Vehicle Emissions and Health:

A Global Perspective on Effects, Placed in an Indian Context

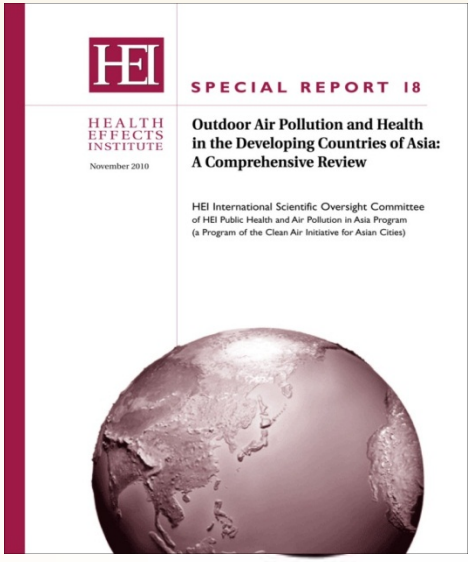
Dan Greenbaum, President
Health Effects Institute

Delhi, India
31 August 2011

The Health Effects Institute

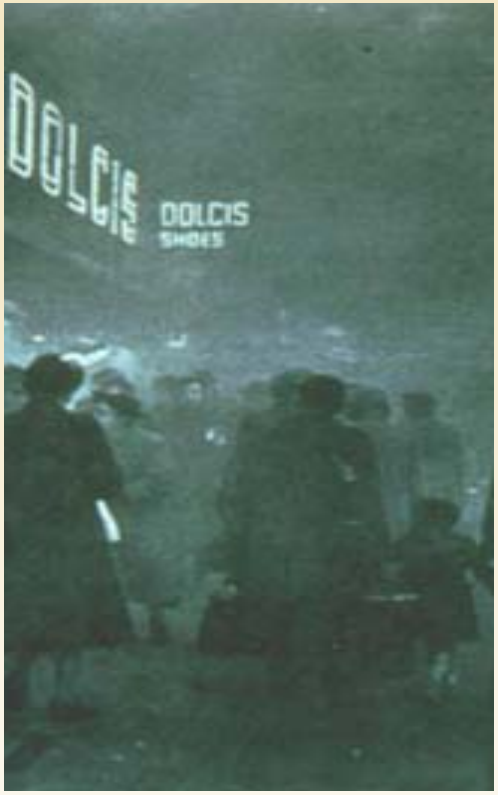
- Non-profit institute providing *trusted science on the health effects of air pollution* for over 30 years
- Joint core support from
  - Government (US EPA) and Industry (Worldwide Motor Vehicle)
  - Partnerships with WHO, ADB, CAI-Asia, EU, US DOE, other industries, Hewlett foundation
- Independent 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*
  - *Comprehensive Review of **Asian Health Science (2010)***
- HEI delivers science
  - no policy positions taken

Understanding local impacts in a global context



Particulate Matter (PM)

- Sources:
  - wide range of combustion sources;
  - vehicles are significant, though not only, contributor
- High levels of PM (> 500 $\mu g/m^3$) 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)

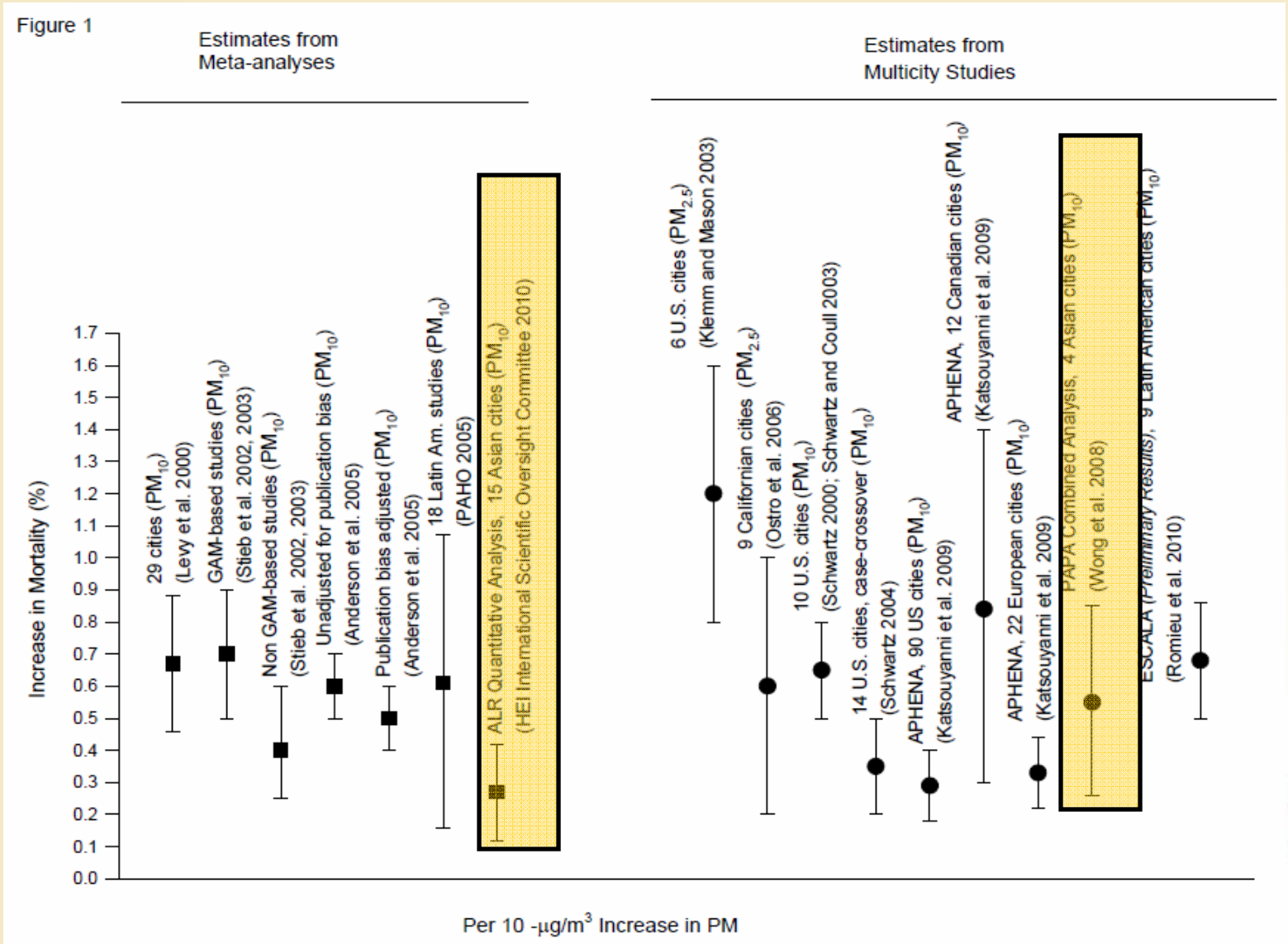


London at Noon, December 1952

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

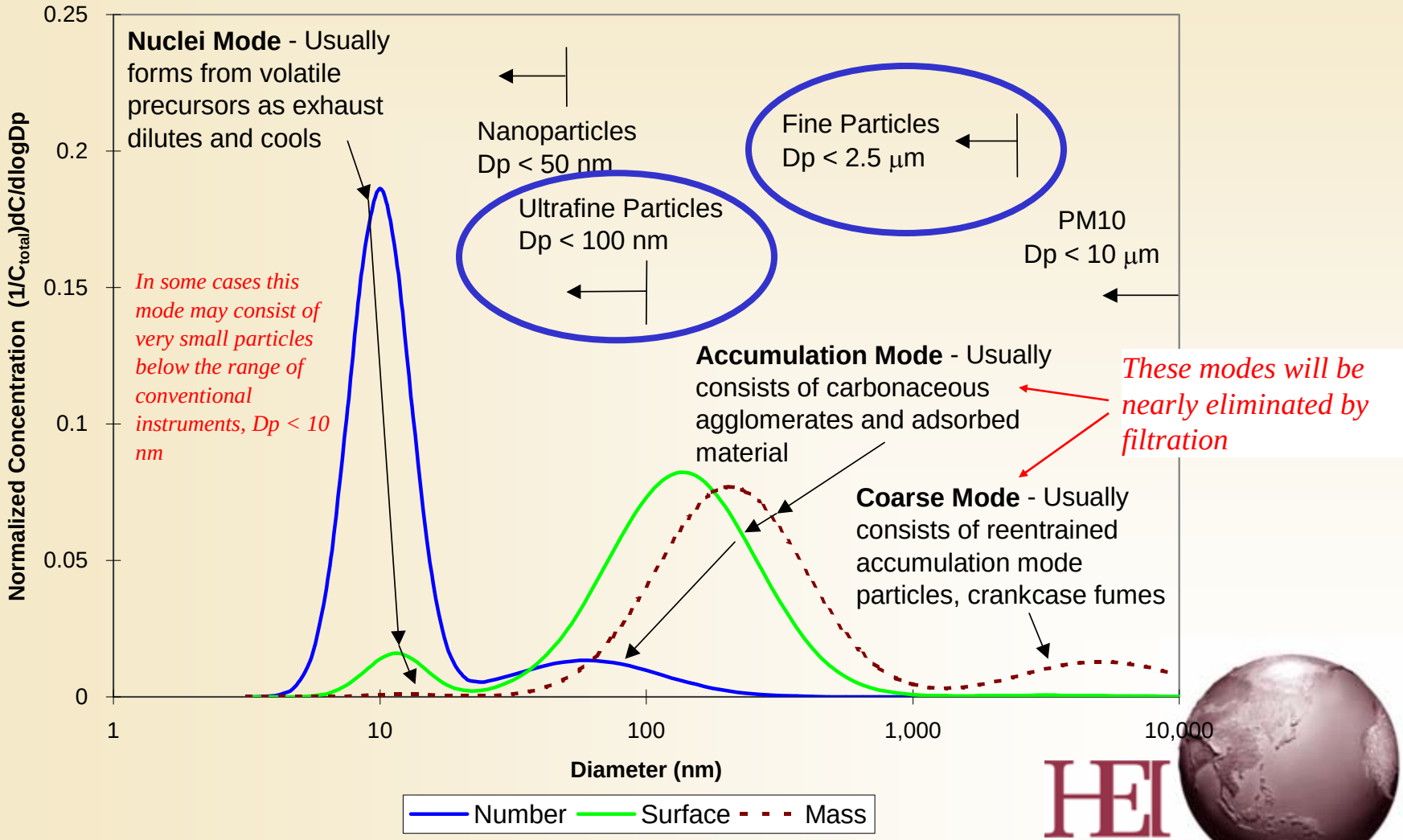


New HEI PAPA Studies in India Find Similar Results to Global Science

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

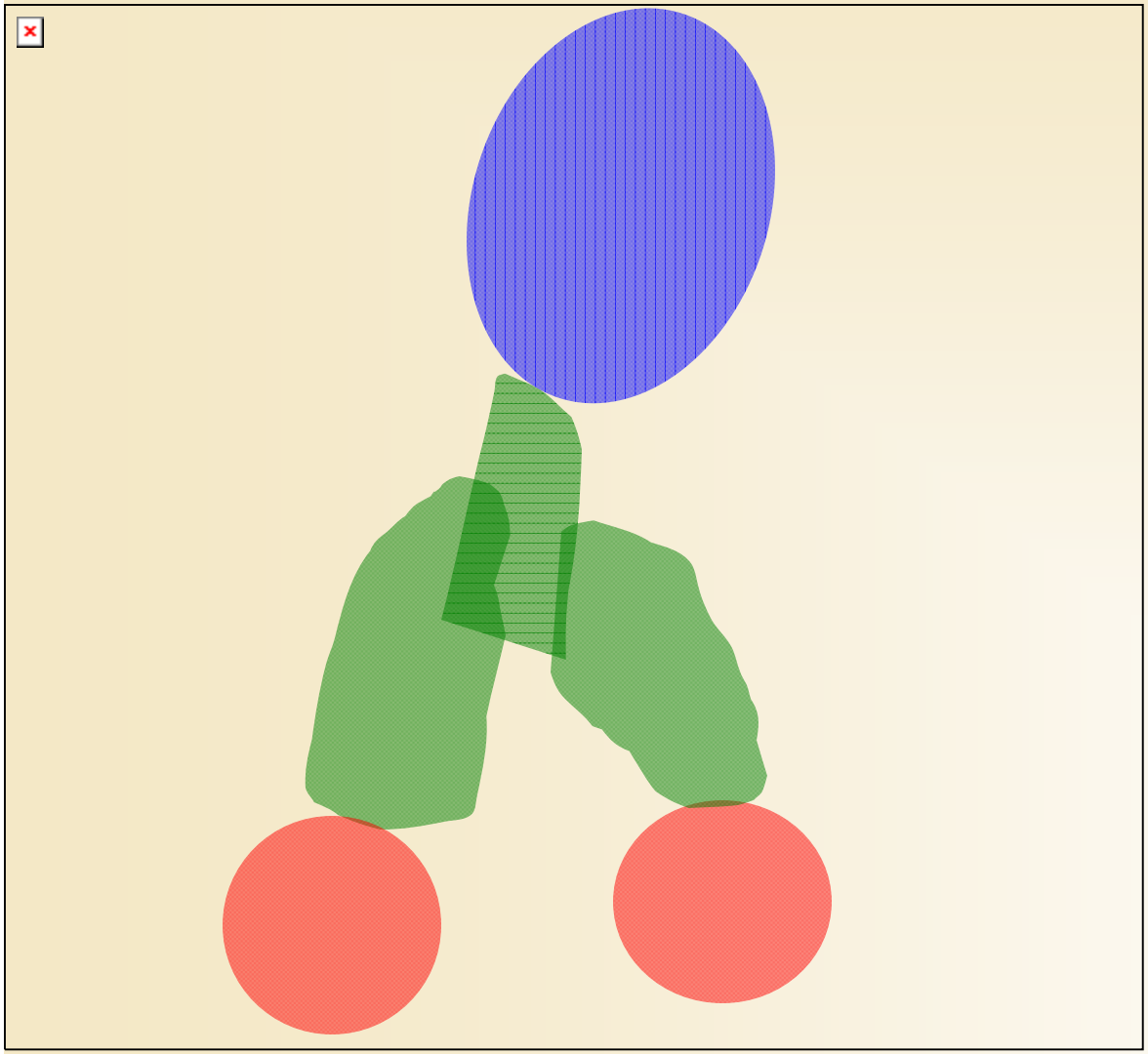
VEHICLE CONTRIBUTIONS ARE LARGELY ULTRAFINE PM (<100 nanometers)

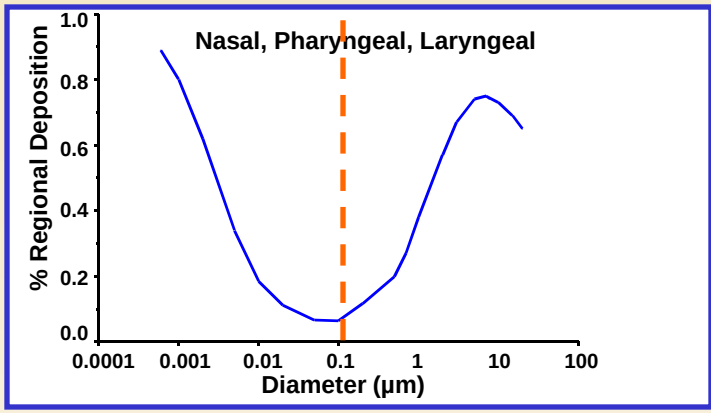
Typical Diesel Particle Size Distributions, Number, Surface Area, and Mass Weightings

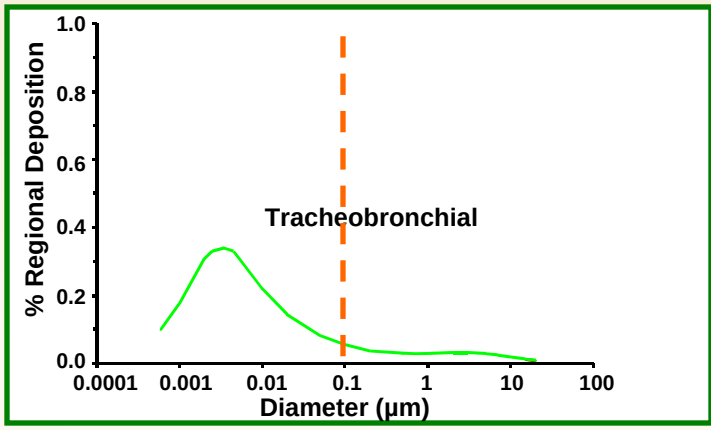


Fractional Deposition of Inhaled Particles in the Human Respiratory Tract

(ICRP Model, 1994; Nose-breathing)







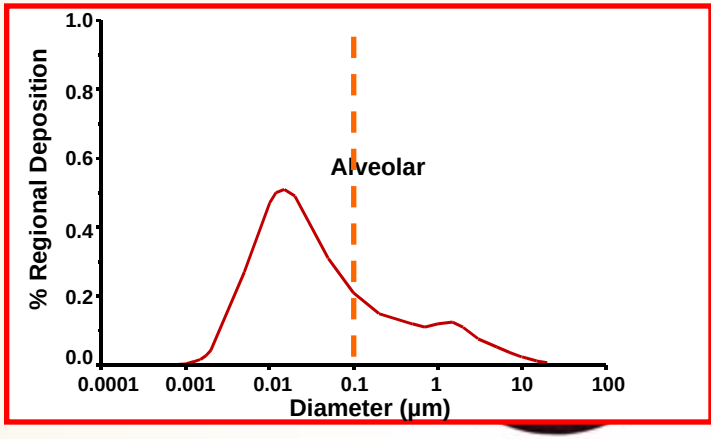


Figure courtesy of J.Harkema

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]

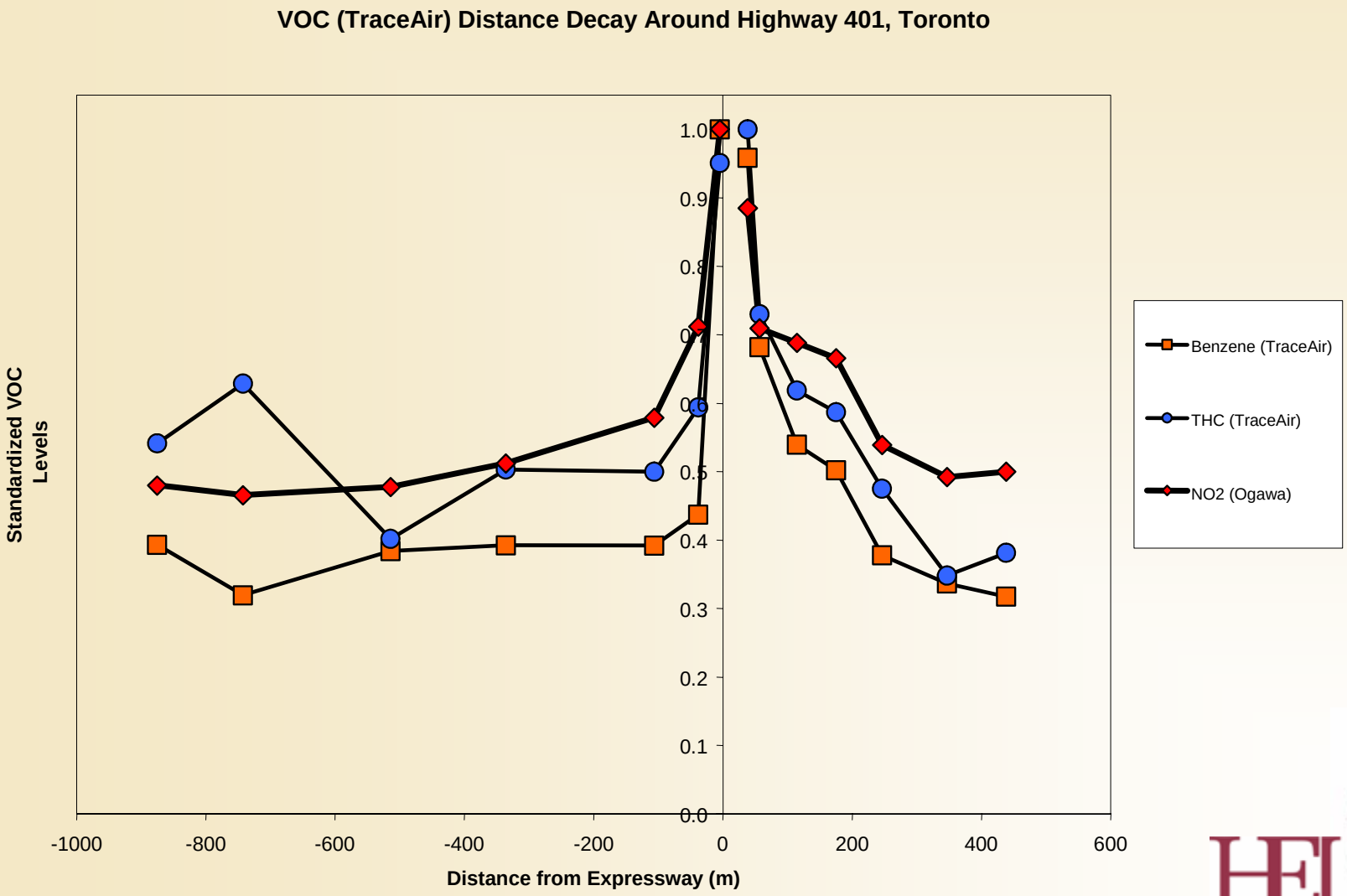
Reference	Health Endpoint	Subjects	Exposure Conditions (concentration, time)	Findings
Traffic Mixture				
(Bräuner et al. 2007)	DNA damage and oxidative stress	29 healthy subjects (20–40 yr)	Particle filtered air (91–542 particles/cm³) or unfiltered air delivered from a busy roadway in Copenhagen (6169–15,362 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 $PM_{10-2.5}$ 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?

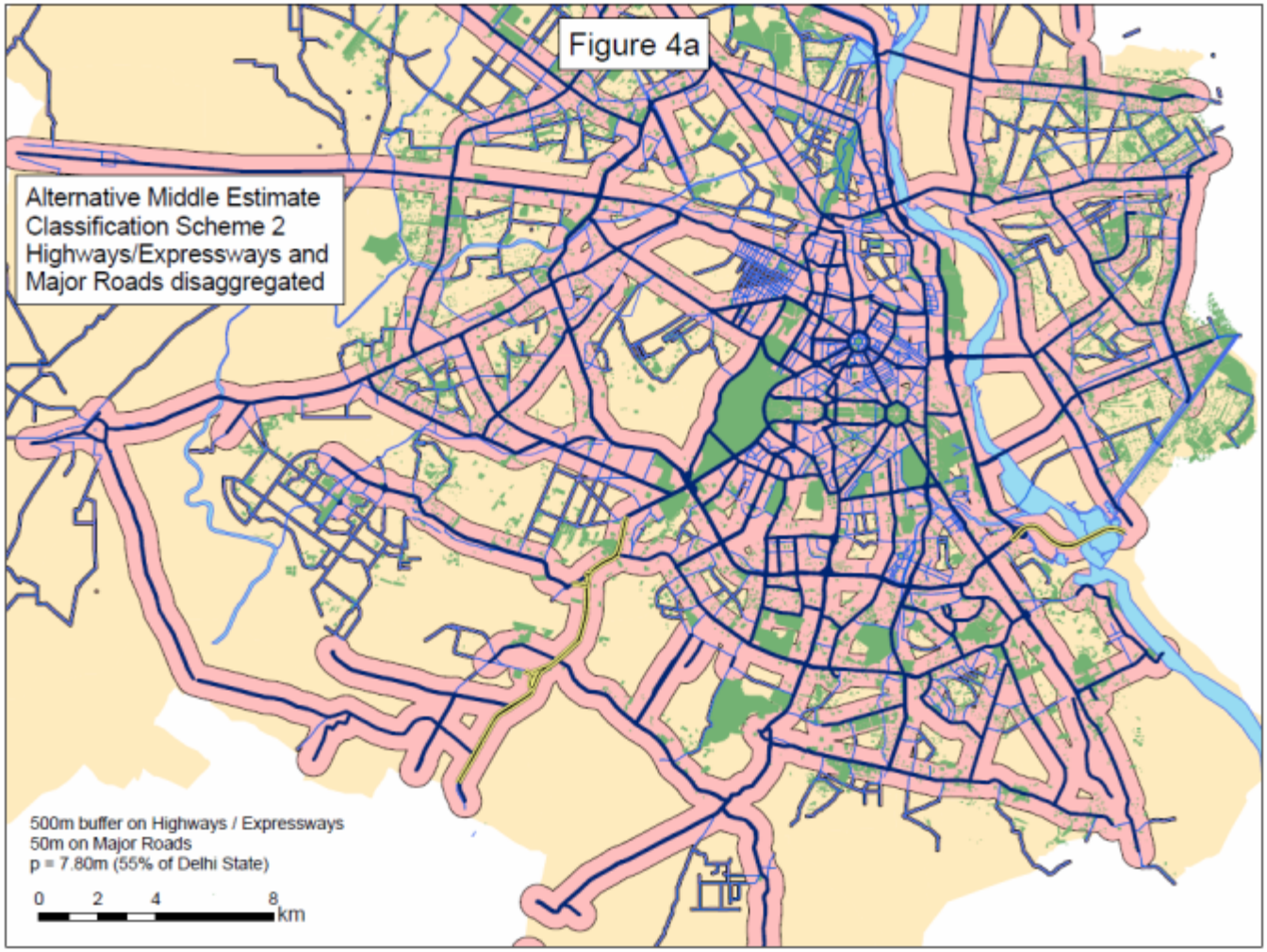
Highest levels within 300 – 500 meters of a major road



Toronto, Beckerman et al. (2008)

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.

Thank You

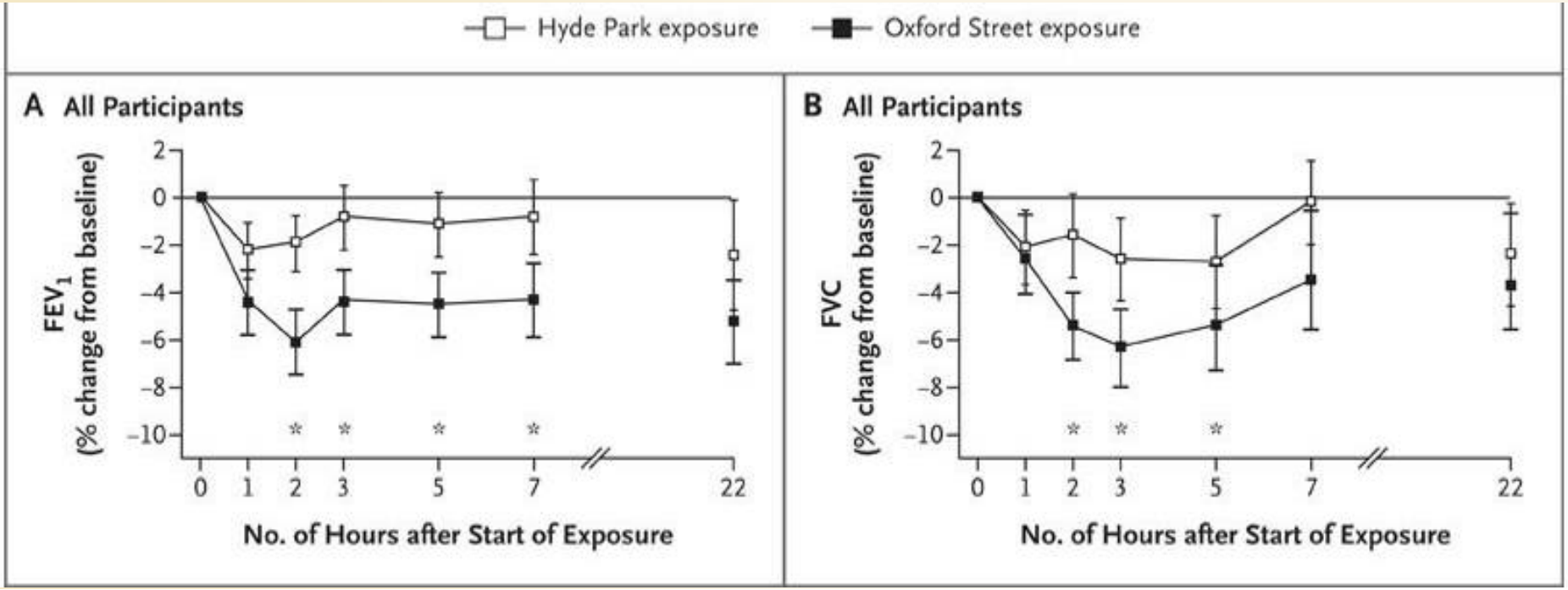
Dan Greenbaum
dgreenbaum@healtheffects.org

What Can We Learn from Toxicology?

(Example from a somewhat limited database):

Effects of Traffic Exposure on Asthmatics
(Zhang HEI 2009)

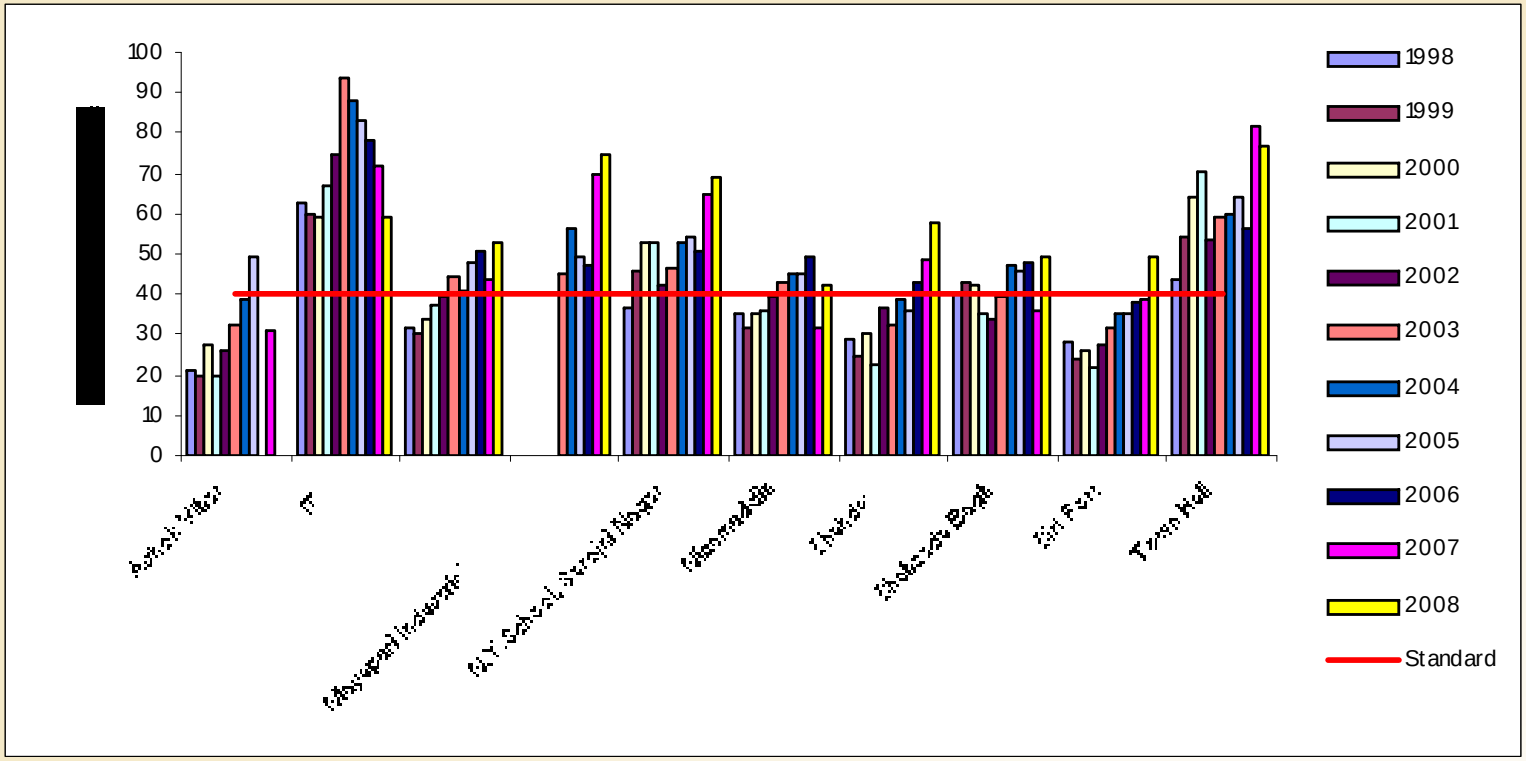
Lung function decline in asthmatics comparing Hyde Park and Oxford Street, London
(*although symptoms did not increase…)*



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.

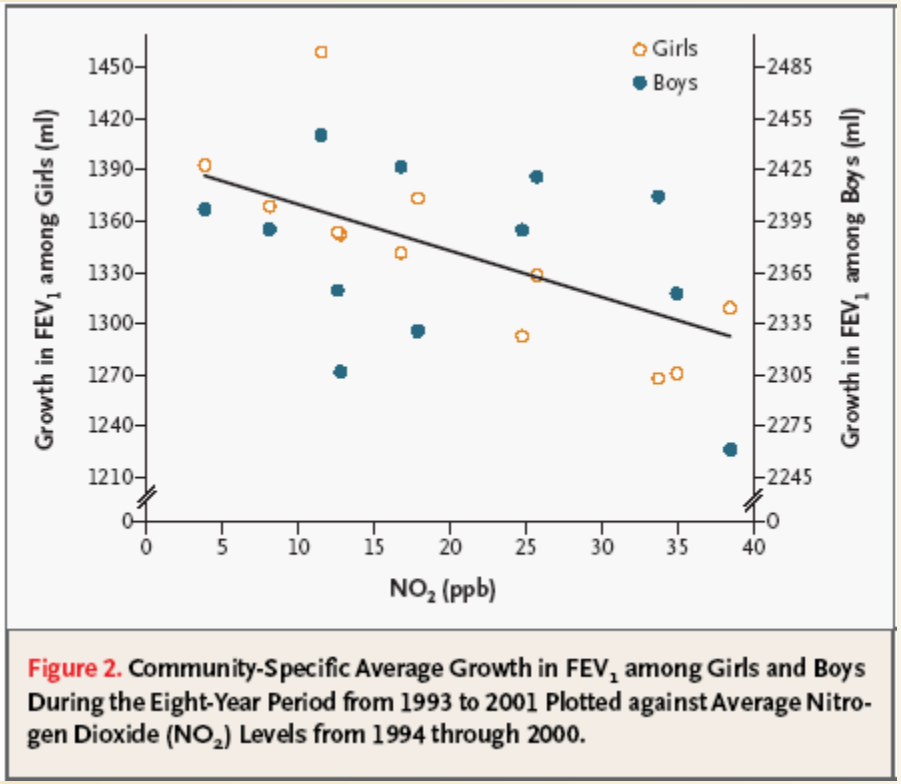


Figure 2. Community-Specific Average Growth in FEV_1 among Girls and Boys During the Eight-Year Period from 1993 to 2001 Plotted against Average Nitrogen Dioxide (NO_2) Levels from 1994 through 2000.

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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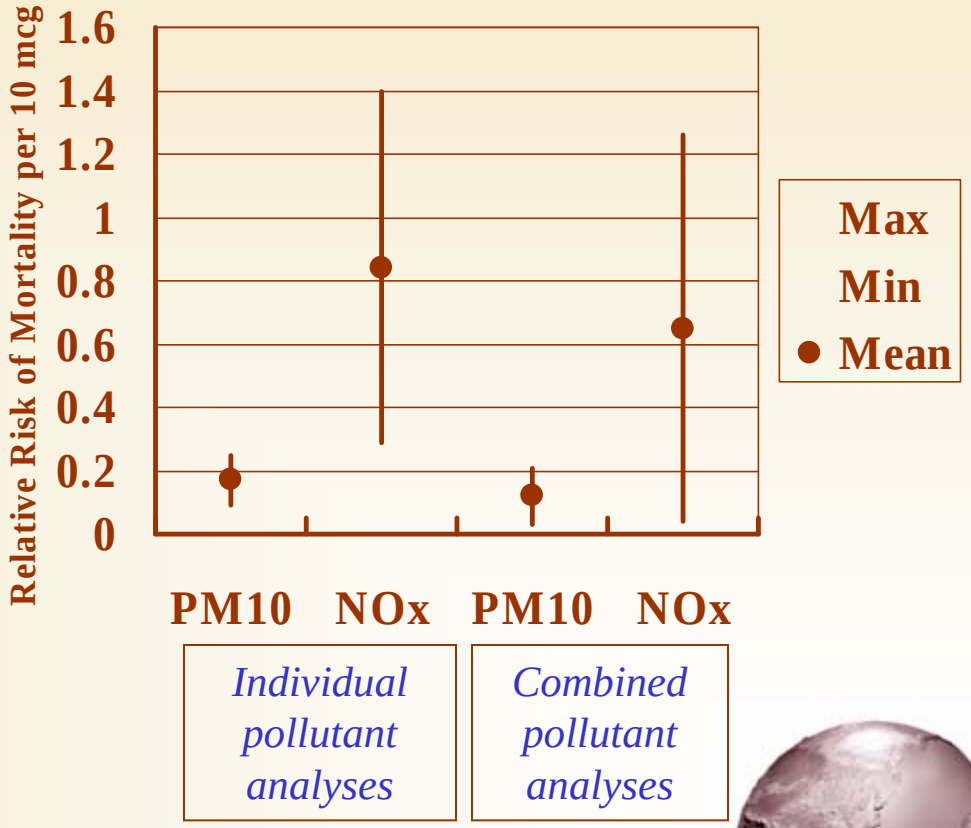
New NOx Results from India:
HEI Study in Delhi

- 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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