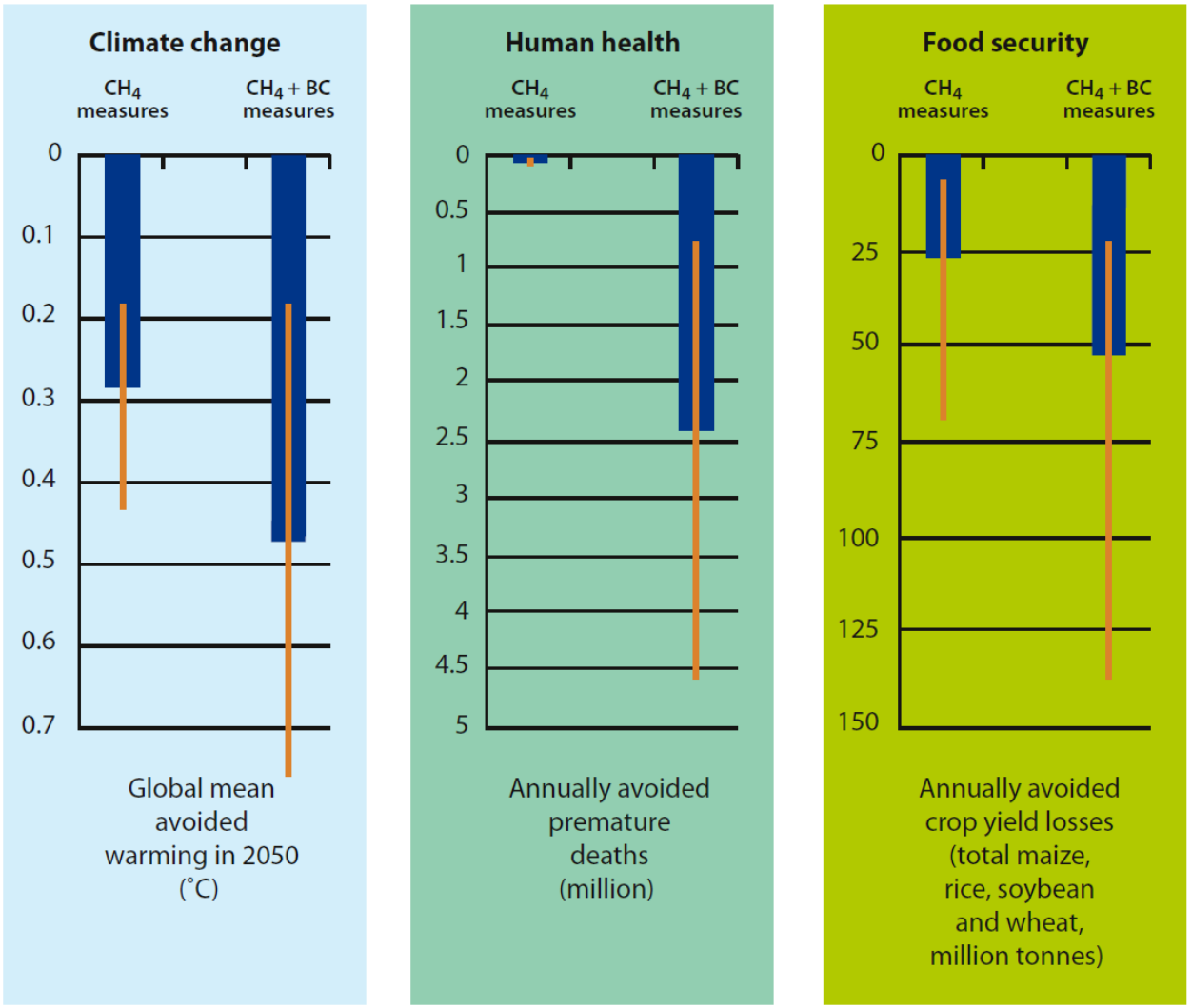


Figure 1. Global benefits from full implementation of the identified measures in 2030 compared to the reference scenario. The climate change benefit is estimated for a given year (2050) and human health and crop benefits are for 2030 and beyond.

UNEP Assessment 2011

Largest regional benefits in Asia

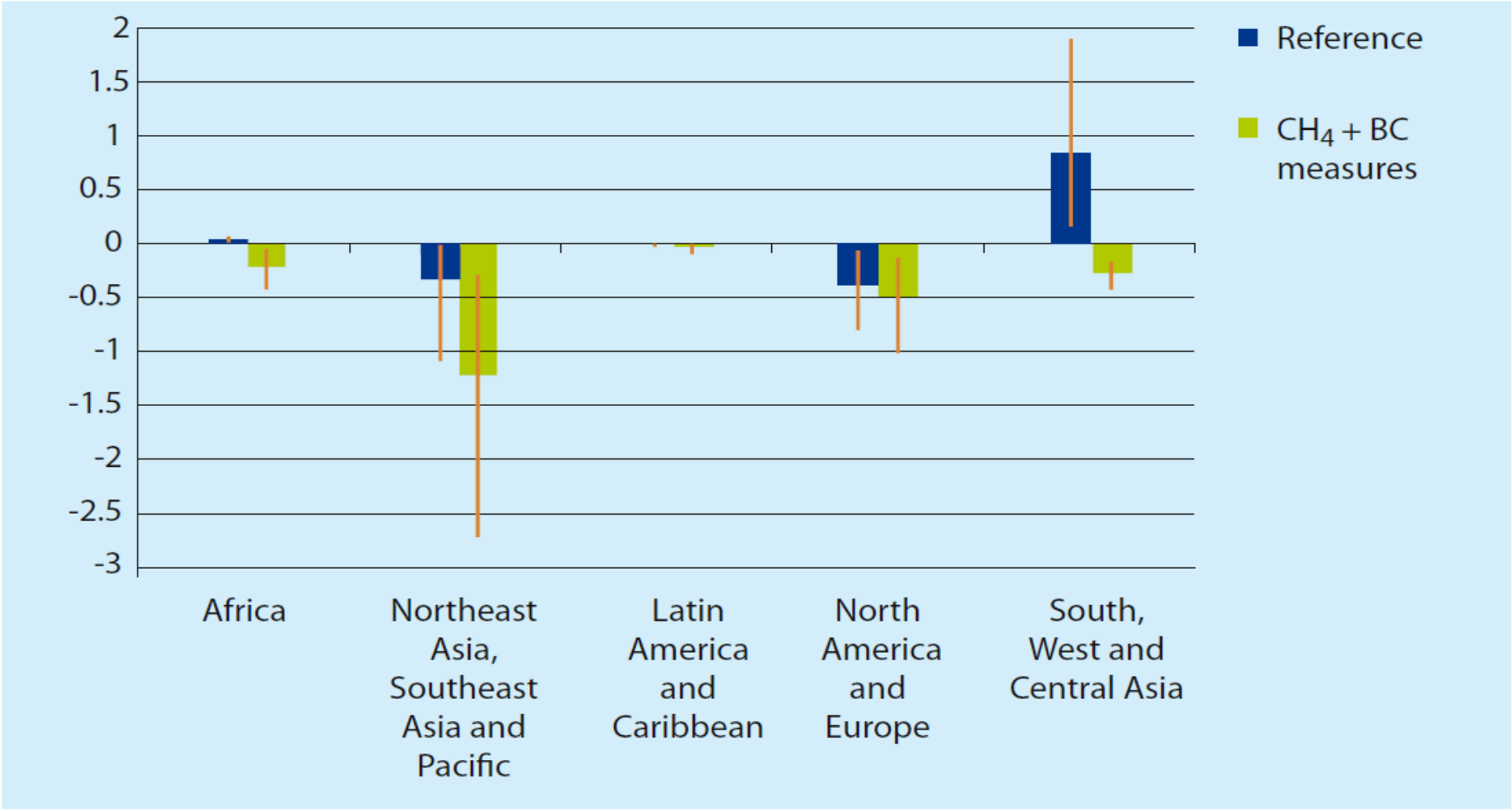


Figure 7. Comparison of premature mortality (millions of premature deaths annually) by region, showing the change in 2030 in comparison with 2005 for the reference scenario emission trends and the reference plus CH_4 + BC measures. The lines on each bar show the range of estimates.

Concluding Thoughts

- Black carbon is an important, though not the only, component of PM
  - Large portions of the globe face significantly elevated PM levels
  - Accurate understanding of source emissions and their contributions to black carbon is critical to health assessments
- There is significant toxicological and epidemiological evidence of black carbon effects
- New estimates of global public health impact are applying enhanced exposure assessment and health knowledge
  - Resulting in substantially increased estimates of PM health impact
  - And of black carbon as one component (though much more needs to be understood on the BC contribution)

Thank You

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