Traffic- Related Air Pollution:
A Critical Review of the Literature on Emissions, Exposure, and Health Effects

Clean Air Act Advisory Committee
Crystal City, VA
October 6 – 7, 2009

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Goals of the Review

Summarize and synthesize relevant information on air pollution from traffic and its health effects, linking:

- Emissions *and* exposure to traffic air pollution
- Exposure to traffic air pollution *and* health effects
- Toxicological data *and* epidemiologic associations

A preprint of the report was released in May 2009

The final Report, following extensive QA/QC, will be published in fall 2009

HEI Traffic Review Panel

Ira Tager—UC Berkeley, *Chair*
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Frederick Lurmann—Sonoma Tech. Inc
Sylvia Richardson—Imperial College
Jon Samet—Johns Hopkins
Michael Walsh—Consultant

Emissions from Motor Vehicles
The Current Context

Significant progress has been made in reduction of pollutant emissions from motor vehicles despite increases in number of vehicles and vehicle miles traveled

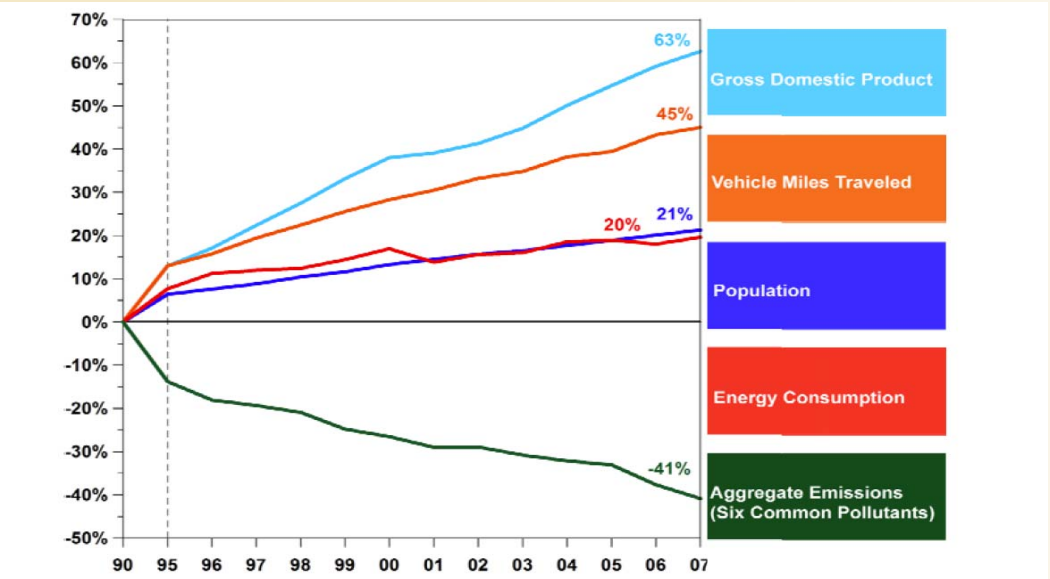


Figure 1.1. Comparison of growth measures and aggregate emissions from all sources, 1990–2007. Adapted from U.S. Environmental Protection Agency, 2008.

Increased urbanization and urban populations have:

- Increased dependence on motor vehicles and traffic congestion
- Changed land use patterns such that *more people are near traffic sources of pollution*

Report Organization

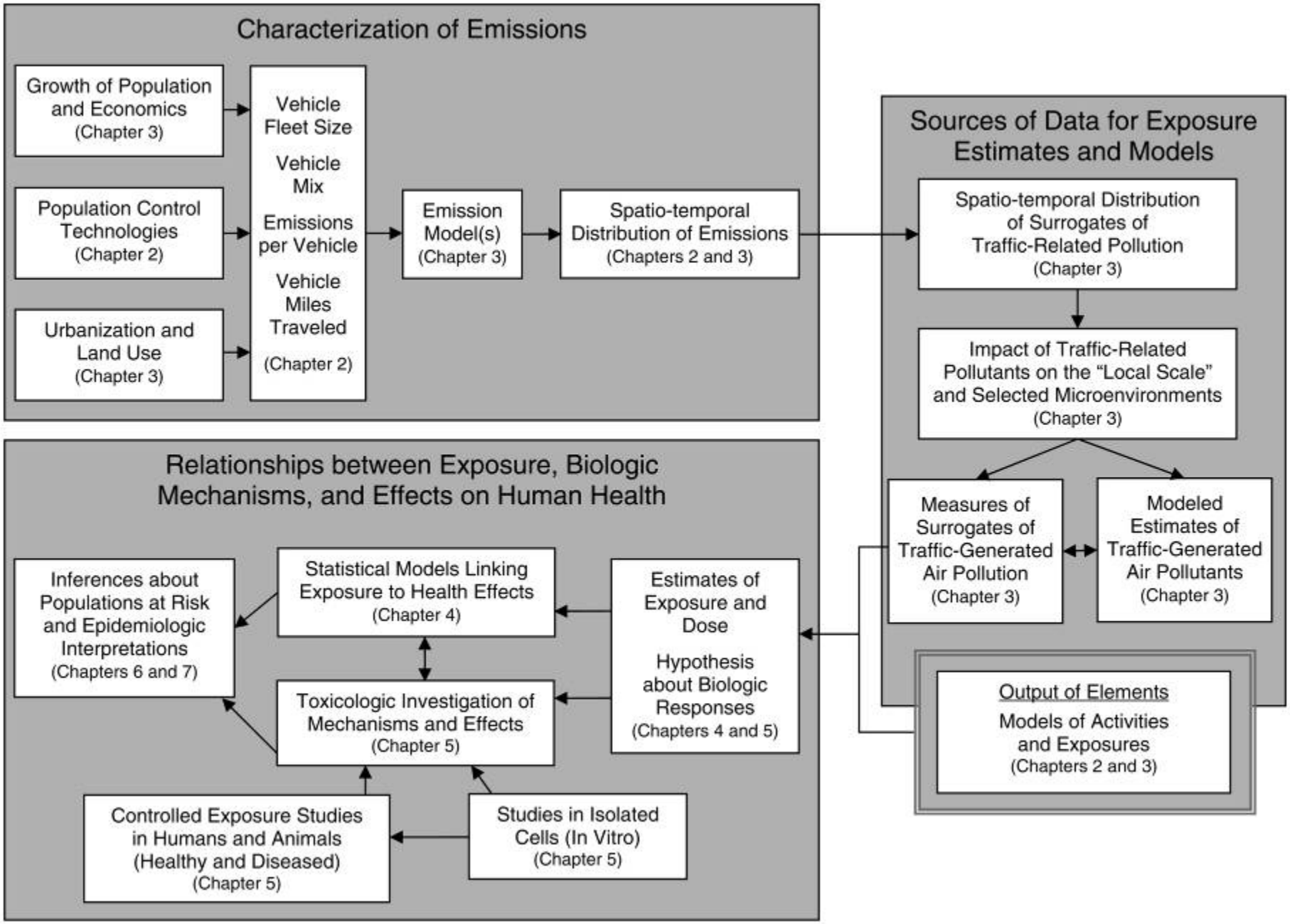


TABLE 5.2. SUMMARY OF HUMAN STUDIES DISCUSSED[2]

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 $PM_{0.1-2.5}$ 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 400) that have attempted to look at traffic exposure and effects

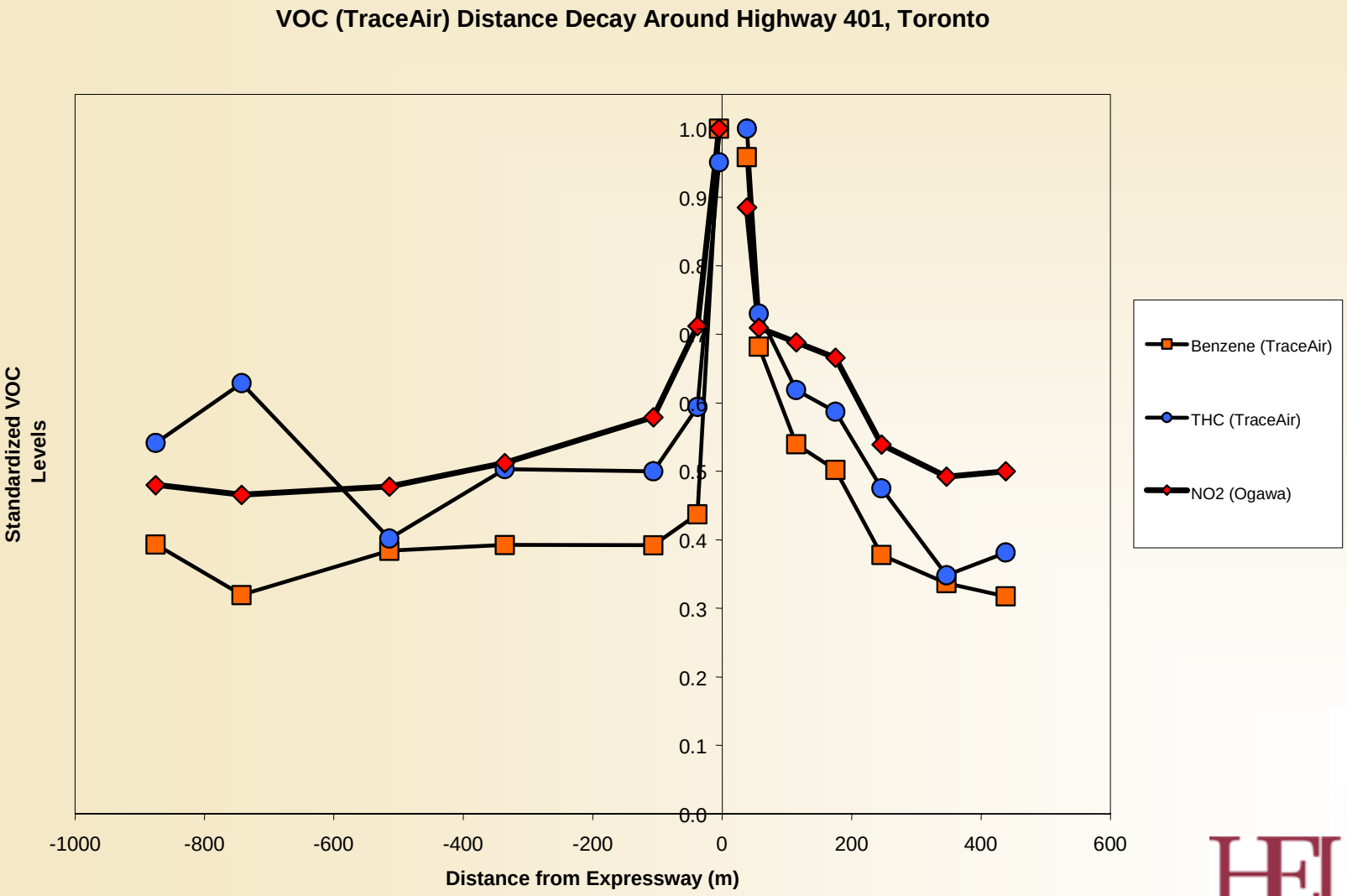
- *However, they are not **all** of equal quality*

1. How should we assess Exposure?

- **Who is likely to be exposed?**
- **What exposure assessment methods used in epidemiologic studies?**
  - Pollutant surrogates for traffic exposures (e.g., NO_2, EC/BS, CO, UFPM, benzene, etc.)
  - Distance from and/or length of roadways
  - Estimate of traffic density or intensity
  - Modeling of primary traffic-generated pollutant exposure

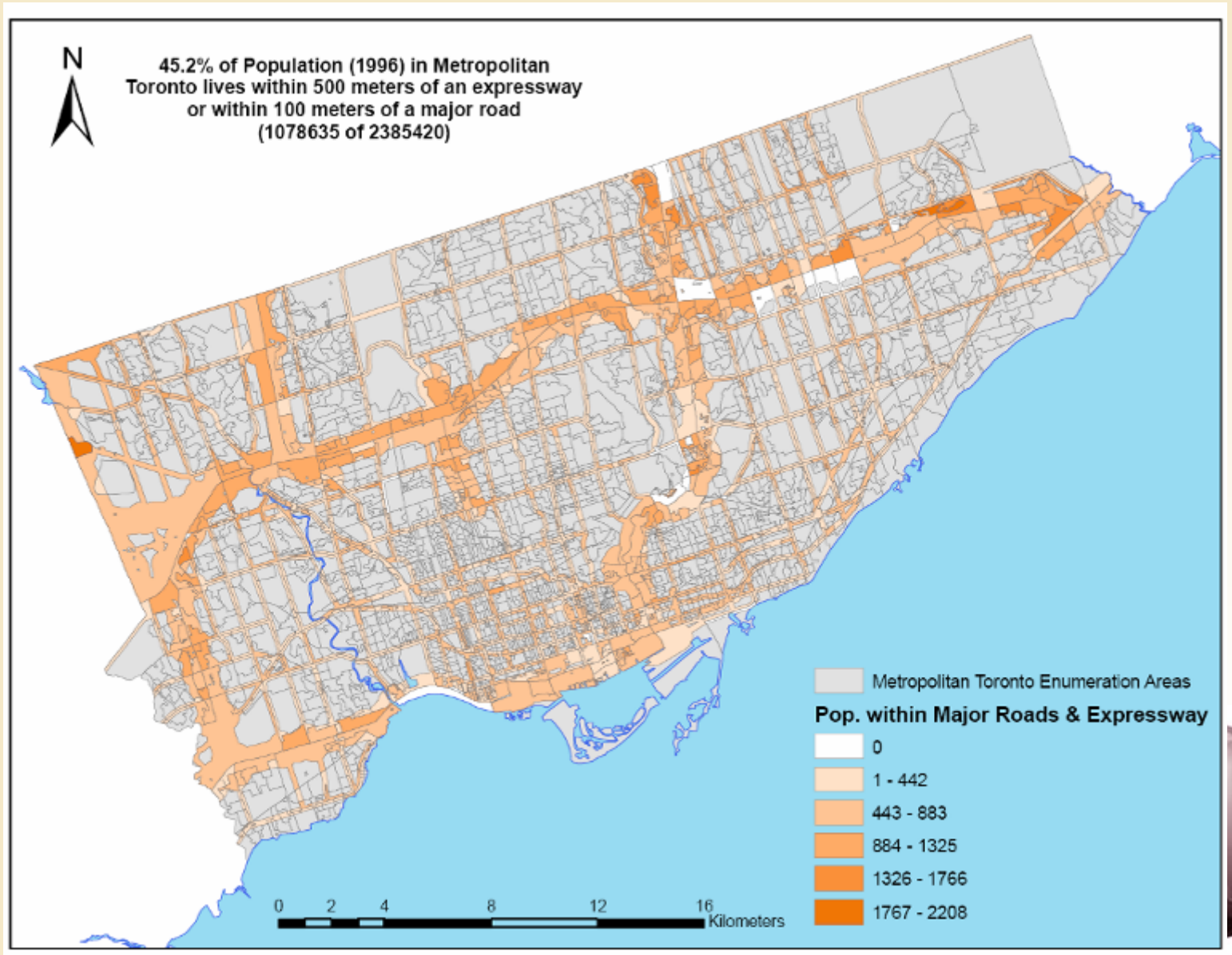
Who is Likely to be Exposed?

Highest levels within 300 – 500 meters of a major road

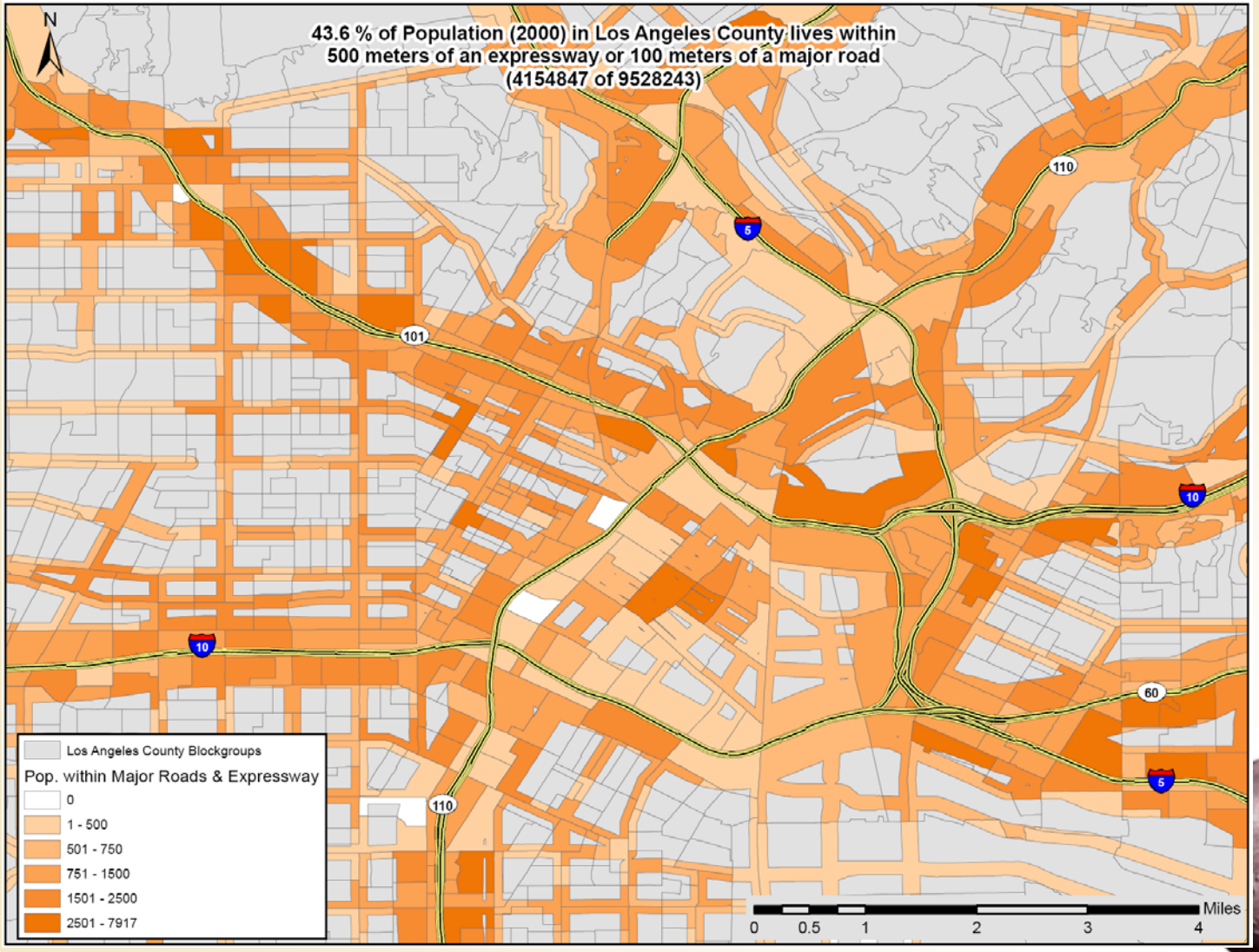


Toronto, Beckerman et al. (2008)

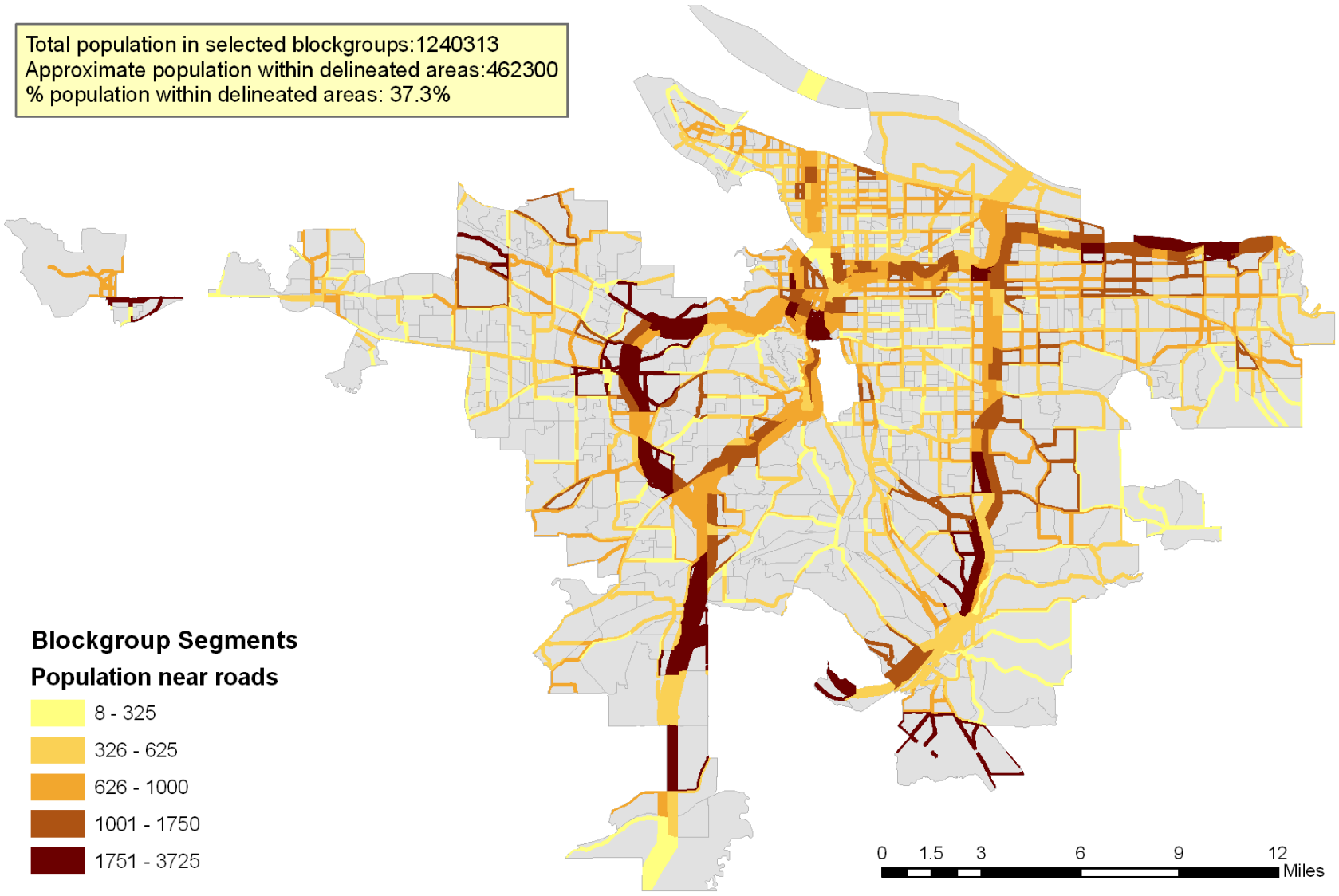
Near Roadway Exposure Can Include Large Populations
Toronto Example: ~45%
(within 500 meters of an expressway; 100 meters of a major road)



Los Angeles Example: (~44%)



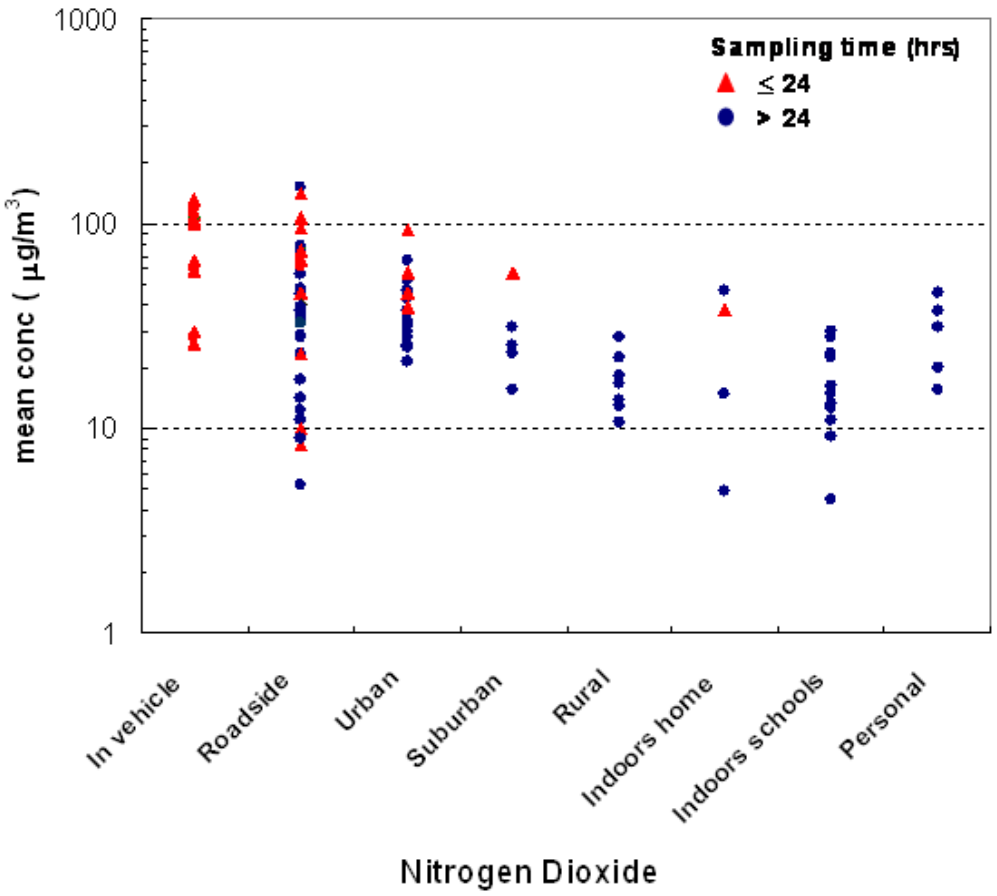
Portland, Oregon ~37%



What Markers or Surrogates?

- Pollutant surrogates for traffic exposures (e.g., NO_2, EC/BS, CO, UFPM, benzene, etc.)
- Criteria for what is a good surrogate:
  1. Traffic as the major source
  2. Emissions vary with other motor vehicle constituents
  3. Can be measured at low concentrations by reasonably inexpensive and accurate methods
  4. Not have independent health effects

NO_2 as a surrogate



- There is substantial variability in average concentrations by locations.

- NO2 is a potential surrogate for vehicle emissions if it is measured on a fine spatial resolution.

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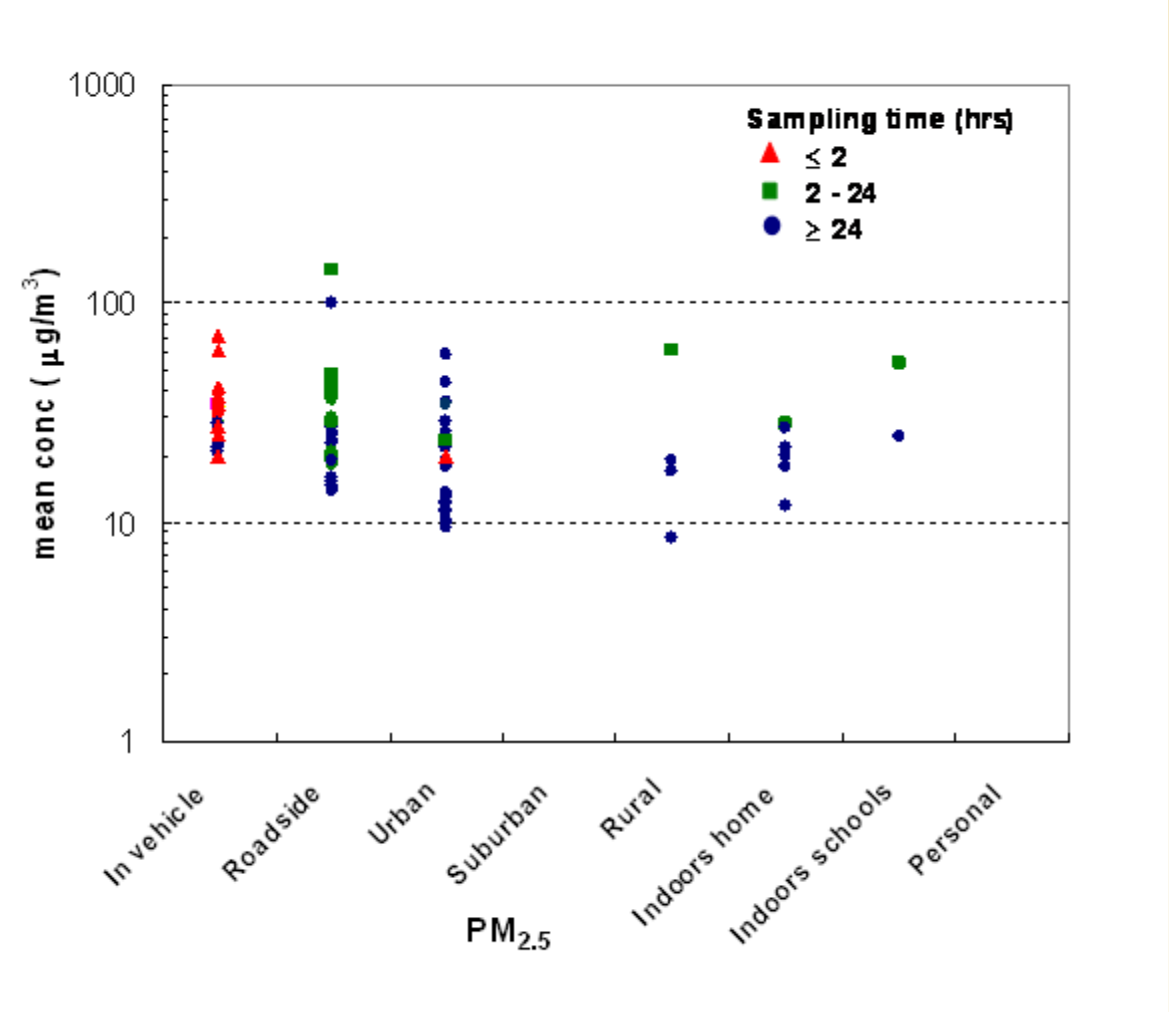
$PM_{2.5}$ as a Surrogate



- Use of $PM_{2.5}$ as a surrogate is of limited value because many sources contribute to urban $PM_{2.5}$ and $PM_{2.5}$ concentrations are well mixed within a region

- Current central monitors do not provide sufficient spatial resolution for assessing the contribution of traffic to ambient $PM_{2.5}$

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What Markers or Surrogates?

- Pollutant surrogates for traffic exposures (e.g., NO_2, EC/BS, CO, UFPM, benzene, etc.)
- Criteria for what is a good surrogate:
  1. Traffic as the major source
  2. Emissions vary with other motor vehicle constituents
  3. Can be measured at low concentrations by reasonably inexpensive and accurate methods
  4. Not have independent health effects
- ***Can provide useful information but none meet all these criteria…***

Can We Use Exposure Models?

Models used

- ~~Proximity models~~
- Geostatistical interpolation models
- Dispersion models
- Land-use regression models
- Hybrid models
  - Combine a model with time-activity data, or personal/microenvironmental monitoring

- Proximity models are least effective:
  - Can be confounded by Socioeconomic Status, Noise, other factors
- Newer models of exposure are better
  - But should be validated against some real-world data.

Criteria for Inclusion of Toxicology and Epidemiology Studies

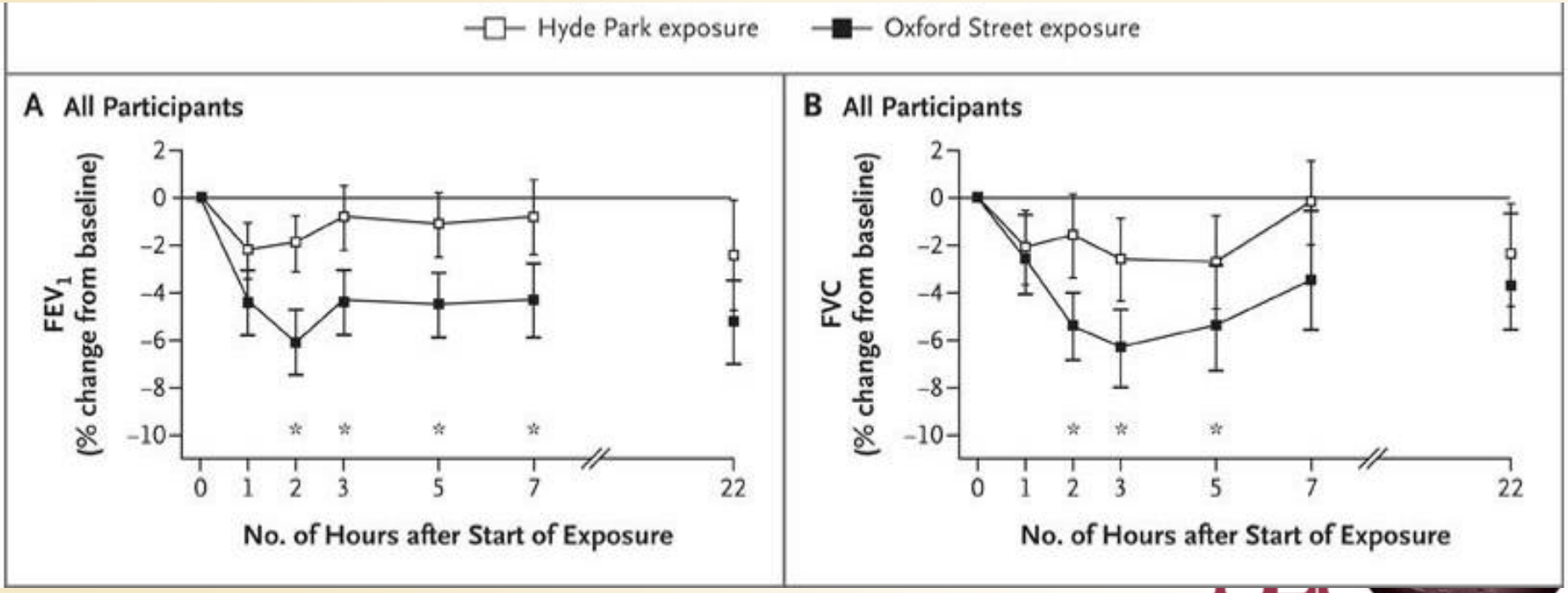
- ***Quality of exposure assessment was key…***
- ***Studies had to include 1 or more of the following exposure methods:***
  - Distance from and/or length of roadways
  - Estimate of traffic density or intensity
  - Modeling of primary traffic-generated pollutant exposure
  - Studies of occupations characterized by exposure to traffic
  - Pollutant surrogates for traffic exposures (e.g., NO_2, EC/BS, CO, benzene, etc.) only if data provided to validate the pollutant as a reasonably specific surrogate for such exposure

2. 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…)*



3. What can we learn from epidemiology?
Criteria for Causal Inference

Four categories to test whether traffic causes effects, based on:

- ***how well studies controlled for confounding***
- ***consistency of the findings with other studies***
- ***quality of the method to estimate exposure***

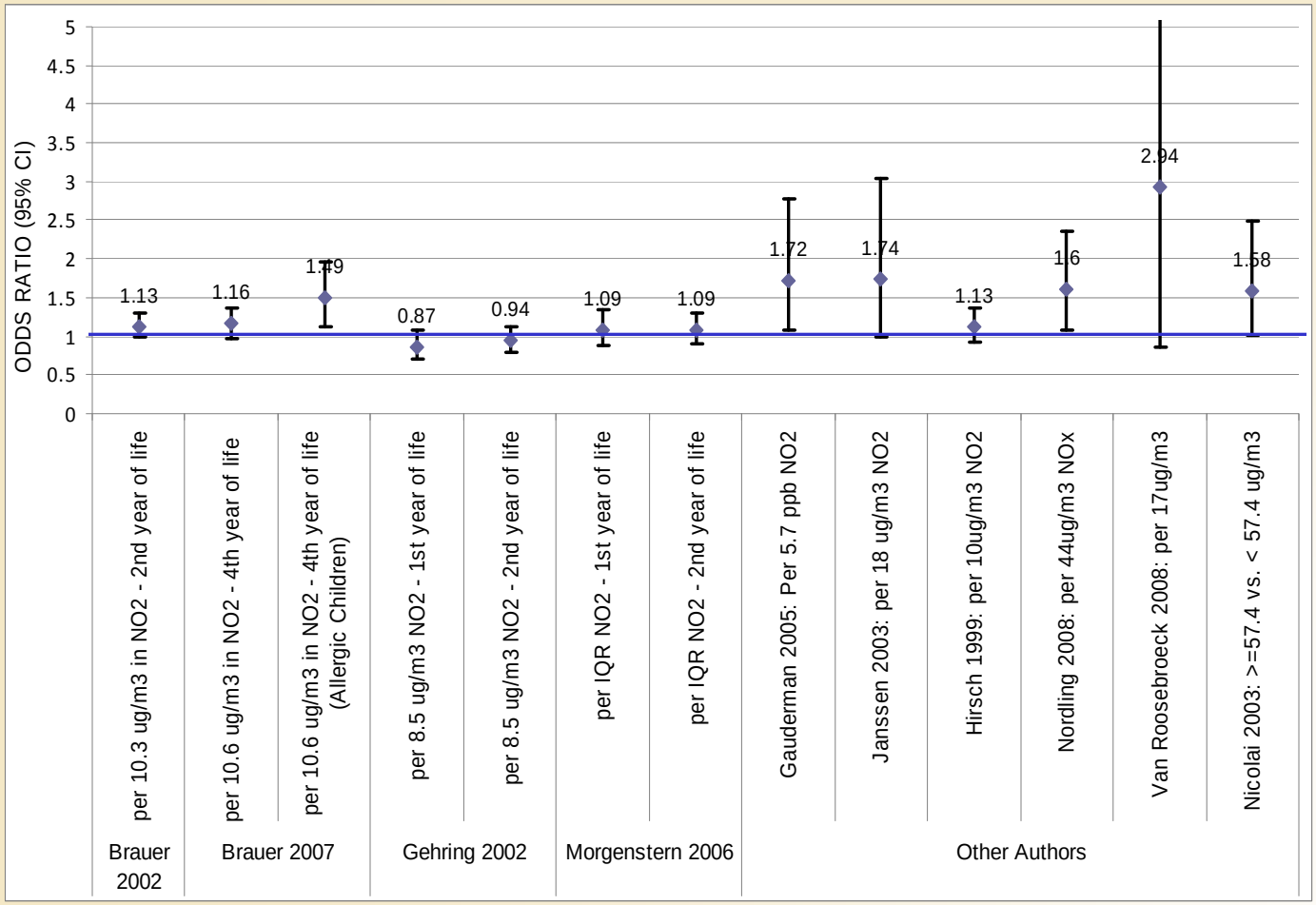
- ***Sufficient*** evidence
- ***Suggestive*** but not sufficient
- ***Inadequate and insufficient*** evidence
- Suggestive of ***no association***

Epidemiology
Health Outcomes Evaluated

- Mortality (all cause, cardiopulmonary)
- Cardiovascular morbidity
- Respiratory disease
  - Asthma—childhood/adult
  - General respiratory symptoms
  - Lung function-childhood/adult/COPD
  - Health care utilization
- Non-asthmatic allergy
- Birth Outcomes
- Cancer

Exacerbation of Asthma Symptoms

Increase in Wheeze Per Increment NO2



Synthesis of Evidence

Exacerbations <u>with</u> asthma—***Sufficient*** for causal association

Reasons

Large number of studies with adequate control for confounding and mostly precise effect estimates

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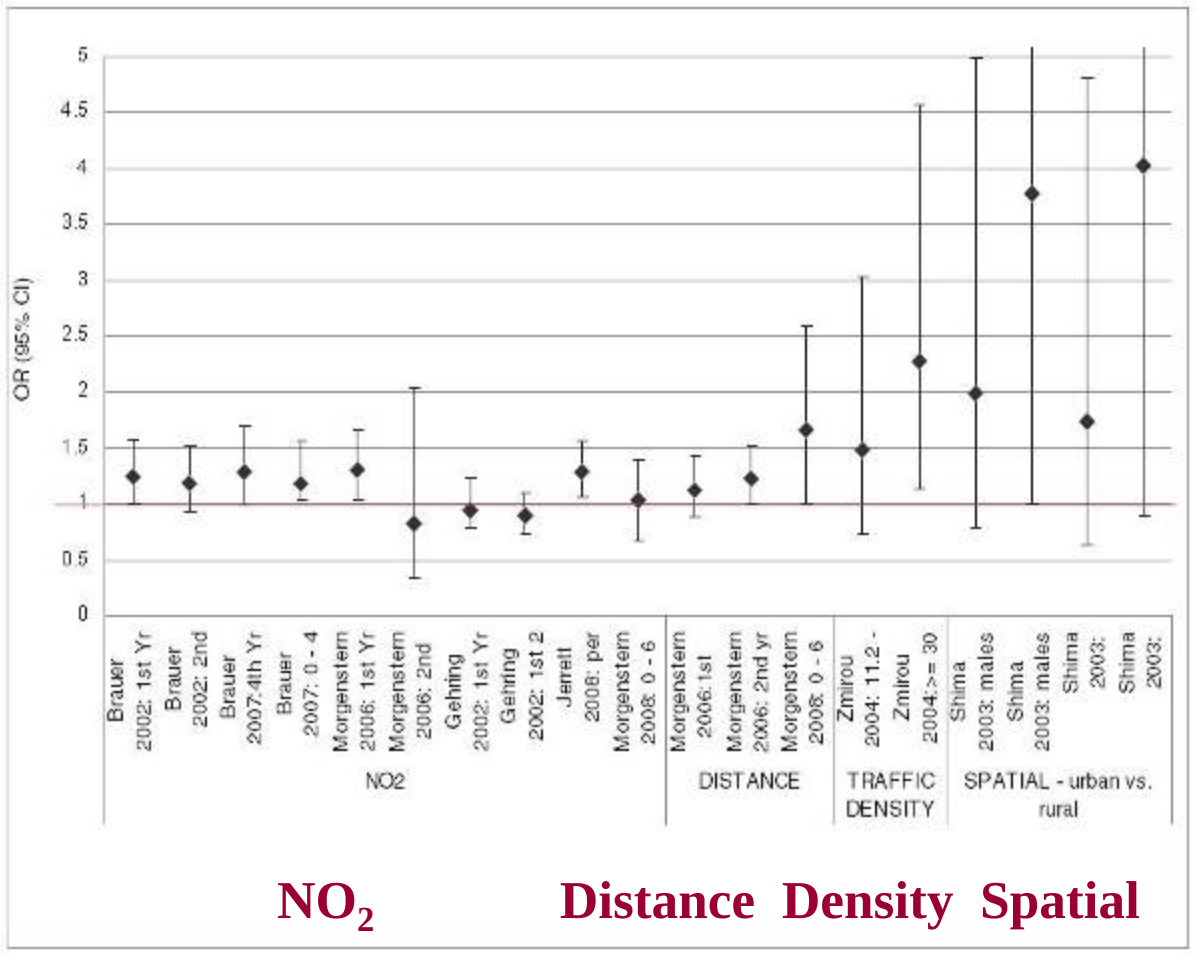
Traffic Exposure and Doctor-Diagnosed Asthma Incidence in Children



NO_2 **Distance** **Density** **Spatial**

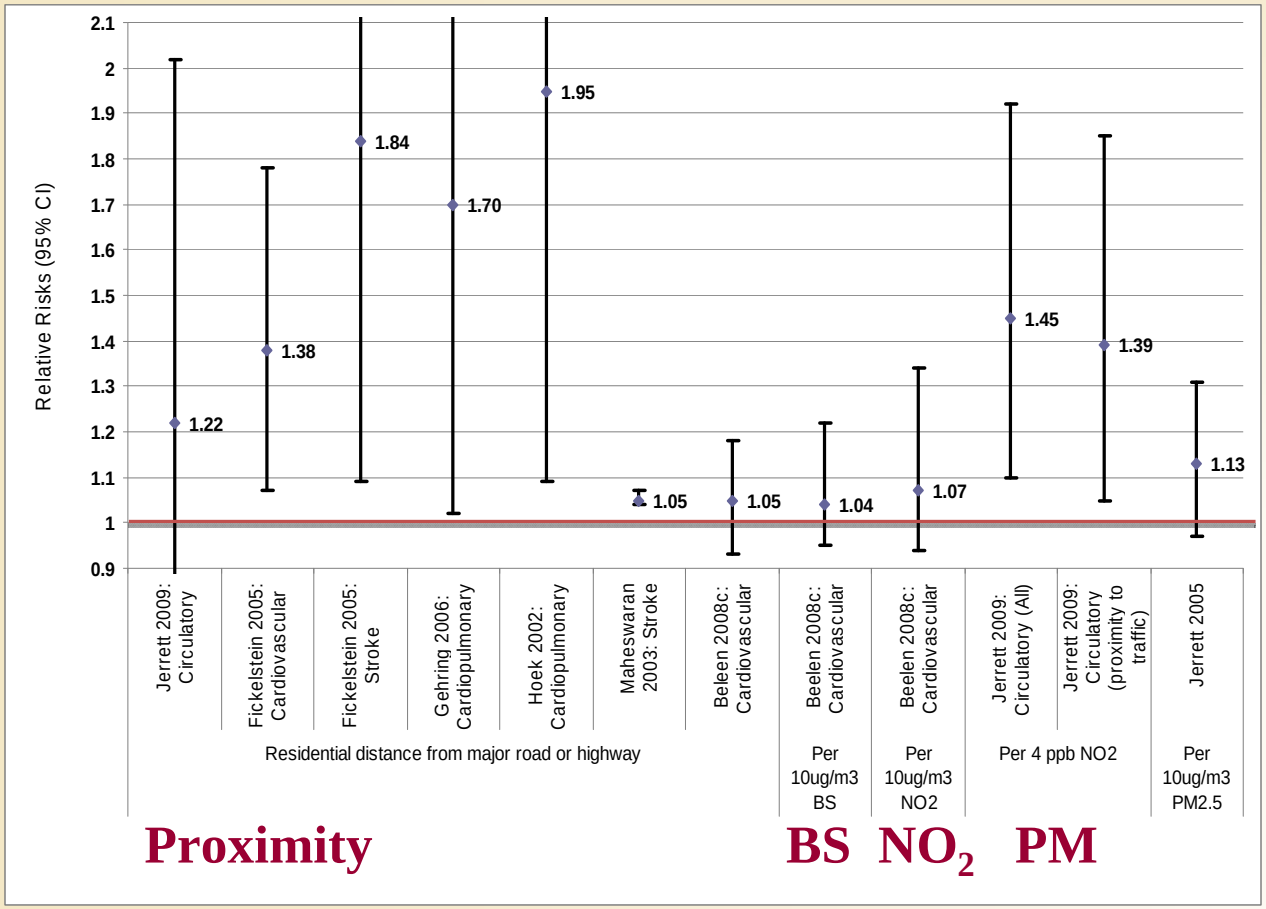
Synthesis of Evidence

Sufficient OR ***suggestive*** evidence

Reasons

Studies that included both traffic-specific pollutants and density measures most consistent

Long-Term Traffic Exposure and Cardiopulmonary Mortality



Synthesis of Evidence

Suggestive to infer causal association but not yet sufficient

Reasons

Too few studies

Relative imprecision of most estimates

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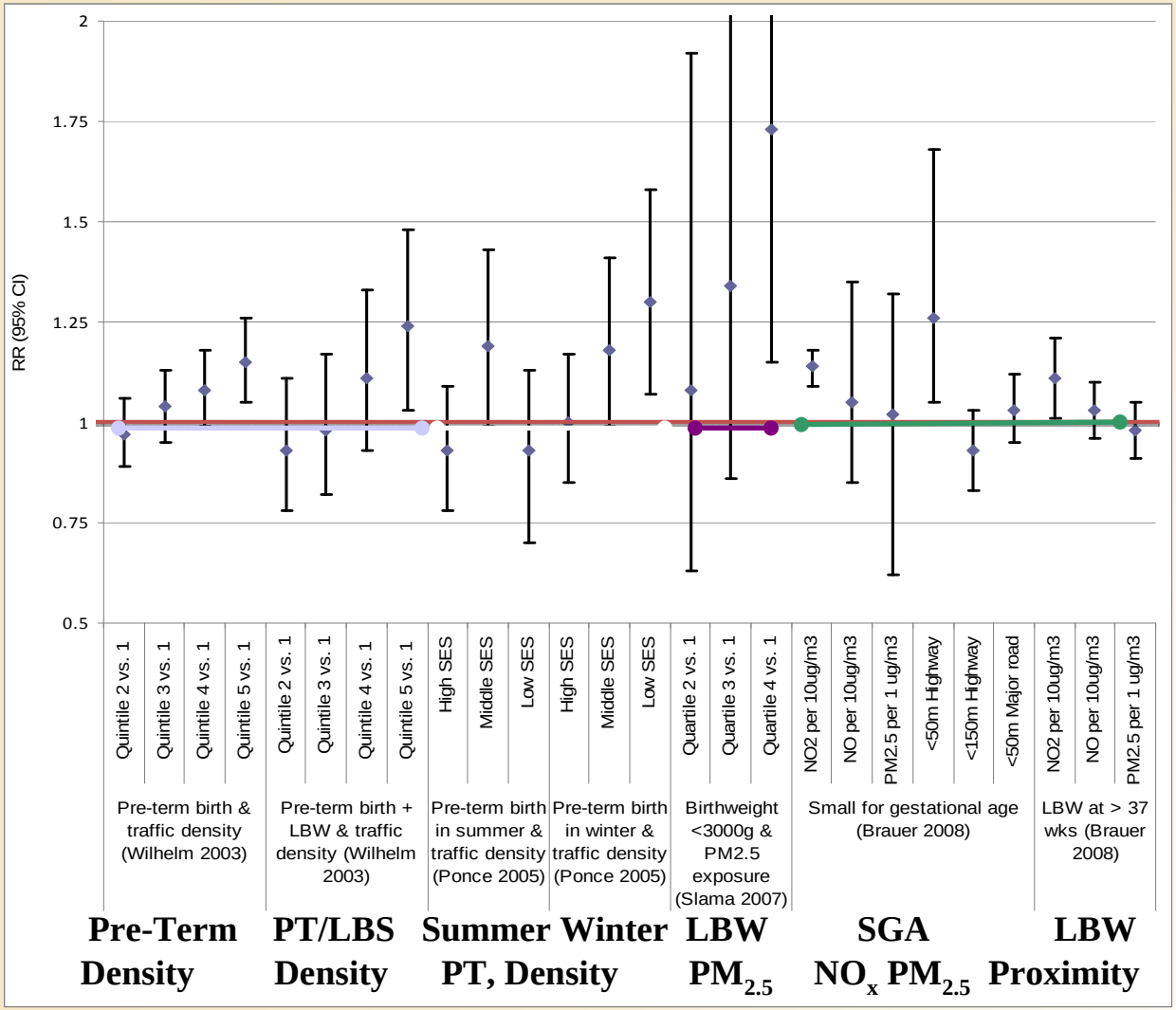
Effects of Traffic Exposure on Birth Outcomes



- **Synthesis of Evidence**
  - ***Insufficient evidence***
- **Reasons**
  - **Only 4 studies met criteria for inclusions**

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Conclusions

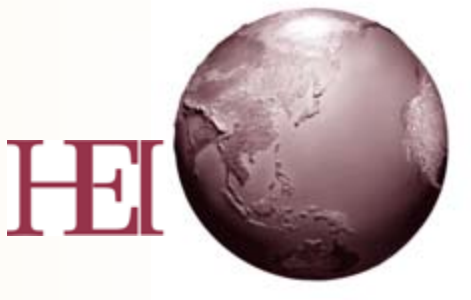
Exposure

- Traffic-related pollutants impact ambient air quality on a broad spatial scale ranging from roadside, to urban, to regional background

- Based on synthesis of evidence, 300 to 500 meters from major road was identified as the near-source area most impacted by traffic;
  - variations exist depending on meteorology, background pollution, and local factors

Issues for Exposure Assessment

- None of the pollutant surrogates considered met all criteria for an ideal surrogate
  - CO, benzene, and NOx [NO_2] found in on-road vehicle emissions are *also major components of emissions from all sources*
  - UF PM has not been used in epidemiologic studies so far. It is difficult to model them because there are no emission inventories
- Exposure models are important, but have various degrees of utility to health studies
  - The proximity model is the most error-prone
  - Other models are better:
    - Dispersion models (need adequate data)
    - Land use regression models
    - Several approaches together (hybrid)



Overall Conclusions

- The data are incomplete on emissions, their transformations, and exposure assessment
- There were, however, enough studies to find
  - ***Sufficient*** evidence for a causal association with exacerbation of asthma
  - ***Suggestive*** evidence for a number of other health effects (mortality, lung function, respiratory symptoms, and others)

Overall Conclusions II

- Limited evidence of effects but *inadequate and insufficient* to infer causal associations:
  - Adult onset asthma
  - Health care utilization
  - COPD
  - Non-asthmatic allergy
  - Birth outcomes
  - Cancers

Overall Conclusions III

- *A caution*: 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!

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