Vehicle Emissions and Health:

A Global Perspective on Effects, Placed in an Indian Context

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Health Effects Institute

Delhi, India
1 September2011

Vehicle Emissions and Health

- What do vehicles contribute to exposure?
- What do we know about health effects?
  - Particulate Matter
  - Ozone and NO2
  - Traffic
- Concluding Thoughts

The Health Effects Institute

- Non-profit institute providing *trusted science on the health effects of air pollution* for over 30 years
- Joint core support from
  - Government (US EPA) and Industry (Worldwide Motor Vehicle)
  - Partnerships with WHO, ADB, CAI-Asia, EU, US DOE, other industries, Hewlett foundation
- Independent Board and Expert Science Committees
  - Including international experts (e.g. India (AIIMS), China, Thailand)
- Over 270 studies, scientific reviews, reanalysis conducted around the world, including:
  - *Public Health and Air Pollution in Asia (PAPA) program*
- HEI delivers science; no policy positions taken

Understanding local impacts in a global context

HEI

Major Report from:

HEI's *P*ublic Health and *A*ir *P*ollution in *A*sia (PAPA)

Nov. 2010

Summary of Current Global Epidemiologic Evidence on Health Effects of Air Pollution: Implications For Asia

PAPA SAN: Overview of all Asian health effects studies identified through 2007

Quantitative review (meta-analysis) of more than 80 time-series studies of daily mortality and hospital admissions

- ***Including 7 NEW PAPA Studies***

First-ever review of over 100 studies of the chronic effects of exposure to air pollution (to be published separately)

For policymakers, scientists and stakeholders

www.healtheffects.org

*Public Health and Air Pollution in Asia – Science Access on the Net (PAPA-SAN)**

Studies of Air Pollution and Health in Asia, 1980–2007

- Web compendium of studies on health effects of air pollution in Asia
- Currently > 420 studies in 11 countries
  - 44 Studies in India

*available at *http://www.healtheffects.org/Asia/papasan-home.htm*

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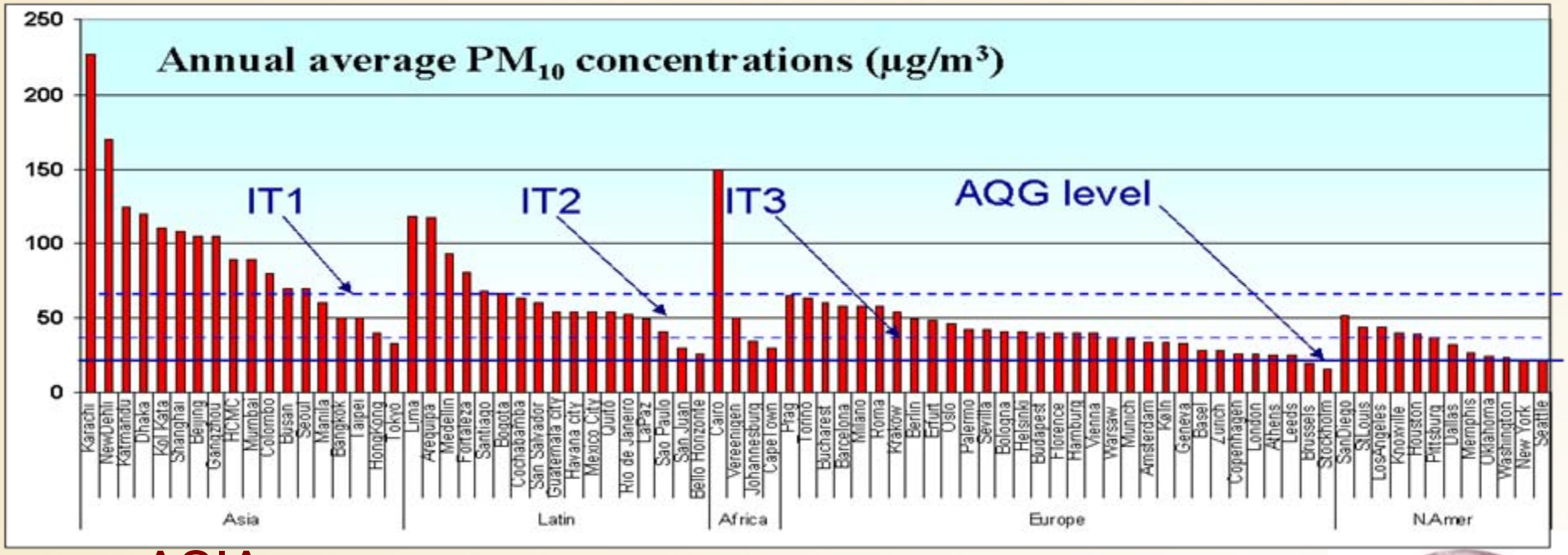
What do vehicles contribute to exposure?

Air Pollution: A Problem Worldwide

Ambient Levels of Particulate Matter (PM) Exceed Current WHO Air Quality Guidelines

Especially in Some Asian Countries



ASIA

World Health Organization 2006

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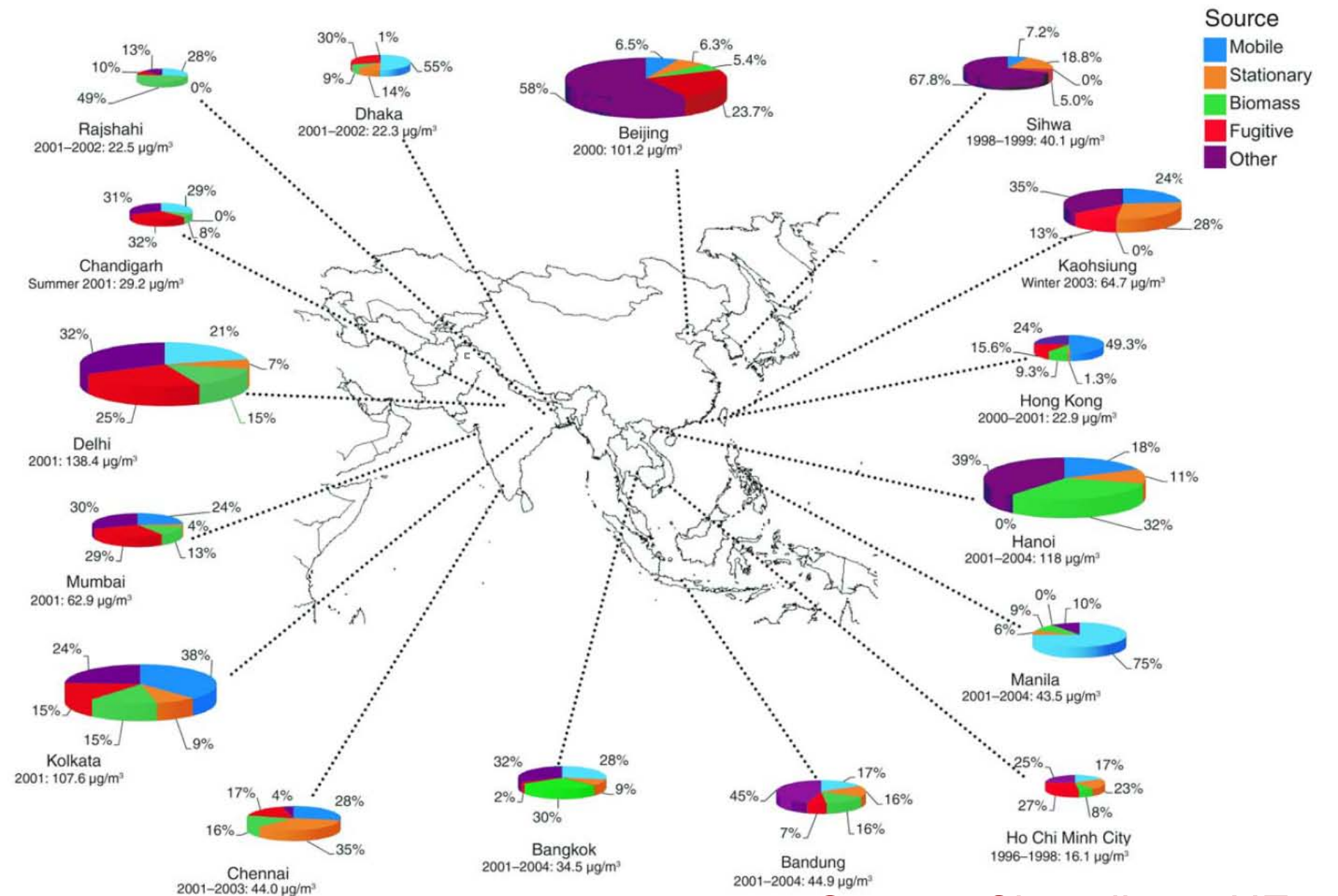
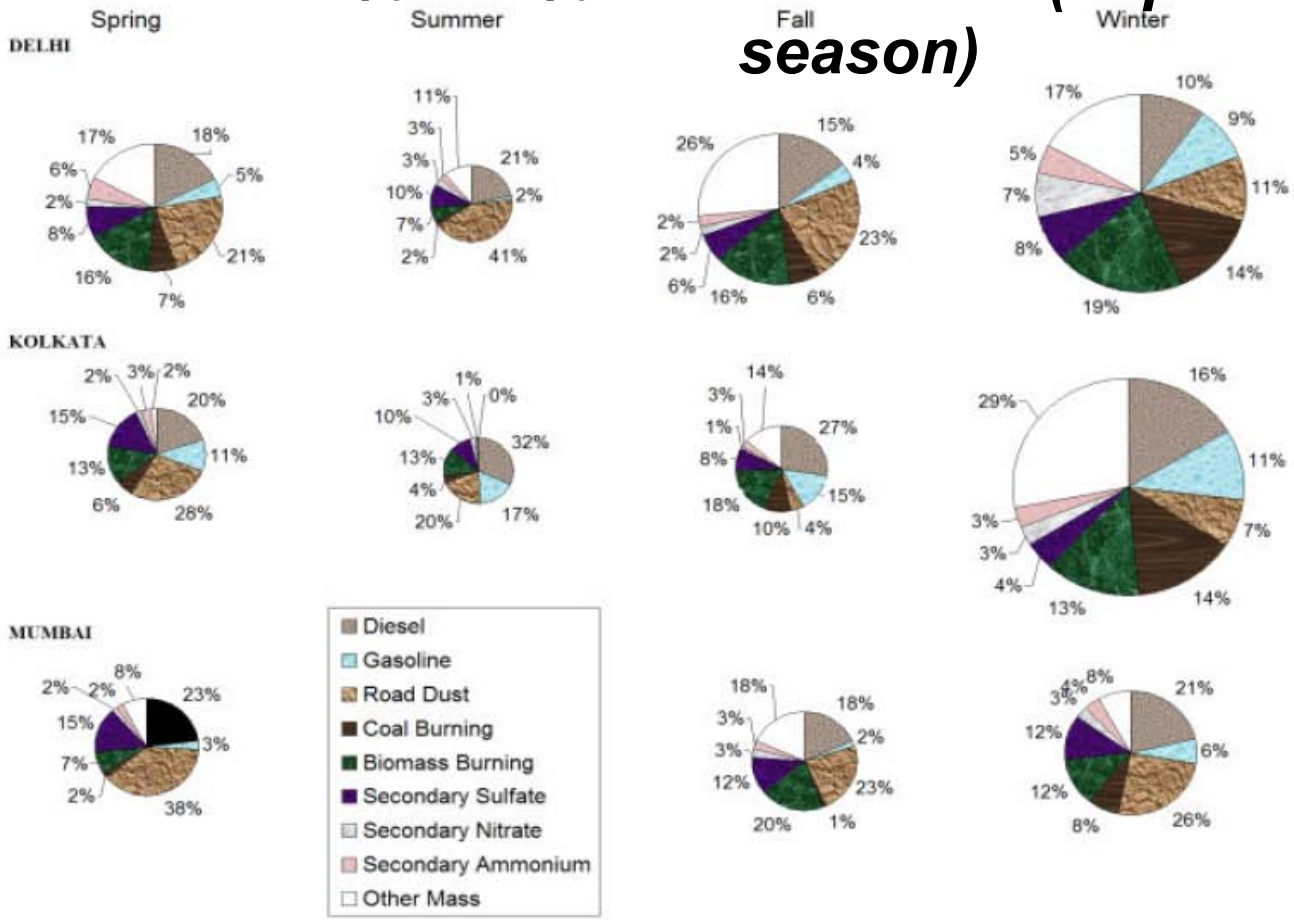
Asia PM 2.5 Source Apportionment: Vehicles ~20% - 35%



Source: Chowdhury, HEI

Seasonality of Sources in India

Vehicles ~20% - 30% of total PM2.5 (depending on city and season)



- Fossil fuel and biomass combustion dominates:
  - Fossil Fuel
  - Del: 25-33%
  - Kol: 37-53%
  - Mum: 21-35%
  - Biomass
  - Del: 7-19%
  - Kol: 13-18%
  - Mum: 7-20%
- Dust dominates during Spring and Summer
  - Long range transport and dust from local construction
- Biomass and coal are high in winter
  - Heating
  - Poor mixing and atmospheric inversion

Road Dust an additional 10% - 30%

Source: Adapted from Chowdhury et al. (2007).

Next Session to Discuss Source Apportionment in Detail

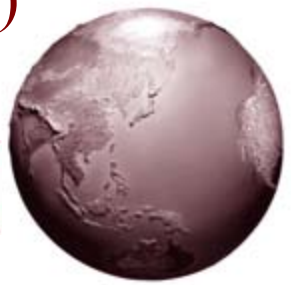
What Do We Know About Health Effects?

- PM and Gases (Ozone and NO2)
- Traffic as a "Source" Exposure

Major Vehicle/Fuel Emissions

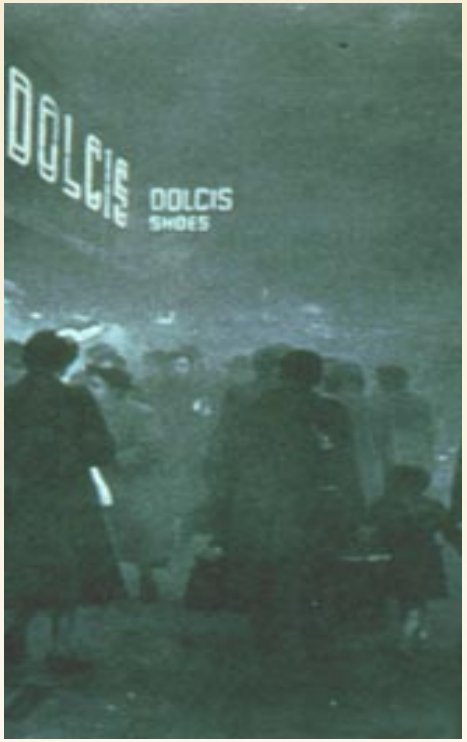
Many substances, not all of equal concern

- Carbon Monoxide
- Carbon Dioxide (Climate Change)
- Diesel Exhaust
- **Particulate Matter (PM)**
- Lead
- **Ozone precursors**
  - **Nitrogen Oxides (NOx) and Hydrocarbons (HC)**
- **Nitrogen Dioxide**
- Air Toxics
  - Aldehydes
    - formaldehyde
    - acetaldehyde
    - others
  - Benzene
  - 1,3-butadiene
  - Metals
  - Polycyclic organic matter (e.g. PAHs)



PM

- Sources:
  - wide range of combustion sources;
  - vehicles are significant, though not only, contributor
- High levels of PM (> 500 μg/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 μ/m^3)
  - no evidence of a "threshold" (safe level)



London at Noon, December 1952

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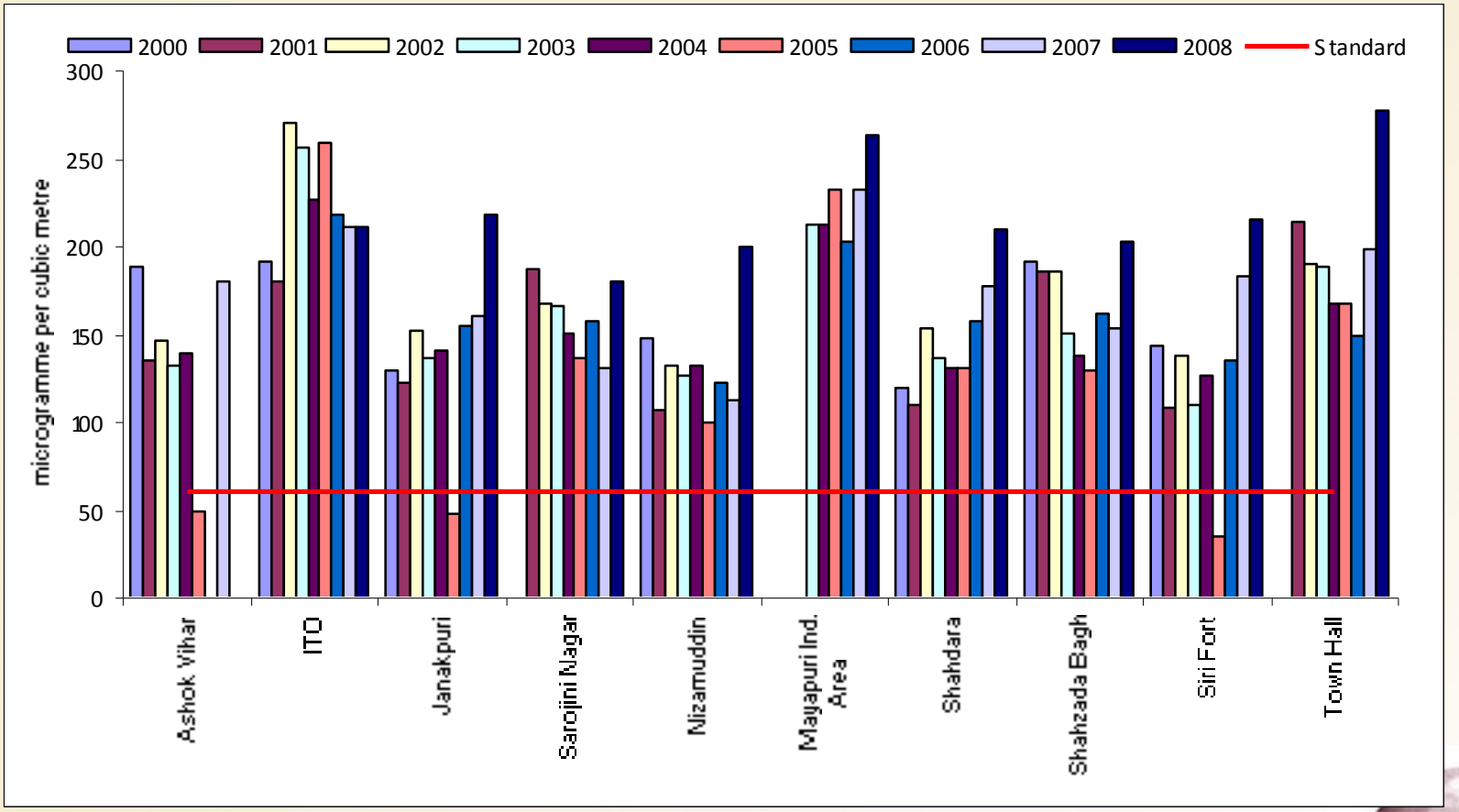
PM10 in Delhi:
Substantially Above Indian NAAQS of 60 µg/m^3

PM10: Annual average levels



Source: CSE analysis based on CPCB air quality data

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 $PM_{2.5}$)

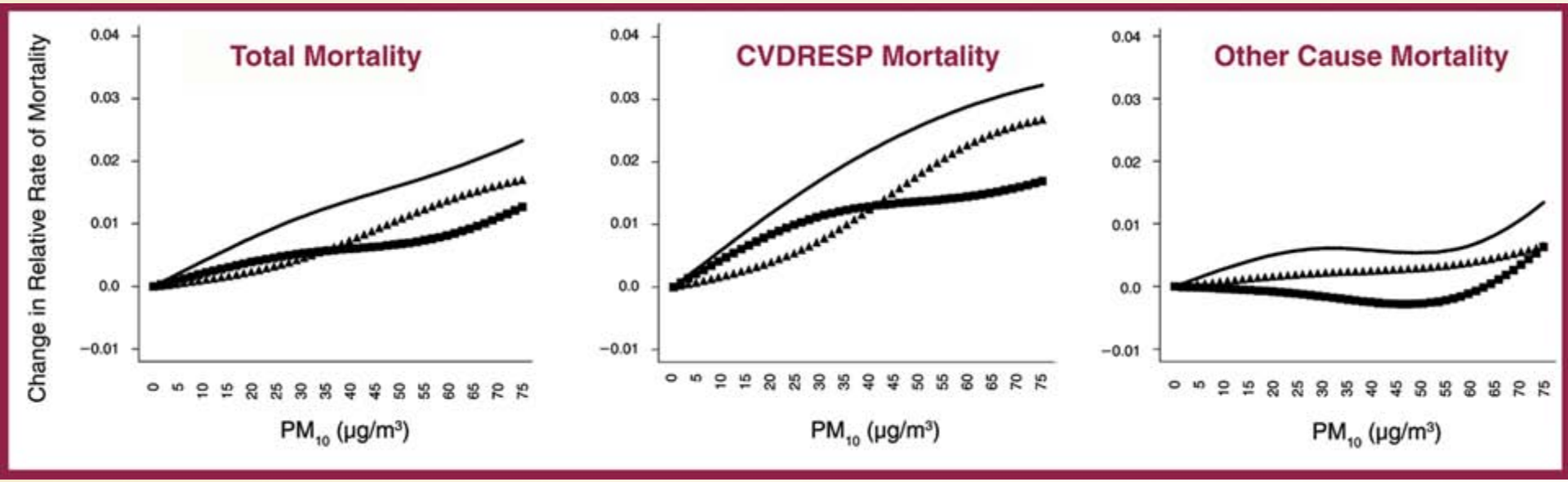
Commentary Table 3. Associations Between Various Causes of Death and Long-Term Exposure to $PM_{2.5}$ in Two Time Periods from the Nationwide Analysis[a]

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)

Short Term (Daily) PM Effects

National Morbidity, Mortality and Air Pollution Study (NMMAPS)

Approximately 0.2% increase in mortality per 10 µg/m^3 PM10



20 largest US cities
(Daniels et al HEI 2004)

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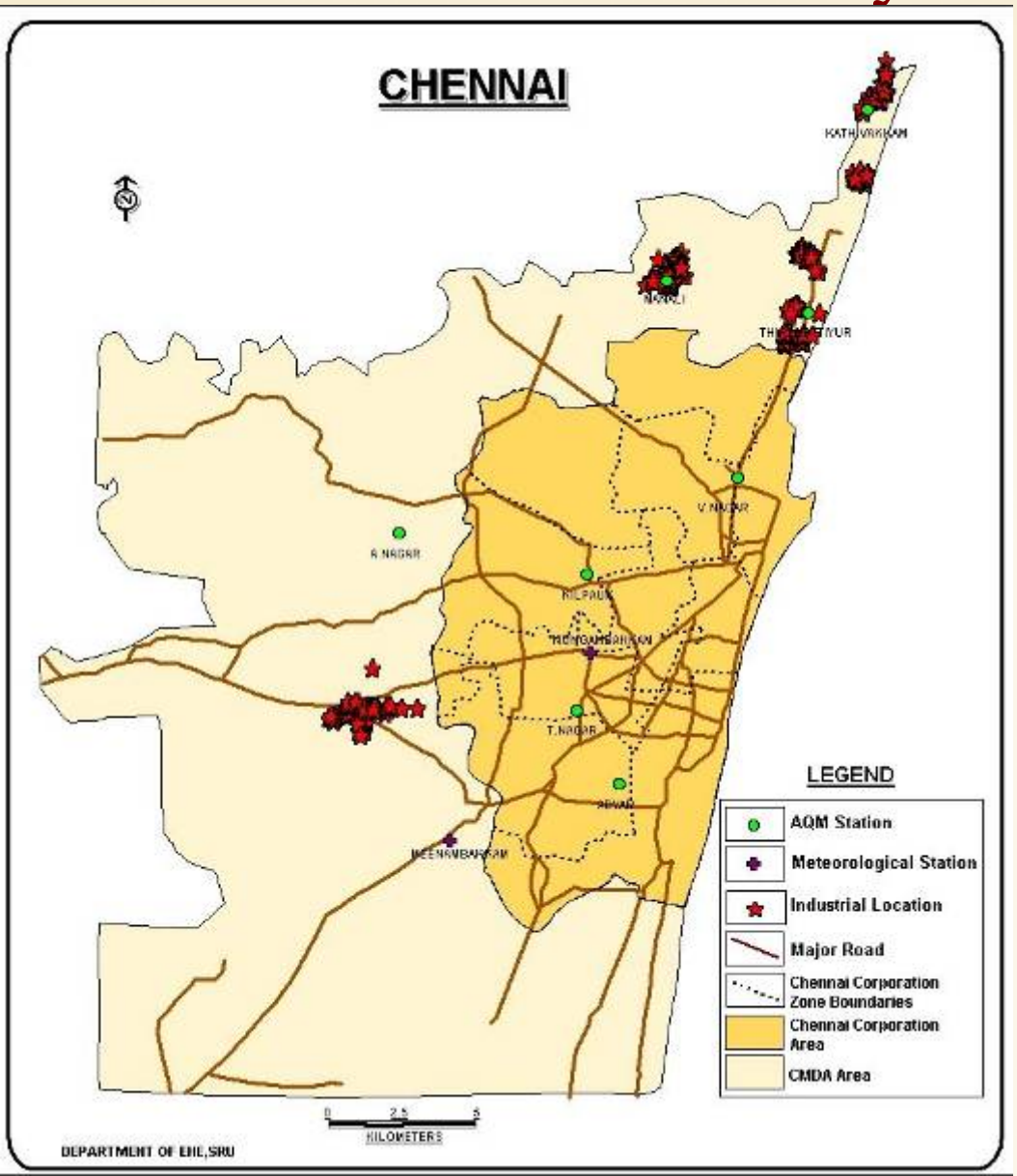
New HEI PAPA Studies in India

- Three studies chosen competitively and overseen by international experts
- Major new HEI Report, March 2011:
  - Chennai – Dr. Kalpana Balakrishnan and colleagues, Sri Ramachandra University
  - Delhi – Dr. Uma Rajarathnan, and colleagues, TERI
- Also,
  - Ludhiana – Dr. Rajesh Kumar, PGI Chandigarh
    - Published in the Indian Journal of Public Health

New Data from India:
HEI Study in Chennai



- Careful analysis of ***daily trends in air pollution and all cause mortality***
- Dr. Kalpana Balakrishnan and colleagues
- Overseen by HEI International Science Oversight Committee
- Independently and Intensively Peer Reviewed

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Chennai Results:

Approximately 0.3% -0.6% increase in mortality per *10 μg/m³* PM10

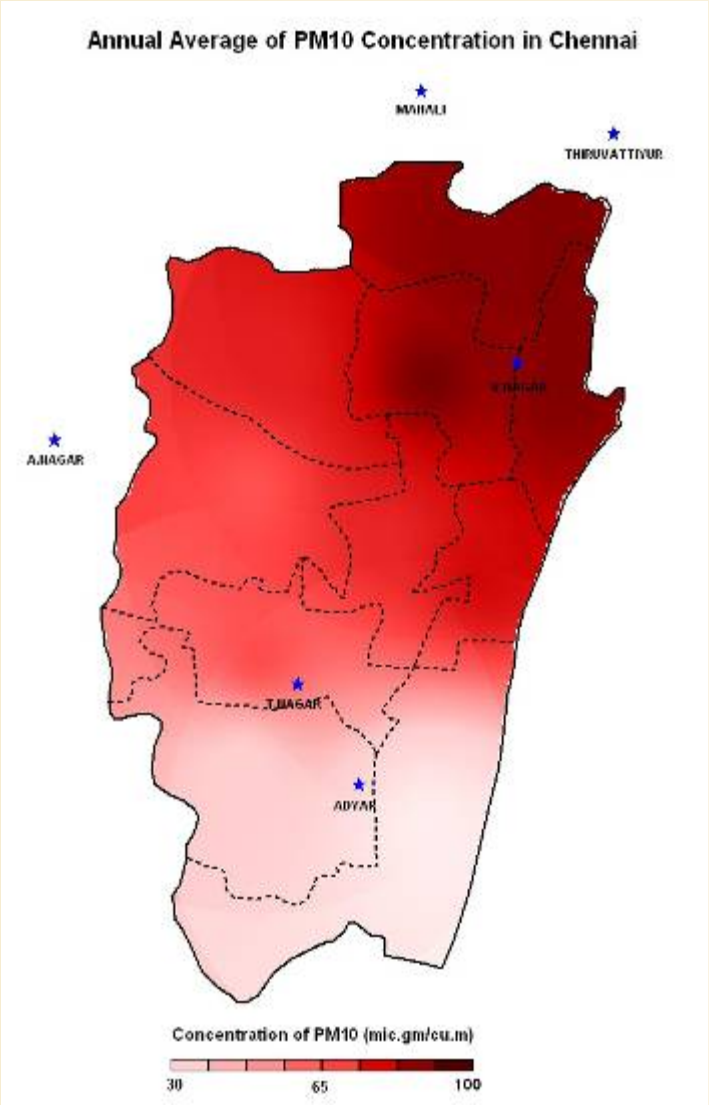
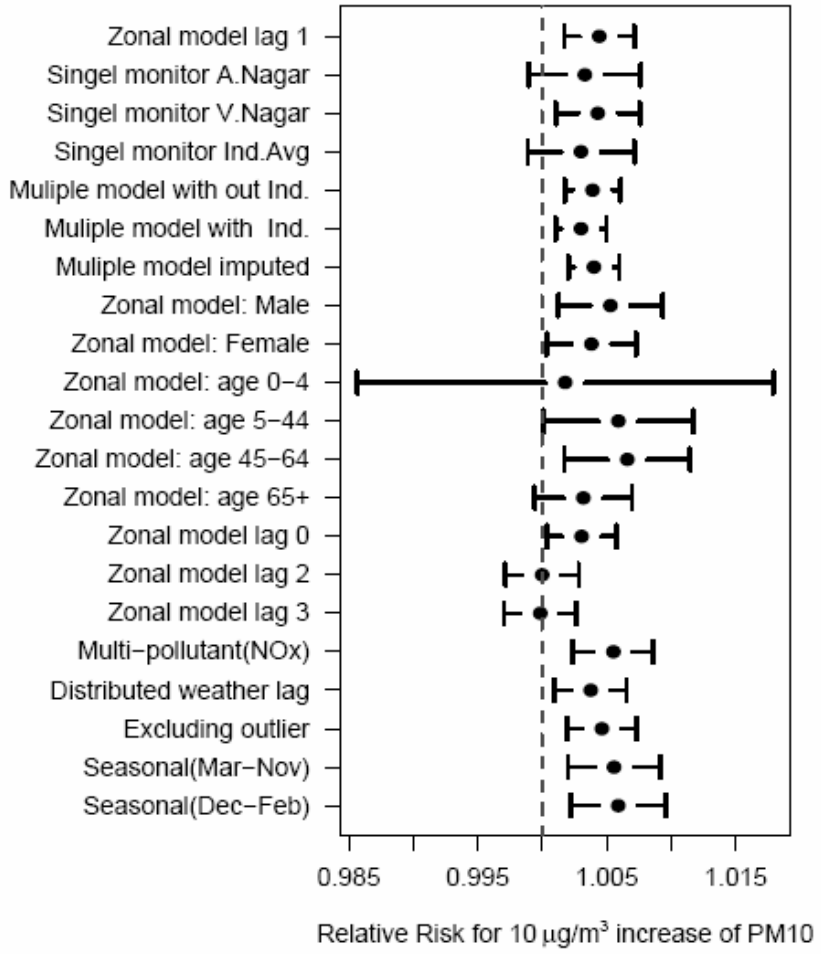


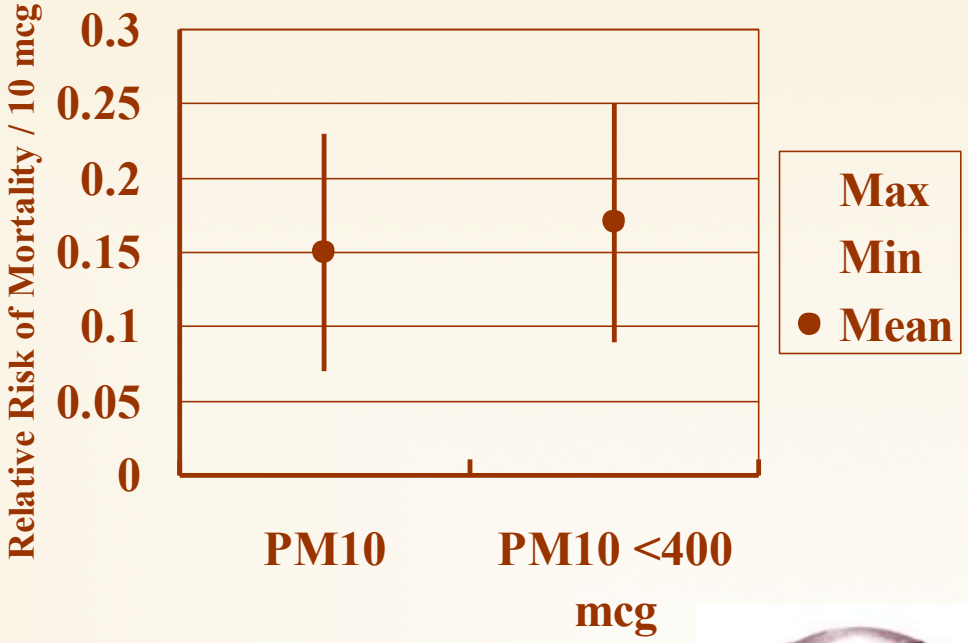
Fig. 23: A comparison of the estimated RR's for PM10 obtained from the core zonal model, alternative models and sensitivity analysis.



New Data from India:
HEI Study in Delhi

Approximately 0.15% to 0.17% increase in mortality per 10 µg/m^3 PM10 (~0.3%/ 20 µg/m^3)

- Careful Analysis of Daily trends in air pollution and all cause mortality
- Dr. Uma Rajarathnam and colleagues at TERI
- Overseen by HEI International Science Oversight Committee
- Independently and Intensively Peer Reviewed



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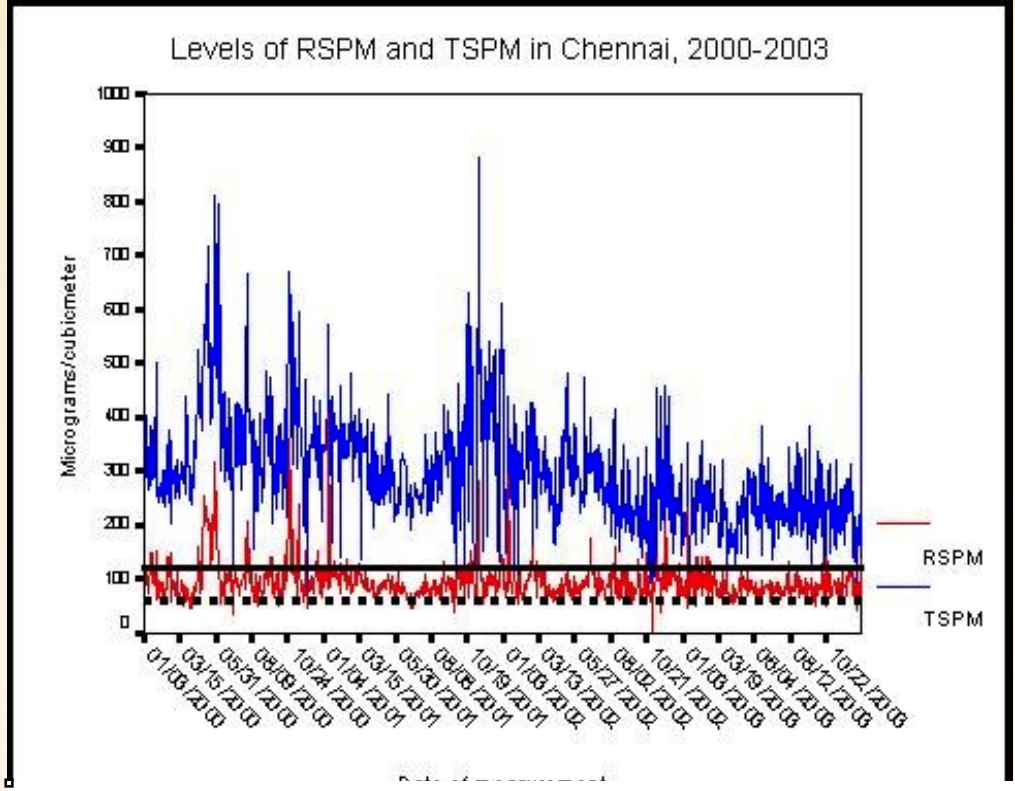
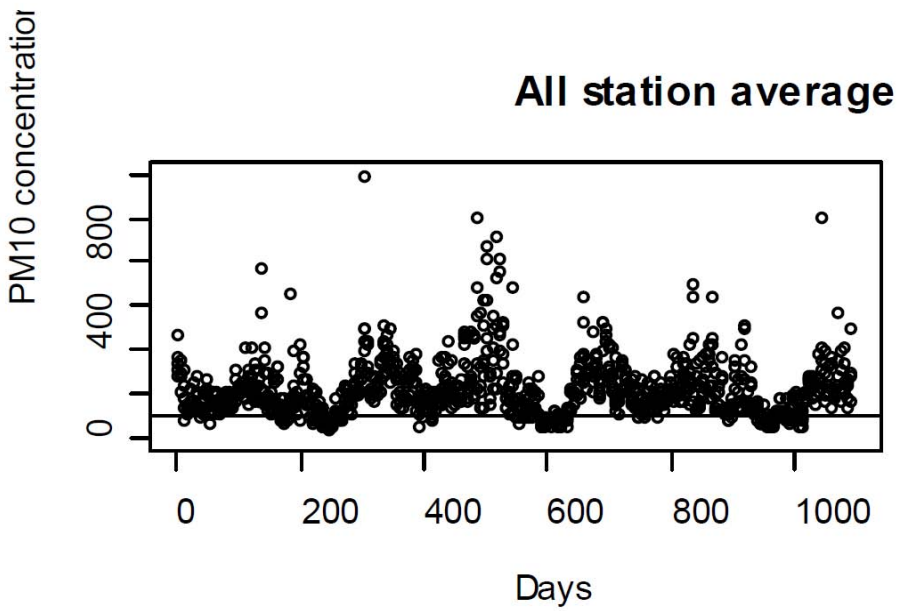
What might "X% increase in risk per 20µg/m³" mean for public health?

- Very high levels of RSPM (PM_{10}):
  - Chennai (red in upper graph);
  - Delhi (black in lower graph)
- Many days above Indian 24-hour RSPM AQ Standard (100µg/m³) (black line)
- Can regularly be as high as 250 – 300µg/m³ with peaks as high as 800µg/m³ in Delhi





What might X% increase in risk per 20µg/m³ mean for public health?

- For example: if annual Delhi RSPM levels are 250µg/m³, *levels* ***are 150µg/m³ higher than the Indian National Ambient Air Quality Standard***

- Using HEI's India results (0.3 - 0.6% increase per 20 µg/m³), ***risk of premature mortality would be ~ 2% - 4.5% higher due to air pollution***
  (i.e. 150/20 = 7.5; 7.5 X 0.3 – 0.6% = 2.25% – 4.5% increase in risk)

- This is a relatively small increase in risk, although if applied to overall deaths in Delhi (~100,000 per year), the public health implications could be much larger
  - ***e.g.~2,250 – 4,500 premature deaths per annum***

- This is based on *short term studies* of daily effects;
  - the ***longer term effects are likely to be significantly larger***

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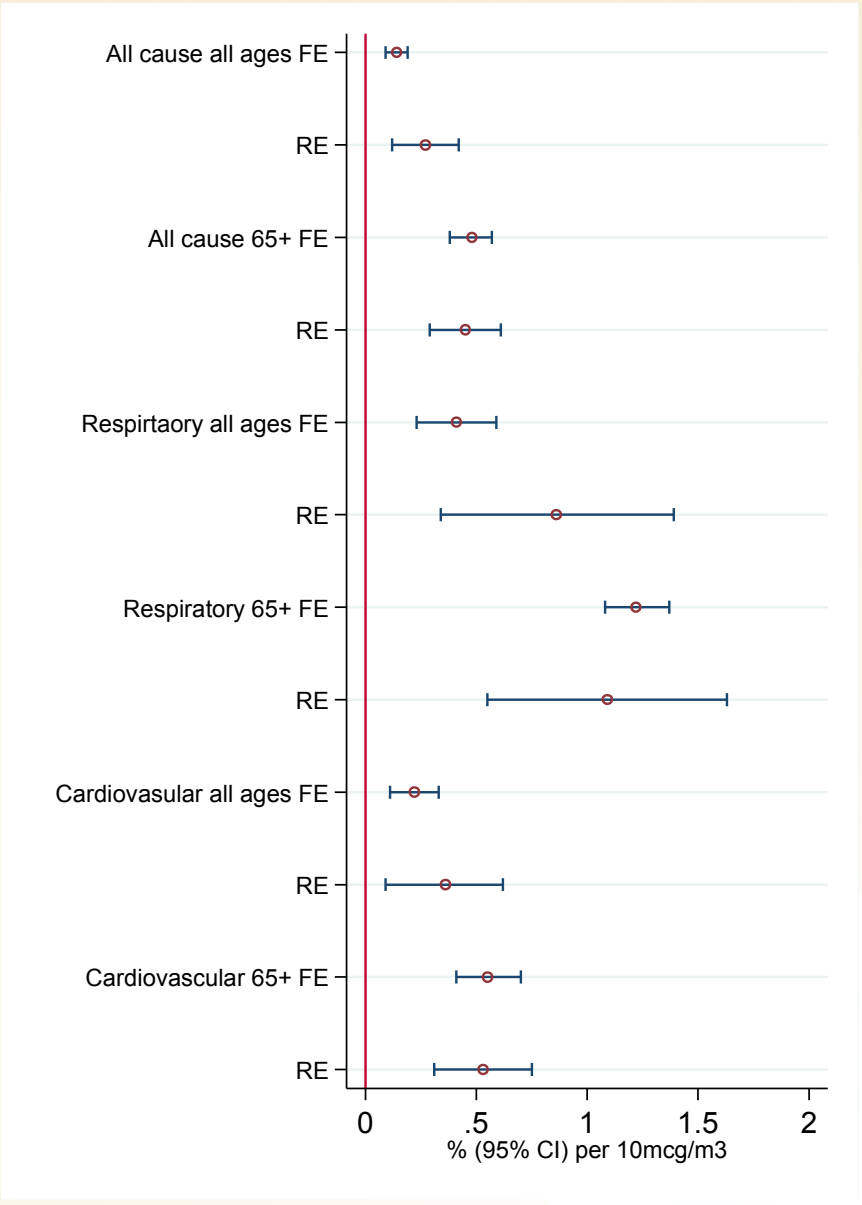
The Broader Asian Evidence (HEI Review 2010):
PM_{10} and daily mortality:
consistent small increase in premature mortality risk

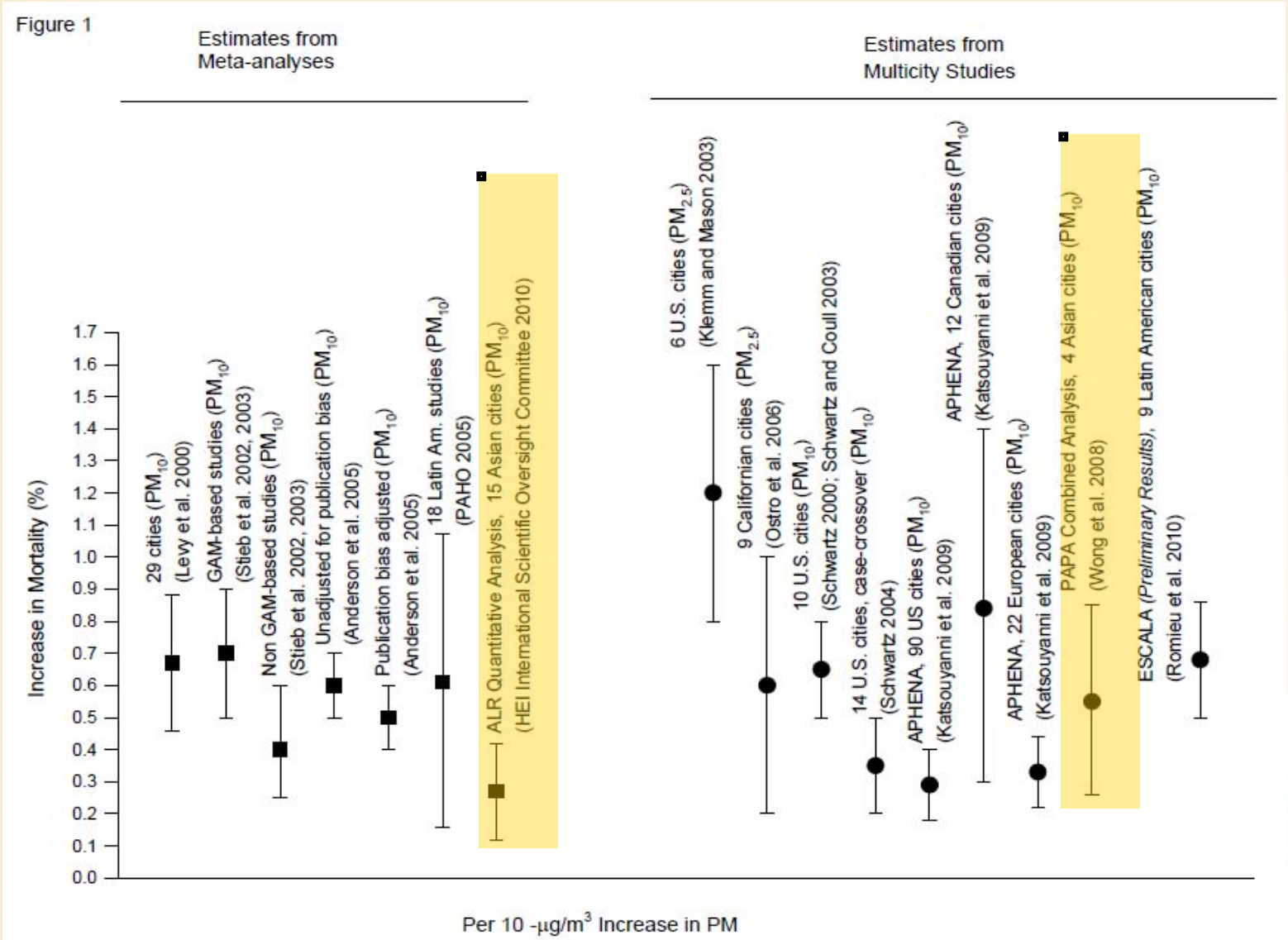
- **Short-term exposure associated with increased daily mortality from all non-accidental causes (0.27%).**
  - ***About halfway between the Chennai and Delhi results***
- **Larger estimates for respiratory (0.84%) and cardiovascular (0.36%) mortality**
- **Somewhat larger estimated effects among those >65 yr.**



Asia in a Global Context

(Risk of Premature Mortality with Increased Exposure to PM10)

Effects of pollution in India and Asia are similar to results around the world



Ozone

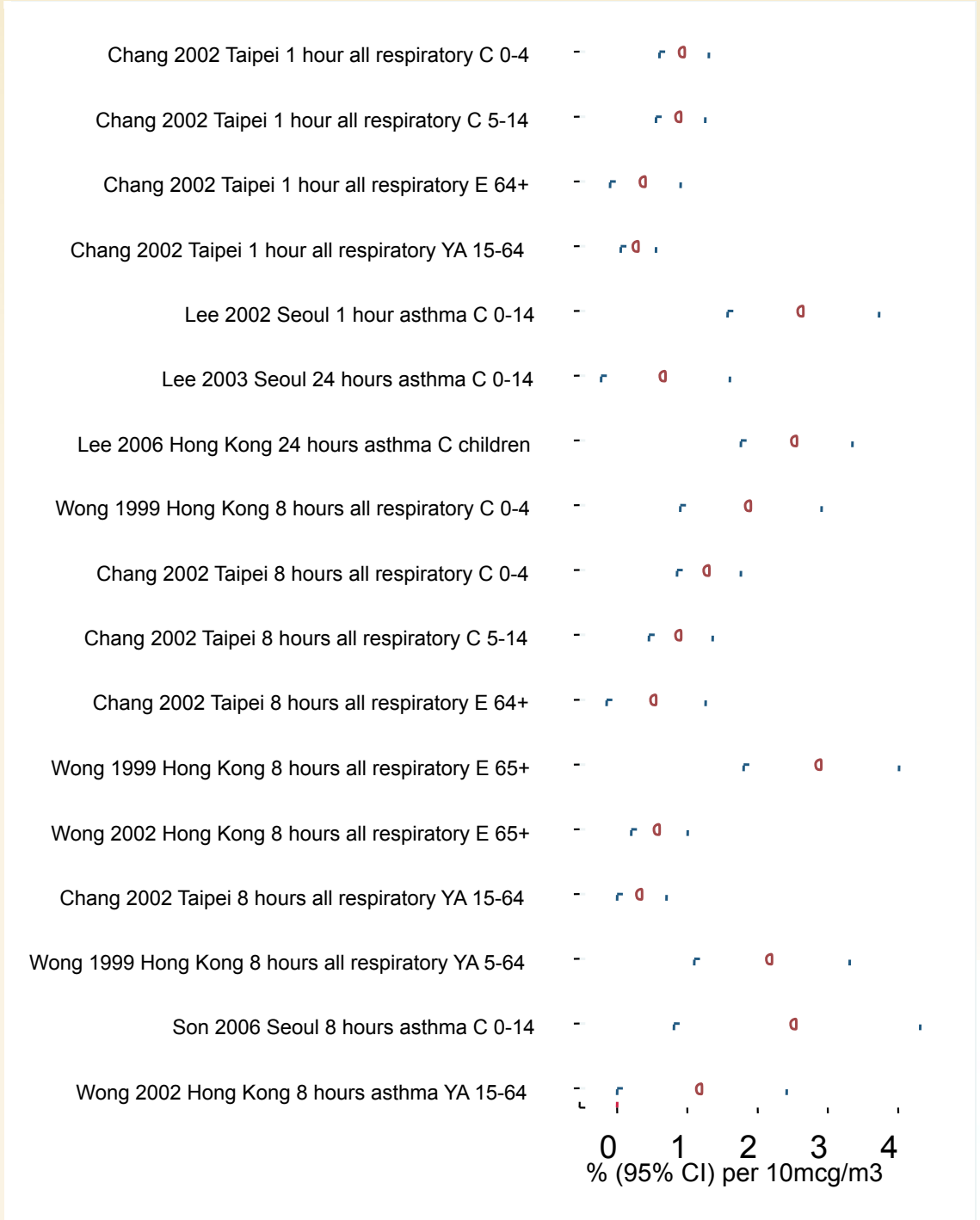
- Sources: VOCs, NOx from mobile and other sources
- Known to:
  - Cause inflammation in respiratory tract
  - Reduce ability to breathe (lung function) for some
  - Increase hospitalization for asthma, other lung diseases
- Recent multi-city evidence of effects on premature mortality
  - *Long term effects are less certain*

Ozone Evidence from Asia: Increased Hospitalization

(HEI Review, 2010)

Ozone and Respiratory Admissions – Specific Ages and Diseases

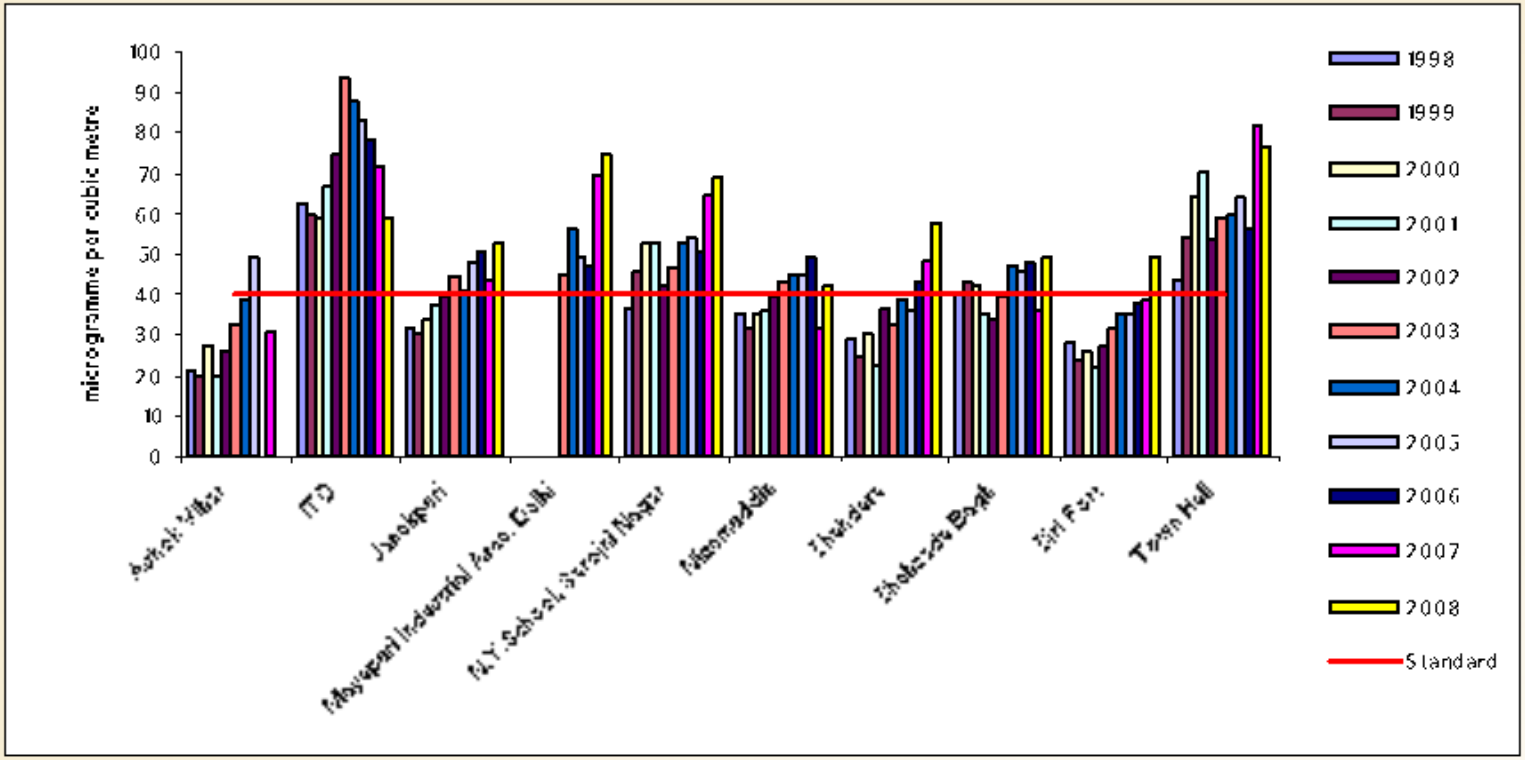
- Consistent small increases in risk



Nitrogen Dioxide (NO2)

- Sources: vehicles a significant source; also thermal power plants
- Known, like many "oxidants" to cause inflammation
- May cause serious problems at lower levels and short, high doses
- Also may be a "marker" for other pollutants (e.g. fine PM)

NOx Levels Rising in Delhi



Nitrogen oxide levels are rising in almost all locations in Delhi.

NOx also contributes to the problem of ozone pollution

Source: CSE analysis based on CPCB air quality data

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 9, 2004 VOL. 351 NO. 11

The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age

W. James Gauderman, Ph.D., Edward Avol, M.S., Frank Gilliland, M.D., Ph.D., Hita Vora, M.S., Duncan Thomas, Ph.D., Kiros Berhane, Ph.D., Rob McConnell, M.D., Nino Kuenzli, M.D., Fred Lurmann, M.S., Edward Rappaport, M.S., Helene Margolis, Ph.D., David Bates, M.D., and John Peters, M.D.

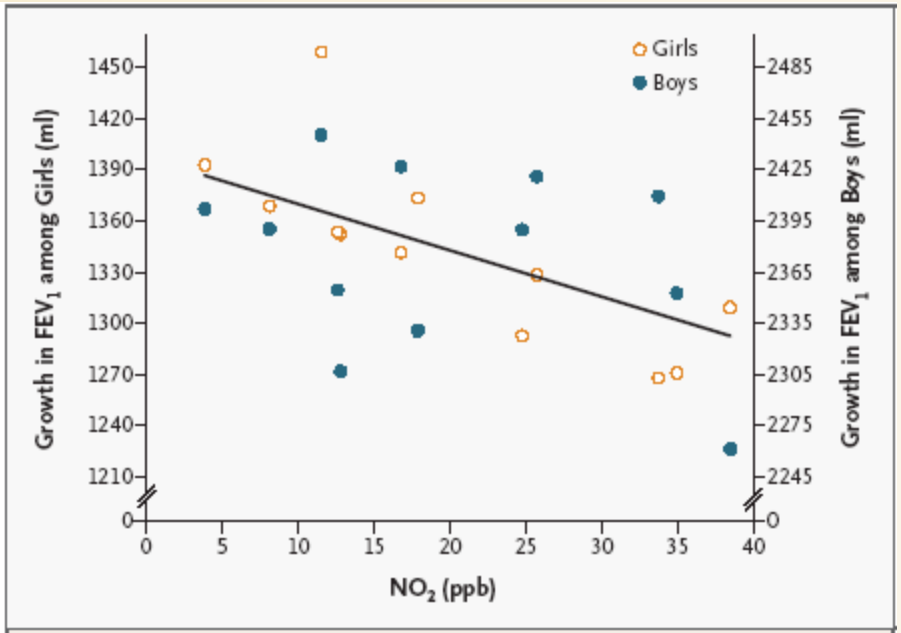


Figure 2. Community-Specific Average Growth in FEV_1 among Girls and Boys During the Eight-Year Period from 1993 to 2001 Plotted against Average Nitrogen Dioxide (NO_2) Levels from 1994 through 2000.

Childhood lung function development reduced in those exposed to higher NO2

Community-specific average growth in FEV1 among Girls and Boys for the period 1993 to 2001 plotted against average nitrogen dioxide (NO2) levels from 1994 to 2000 (Gauderman 2004)

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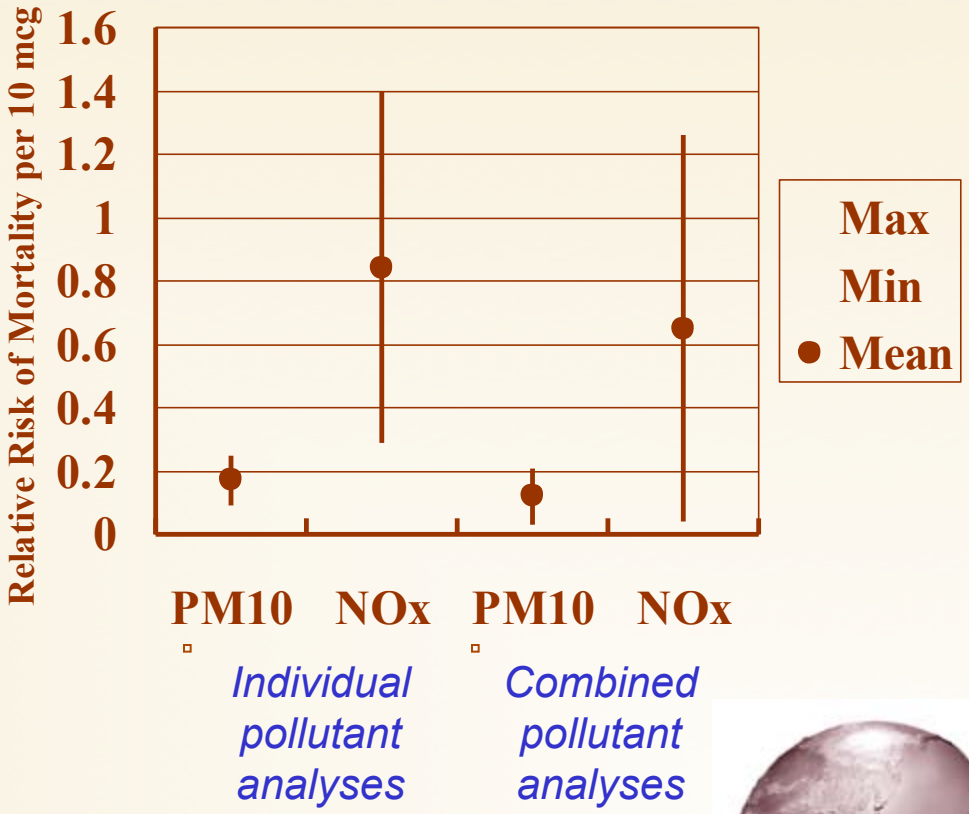
New NOx Results from India:
HEI Study in Delhi

- Delhi study also tested Nitrogen Oxide associations
  - Independently and with PM10
- Found higher estimates of risk for NOx (0.65%/ 10 µg/m^3) than for PM10



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Traffic- Related Air Pollution:

A Critical Review of the Literature on Emissions, Exposure, and Health Effects

HEI Expert Panel
Dr. Ira Tager, UC Berkeley, Chair

January 2010

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TABLE 5.2. SUMMARY OF HUMAN STUDIES DISCUSSED[2]

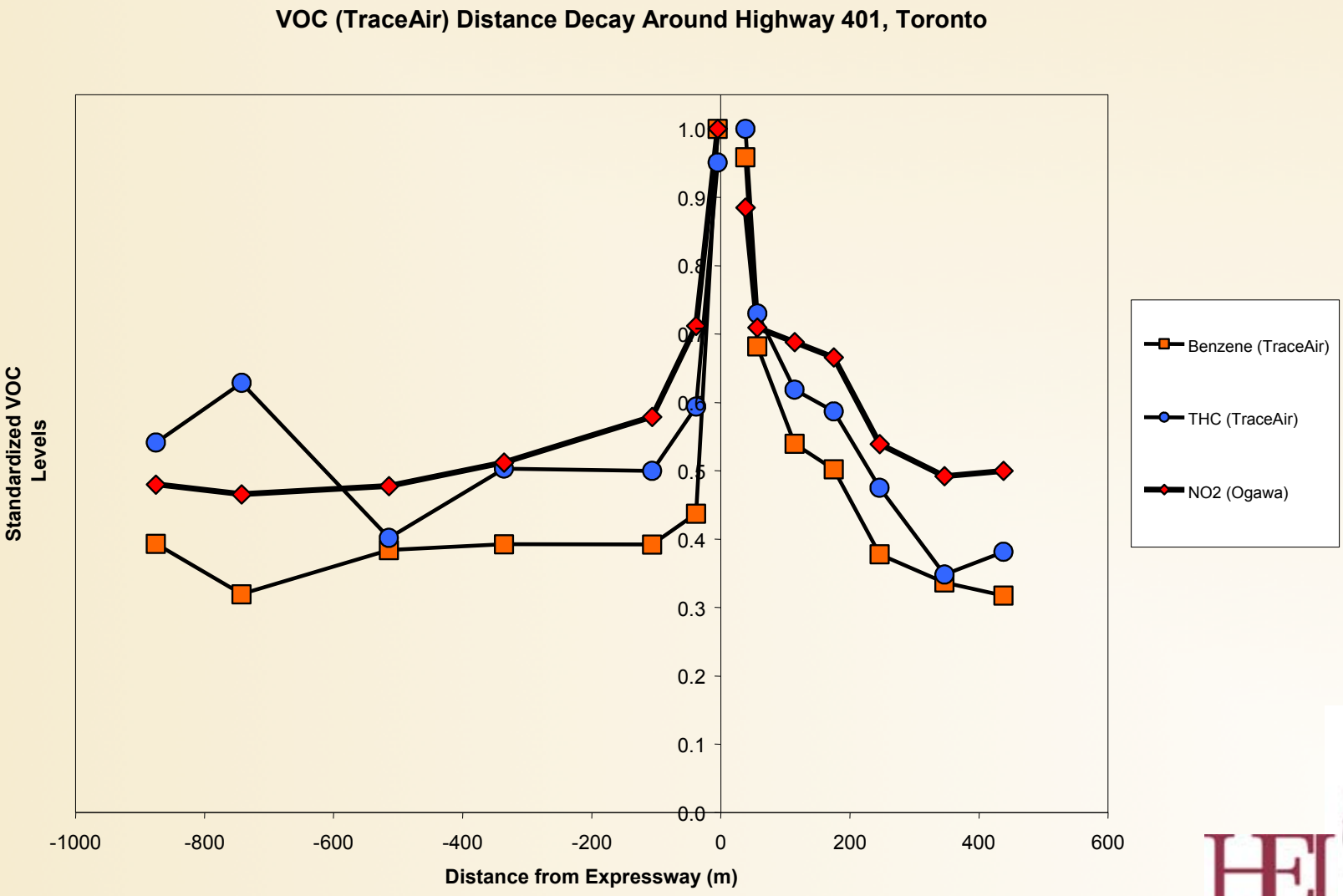
Reference	Health Endpoint	Subjects	Exposure Conditions (concentration, time)	Findings
Traffic Mixture				
(Bräuner et al. 2007)	DNA damage and oxidative stress	29 healthy subjects (20–40 yr)	Particle filtered air (91–542 particles/cm³) or unfiltered air delivered from a busy roadway in Copenhagen (6169–15,362 particles/cm³) for 24 hr, with two 90-min episodes of exercise	Particle exposure associated with increased strand breaks and oxidized purines. Dose–response relation between particle number and DNA damage.
(Bräuner et al. 2008a)	Microvascular function, markers of systemic inflammation and coagulation	41 healthy subjects (60–75 yr)	Indoor air (7,718–12,988 particles/cm³) or filtered air (2,533–4,058 particles/cm³) in homes within 350 m of major roads for two consecutive 48-hr exposures	8.1% improvement in digital peripheral arterial tone following ischemia after particle filtration, compared with no filtration. No differences in blood markers.
(Bräuner et al. 2008b)	Microvascular function, markers of systemic inflammation and coagulation	29 healthy subjects (20–40 yr) (same volunteers as Bräuner et al. 2007)	Filtered air (~555 particles/cm³) or air delivered from near a busy roadway (~11600 particles/cm³, 13.8 μg/m³ $PM_{0.2-2.5}$, and 10.5 μg/m³ $PM_{2.5}$) for 24 hr with 2 90-min episodes of exercise	No significant effects on peripheral vascular function or blood markers.
(Larsson et al. 2007)	Pulmonary cellular inflammation response	16 healthy subjects (19–59 yr)	Exposure in a busy road tunnel (median concentrations of 64 μg/m³ $PM_{2.5}$, 176 μg/m³ PM_{10}, 230 μg/m³ NO_2) or urban air for 2 hr during normal activity	Significantly higher numbers of bronchoalveolar lavage fluid total cells, lymphocytes, alveolar macrophages, and nuclear expression of transcription factor component c-Jun; no increase in neutrophils
(McCreanor et al. 2007)	FEV_1 and FVC measurement	60 adults with mild or moderate asthma (19–55 yr)	Walking on low–traffic street (median concentration of 11.9 μg/m³ $PM_{2.5}$, 72 μg/m³ PM_{10}, 21.7 μg/m³ NO_2) or high-traffic street (median concentration of 28.3 μg/m³ $PM_{2.5}$, 125 μg/m³ PM_{10}, 142 μg/m³ NO_2) in London	High-traffic group had significant reductions in FEV_1 and FVC compared to low-traffic group and increases in neutrophilic inflammation and airway acidification
(Rundell et al. 2007)	Flow-mediated dilatation (FMD) and near-infrared light absorption (NIR) (indicators of endothelial function)	16 male collegiate athletes (18–22 yr)	Exposure adjacent to highway (PM_{10}, 143,501 ± 58,565 particles/cm³) or low traffic area (PM_{10}, 5,309 ± 1,942 particles/cm³) while running for 30 min at 85–90% of maximum	FMD and NIR were ablated after exercise near high traffic, and were unchanged near low traffic.
(Svartengren et al. 2000)	Asthmatic reactions	20 subjects with mild allergic asthma	Exposure inside a car in a Stockholm city road tunnel for 30 min (~300 μg/m³ NO_2) or in a suburban area, inhalation of a low-dose allergen 4 hr after exposure	Tunnel-exposed subjects had a significantly greater early reaction to allergen, lower lung function, and more asthma symptoms during the late phase.
Concentrated Ambient Particles (CAPs)				
(Brook et al. 2002)	Systemic vascular function assessed using ultrasound measurement of brachial (forearm) artery diameter and flow-mediated dilatation (FMD)	25 healthy subjects (18–50 yr)	Filtered air (zero $PM_{2.5}$, low O_3) or a mixture of CAPs (in Toronto, $PM_{2.5}$, ~150 μg/m³) and O_3 (0.12 ppm) for 2 hr at rest	Brachial artery constriction 10 min after exposure to pollutants, not after exposure to air. No change in FMD or blood pressure measured at the same time.
(Devlin et al. 2003)	Heart rate variability (HRV)	10 healthy subjects (60–80 yr)	Filtered air or fine CAPs (Chapel Hill, N.C., 0.1–2.5 μm, mean concentration 40.5 μg/m³, range of 21.2–80.3 μg/m³) for 2 hr at rest	Particle-associated reductions in pNN50 and high frequency HRV.
(Ghio et al. 2000a)	Lung function, airway inflammation, blood markers	38 healthy subjects (18–40 yr) (36 males and 2 females)	Filtered air or fine CAPs (Chapel Hill, N.C., 0.1–2.5 μm, mean mass 120 μg/m³, range 23.1–311.1 μg/m³) for 2 hr with intermittent exercise	Mild airway inflammation, increased plasma fibrinogen. No symptoms noted by volunteers or decrements in pulmonary function, mild increase in neutrophils in bronchial and alveolar fractions taken 18 hr after exposure.
(Gong Jr et al. 2003)	Lung function, airway and systemic inflammation, heart rate variability (HRV)	12 healthy subjects and 12 asthmatic subjects with COPD (18–45 yr)	Filtered air or fine CAPs (Los Angeles, < 2.5 μm in diameter, mean mass 174 μg/m³, range 99–224 μg/m³) for 2 hr with intermittent exercise	Systolic blood pressure decreased in asthmatics and increased in healthy subjects during particle exposure, compared with air. Plasma levels of ICAM-1 increased 4 hr post-exposure. PM exposure was associated with HRV effects. Overall changes observed were small and not always consistent across different parameters.
(Gong Jr et al. 2004a)	Lung function, airway and systemic inflammation, HRV	13 elderly patients with COPD (54–85 yr) 6 age-matched healthy adults	Filtered air or fine CAPs (Los Angeles, < 2.5 μm in diameter, mean mass 194 ± 26 μg/m³) for 2 hr with intermittent exercise	Ectopic heart beats increased with particles in the healthy subjects, but decreased in the COPD subjects. HRV decreased with PM in the healthy but not in the COPD subjects. The COPD subjects appeared to be less susceptible than the healthy subjects, although effects were modest.
(Gong Jr et al. 2004b)	Lung function, airway and systemic inflammation, HRV	4 healthy and 12 mildly asthmatic subjects (19–51 yr)	Filtered air or coarse CAPs (Los Angeles, 2.5–10 μm in diameter, mean mass 157 μg/m³, range 56–218 μg/m³) for 2 hr with intermittent exercise	Heart rate increased and HRV decreased, without effects on cardiac ectopy; effects were generally larger in the healthy subjects compared to the asthmatics.
(Gong Jr et al. 2008)	Lung function, exhaled nitric oxide, inflammatory markers, Holter electrocardiography	17 healthy and 14 asthmatic adults (18–50 yr)	Filtered air or concentrated UFP (Los Angeles, 0.1–2.5 μm in diameter, mean counts 145,000 particles/cm³, range 39,000–312,000, mean mass 100 μg/m³, range 13–277, for 2 hr with intermittent exercise	UFP exposures were associated with some mild acute cardiopulmonary responses (0.5% mean fall in arterial O_2 saturation, 2% mean fall in FEV_1 the morning after exposure, slight decrease in low frequency power in Holter readings during rest periods).
(Harder et al. 2001)	Airway and blood immune cell function	38 healthy young adults (18–40 yr) (36 males, 2 females)	Filtered air or CAPs (Chapel Hill, N.C., 0.1–2.5 μm in diameter, mean mass 120.5 ± 14.0 μg/m³, range 23.1 to 311.1 μg/m³) for 2 hr with intermittent exercise	CAPs did not alter distribution or function of immune cells in lung or blood.
(Mills et al. 2008)	Peripheral vascular vasomotor and fibrinolytic function, inflammation	12 male adults with stable coronary heart disease and 12 age-matched healthy adults	Filtered air or CAPs (Edinburgh, U.K., mean mass 190 ± 37 μg/m³, range 50–682 μg/m³) for 2 hr with intermittent exercise	No effect on vascular function or markers of systemic inflammation, dose-dependent significant increase in blood flow and plasma tissue plasminogen activator release.
(Samet et al. 2007)	Lung function, airway inflammation, blood markers, HRV measured with an ECG	72 healthy adults (18–35 yr) (38 adults exposed to fine, 14 to coarse, and 20 to ultrafine)	CAPs (Chapel Hill, N.C., mean mass 120.4 μg/m³ (fine), 89.0 μg/m³ (coarse) ultrafine PM number concentration: 151.8 × 10^3/mL)	Mild airway inflammation with fine and coarse, but not ultrafine CAPs. Reductions in HRV with coarse and ultrafine CAPs. Changes in measures of blood clotting with fine and ultrafine CAPs.
(Urch et al. 2004)	Systemic vascular function (ultrasound measurement of brachial (forearm) artery diameter and flow-mediated dilatation [FMD])	24 healthy subjects (35 ± 10 yr) (same subjects as Brook et al. 2002 study)	Filtered air or a mixture of CAPs (Toronto, median total mass 147.4 μg/m³, range 101.5–257.3 μg/m³) and O_3 (0.12 ppm) for 2 hr at rest	Analysis of day-to-day variability in PM composition in relation to this effect suggested a role for both organic and inorganic elemental carbon. There was no pollutant effect on FMD.
(Urch et al. 2005)	Blood pressure	23 healthy subjects _18–50 yr) (same subjects as the Brook et al. 2002 study with 3+ subjects)	Filtered air or a mixture of fine CAPs (Toronto, <2.5 μm in diameter, mean concentrations 147 ± 27μg/m³) and O_3 (0.121 ppm) for 2 hr at rest	Increased diastolic blood pressure at the end of the 2-hour CAPs + ozone exposures.

There are many studies (over 700) that have attempted to look at traffic exposure and effects

- *However, they are not **all** of equal quality*

Who is Likely to be Exposed?

Highest levels within 300 – 500 meters of a major road

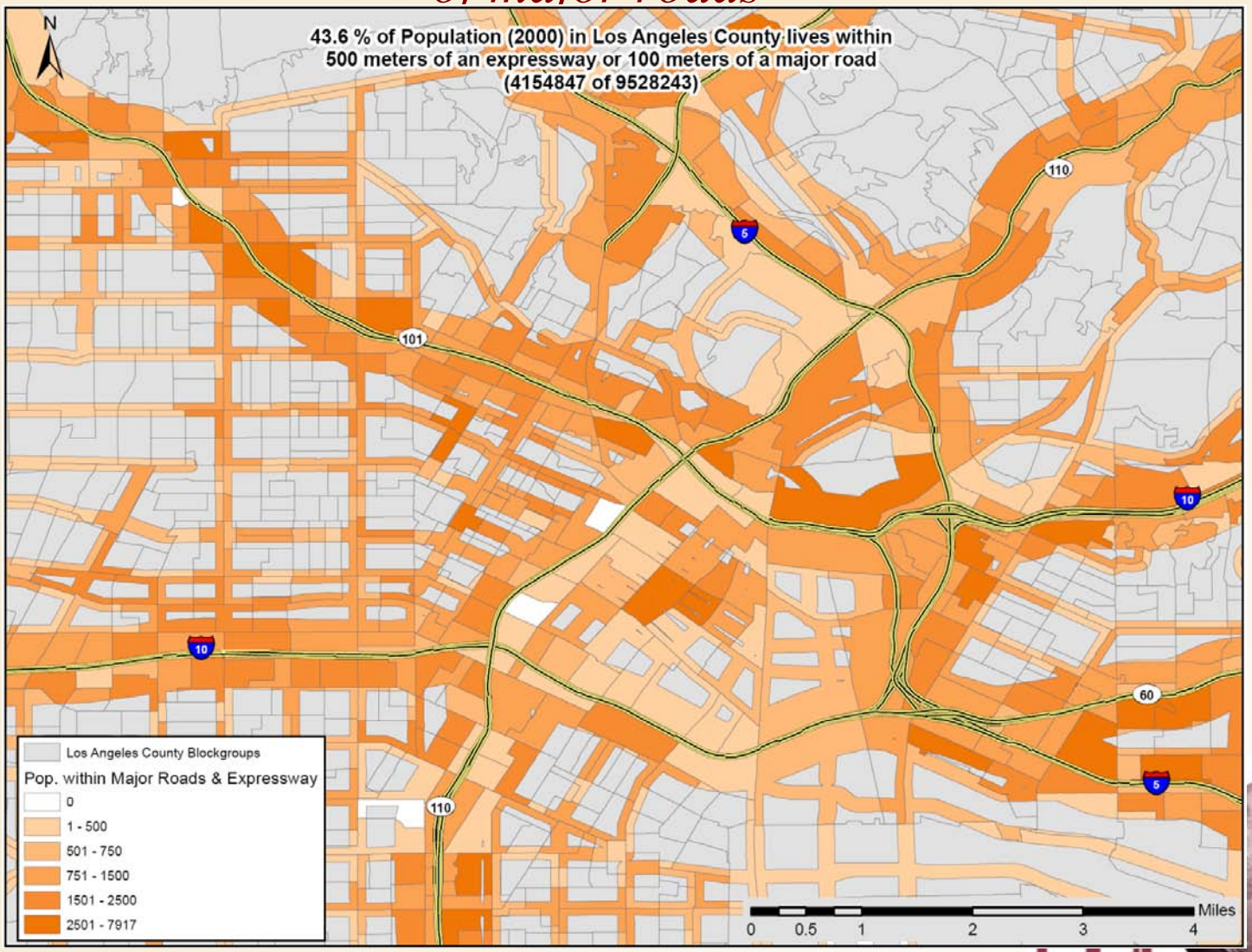


Toronto, Beckerman et al. (2008)

HEI

The HEI Traffic Review:

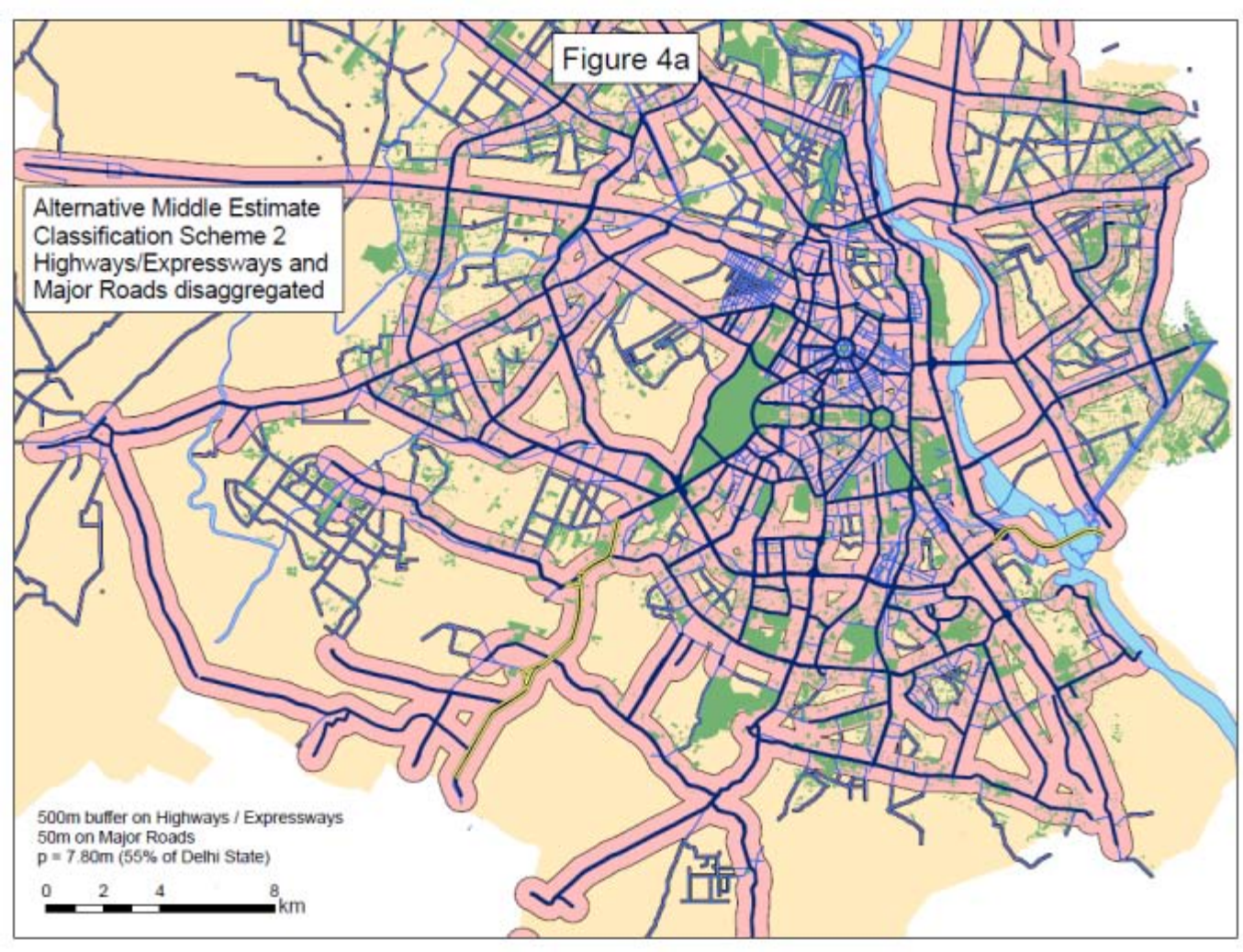
*In **Los Angeles**, **44% of population** live in the maximum zone of impact of major roads*



(within 500 meters of an expressway; 100 meters of a major road)

The Traffic Impact Area in Delhi:

New HEI Analysis: ***55% of the Population*** *within 500 meters of a Freeway; 50 meters of a Major Road*



Overall Traffic Conclusions

- The data are incomplete on emissions, their transformations, and exposure assessment
- There were enough studies to find:
  - ***Sufficient*** evidence that exposure to traffic can cause exacerbation of asthma, especially in children
  - ***Suggestive*** evidence for other health effects (premature mortality, lung function, respiratory symptoms, and others)
  - But only ***limited evidence*** of effects for: Adult onset asthma; Health care utilization; COPD; Non-asthmatic allergy; Birth outcomes; Cancers

Overall Traffic Conclusions II

- Epidemiology studies are based on past estimates of exposure
  - they may not provide an accurate guide to estimating health associations in the future
- However, given the large number of people living within 300- 500 meters of a major road, the Panel concluded that exposures to primary traffic generated pollutants are likely to be of public health concern and deserve attention.

Concluding Thoughts

- Traffic is a significant but *not the only* contributor to major air pollutant exposure
  - Key pollutants: PM, Ozone, NO2
  - Traffic as a "source" exposure
- Strong body of evidence of effects for most of these pollutants
  - Growing Asian and Indian evidence suggesting similar effects
- Significant fuel and technology enhancements underway:
  - HEI Vice President Bob O'Keefe will describe these tomorrow

Thank You

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