

# Health risks of exposure to traffic air pollution and noise

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# Plan

- Health risks from exposure to road traffic **air** emissions
- Health risks from **noise** associated with road traffic
- Is the effect of road noise due to traffic emissions of air pollutants (**confounding**)?
- Health **impacts** of traffic related air and noise emissions

# Health risks from exposure to road traffic **air** emissions (from HEI 2010)

- Traffic-related exposure:
  - Exposure to primary emissions from motor vehicles
  - Motor vehicles emit : CO<sub>2</sub>, CO, HC (including benzene, formaldehyde, acetaldehyde, 1,3-butadiene), NO<sub>x</sub>, PM, and other substances known as mobile source air toxics , such as, and lead where leaded gasoline is still in use.
  - Each of these, along with secondary by-products, such as ozone and secondary aerosols (e.g., nitrates and inorganic and organic acids), can cause adverse effects on health and the environment.

# Health risks from exposure to road traffic **air** emissions (from HEI 2009)

Gradients in pollutants as a function of distance from busy roadways :

- Exposure zones for traffic-related air pollution in the range of 50 to 1500 m from highways and major roads, depending on the pollutant and the meteorologic conditions.
- Based on a synthesis of the best available evidence, the HEI Panel identified an exposure zone within a range of up to 300 to 500 m from a highway or a major road.
- The HEI panel estimated that 30% to 45% of people living in large North American cities live within such zones.

# Health risks from exposure to road traffic **air** emissions (from HEI 2009)

- Because it is not practical or feasible to measure all the components of the traffic-pollutant mix, in health studies, surrogates of traffic related pollution have been used as a reasonable compromise for estimating traffic exposure.
- Two broad categories of surrogates have been used in epidemiology studies to estimate traffic exposure:
  - (1) measured or modeled (geostatistical, LUR, dispersion, etc) concentrations of pollutant surrogates - main surrogates include CO, NO<sub>2</sub>, elemental carbon (EC; or black carbon [BC] or black smoke [BS]), PM, benzene, and ultrafine particles (UFP)

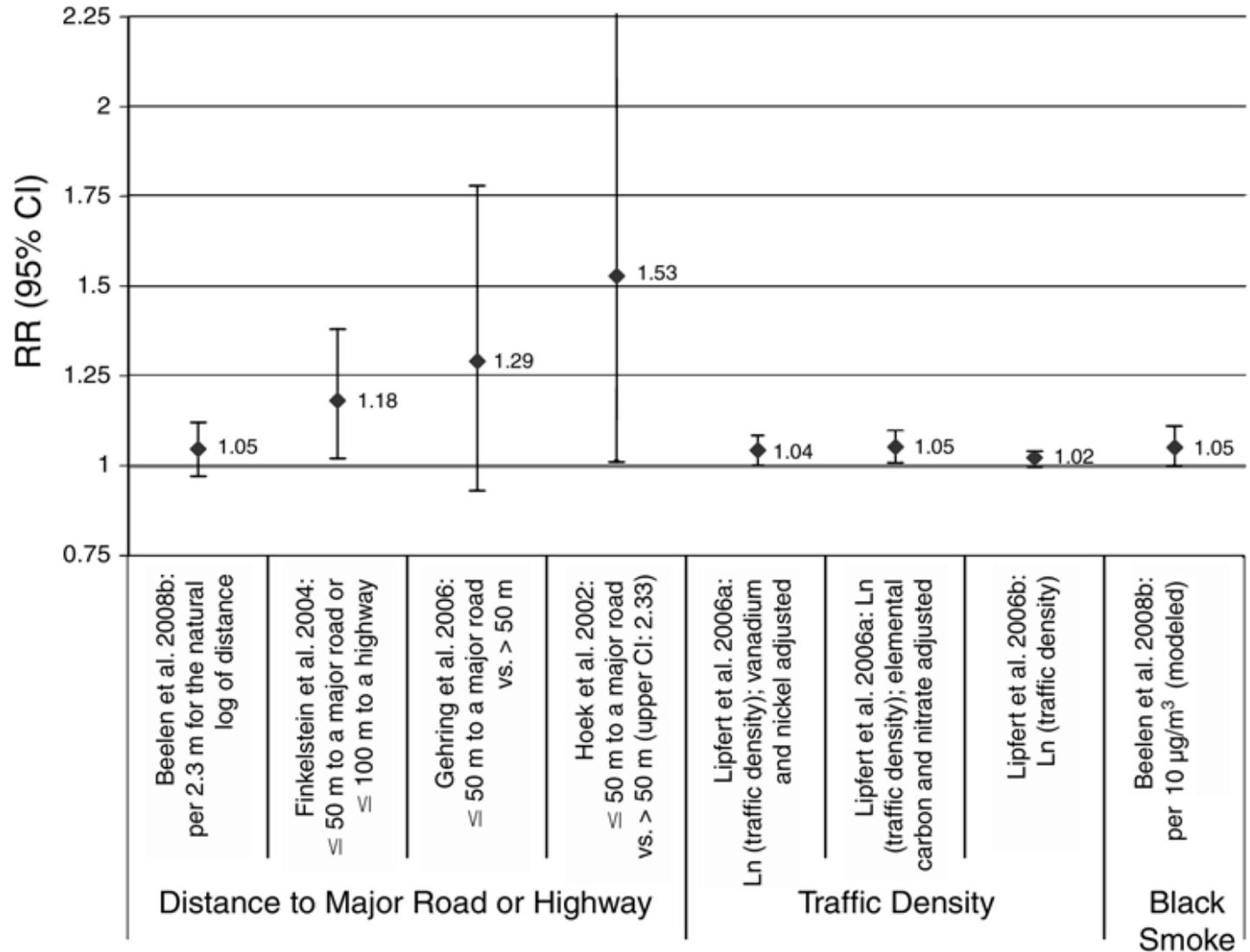
# Health risks from exposure to road traffic **air** emissions (from HEI 2009)

- Two broad categories of surrogates have been used in epidemiology studies to estimate traffic exposure:
  - (2) direct measures of traffic itself (such as proximity, or distance, of the residence to the nearest road and traffic volume within buffers)
- Proximity ignores the parameters that affect the dispersion and physicochemical activity of the pollutants. Moreover, estimates based on proximity can be confounded by factors such as noise.

# Health risks from exposure to road traffic **air** emissions (from HEI 2009)

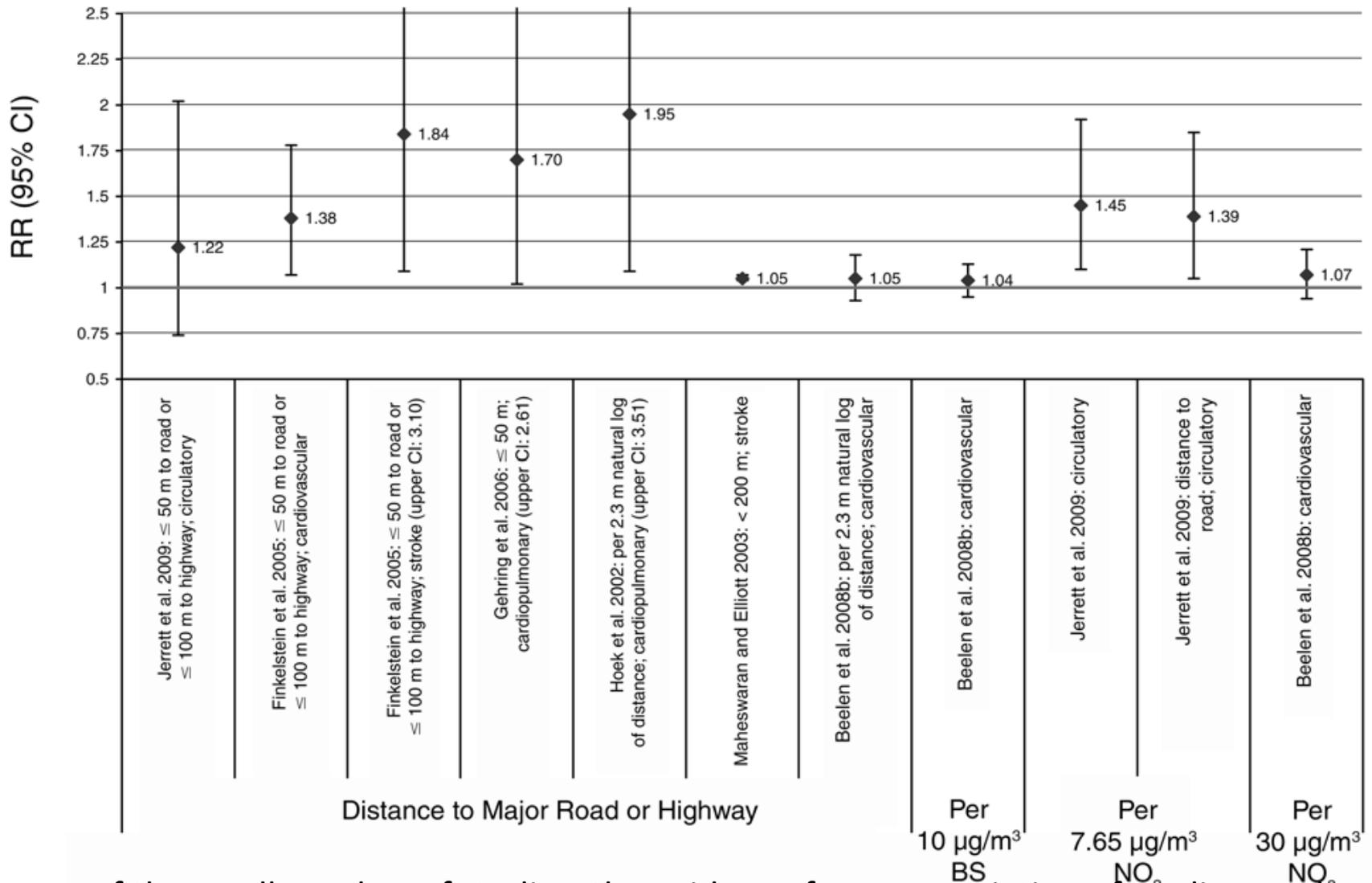
- The Panel included only studies that investigated associations between primary emissions from traffic and human health. Studies with measurements from a central monitoring site were not included unless the site was in proximity to traffic.
- In order to deem the evidence sufficient to conclude that association between a metric of traffic exposure and an outcome was causal, the Panel decided that it was necessary:
  - for the magnitude and direction of the effect estimates to be consistent across different populations and times
  - to rule out with reasonable confidence chance, bias in subject selection, and confounding (in particular, socioeconomic status).
  - a traffic-specific coherence criterion to account for the degree of validity of the traffic-specific exposure metrics was also added.

Figure 4.1. Studies of long-term exposure to traffic pollution and all-cause mortality \*



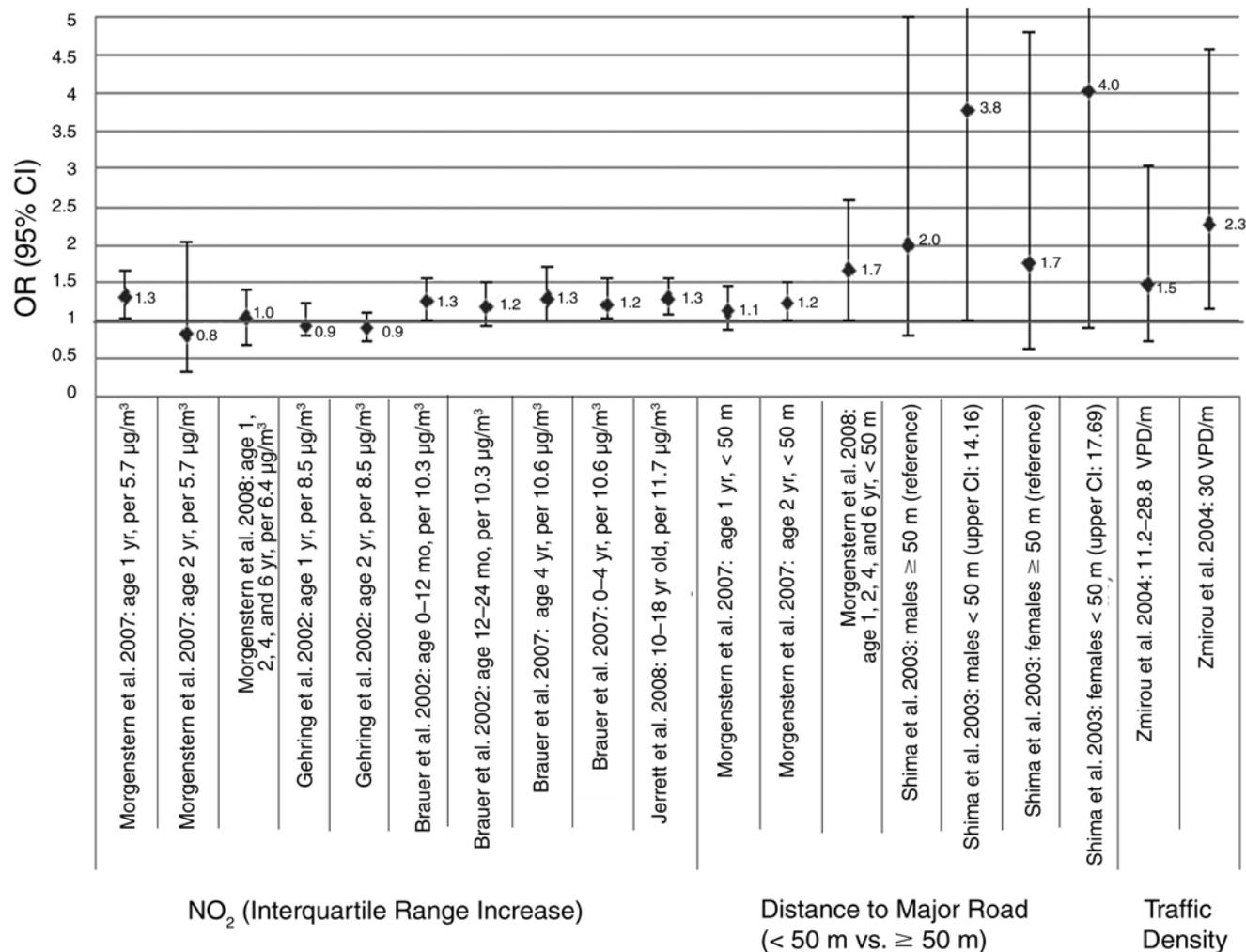
Because of the small number of studies, the evidence for an association of all cause mortality with long-term exposure was classified as “**suggestive but not sufficient**” to infer a causal association.

**Figure 4.3. Studies of long-term exposure to traffic pollution and cardiovascular and cardiopulmonary mortality**



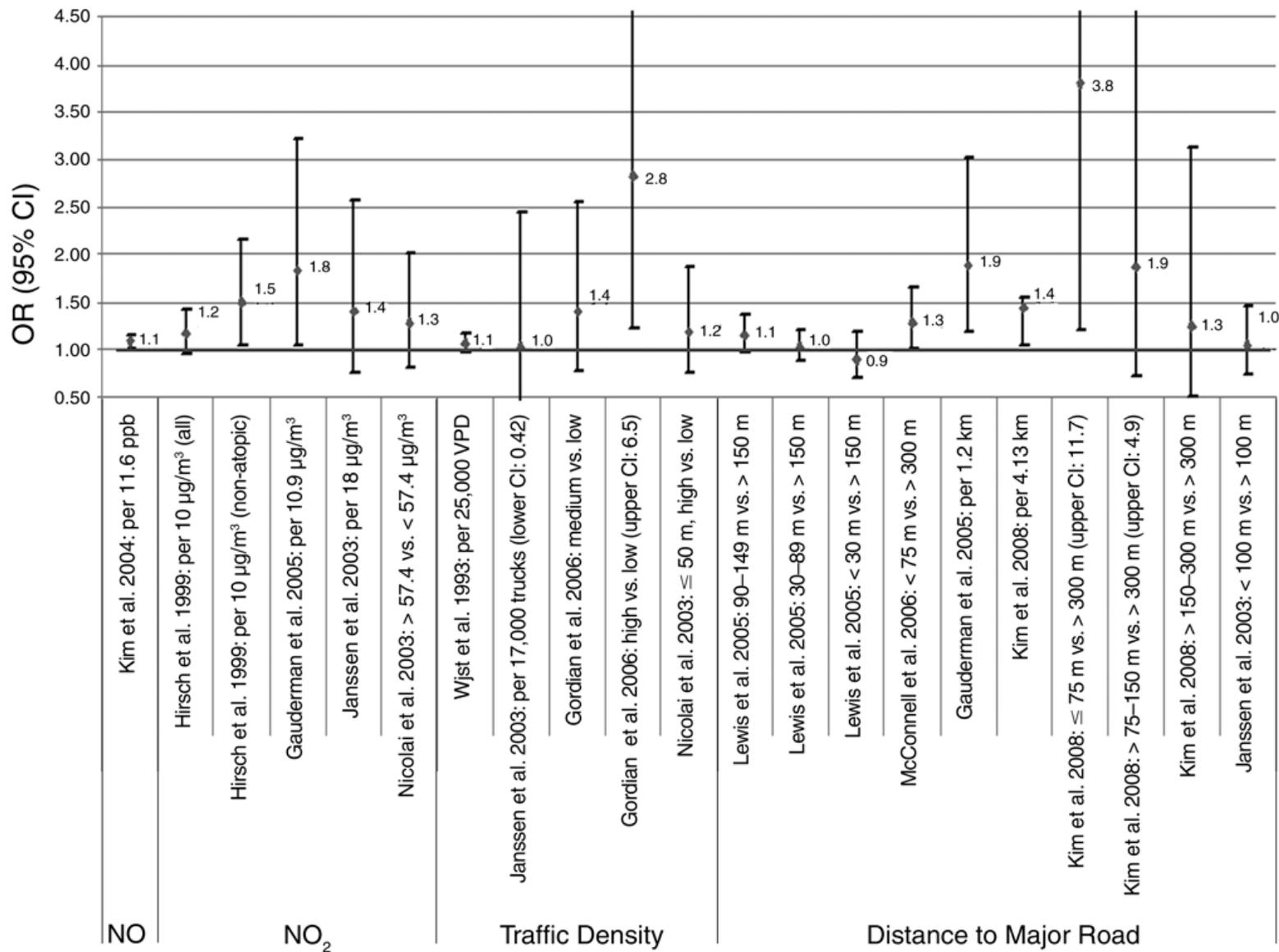
Because of the small number of studies, the evidence for an association of cardiovascular mortality with long-term exposure was classified as “**suggestive but not sufficient**” to infer a causal association.

**Figure 4.5. Studies of exposure to traffic pollution and **incidence** of doctor-diagnosed asthma in children**

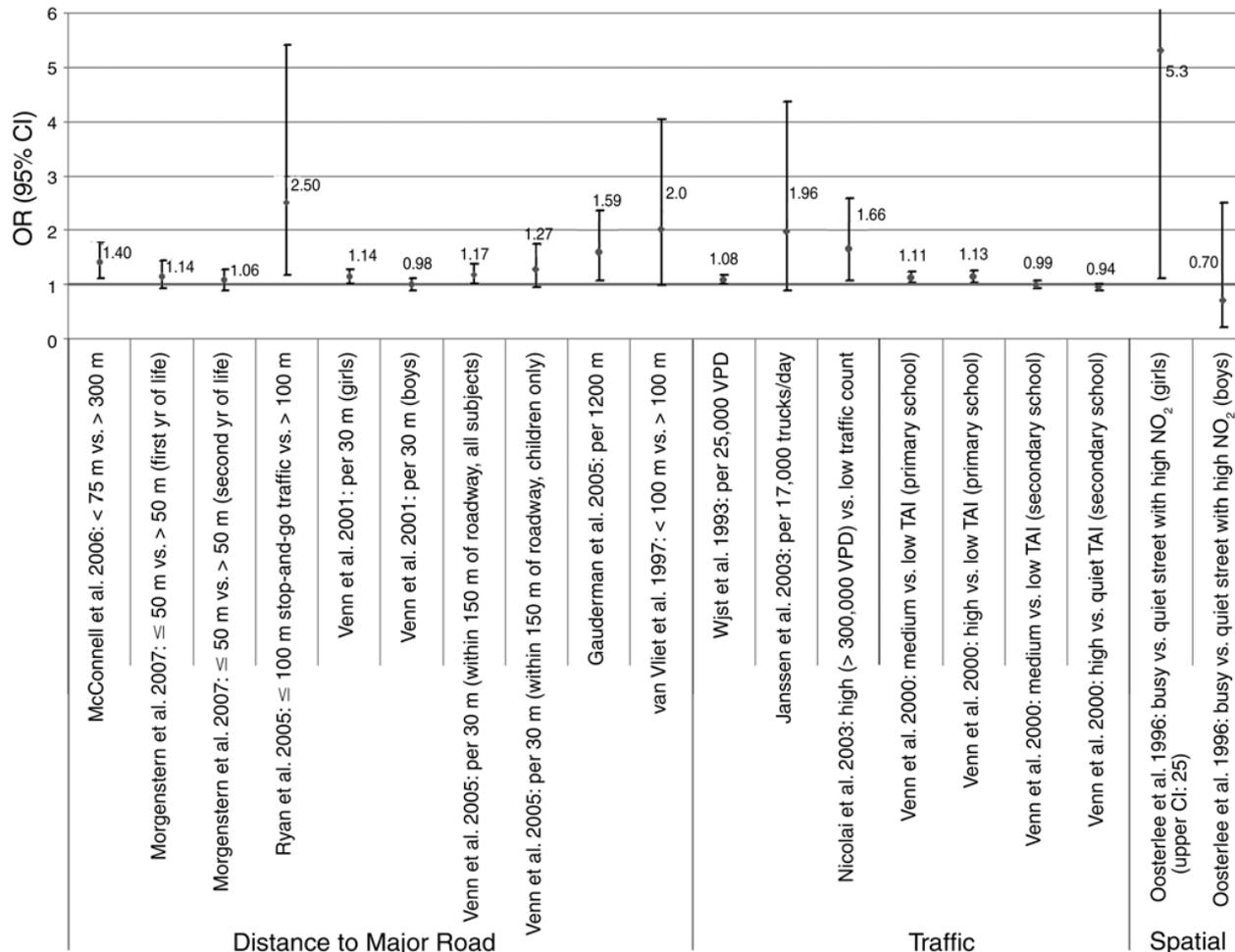


Living close to busy roads appears to be an independent risk factor for the onset of childhood asthma. The Panel considered the evidence for a causal relation to be in a gray zone between “**sufficient**” and “**suggestive but not sufficient.**”

Figure 4.6. Studies of exposure to traffic pollution and **prevalence** of doctor-diagnosed asthma in children



**Figure 4.7a. Studies of exposure to traffic pollution (traffic distance and density) and wheeze in children**



There was a high degree of consistency in finding positive associations with wheezing. The Panel concluded that the evidence is “sufficient” to infer a causal association between traffic exposure and exacerbations of asthma but that it is “inadequate and insufficient” to infer a causal association between exposure and respiratory symptoms in children without asthma.

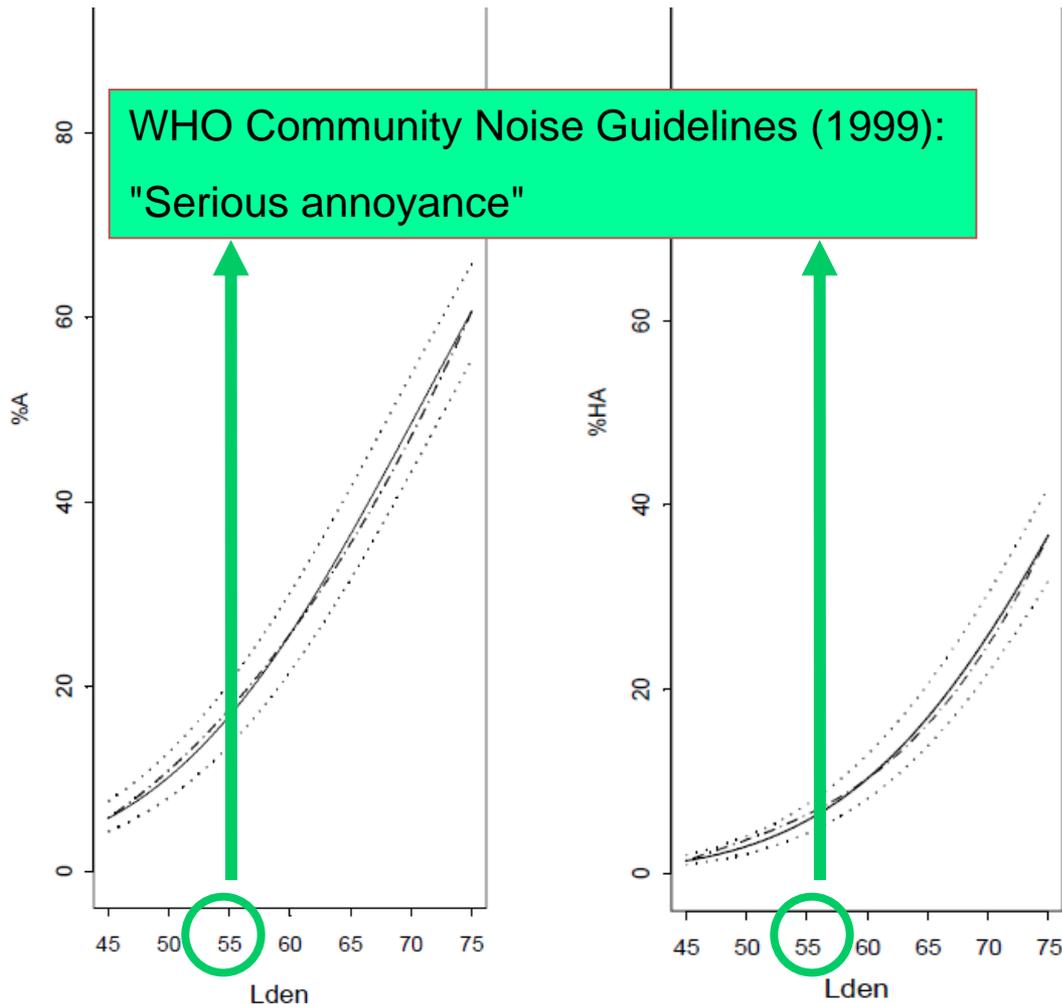
# Health risks from exposure to road traffic air emissions, according to the HEI panel, 2010

- The evidence for an association of all cause mortality and of cardiovascular mortality with long-term exposure was classified as “**suggestive but not sufficient**” to infer a causal association.
- The evidence for a causal relation between exposure and the onset of childhood asthma (based on prevalence and incidence) was said to be in a gray zone between “**sufficient**” and “**suggestive but not sufficient.**”
- The evidence is “**sufficient**” to infer a causal association between traffic exposure and exacerbations of asthma.

# Health risks from **noise** associated with road traffic

- ~~Hearing loss~~
- Annoyance
- Sleep disturbance (and associated effects such as decreased performance)
- Cardiovascular risk (hypertension, infarction – long term exposure)
- *Cognitive impairment (children)*

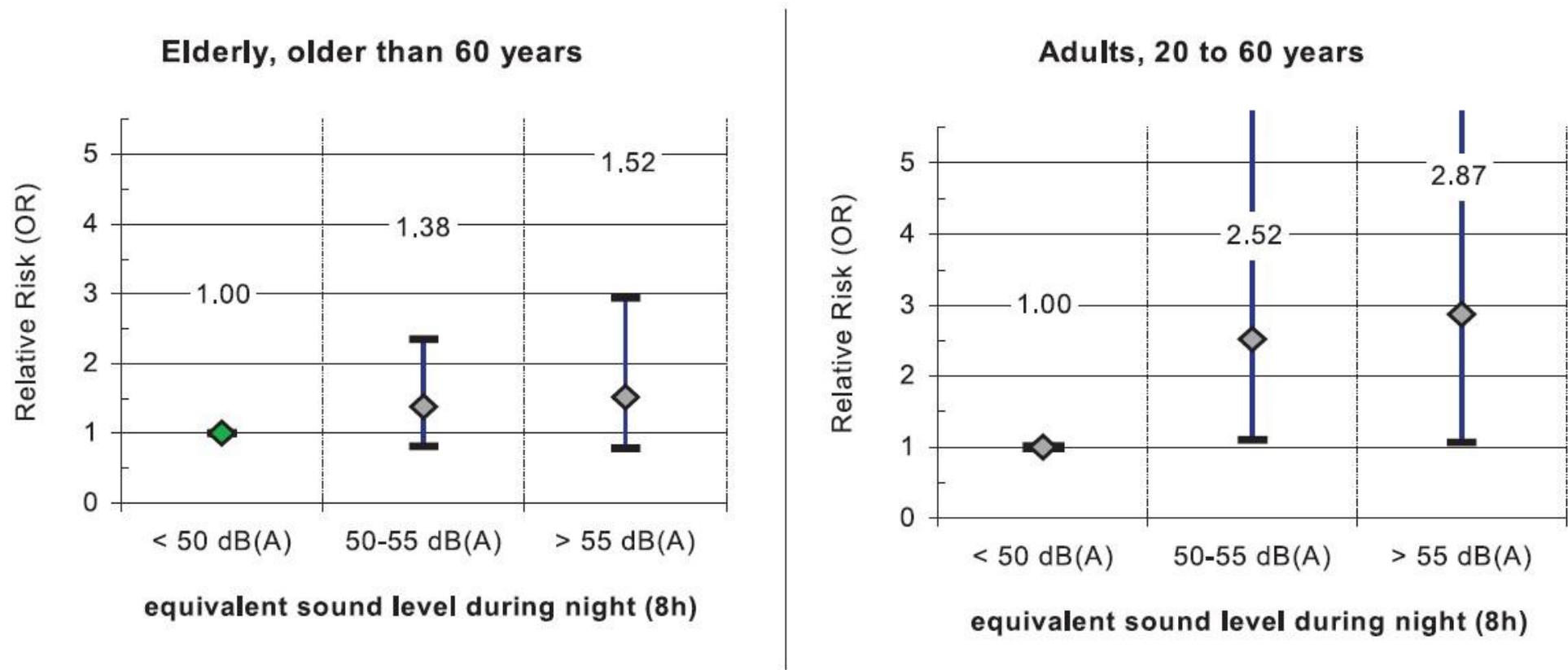
Figure 3.1 % **Annoyed** (% A) and % **highly annoyed** (% HA) for road traffic noise with 95 % confidence (EEA Technical report No 11/2010, from Miedema et al 2001)



# Sleep disturbance and road traffic

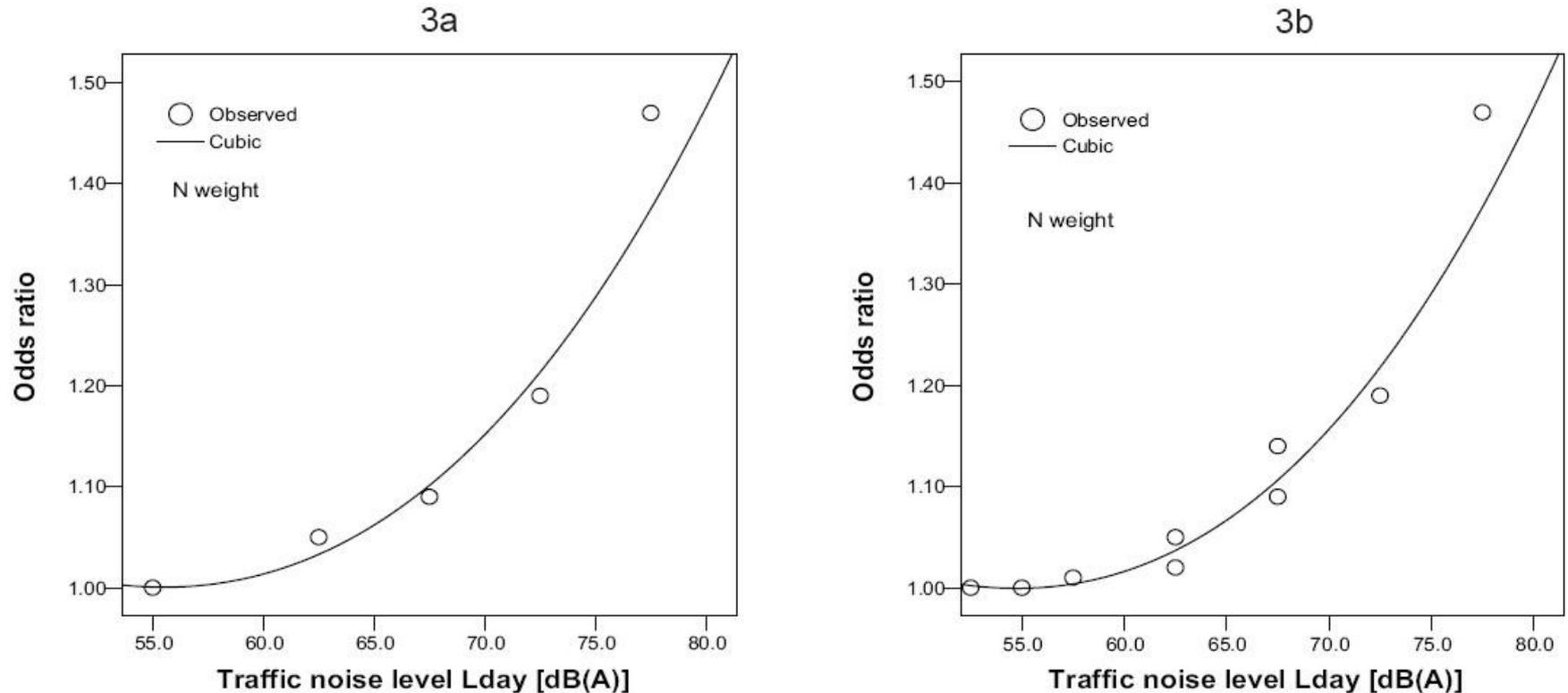
- According to Hume et al., 2012
  - Noise events induce arousals at relatively low exposure levels, and independent of the noise source (air, road, and rail traffic, neighbors, church bells).
  - Studies support evidence that night-time noise is likely associated with cardiovascular disease and stroke in the elderly.

Figure 20: Statistical association between **nightly** road traffic noise and **hypertension** treatments in the 9th repetition cycle of the Spandau Health Survey (adjusted OR) (Niemann et al, 2006, Noise and Health – LARES)



# Exposure-Response Curve: Myocardial Infarction

## Meta-Analysis: Road Traffic Noise



**Figure 3 (a-b): Polynomial fits of the exposure-response relationship between road traffic noise and myocardial infarction. The left graph (3a) refers case-control or cohort studies (analytic studies), the right graph (3b) to cross-sectional, case-control or cohort studies (descriptive and analytic studies), Babisch, Noise and Health 2008**

# Is the effect of road noise due to traffic emissions of air pollutants (**confounding**)

- Studies have reported associations between exposure to road traffic noise and air pollutant emissions and cardiovascular (CV) outcomes.
- CV health effects could be explained either by noise or air pollution associated with road traffic.

# Review of the epidemiological evidences on confounding effects (Tetreault et al., submitted)

- Due to limitations of the literature, we could not conclusively ascertain the independence of effect of both risks on any cardiovascular health outcome. However, the results reported tend to indicate that the impacts of traffic noise and air pollution on cardiovascular outcome are distinct, or at least that they are not completely dependent on one another.

# Health **impacts** of traffic related air and noise emissions (Price et al., 2012)

- HEI 2010 Document
  - The evidence almost sufficient for a causal relation between exposure and the onset of childhood asthma
  - We used 4 studies on asthma prevalence and proximity to major roads
    - *Morgenstern et al. 2007 et 2008*
    - *McConnell et al. 2006*
    - *Kim et al. 2006*

# Exposure in selected studies

- Distance to major roads :
  - < **50 m** (2, 4, 6 years old) or
  - < **75 m** (5-7 and 8-10 years old)

# Attributable fraction

## Population attributable risk

$$\text{Attributable Fraction} = \frac{p_e(RR - 1)}{p_e(RR - 1) + 1}$$

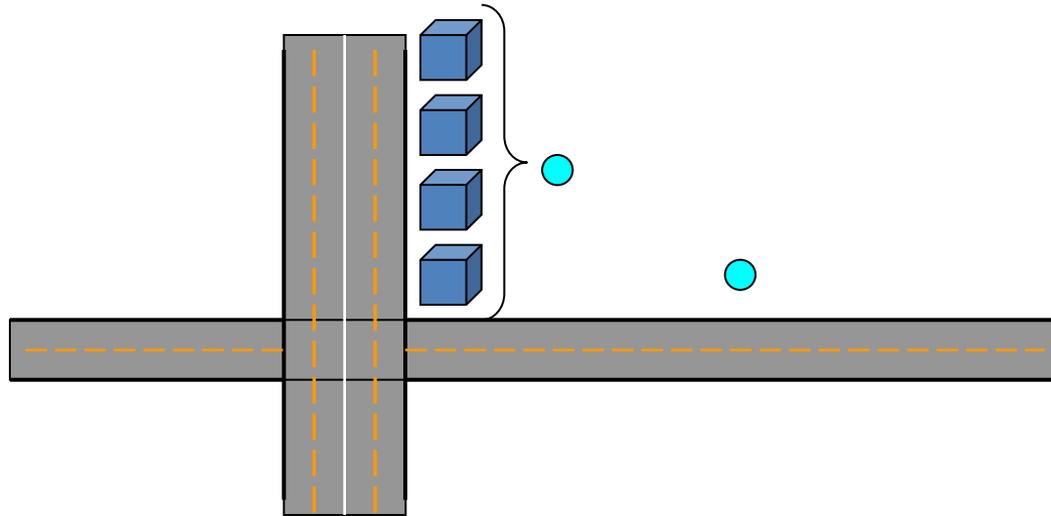
and

$$\text{Nb.cases.attributable} = \underbrace{pop * prev}_{\text{Nb asthmatics}} * AF$$

$p_e$  = proportion of the population that is exposed

# Population exposed

- Traffic categories (DMTI)
- Nombre of people living in proximity to major roads: based on centroid of postal codes



# Population exposed

Proportion of children  
of different age groups  
(Census 2006)

x

Total  
population  
along major  
roads  
  
(PC)

=

Population  
of children in  
proximity to  
major roads

# Nombre of asthmatics

- Population of asthmatics on the island of Montréal:
  - Prevalence of asthma in Montréal
  - Prevalence of asthme in Ontario  
(comparison)

# Results

Age (years)	Distance (m)	OR (95%CI)	Pop. In proximity to roads	Prevalence asthma on Island	Attributable cases
2	<50	1.23 (1.00, 1.51)	2973	1855	<b>57</b>
4	<50	1.66 (1.01, 2.59)	2830	2388	<b>183</b>
6	<50	1.66 (1.01, 2.59)	2887	3367	<b>234</b>
5-7	<75	1.29 (1.01, 1.66)	11809	10013	<b>751</b>
8-10	<75	3.8 (1.20, 11.71)	650	10735	<b>178</b>

# Public health benefits of strategies to reduce greenhouse-gas emissions: urban land transport

James Woodcock, Phil Edwards, Cathryn Tonne, Ben G Armstrong, Olu Ashiru, David Banister, Sean Beevers, Zaid Chalabi, Zohir Chowdhury, Aaron Cohen, Oscar H Franco, Andy Haines, Robin Hickman, Graeme Lindsay, Ishaan Mittal, Dinesh Mohan, Geetam Tiwari, Alistair Woodward, Ian Roberts

## Co-benefits

	Delhi			London		
	Lower-carbon-emission motor vehicles	Increased active travel	Towards sustainable transport	Lower-carbon-emission motor vehicles	Increased active travel	Towards sustainable transport
<b>Physical activity</b>						
<i>Per million per year vs "business as usual"</i>						
Premature deaths	0	-352	-352	0	-528	-528
YLL	0	-6040	-6040	0	-5496	-5496
YLD	0	-816	-816	0	-2245	-2245
DALYs	0	-6857	-6857	0	-7742	-7742
<b>Air pollution</b>						
Premature deaths	-74	-99	-122	-17	-21	-33
YLL	-1696	-2240	-2749	-160	-200	-319
YLD	0	0	0	0	0	0
DALYs	-1696	-2240	-2749	-160	-200	-319
<b>Road traffic crashes*</b>						
Premature deaths	0	-67	-67	0	11	11
YLL	0	-2809	-2809	0	418	418
YLD	0	-730	-730	0	101	101
DALYs	0	-3540	-3540	0	519	519
<b>Total†</b>						

# Summary

- Air and noise emissions from road traffic are associated with various health outcomes
- Comparative health risk assessment approaches can be used to quantify health impacts-gains of various transportation scenarios  
(for health risks for which the evidence is sufficient)



# Methods

- Comparative Risk Assessment

- ✓  $\Delta$  Disease Burden = Attributable Fraction  $\times$  Disease Burden



Percent change in disease rates from  
shift in exposure distribution in the alternative scenario

$$AF = \frac{\sum_x RR_x \times Population(BAU)_x - \sum_x RR_x \times Population(Alt.)_x}{\sum_x RR_x \times Population(BAU)_x}$$

*RR is the relative risk of the health outcome at the given exposure level*

- For physical activity, exposure,  $x$ , is the hours per week spent in walking and bicycling (and all other physical activity),
- Burden of Disease
  - ✓ Disability Adjusted Life Year, DALY, is a measure of premature mortality and disability based on the years of life lost, YLL (years of expected life - age at death) + years lived with a disability, YLD

$$DALY = YLL + YLD$$

# DALY (Disability adjusted life years)

- $DALY = YLL + YLD$
- YLL (Years of Life Loss)
- YLD (Years lived with disability)

*where:*

N = number of deaths.

L = standard life expectancy at age of death (in years).

$$YLL = N \times L$$

*where:*

YLD = years lived with disability.

I = number of incident cases.

DW = disability weight.

L = average duration of disability (years)

$$YLD = I \times DW \times L$$