Cardiovascular Burden of Disease from Environmental Noise

Evidence, Uncertainties and Public Health Implications

Wolfgang Babisch

Department of Environmental Hygiene

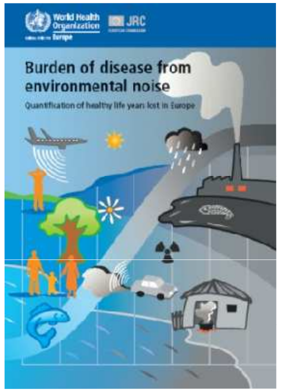
Federal Environment Agency

Berlin, Germany

Umwelt Bundes Amt
Für Mensch und Umwelt

Environmental Noise Burden Of Disease

Disability Adjusted Life Years (Western Europe, Major Agglomerations)



WHO 2011

Every year in the EU cities, at least:

- 61 000 DALYs for ischaemic heart disease
- 45 000 DALYs for cognitive impairment
- 903 000 DALYs for sleep disturbance
- 22 000 DALYs for tinnitus
- 654 000 DALYs for annoyance

1~1.6 million healthy life years are lost every year from traffic noise in the EU cities.

Sleep disturbance and annoyance related to road traffic noise comprise the main burden.

WHO European Centre for Environment and Health, 2011

Noise- DALYS

EU-27 Member States

DALYS = YLL + YLD

The sum of years of potential life lost due to premature mortality and the years of productive life lost due to poor health or disability

	YLL	YLD
Annoyance	0	654,086
Self-reported sleep disturbance	0	903,000
Heart disease	29,488	30,147

WHO European Centre for Environment and Health, 2011

History Of Noise And CVD Research

~ 1950:	Laboratory experiments with humans
~ 1960:	Animal experiments
~ 1970:	Occupational epidemiology
~ 1980:	Environmental epidemiology
~ 2000:	Quantitative risk assessment
~ 2005:	Combined effects (e. g. air pollution)

Laboratory Studies

(1950s to 1970s)

Circulatory system, heart rate, stroke volume, cardiac output, blood pressure, peripheral vascular resistance (finger pulse amplitude), endocrine system: catecholamines (epinephrine, norepinephrine), ACTH, corticosteroids (cortisol), plasma triglycerides, leucocyte count, cardiovascular changes, ECG (ischemia, bradycardia), cerebral blood flow (carotid artery), vasoconstriction, endocrine system, sexual hormones (inhibition), growth hormone (increase), salivary and gastric secretions (reduction), electrolytes (imbalance), whole blood glucose, free fatty acids, plasma cholesterol, uric acid, cardio-respiratory efficiency, vital capacity, apnoea/pulse rate, respiratory rate (increase), sinus arrhythmia, sleep research (EEG, heart rate, blood pressure).

Lehmann and Tamm (1956), Levi (1961, 1967), Arguelles (1962), Glas, Singer and Friedman (1969), Anticaglia and Cohen (1970), Welch and Welch (1970), Kryter (1970,1972), Miyazaki (1971), Semzcuk (1971), Favino (1973), Verdun di Cantogno et al. (1976), Griefahn (1977), Mosskov and Ettema (1977), Griefahn and Muzet (1978)

Endothelial Dysfunktion

Schmidt et al. 2013

Aircraft noise during the night

- Dose-dependent decrease of brachial artery diameter (increased wall thickness)
- Non-inflammatory accelerated growths of connective tissue in smooth muscle cells of arteries (fibro-muscular dysplasia)
- Decrease of the endothelial release of vasodilatory substances (NO-synthase)
- Stiffening of vascular walls
- Early indicator of atherosclerosis

General Stress Model

Chronic (Longterm) Effects

Dysregulation, disturbed biorhythm, physiologic and metabolic imbalance!

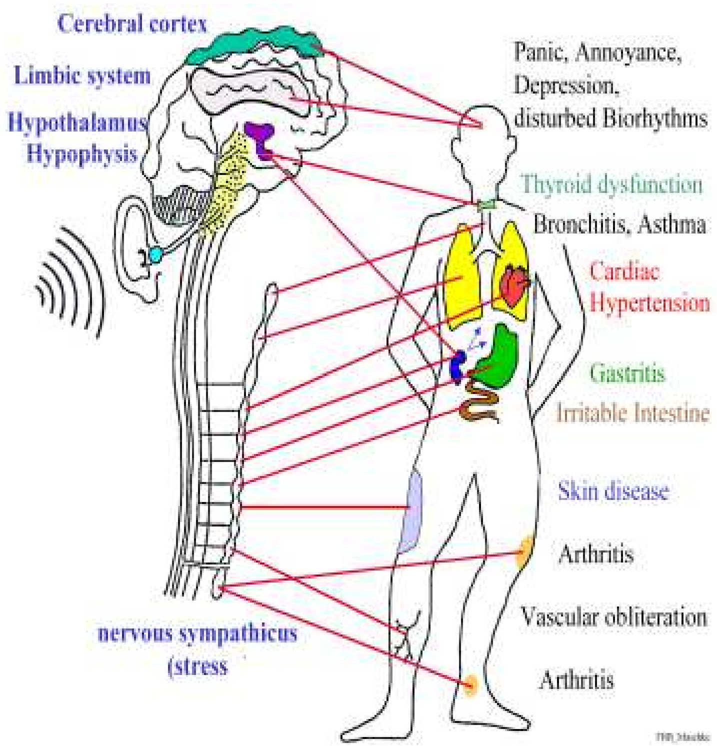
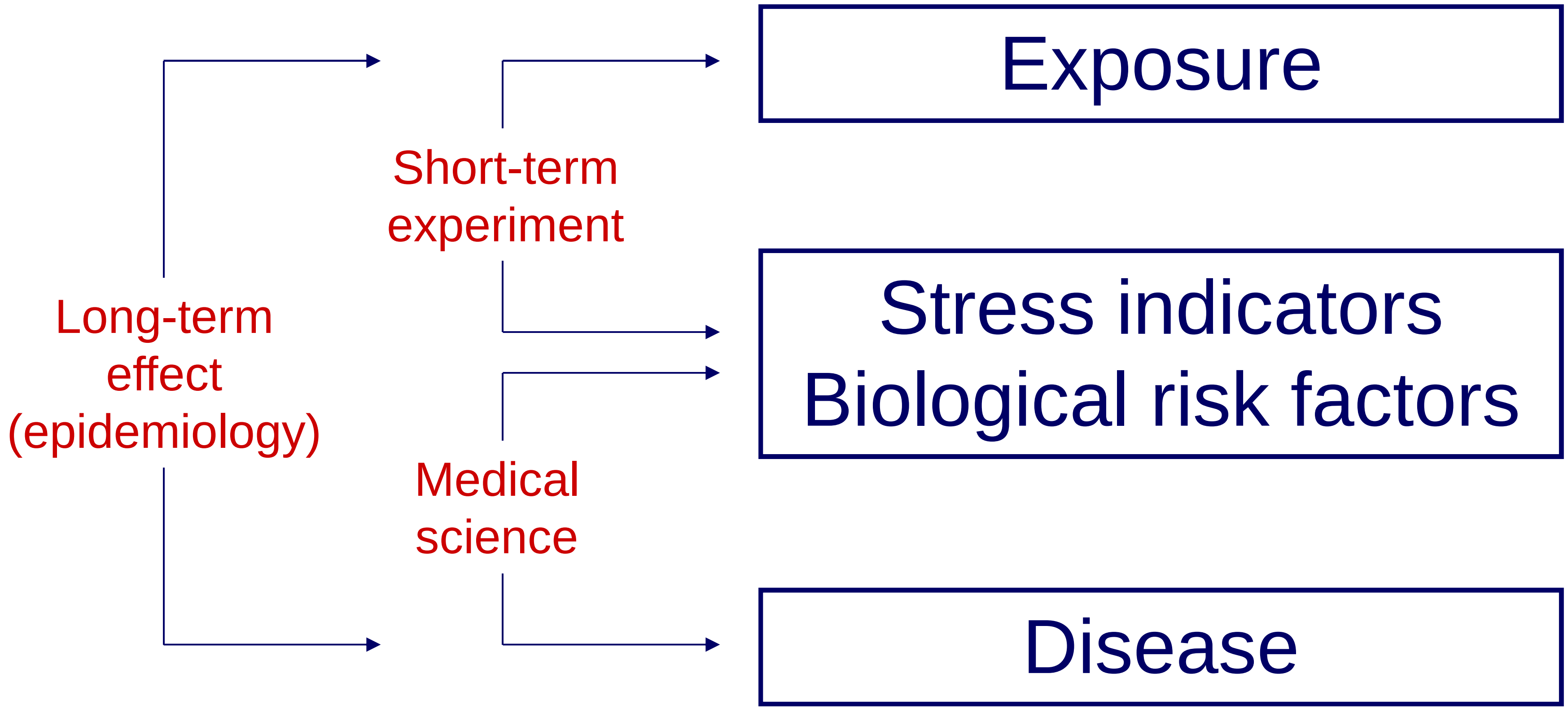


Image: Maschke, 2004

Short-term – Long-term Effects



What Have We Learned From Short-term Experiments?

- Noise is an unspecific stressor.
- Adverse health effects occur, in particular, when noise interferes with intended activities (e.g. communication, concentration, relaxation, sleep).
- The toxicological principle does not apply.
- Situational context – not the 24 hour daily noise-dose per se.
- "Decibels do not behave like µg/m^3".
- No or incomplete habituation/adoption.

Habituation / Adaption

- Subjects that have been working or living for many years in noise-exposed environments show physiological stress reaction in response to acute noise events.
- During sleep, even subjects who are subjectively not disturbed by the noise show acute electrophysiological responses (EEG, EMG, ECG) and vegetative responses (blood pressure, heart rate) to single noise events.
- Such vegetative reactions (blood pressure, heart rate) occur even in the absence of cortical activation (EEG) – no cognitive control (non-conscious reactions).

Ill-Health

Cardiovascular Effects Of Noise

Sound level

Direct pathway

Indirect pathway

Nerval interaction
(pysiological stress)

Cortical perception
(emotional stress)

Autonomic nervous system
Endocrine system

Dysregulation
Long-term health effects

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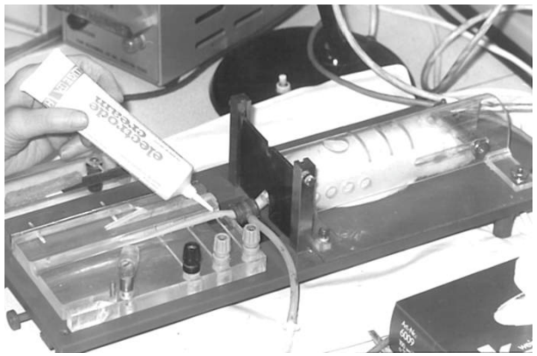
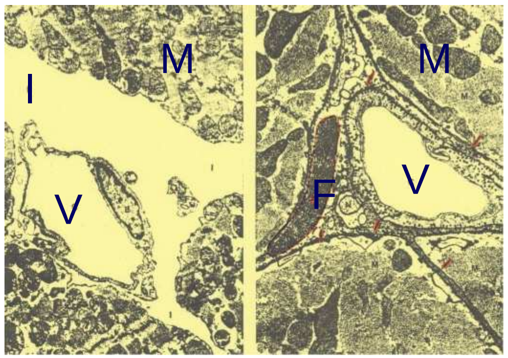
Animal Experiments

- Circulation (peripheral blood vessels and arteries)

+ Chronic blood pressure increase
+ Collagen (connective tissue) in heart muscle
+ Aging of the heart

Stronger effects in SHR rats





I=intercellular spaces, M=muscle cell, V=capillary vessel, F=collagen fibrils

History Of Noise And CVD Research

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Occupational Noise Studies

<u>Reviews – Hypertension</u>

Thompson (1993):	prevalence ratios ranged from 2.0 to 2.8
v. Kempen et al. (2002):	meta-analysis (9 studies) **$OR_{10\ dB(A)}$ = 1.30 (95% CI = 1.02-1.66)** range L_{Aeq8h} ~ 55-116 dB(A)
Tomei et al. (2010):	meta-analysis (15 studies) **prevalence ratio 2.56 (95% CI = 2.01-3.27)** high (92 ± 7 dB(A)) vs. low (62 ± 29 dB(A))

<u>Studies – Myocardial Infarction / Coronary Heart Disease</u>

Davies et al. (2005):	>100 dB(A), no hearing protection incidence rate ratio 1.2 to 1.5 (length of exposure)
Gan et al. (2011):	**prevalence ratio 2.04 (95% CI = 1.16-3.58)***

*Note: disease ratios refer to (extreme) group comparisons

Occupational Noise Studies

Reviews – Hypertension

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$OR_{10\ dB(A)}$ = 1.30 (95% CI = 1.02-1.66)
range $L_{A[illegible]}$ [illegible] 5[illegible]-116 dB(A)

Tomei et al. (2010): meta-analysis (15 studies)
prevalence ratio 2.[illegible]6 (95% CI = 2.01-3.27)
high (9[illegible] ± [illegible]7 dB(A)) vs. low (62 ± 29 dB(A))

L_{Aeq8h} ≥ 95 dB(A):
50-150 % increase in risk

Studies – Myocardial Infarction / Coronary Heart Disease

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History Of Noise And CVD Research

~ 1960: Laboratory experiments with humans

~ 1970: Animal experiments

~ 1980: Occupational epidemiology

~ 1990: Environmental epidemiology

~ 2000: Quantitative risk assessment

~ 2010: Combined effects (e. g. air pollution)

Health Endpoints

- Hypertension
- Myocardial infarction
- Ischaemic heart disease (coronary heart disease)
- Cerebrovascular diseases (stroke)
- Metabolic syndrome (diabetes mellitus type 2)
- Obesity (sleep disturbance)
- Altzheimer disease and other dementia (blood pressure)

Short-term Experiments

Acute Effects

- \+ Muscle tension
- \+ Vasoconstriction of peripheral blood vessels
- \+ Stroke volume
- +/- Heart beat frequency
- +/- Cardiac output (heart-minute volume)
- \+ Stress hormones
- \+ Blood pressure
- \- Endothelial function

Global Burden Of Disease

WHO 2004

- 13.5% of deaths are attributable to high blood pressure (hypertension)
- 12.2% of deaths are caused by IHD (leading cause);
  high-income (16.3%) and low-income countries (9.4%)
- 9.7% of deaths are caused by cerebrovascular diseases (stroke)

Sources: Lopez et al. (2006) , WHO (2008)

Relative Risk (RR)

- Number of cases due to noise either directly or indirectly (population attributable risk)
- Example: reported annoyance or sleep disturbance due to noise
- Reported prevalence or incidence of a disease due to noise?
- → Relative risk: disease occurrence
  exposed vs. non-exposed
- Estimates: odds ratio (OR), incidence risk ratio (iRR), hazard ratio (HR), prevalence ratio (PR)

Quantitative Risk Assessment

Heath Impact Assessment

Hazard identification

Exposure assessment

Exposure-response relationship

Risk characterization

Attributable risk concept

Risk management

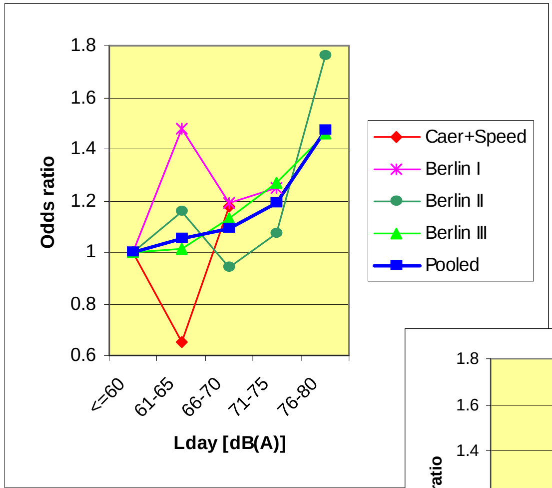
Regulatory options

Road Traffic Noise Studies – Myocardial Infarction

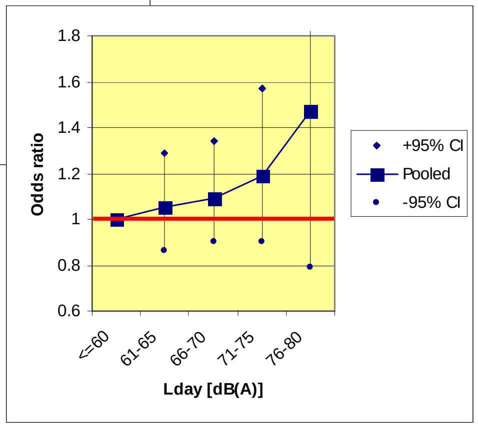
Meta-Analyses (2008)

Exposure-response function:

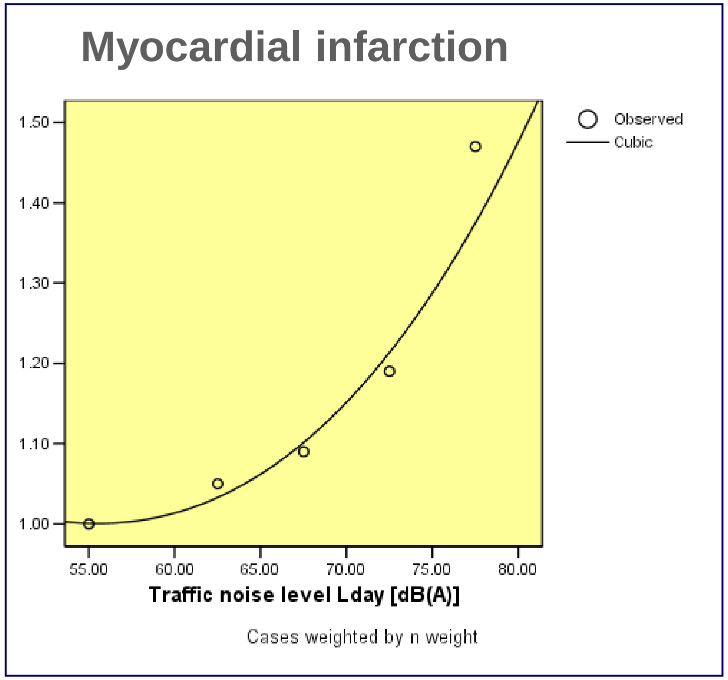
OR = 1.629657 – 0.000613*$(L_{day,16h})^2$ + 0.000007357*$(L_{day16h})^3$, R^2 = 0.96



Heterogeneity: p = 0.943



Analytic studies (CC, CO)



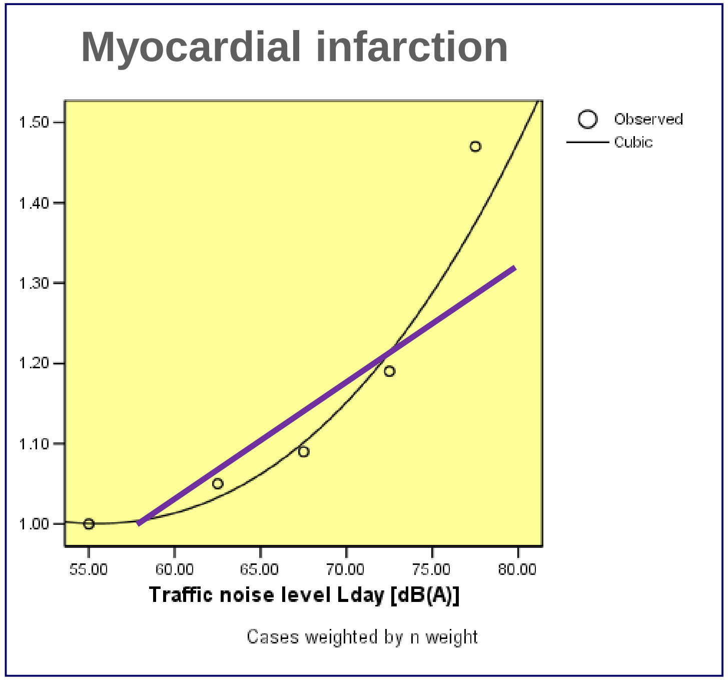
Source: Babisch, 2008

Road Traffic Noise Studies – Myocardial Infarction

Meta-Analyses (2008)

Exposure-response function:

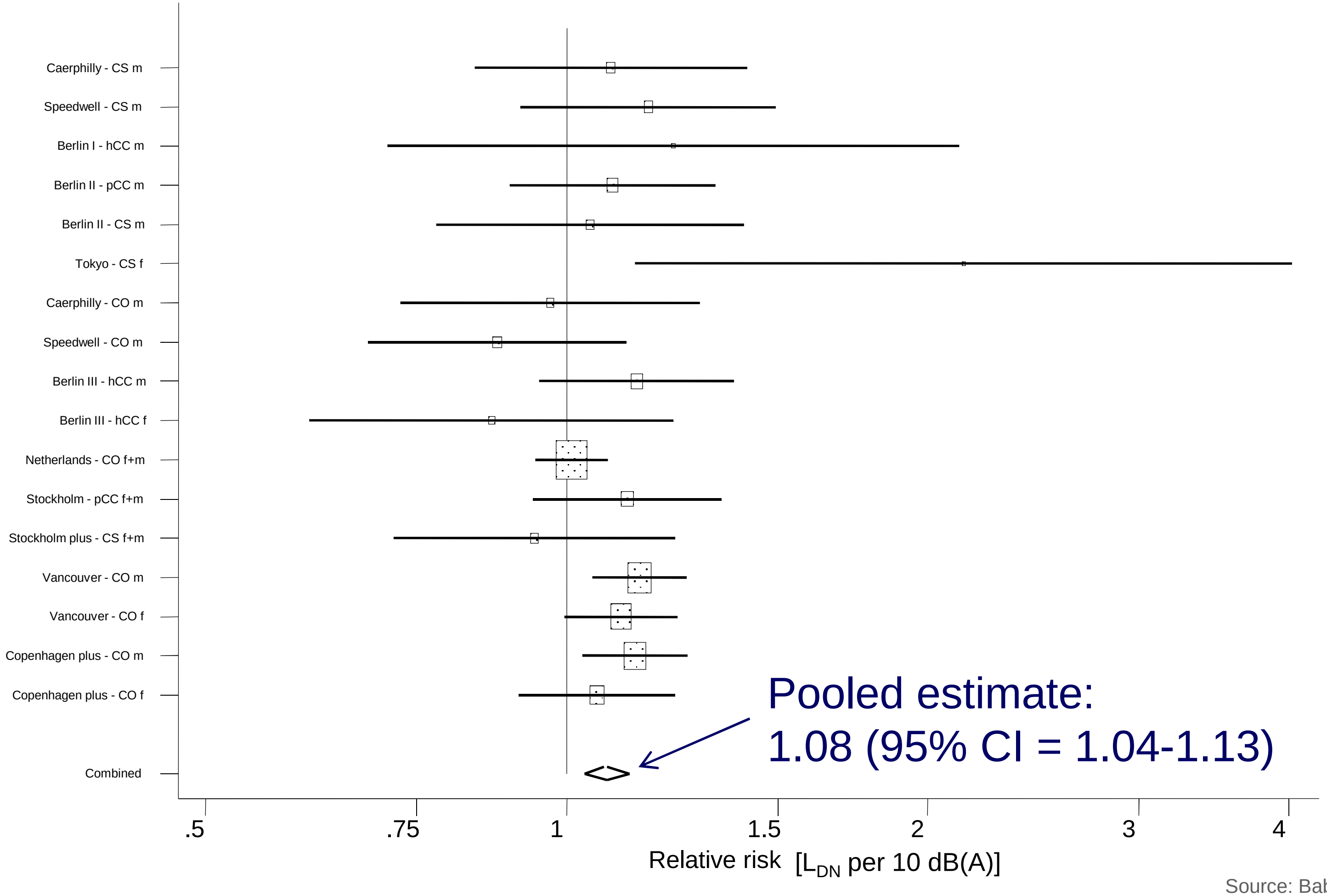
OR per 10 dB(A) = 1.17, 95% CI = 0.87-1.57, range L_{Aeq16h} = 58-78 dB(A)



Source: Babisch, 2008

Road Traffic Noise – Coronary Heart Disease

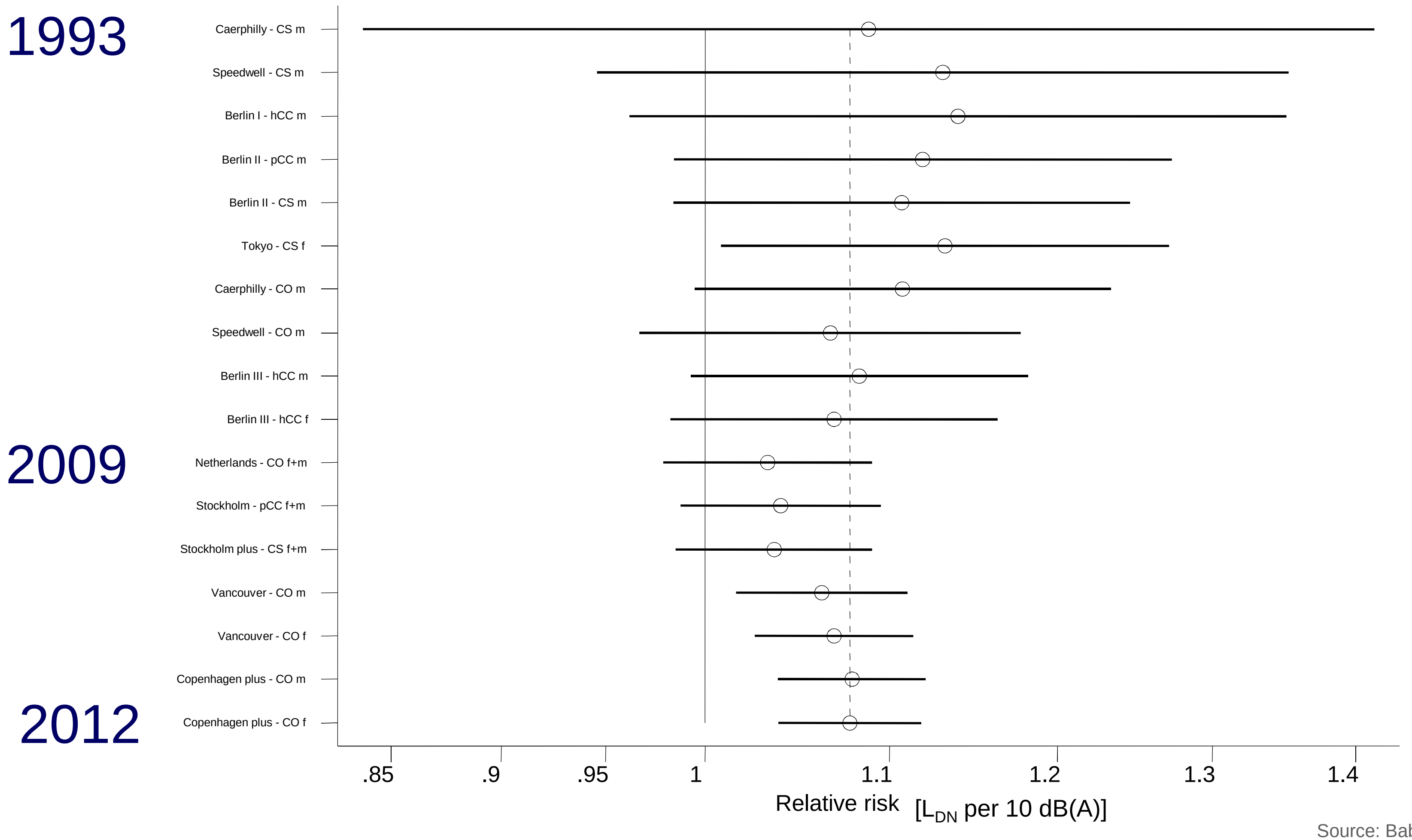
Updated Meta-Analysis (Forest Plot)



Source: Babisch, 2014

Cumulative Risk Of Studies

By Year - Road Traffic Noise



Source: Babisch, 2014

Road Traffic Noise – Coronary Heart Disease

Exposure-Response Relationship

<u>Myocardial Infarction (5 studies)</u> *– Babisch (2008)*

L_{Aeq16h}: range ~ <u>58</u>-78 dB(A), $OR_{10\ dB(A)}$ = 1.17 (95% CI = 0.87-1.57)

- **17%** increase in risk per 10 dB(A) increase in noise level

<u>Myocardial Infarction (14 studies)</u> *– Babisch (2014)*

L_{DN}: range ~ <u>53</u>-78 dB(A), $OR_{10\ dB(A)}$ = 1.08 (95% CI = 1.04-1.13)

- **8%** increase in risk per 10 dB(A) increase in noise level

OR = Odds ratio = estimate of the relative risk

CVD Studies

Road Traffic Noise

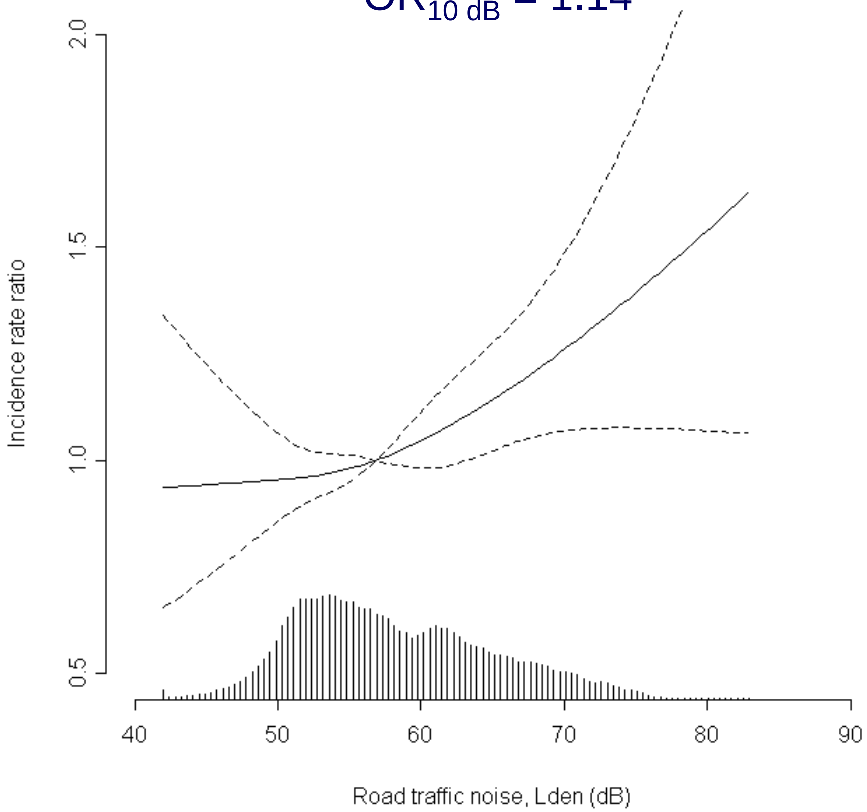
Study - type (Country)	Reference	Number of subjects	Number of cases	Response rate	Covariates	Age	Exposure assessment	Health outcome
Caerphilly - CS (United Kingdom)	Babisch et al. 1993[30]	M: 2.512	M: 438	89	A, G, B, C, S, E, P, M, F, O	45-59	$L_{Aeq16hr}$ ≤55 to >65	Prevalent IHD (clinical)
Speedwell - CS (United Kingdom)	Babisch et al. 1993[30]	M: 2.348	M: 340	92	A, G, B, C, S, P, F, O	46-63	$L_{Aeq16hr}$ ≤55 to >65	Prevalent IHD (clinical)
Berlin I - hCC (Germany) - hCC	Babisch et al. 1994[27]	M: 243	M: 109	Cases: 89 Controls: 87	A, G, B, C, S	41-70	$L_{Aeq16hr}$ ≤60 to >75	Incident MI (clinical)
Berlin II - pCC (Germany)	Babisch et al. 1994[27]	M: 4.035	M: 645	Cases: 91 Controls: 64	A, G, B, C, S, M, O	31-70	$L_{Aeq16hr}$ ≤60 to >75	Incident MI (clinical)
Berlin II - CS (Germany)	Babisch et al. 1994[27]	M: 2.375	M: 206	64	A, G, B, C, S, M, O	31-70	$L_{Aeq16hr}$ ≤60 to >75	Prevalent MI (self-reported)
Tokyo - CS (Japan)	Yoshida et al. 1997[31]	F: 3.950	F: 305	73	A, G	20-60	$L_{Aeq16hr}$ ≤55 to >70	Prevalent CHD (self-reported)
Caerphilly - CO (United Kingdom)	Babisch et al. 1999[26]	M: 2.369	M: 281	Follow-up: 94	A, G, B, C, S, E, P, M, F, O	45-59	$L_{Aeq16hr}$ ≤55 to 70	Incident MI (clinical)
Speedwell - CO (United Kingdom)	Babisch et al. 1999[26]	M: 2.330	M: 290	Follow-up: 78	A, G, B, C, S, P, F, O	46-63	$L_{Aeq16hr}$ ≤55 to 70	Incident MI (clinical)
Berlin III - hCC (Germany)	Babisch et al. 2005[28]	M: 3.054 F: 1.061	M: 1.528 F: 354	Cases + Controls: 84	A, G, B, C, S, M, F, O	20-69	$L_{Aeq16hr}$ ≤60 to >70	Incident MI (clinical)
Netherlands - CO (The Netherlands)	Beelen et al. 2009[15]	M+F: 105.269	M+F: 3.089	Follow-up: high (death register)	A, G, C, S, O, N[2)]	55-69	$L_{Aeq,max}$[1)] ≤50 to >65	Incident IHD (mortality)
Stockholm - pCC (Sweden)	Selander et al. 2009[14]	M+F: 3.518	M+F: 1.466	Cases: 72 Controls: 70	A, G, C, B, S, P, N, O	45-70	$L_{Aeq24hr}$ <50 to ≥60	Incident MI (clinical)
Stockholm/Gothenborg/ Malmö - CS (Sweden)	Eriksson et al. 2012[16]	M+F: 2.498	M+F: 161	59	A, G, C, S, N	18-80	L_{denr} <50 to ≥65	Prevalent CVD (self-reported)
Vancouver - CO (Canada)	Gan et al. 2012[17]	M: 189.713 F: 222.707	M+F: 3.095	Follow-up: high (death register)	A, G, C, O, N[2)]	45-85	L_{den} ≤58 to >70	Incident CHD (mortality)
Copenhagen/Aarhus - CO (Denmark)	Sørensen et al. 2012[18]	M: 24.294 F: 26.319	M: 1.184 F: 416	Follow-up: 89	A, G, B, C, S, E, P, N, O	50-64	L_{den} ≤50 to >70	Incident MI (clinical)

Danish Road Traffic Noise Studies

Stroke And Heart Attack, N = 57,053 subjects

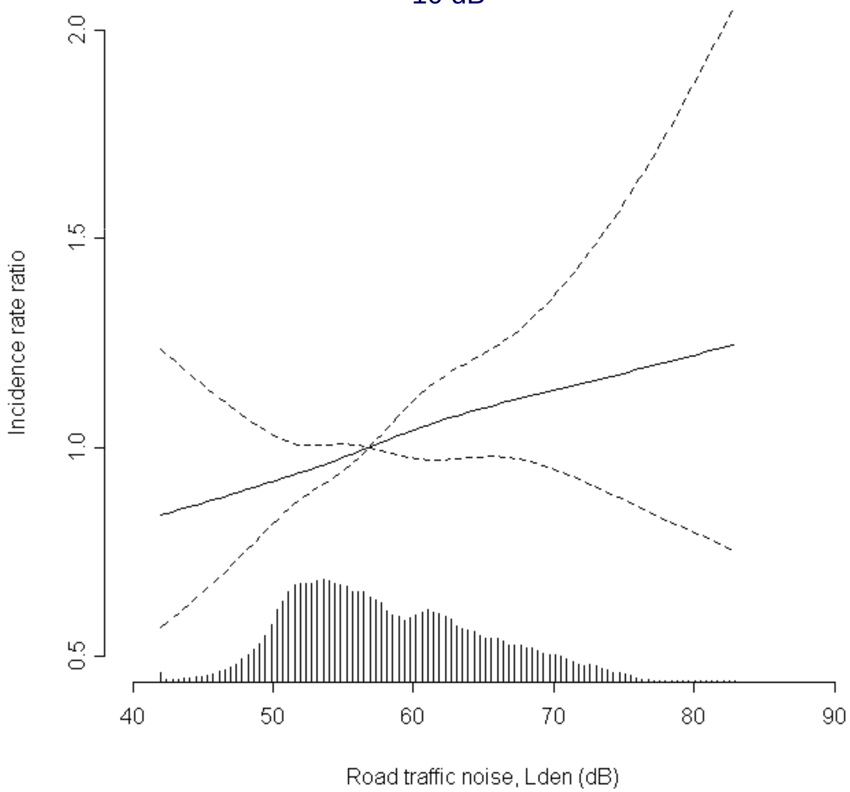
Stroke

$OR_{10\ dB} = 1.14$



Heart attack

$OR_{10\ dB} = 1.12$



Sources: Sørensen et al, (2011); Sørensen et al, (2012)

Adjustment
(Confounding)

- Age, gender
- Socio-economic status
- Smoking, alcohol,
- Physical activity, body mass index
- Family history
- Air pollution
- Other

Road Traffic Noise – Hypertension

Exposure-Response Relationship

<u>Hypertension (24 studies</u>) *– Van Kempen and Babisch (2012)*

L_{Aeq16h}: range ~ 48-75 dB(A), $OR_{10\ dB(A)}$ = 1.07 (95% CI = 1.02-1.12)

- **7%** increase in risk per 10 dB(A) increase in noise level

OR = Odds ratio = estimate of the relative risk

Road Traffic Noise – Other Health Endpoints

Single Large Study Results

Stroke (1 study) *– Sørensen et al. (2011)*

L_{DEN}: range ~ 53-73 dB(A), $RR_{10\ dB(A)}$ = 1.14 (95% CI = 1.04-1.25)

- **14%** increase in risk per 10 dB(A) increase in noise level

Diabetes mellitus (1 study) *– Sørensen et al. (2012)*

L_{DEN}: range ~ 53-73 dB(A), $OR_{10\ dB(A)}$ = 1.11 (95% CI = 1.05-1.18)

- **11%** increase in risk per 10 dB(A) increase in noise level

OR = Odds ratio = estimate of the relative risk

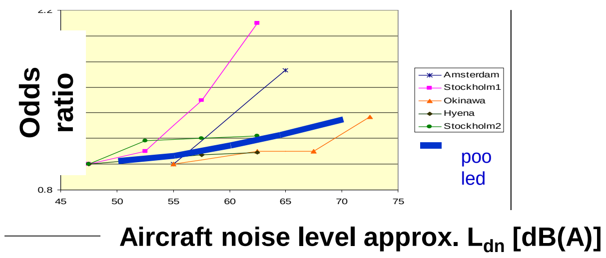
Aircraft Noise – Cardiovascular Diseases

Exposure-Response Relationship

<u>Hypertension (5 studies)</u> *– Babisch and van Kamp (2009)*

L_{DN}: range ~ 48-68 dB(A), $OR_{10\ dB(A)}$ = 1.13 (95% CI = 1.00-1.28)

- **13%** increase in risk per 10 dB(A) increase in noise level



<u>Myocardial Infarction (1 study)</u> *– Huss et al. (2010)*

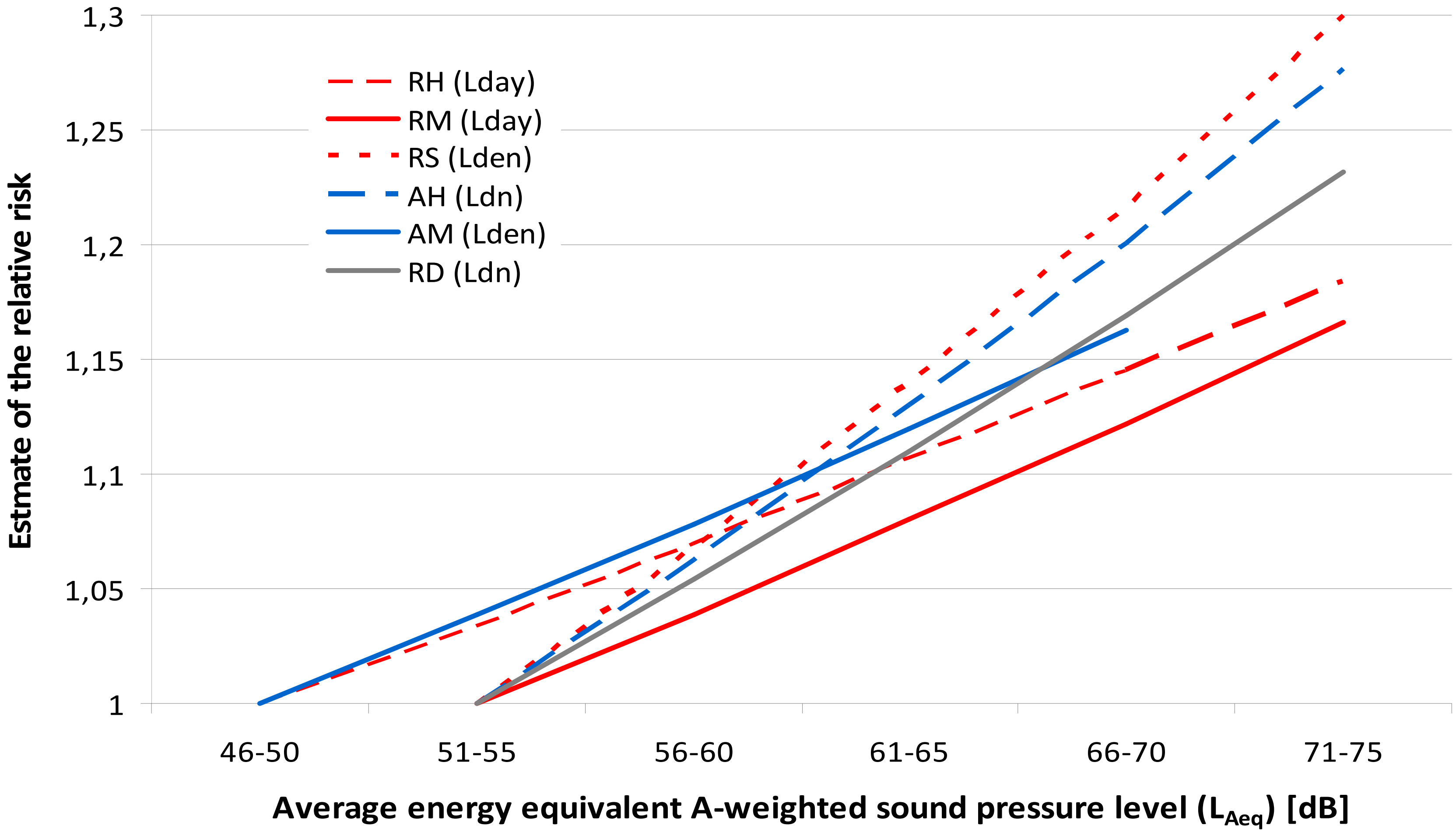
L_{DN}: range ~ 48-63 dB(A), $HR_{10\ dB(A)}$ ~ 1.07 (95% CI = 0.94-1.23)

- **7%** increase in risk per 10 dB(A) increase in noise level

OR = Odds ratio = estimate of the relative risk

Synthesis Of Cardiovascular Risk Curves

Environmental Noise



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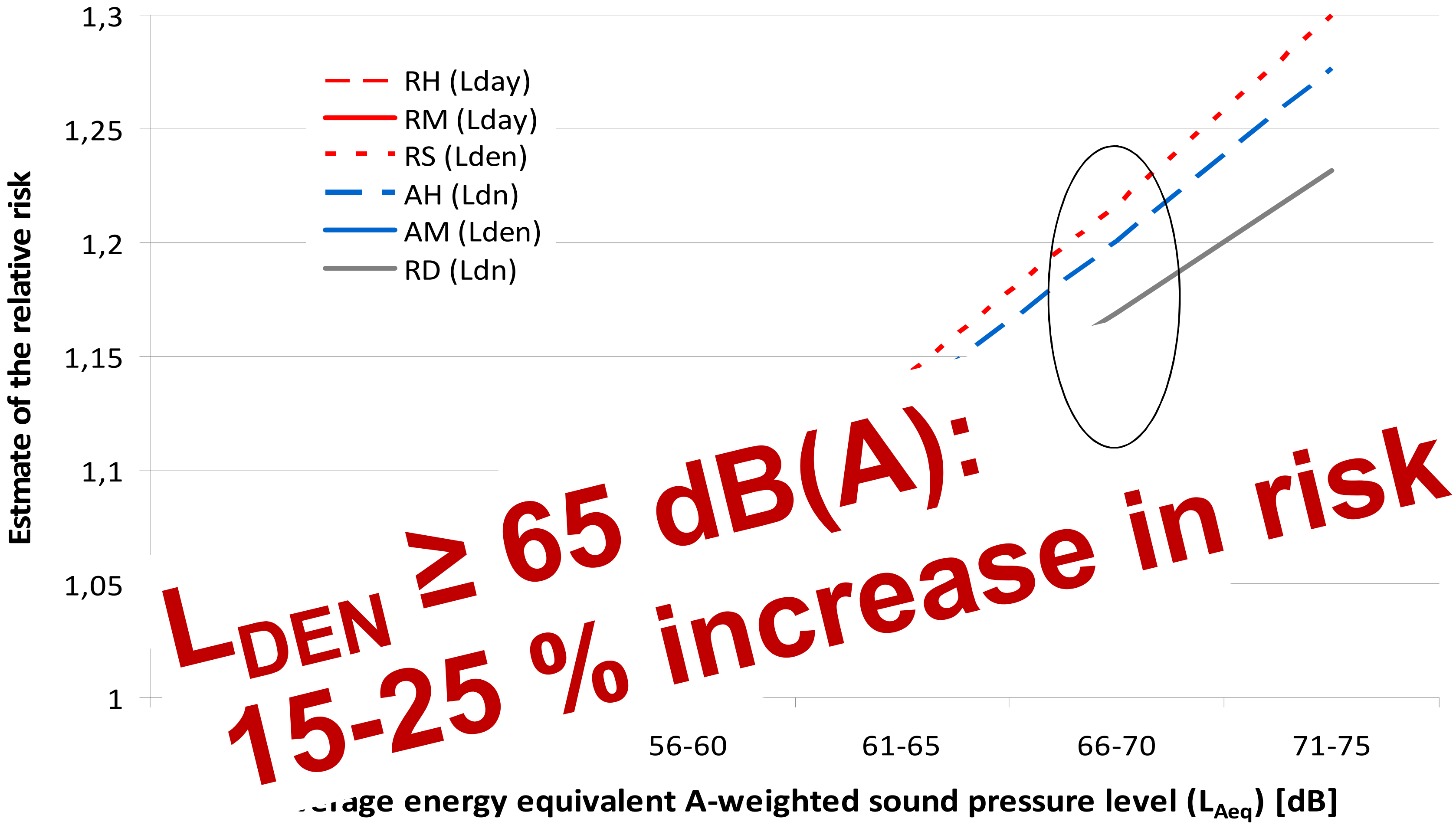
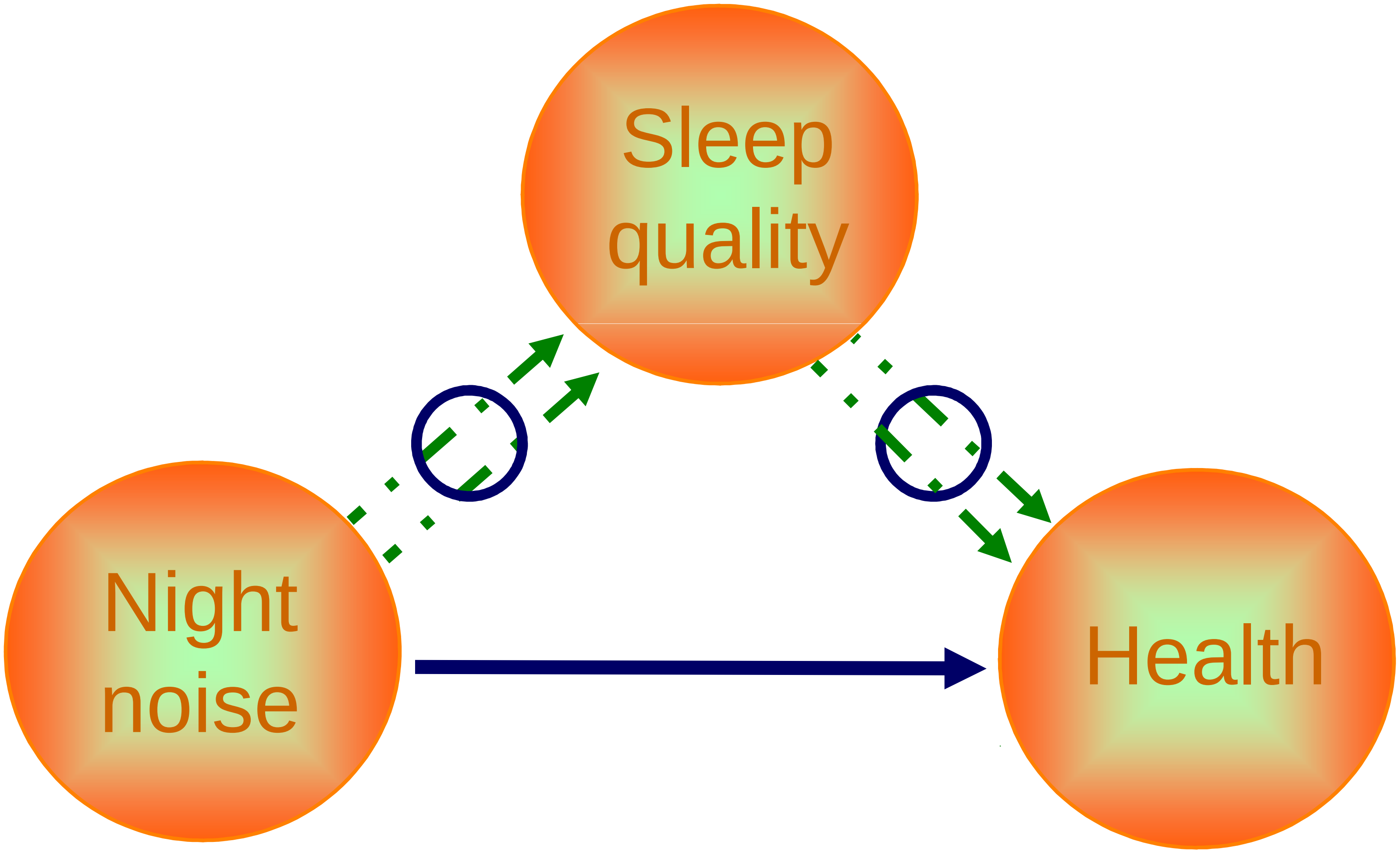


Noise, Sleep And Health



Source: WHO, 2009

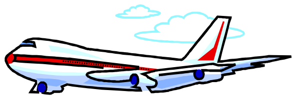
Day / Night

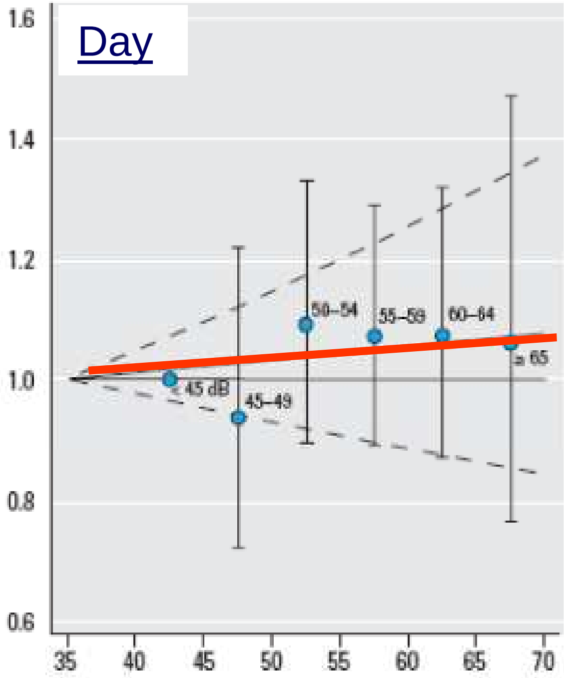
Six Airports Study ('HYENA')

Cross-sectional study, 4861males+females, aged 45-70 yrs

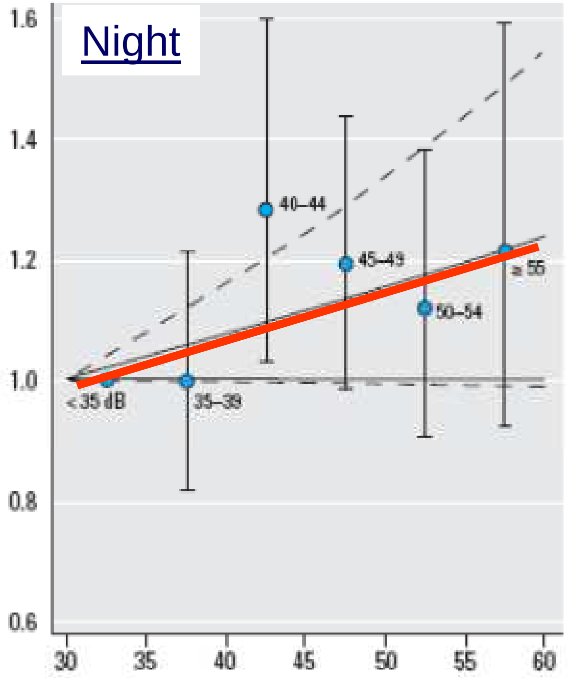
Prevalence of high blood pressure

Per 10 dB(A): OR_{Day} = 1.02 (0.95-1.10) **OR_{Night} = 1.07 (1.00-1.15)**





L_{Aeq16h} [dB(A)]



L_{Aeq8h} [dB(A)]

OR = Odds ratio = estimate of the relative risk

Source: Jarup et al., 2008

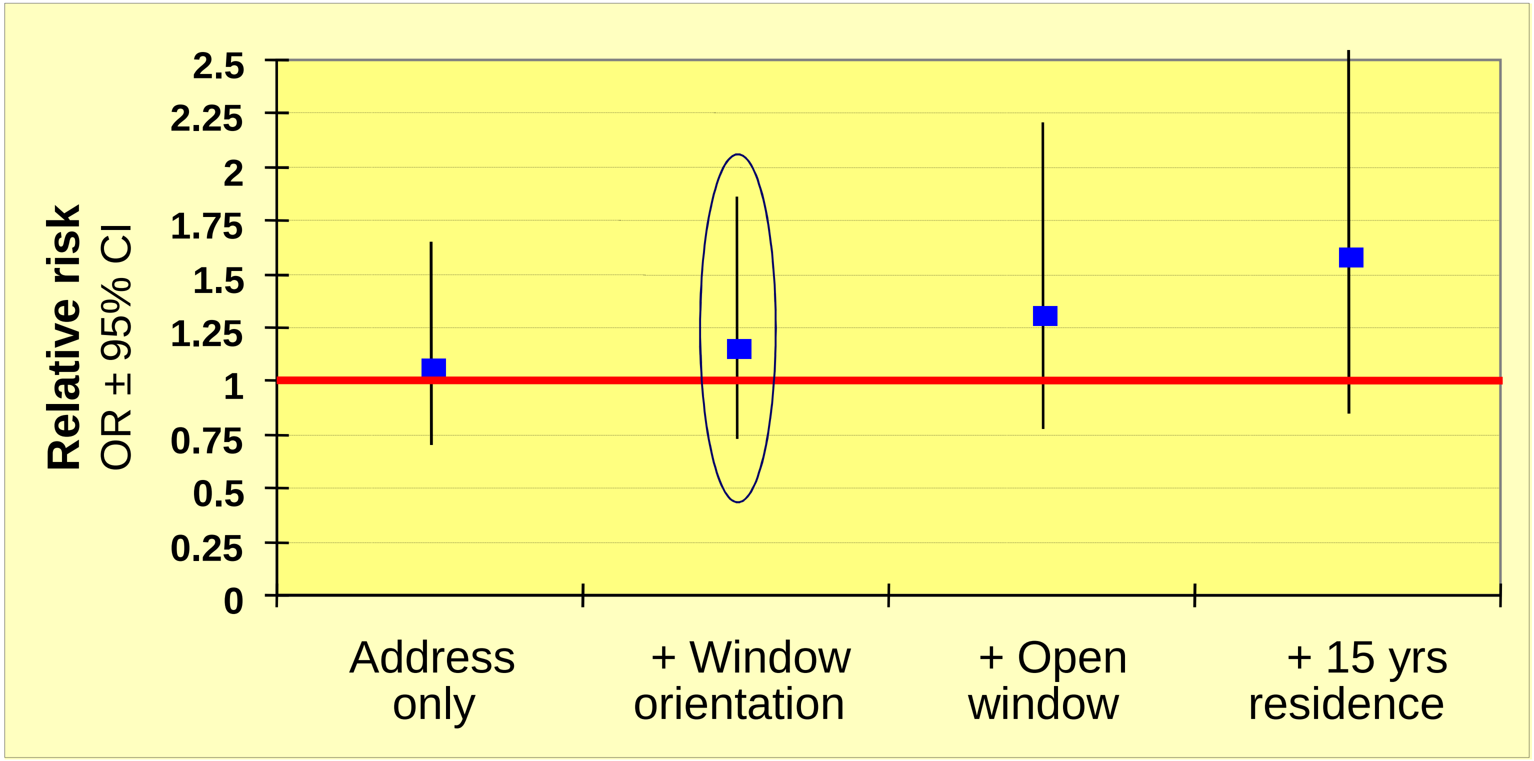
Exposure Modifiers

Caerphilly & Speedwell Studies

Prospective cohort study, 3950 males, aged 45-63 yrs

Extreme group comparison: L_{Aeq16h} = 66-70 dB(A) vs. 51-55 dB(A)

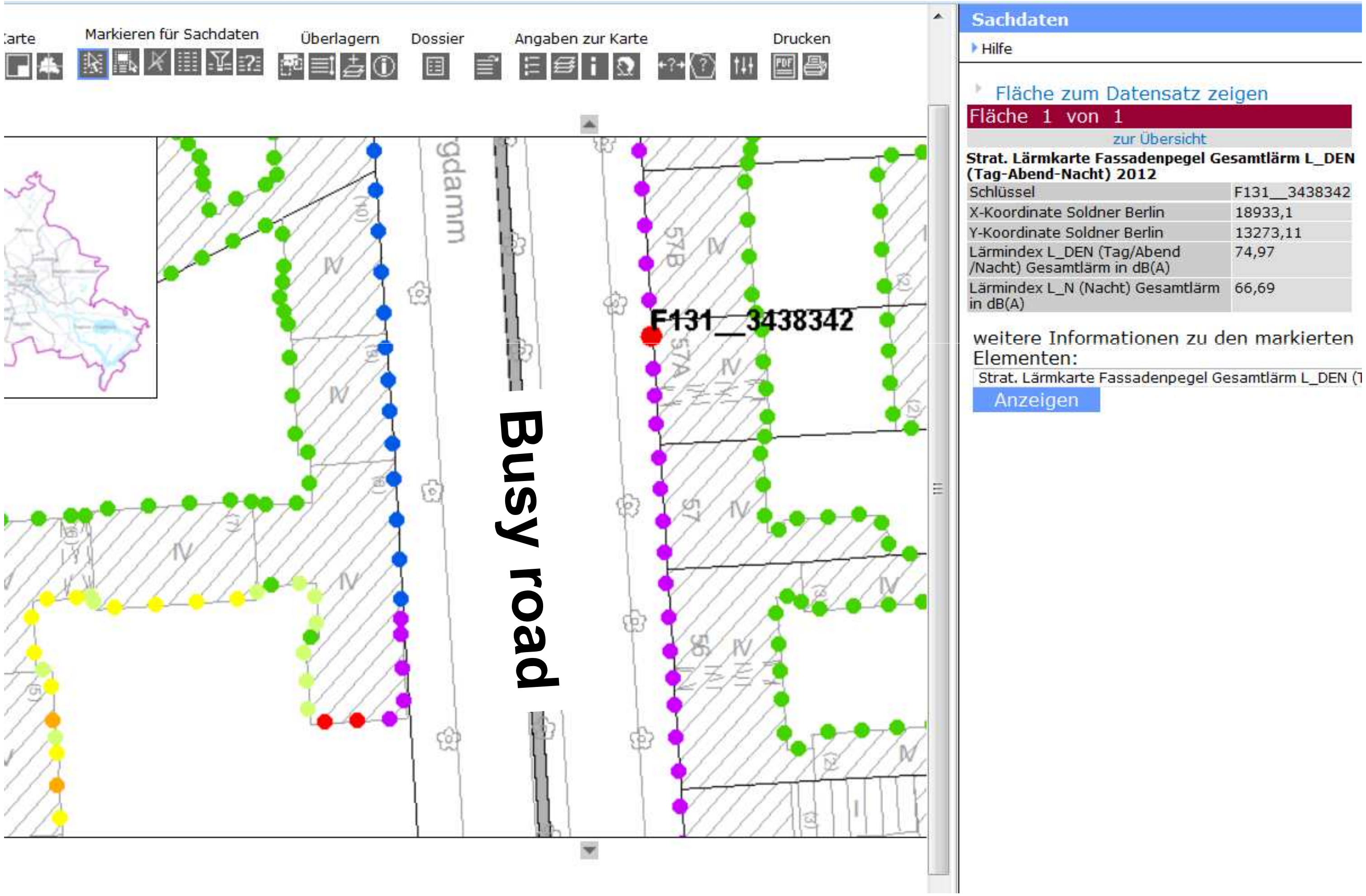
Incidence of major ischaemic heart diseases by road traffic noise



OR = Odds ratio = estimate of the relative risk

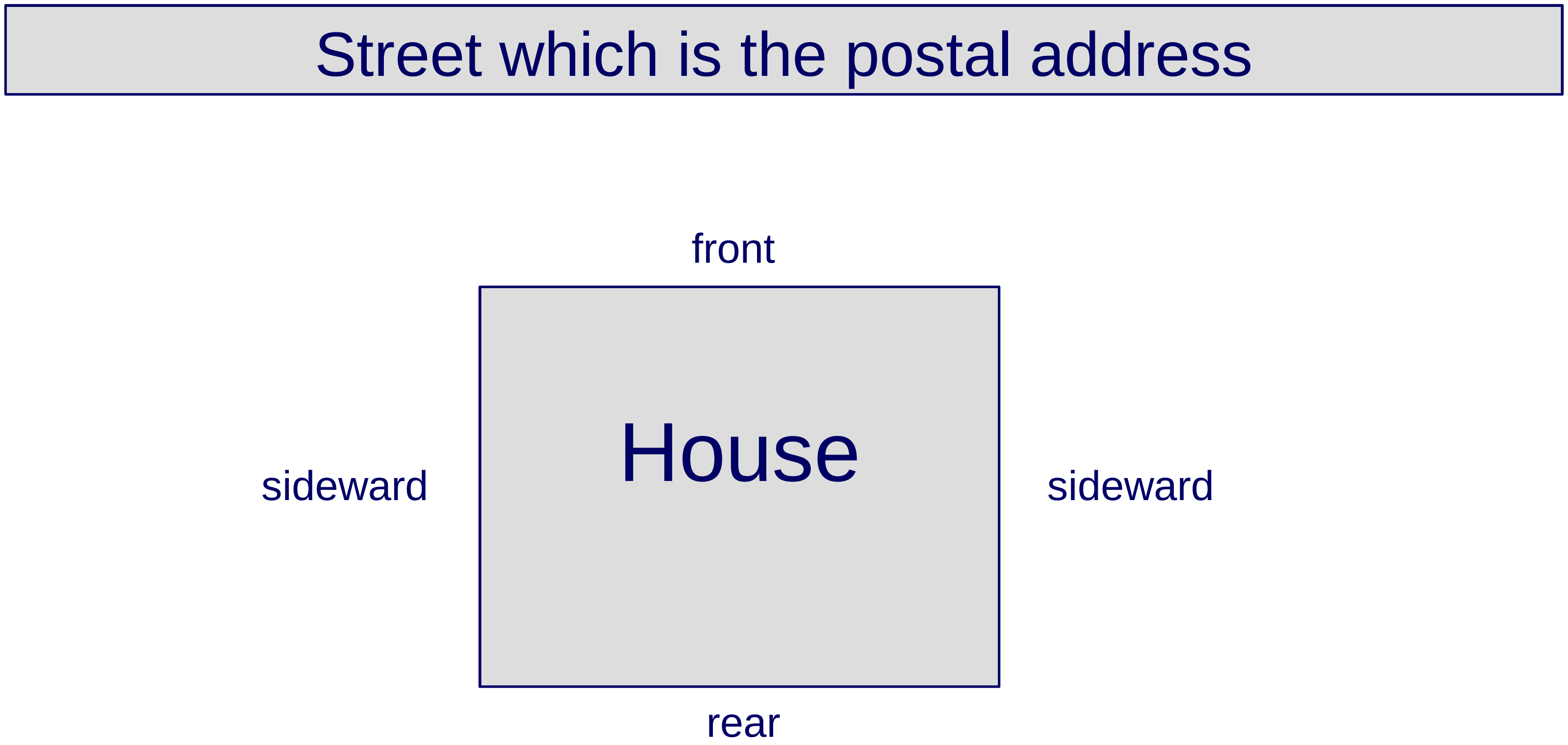
Source: Babisch et al. (1999)

Berlin Noise Map



Location Of Rooms

Exposure Modifiers



Source: Babisch et al., 2014 in print

Location Of Rooms

Berlin-4 Study

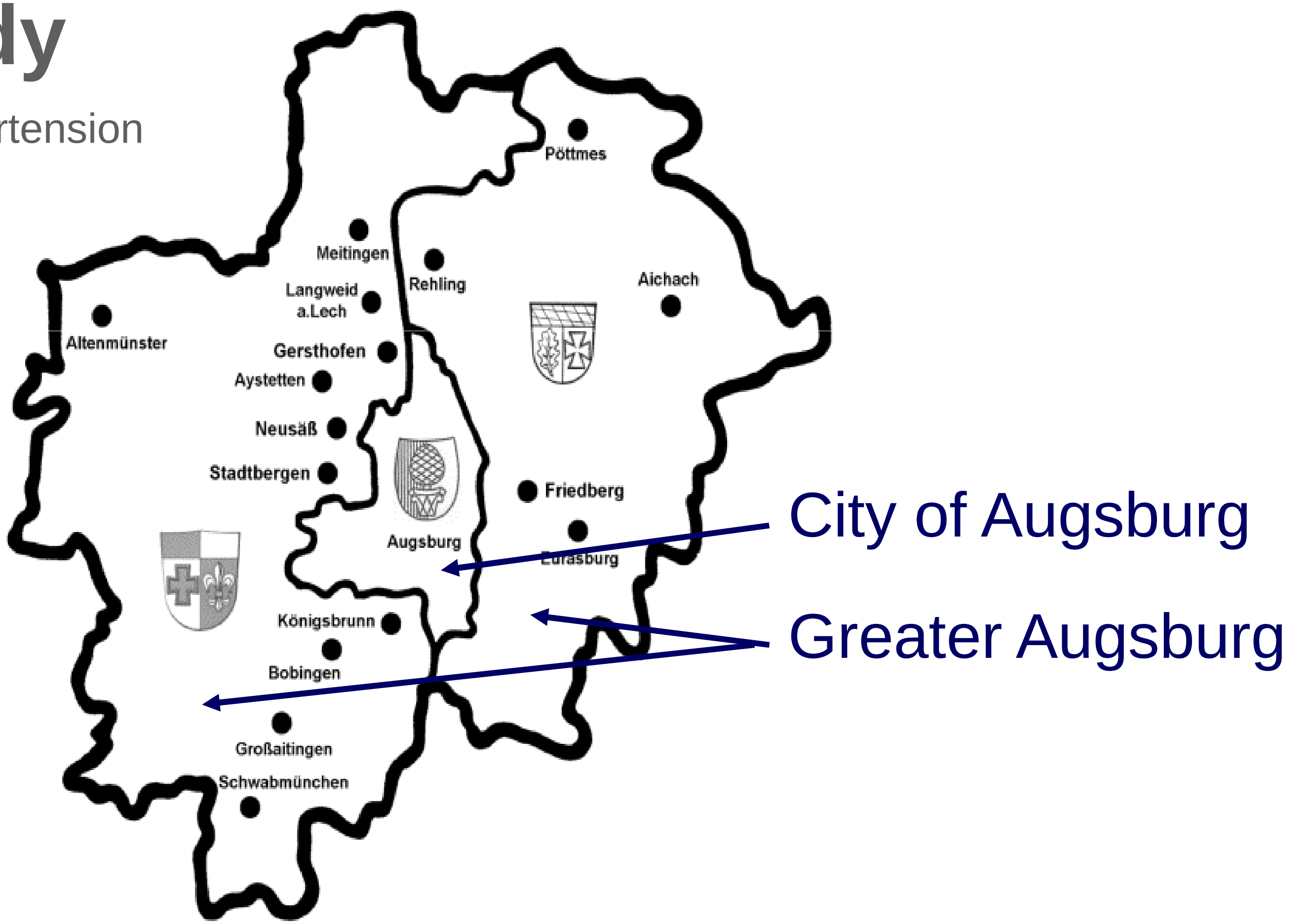
Model Logistic regression	Noise indicator	Number of subjects N	OR (95% CI) Per 10 dB(A)	P-Value
- Total Sample	L_{DEN}	1766	1.11 (1.00-1.23)	0.043
- Living room or bedroom facing the road	L_{DEN}	1016	1.21 (1.06-1.38)	0.004
- Living room and bedroom on the rear side of the house	L_{DEN}	248	0.98 (0.75-1.29)	0.906
- Living room and bedroom facing the road	L_{DEN}	354	1.21 (0.95-1.54)	0.132
- Either living room or bedroom facing the road	L_{DEN}	662	1.23 (1.04-1.44)	0.013

Source: Babisch et al., 2014 in print

Exposure Assessment

KORA Study

Road Traffic Noise – Hypertension
N = 4,166



Source: Babisch et al., 2014

KORA Study

Road Traffic Noise – (Systolic) Hypertension

Hypertension

City ofAugsburg: $OR_{10\ dB(A)}$ = 1.16 (1.00-1.35)
Greater Augsburg: $OR_{10\ dB(A)}$ = 0.94 (0.81-1.09)

Isolated systolic hypertension

City of Augsburg: $OR_{10\ dB(A)}$ = 1.48 (1.16-1.89)
Greater Augsburg: $OR_{10\ dB(A)}$ = 0.88 (0.69-1.12)

City of Augsburg: All streets, shielding due to houses
Greater Augsburg: Primary road network, free sound propagation

Source: Babisch et al., 2014

History Of Noise And CVD Research

~ 1960:	Laboratory experiments with humans
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~ 1990:	Environmental epidemiology
~ 2000:	Quantitative risk assessment
~ 2010:	Combined effects (e. g. air pollution)

Quantitative Risk Assessment

Heath Impact Assessment

Hazard identification

Exposure assessment

Exposure-response relationship

Risk characterization

Attributable risk concept

Risk management

Regulatory options

Population Attributable Risk Percentage

Formula

Attrubutable fraction

Impact fraction

$$PAR\% = \{\Sigma(P_i * RR_i) - 1\} / \Sigma (P_i * RR_i) * 100 [\%]$$

$$\mathbf{PAR = PAR\% * N_d}$$

where: P_i = Proportion of the population in exposure category i

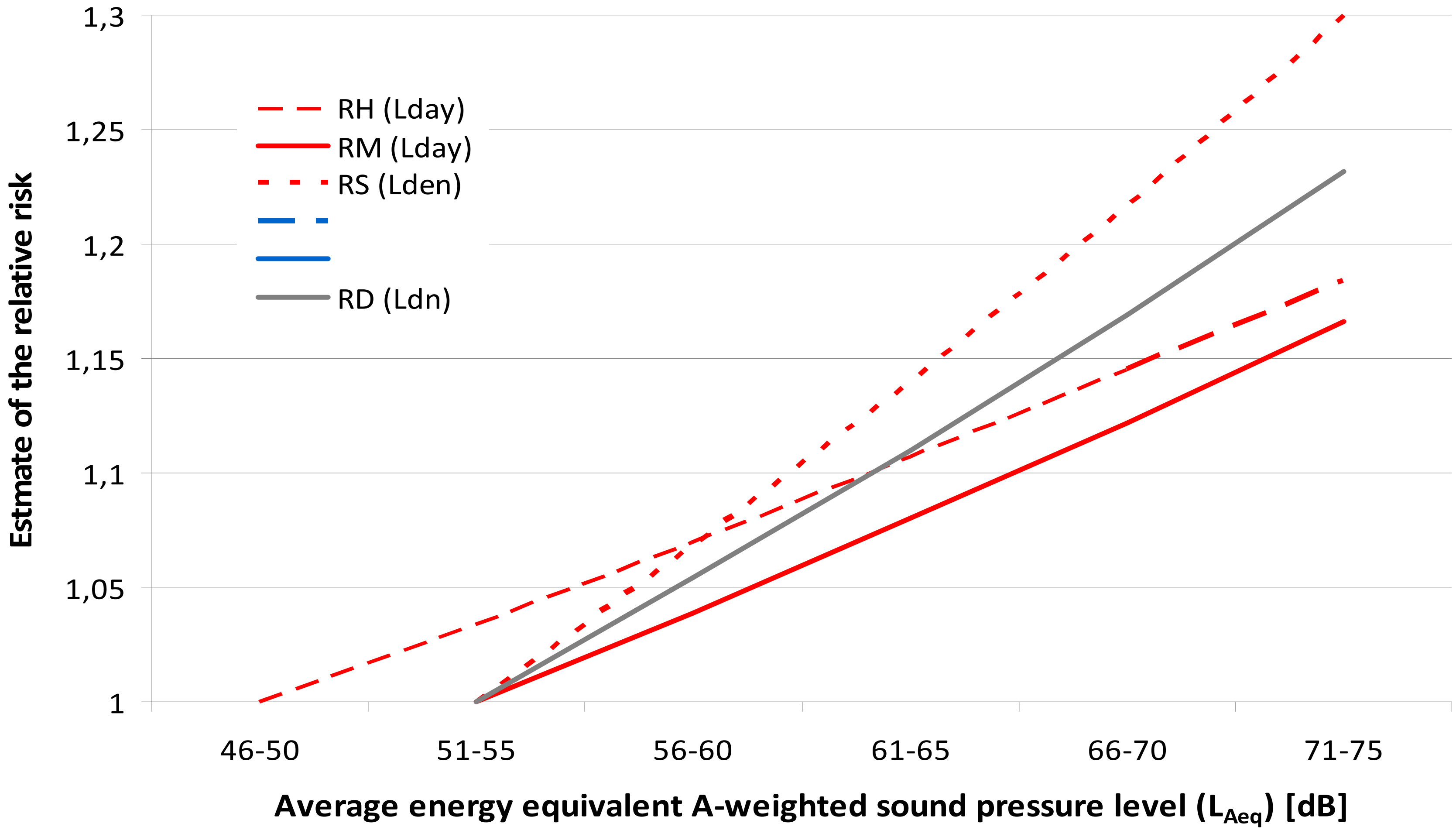
RR_i = relative risk at exposure category i compared to the reference level

N_d = number of subjects with disease in the population (disease occurrence)

Source: Prüss-Üstün et al. 2003

Exposure-Response Curves Used In The Following

For Burden Of Disease Estimation Due to Road Traffic Noise



Exposure-Response Curves Used In The Following

For Burden Of Disease Estimation Due to Road Traffic Noise

Hypertensive Heart Disease (Hypertension) *– Van Kempen and Babisch (2012)*

L_{Aeq16h}: range ~ 48-75 dB(A), $OR_{10\ dB(A)}$ = 1.07 (95% CI = 1.02-1.12)

Ischaemic Heart Disease (Myocardial Infarction) *– Babisch (2014)*

L_{DN}: range ~ 53-78 dB(A), $OR_{10\ dB(A)}$ = 1.08 (95% CI = 1.04-1.13)

Stroke *– Sørensen et al. (2011)*

L_{DEN}: range ~ 53-73 dB(A), $RR_{10\ dB(A)}$ = 1.14 (95% CI = 1.04-1.25)

Diabetes mellitus *– Sørensen et al. (2012)*

L_{DEN}: range ~ 53-73 dB(A), $OR_{10\ dB(A)}$ = 1.11 (95% CI = 1.05-1.18)

OR = Odds ratio = estimate of the relative risk

Population Attributable Risk Percentage (PAR%)

EU 27, Large Agglomerations

Disease	L_{den}<60 dB(A)	L_{den}<55 dB(A)	L_{den}<50 dB(A)
Hypertension	1.93 %	3.73 %	6.22 %
Myocardial infarction	2.22 % (1.80 %)*	4.28 %	7.12 %
Stroke	3.84 %	7.34 %	12.02 %
Diabetes mellitus	2.65 %	5.17 %	8.83 %

* WHO 2011

Noise distribution (L_{DEN}), EU-27, Major agglomerations, 2009

Burden Of Disease (Annual Rates 2004)

European Region, High Income (Total deaths = 3,809,000; DALYs = 49,331,000)

Disease	Deaths x 1,000	DALYs x 1,000
Hypertensive heart disease	69 (1.8%)	294 (0.6%)
Ischaemic heart disease	622 (16.3%)	3,376 (6.8%)
Stroke	380 (10.0%)	2,037 (4.1%)
Diabetes mellitus	98 (2.6%)	1,31 (2.7%)

25 countries, population 407,000,000
World Bank 2004 high income: gross national income per capita (GNI) ≥ 10,099 US$
% of total deaths/Dalys
Disease-specific mortality - no double counting (different ICD coding on death certificates)

Source: WHO Global burden of disease 2004 update (2008)

Attributable Mortality (2004)

European Region, High Income* (Population: N = 407,000,000)

Disease	**WHO 2012** L_{den} >60 dB(A)	**Update 2014** L_{den} >60 dB(A)	**Update 2014** L_{den} >55 dB(A)	**Update 2014** L_{den} >50 dB(A)
Hypertensive heart disease	---	1,332	2,574	**4,292**
Ischaemic heart disease	**11,196**	13,808	**26,622**	44,286
Stroke	---	14,592	**27,892**	45,676
Diabetes mellitus	---	2,597	**5,067**	8,653

Note: no double counting („one can die only once“).

$PAR = PAR\% * N_d$

63,873 Cases

* 25 countries (EU 27 = 27 countries)

Attributable DALYS (2004)

European Region, High Income* (Population: N = 407,000,000)

Disease	WHO 2012 L_{den} >60 dB(A)	Update 2014 L_{den} >60 dB(A)	Update 2014 L_{den} >55 dB(A)	Update 2014 L_{den} >50 dB(A)
Hypertensive heart disease	---	5,674	10,966	**18,287**
Ischaemic heart disease	**60,786**	74,947	**144,493**	240,371
Stroke	---	78,221	**149,516**	244,847
Diabetes mellitus	---	34,742	**67,779**	112,761

$$PAR = PAR\% * N_d$$

380,075 DALYs

* 25 countries (EU 27 = 27 countries)

History Of Noise And CVD Research

~ 1960:	Laboratory experiments with humans
~ 1970:	Animal experiments
~ 1980:	Occupational epidemiology
~ 1990:	Environmental epidemiology
~ 2000:	Quantitative risk assessment
~ 2010:	Combined effects (e. g. air pollution)

Correlation Between Noise And Air Pollutants

Roadside Measurements

Davies et al. (2009), Allen (2009) – 103 urban sites

- 2 week average NO_2, NO_x passive sampler, black smoke (BS)
- $L_{Aeq,5\ minutes}$ short-term noise measurement, distance from major road
- r = 0.53 (NO_2), r = 0.64 (NO_x), r = 0.39 (NO), r = 0.44 (BS)
- Proximity from major road was not significantly associated with NO_x.
- Ultrafine particles did not differ by road proximity.

Correlation Between Noise And Air Pollutants

Modeled Air Pollution (Land-use Regression,Dispersion Modeling)

Selander et al. (2009) – spatial exposure

Long-term exposure (NO_2) from heating and traffic, $L_{Aeq,\ 24h}$ long-term noise exposure:

r = 0.60 (noise – NO_2)

Fuks et al. (2011) – spatial exposure

Annual mean ($PM_{2.5}$, PM_{10}), L_{DEN}, Proximity to road:

r = 0.07 (noise – $PM_{2.5}$), r = 0.13 (noise – PM_{10}),
r = 0.31 (noise – proximity),
r = 0.04 ($PM_{2.5}$ – proximity), r = 0.08 (PM_{10} – proximity).

Systematic Reviews

Noise And Air Pollution

- Lekaviciute et al. (2012), Internoise 2012, Paper No. 590

  "The results suggest that noise and air pollution exert independent effects on cardiovascular health, but the evidence for any interactive effects is still limited..."

- Tétreault at al. (2013), Int. J. Public Health 58: 649-666

  "For most studies, the specific confounders produced changes in estimates <10 %. The correlation between noise and pollutants, the quality of the study and the exposure assessment do not seem to influence the confounding effects."

- Floud et al. (2013), Foraster et al. (2014)

  Noise effect disappeared after adjustment for NO_2. Collinearity-statistic (variance inflation factor) is questionable. It does not consider the meaning/rationale of a variable (e.g. modeled road noise and modeled NO_2 are both determined by traffic volume ➔ over-adjustment (“NO_2 has a something meaning as road traffic noise”).

Noise Versus Air Pollution

- Different mechanisms: PM - inflammation / noise - stress
- Noise assessment is less affected by background exposures than air pollution assessment (no long-range transport)
- No accumulation or latency in the atmosphere ("no cars, no noise")
- Proximity to the road is not exclusively an indicator of the exposure to air pollutants (noise correlates better)
- Well defined physical propagation rules for noise (individual exposure)
- Noise independent of meteorology (in urban distances)
- Obstacles (orientation of rooms) may be more effective for noise
- Effects were found with respect to sound sources other than road traffic (aircraft noise, occupational noise)
- Day-night differences (stronger effects for night noise / less air pollution)

Evidence

- Laboratory experiments on humans
  (acute effects, high and moderate noise levels)
- Animal experiments
  (long-term effects, high noise levels)
- Occupational noise studies
  (long-term effects on humans, high noise levels)
- Environmental noise studies
  (long-term effects on humans, moderate noise levels)

Epidemiological Reasoning (Causality)

- Hypothesis
- Biological plausibility
- Consistency of study results
  (different populations, different methodology)
- Temporality
- Exposure-response relationship
- Magnitude of effect (public health relevance)

Source-specific Exposure-response Curves

Different sound characteristics

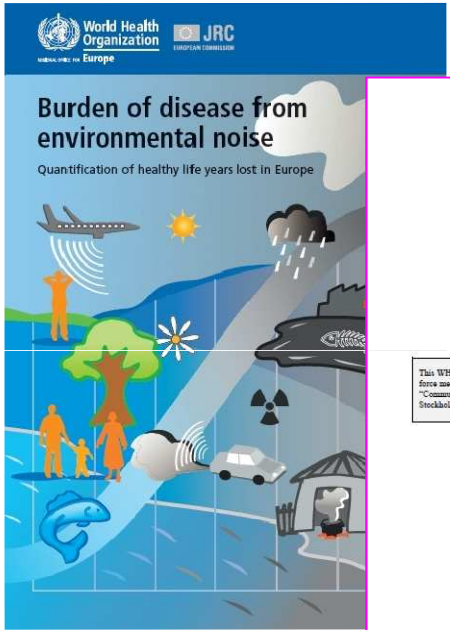
- (Maximum) sound levels
- Noise level rise time
- Time course
- Frequency spectrum
- Tonality
- Informational content

Research Needs

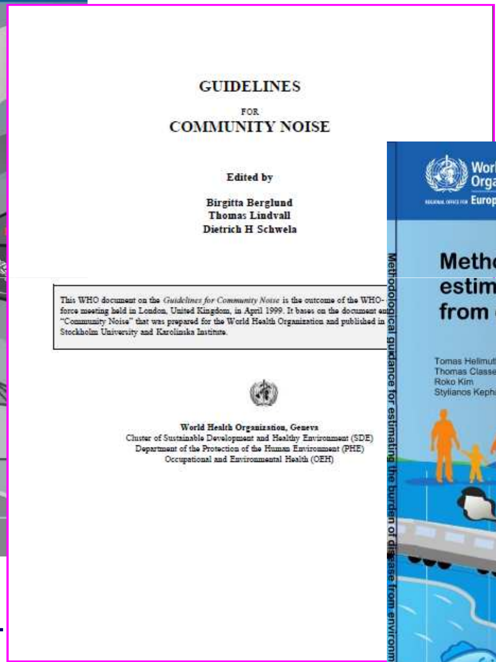
- Refine exposure-response curves
- Threshold of effect?
- Sound levels below 50 dB(A) - reference
- Quiet side / sound insulation
- Day/night differences
- New endpoints (metabolic syndrome, dementia)
- Other noise sources (rail, low frequencies)
- Role of air pollutants (road traffic)
- Combined exposures (noise and other)
- Impact on public health (country-specific)

Thank You !

Important Documents



WHO, 2011


GUIDELINES
FOR
COMMUNITY NOISE

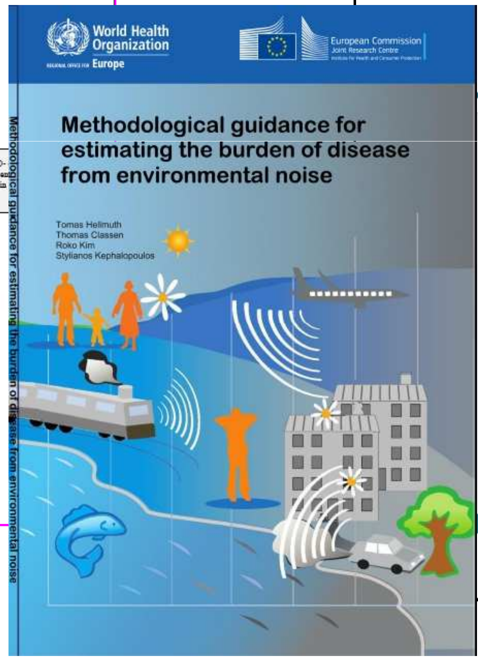
Edited by

Birgitta Berglund
Thomas Lindvall
Dietrich H Schwela

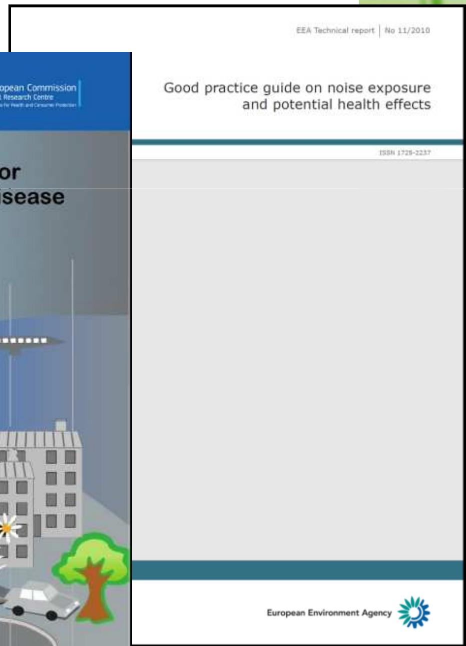
This WHO document on the *Guidelines for Community Noise* is the outcome of the WHO-force meeting held in London, United Kingdom, in April 1999. It bases on the document entitled "Community Noise" that was prepared for the World Health Organization and published in Stockholm University and Karolinska Institute.

World Health Organization, Geneva
Cluster of Sustainable Development and Healthy Environment (SDE)
Department of the Protection of the Human Environment (PHE)
Occupational and Environmental Health (OEH)

WHO, 1999



WHO, 2012



EEA, 2010

WHO, 2009