Cardiovascular Burden of Disease from Environmental Noise
Evidence, Uncertainties and Public Health Implications

Wolfgang Babisch
Department of Environmental Hygiene
Federal Environment Agency
Berlin, Germany
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

1.6 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.
**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

<table>
<thead>
<tr>
<th></th>
<th>YLL</th>
<th>YLD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annoyance</td>
<td>0</td>
<td>654,086</td>
</tr>
<tr>
<td>Self-reported sleep disturbance</td>
<td>0</td>
<td>903,000</td>
</tr>
<tr>
<td>Heart disease</td>
<td>29,488</td>
<td>30,147</td>
</tr>
</tbody>
</table>

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).

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!
Short-term – Long-term Effects

Exposure

Stress indicators
Biological risk factors

Disease

Long-term effect (epidemiology)

Medical science

Short-term experiment
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 $\mu$g/m$^3$".

No or incomplete habituation/adoptive.
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
  - Nerval interaction
    (physiological stress)
  - Autonomic nervous system
  - Endocrine system
    ↓
  - Dysregulation
    Long-term health effects

- Indirect pathway
  - Cortical perception
    (emotional stress)
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)
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

~ 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)
**Occupational Noise Studies**

**Reviews – Hypertension**

Thompson (1993): prevalence ratios ranged from 2.0 to 2.8

v. Kempen et al. (2002): meta-analysis (9 studies)

\[ \text{OR}_{10 \text{ dB}(A)} = 1.30 \ (95\% \ CI = 1.02-1.66) \]

range \( L_{\text{Aeq8h}} \) ~ 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))

**Studies – Myocardial Infarction / Coronary Heart Disease**

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

Thompson (1993): prevalence ratios ranged from 2.0 to 2.8
v. Kempen et al. (2002): meta-analysis (9 studies)
\[ \text{OR}_{10\text{ dB}(A)} = 1.30 \ (95\% \ CI = 1.02-1.66) \]
range L\text{Aeq8h} \sim 55-116 \text{ 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))

Studies – Myocardial Infarction / Coronary Heart Disease

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

L_{\text{Aeq8h}} \geq 95 \text{ dB(A)}: 50-150% increase in risk
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)
- Alzheimer 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
• 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)
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:**

\[
OR = 1.629657 - 0.000613(L_{\text{day,16h}})^2 + 0.000007357(L_{\text{day16h}})^3, \quad R^2 = 0.96
\]

Source: Babisch, 2008
**Exposure-response function:**

OR per 10 dB(A) = 1.17, 95% CI = 0.87-1.57, range $L_{Aeq16h} = 58-78$ dB(A)
Road Traffic Noise – Coronary Heart Disease
Updated Meta-Analysis (Forest Plot)

Pooled estimate: 1.08 (95% CI = 1.04-1.13)

Source: Babisch, 2014
Cumulative Risk Of Studies
By Year - Road Traffic Noise

1993
- Caerphilly - CS m
- Speedwell - CS m
- Berlin I - hCC m
- Berlin II - pCC m
- Berlin II - CS m
- Tokyo - CS f
- Caerphilly - CO f
- Speedwell - CO m
- Berlin III - hCC f
- Berlin III - hCC m
- Copenhagen plus - CO f
- Stockholm - pCC f+m
- Stockholm plus - CS f+m
- Vancouver - CO m
- Vancouver - CO f
- Netherlands - CO f+m

2009

2012

Relative risk [L_DN per 10 dB(A)]

Source: Babisch, 2014
**Myocardial Infarction (5 studies)**  – Babisch (2008)

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

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

**Myocardial Infarction (14 studies)**  – Babisch (2014)

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

• **8%** increase in risk per 10 dB(A) increase in noise level
<table>
<thead>
<tr>
<th>Study - type (Country)</th>
<th>Reference</th>
<th>Number of subjects</th>
<th>Number of cases</th>
<th>Response rate</th>
<th>Covariates</th>
<th>Age</th>
<th>Exposure assessment</th>
<th>Health outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berlin I - hCC (Germany) - hCC</td>
<td>Babisch et al. 1994[27]</td>
<td>M: 243</td>
<td>M: 109</td>
<td>Cases: 89</td>
<td>A, G, B, C, S</td>
<td>41-70</td>
<td>$L_{A_{eq,16hr}} \leq 55$ to $&gt;65$</td>
<td>Incident MI (clinical)</td>
</tr>
<tr>
<td>Berlin II - pCC (Germany)</td>
<td>Babisch et al. 1994[27]</td>
<td>M: 4.035</td>
<td>M: 645</td>
<td>Cases: 91</td>
<td>A, G, B, C, S</td>
<td>31-70</td>
<td>$L_{A_{eq,16hr}} \leq 60$ to $&gt;75$</td>
<td>Incident MI (clinical)</td>
</tr>
<tr>
<td>Berlin II - CS (Germany)</td>
<td>Babisch et al. 1994[27]</td>
<td>M: 2.375</td>
<td>M: 206</td>
<td>Controls: 64</td>
<td>A, G, B, C, S</td>
<td>31-70</td>
<td>$L_{A_{eq,16hr}} \leq 60$ to $&gt;75$</td>
<td>Prevalent MI (self-reported)</td>
</tr>
<tr>
<td>Tokyo - CS (Japan)</td>
<td>Yoshida et al. 1997[31]</td>
<td>F: 3.950</td>
<td>F: 305</td>
<td>73</td>
<td>A, G</td>
<td>20-60</td>
<td>$L_{A_{eq,16hr}} \leq 55$ to $&gt;70$</td>
<td>Prevalent CHD (self-reported)</td>
</tr>
</tbody>
</table>
Danish Road Traffic Noise Studies
Stroke And Heart Attack, N = 57,053 subjects

**Stroke**
\[ \text{OR}_{10 \, \text{dB}} = 1.14 \]

**Heart attack**
\[ \text{OR}_{10 \, \text{dB}} = 1.12 \]

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
Hypertension (24 studies) – Van Kempen and Babisch (2012)

$L_{\text{Aeq}16\text{h}}$: range ~ 48-75 dB(A), $\text{OR}_{10 \text{ dB(A)}} = 1.07$ (95% CI = 1.02-1.12)

• 7% increase in risk per 10 dB(A) increase in noise level
Road Traffic Noise – Other Health Endpoints
Single Large Study Results

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

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

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

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

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

• **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

**Hypertension (5 studies)** – Babisch and van Kamp (2009)

L\(_{DN}\): range ~ 48-68 dB(A), OR\(_{10 \text{dB}(A)}\) = 1.13 (95% CI = 1.00-1.28)

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

**Myocardial Infarction (1 study)** – Huss et al. (2010)

L\(_{DN}\): range ~ 48-63 dB(A), HR\(_{10 \text{dB}(A)}\) ~ 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

Armagh, 25 October 2012
Synthesis Of Cardiovascular Risk Curves
Environmental Noise

Estimate of the relative risk

Average energy equivalent A-weighted sound pressure level ($L_{Aeq}$) [dB]
Estimate of the relative risk

\[ \text{Relative Risk (Lday)} \]

\[ \text{Relative Risk (Lden)} \]

\[ \text{Relative Risk (Ldn)} \]

\[ \text{Relative Risk (A-weighted sound pressure level} (L_{Aeq}) \text{ dB)} \]

\[ L_{DEN} \geq 65 \text{ dB(A)}: \text{ 15-25% increase in risk} \]
Noise, Sleep And Health

Night noise → Sleep quality → Health

Source: WHO, 2009
Day / Night
Six Airports Study ('HYENA')

Cross-sectional study, 4861 males + females, aged 45-70 yrs
Prevalence of high blood pressure

Per 10 dB(A):

\[ \text{OR}_{\text{Day}} = 1.02 (0.95-1.10) \]

\[ \text{OR}_{\text{Night}} = 1.07 (1.00-1.15) \]

Source: Jarup et al., 2008
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

Exposure Modifiers
Caerphilly & Speedwell Studies

Relative risk
OR ± 95% CI

0
0.25
0.5
0.75
1
1.25
1.5
1.75
2
2.25
2.5

Address only
+ Window orientation
+ Open window
+ 15 yrs residence

OR = Odds ratio = estimate of the relative risk

Source: Babisch et al. (1999)

ICBEN 2014

Babisch - 38
Berlin Noise Map
Location Of Rooms
Exposure Modifiers

Street which is the postal address

Source: Babisch et al., 2014 in print
## Location Of Rooms
### Berlin-4 Study

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

Source: Babisch et al., 2014 in print
Exposure Assessment

KORA Study
Road Traffic Noise – Hypertension
N = 4,166

Source: Babisch et al., 2014

City of Augsburg
Greater Augsburg

Source: Babisch et al., 2014
**KORA Study**
Road Traffic Noise – (Systolic) Hypertension

<table>
<thead>
<tr>
<th>Hypertension</th>
<th>OR&lt;sub&gt;10 dB(A)&lt;/sub&gt;</th>
<th>CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>City of Augsburg:</td>
<td>1.16</td>
<td>1.00-1.35</td>
</tr>
<tr>
<td>Greater Augsburg:</td>
<td>0.94</td>
<td>0.81-1.09</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Isolated systolic hypertension</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>City of Augsburg:</td>
<td>1.48</td>
<td>1.16-1.89</td>
</tr>
<tr>
<td>Greater Augsburg:</td>
<td>0.88</td>
<td>0.69-1.12</td>
</tr>
</tbody>
</table>

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
~ 1970: Animal experiments
~ 1980: Occupational epidemiology
~ 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

\[ \text{PAR}\% = \frac{\sum (P_i \times RR_i) - 1}{\sum (P_i \times RR_i)} \times 100\% ] \]

\[ \text{PAR} = \text{PAR}\% \times N_d \]

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

![Graph showing exposure-response curves]

- RH (Lday)
- RM (Lday)
- RS (Lden)
- RD (Ldn)

Estimate of the relative risk

<table>
<thead>
<tr>
<th>Average energy equivalent A-weighted sound pressure level ($L_{Aeq}$) [dB]</th>
<th>Estimate of the relative risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>46-50</td>
<td>1.00</td>
</tr>
<tr>
<td>51-55</td>
<td>1.05</td>
</tr>
<tr>
<td>56-60</td>
<td>1.10</td>
</tr>
<tr>
<td>61-65</td>
<td>1.15</td>
</tr>
<tr>
<td>66-70</td>
<td>1.20</td>
</tr>
<tr>
<td>71-75</td>
<td>1.25</td>
</tr>
</tbody>
</table>
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)

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

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

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

**Stroke** – Sørensen et al. (2011)

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

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

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

OR = Odds ratio = estimate of the relative risk
### Population Attributable Risk Percentage (PAR%)

**EU 27, Large Agglomerations**

<table>
<thead>
<tr>
<th>Disease</th>
<th>$L_{\text{den}&lt;60 \text{ dB(A)}}$</th>
<th>$L_{\text{den}&lt;55 \text{ dB(A)}}$</th>
<th>$L_{\text{den}&lt;50 \text{ dB(A)}}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>1.93 %</td>
<td>3.73 %</td>
<td>6.22 %</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>2.22 % (1.80 %)*</td>
<td>4.28 %</td>
<td>7.12 %</td>
</tr>
<tr>
<td>Stroke</td>
<td>3.84 %</td>
<td>7.34 %</td>
<td>12.02 %</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2.65 %</td>
<td>5.17 %</td>
<td>8.83 %</td>
</tr>
</tbody>
</table>

*WHO 2011

Noise distribution ($L_{\text{DEN}}$), EU-27, Major agglomerations, 2009
Burden Of Disease (Annual Rates 2004)
European Region, High Income (Total deaths = 3,809,000; DALYs = 49,331,000)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Deaths x 1,000</th>
<th>DALYs x 1,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive heart disease</td>
<td>69 (1.8%)</td>
<td>294 (0.6%)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>622 (16.3%)</td>
<td>3,376 (6.8%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>380 (10.0%)</td>
<td>2,037 (4.1%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>98 (2.6%)</td>
<td>1,311 (2.7%)</td>
</tr>
</tbody>
</table>


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)
# Attributable Mortality (2004)

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

<table>
<thead>
<tr>
<th>Disease</th>
<th>WHO 2012 $L_{den} &gt; 60$ dB(A)</th>
<th>Update 2014 $L_{den} &gt; 60$ dB(A)</th>
<th>Update 2014 $L_{den} &gt; 55$ dB(A)</th>
<th>Update 2014 $L_{den} &gt; 50$ dB(A)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive heart disease</td>
<td>---</td>
<td>1,332</td>
<td>2,574</td>
<td>4,292</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>11,196</td>
<td>13,808</td>
<td>26,622</td>
<td>44,286</td>
</tr>
<tr>
<td>Stroke</td>
<td>---</td>
<td>14,592</td>
<td>27,892</td>
<td>45,676</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>---</td>
<td>2,597</td>
<td>5,067</td>
<td>8,653</td>
</tr>
</tbody>
</table>

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

\[
PAR = PAR\% \times N_d
\]

63,873 Cases

* 25 countries (EU 27 = 27 countries)
### Attributable DALYS (2004)

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

<table>
<thead>
<tr>
<th>Disease</th>
<th>WHO 2012 $L_{\text{den}} &gt; 60$ dB(A)</th>
<th>Update 2014 $L_{\text{den}} &gt; 60$ dB(A)</th>
<th>Update 2014 $L_{\text{den}} &gt; 55$ dB(A)</th>
<th>Update 2014 $L_{\text{den}} &gt; 50$ dB(A)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive heart disease</td>
<td></td>
<td>5,674</td>
<td>10,966</td>
<td>18,287</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>60,786</td>
<td></td>
<td>144,493</td>
<td>240,371</td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td>78,221</td>
<td>149,516</td>
<td>244,847</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td>34,742</td>
<td>67,779</td>
<td>112,761</td>
</tr>
</tbody>
</table>

\[
\text{PAR} = \text{PAR}\% \times N_d
\]

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 NO₂, NOₓ passive sampler, black smoke (BS)
- $L_{Aeq,5 \text{ minutes}}$ short-term noise measurement, distance from major road
- $r = 0.53$ (NO₂), $r = 0.64$ (NOₓ), $r = 0.39$ (NO), $r = 0.44$ (BS)
- Proximity from major road was not significantly associated with NOₓ.
- 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, $L_{\text{Aeq},24h}$ long-term noise exposure:
$r = 0.60$ (noise – NO$_2$)

Fuks et al. (2011) – spatial exposure
Annual mean (PM$_{2.5}$, PM$_{10}$), $L_{\text{DEN}}$, 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).
Lekaviciute et al. (2012), Interoise 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 NO₂. Collinearity-statistic (variance inflation factor) is questionable. It does not consider the meaning/rationale of a variable (e.g. modeled road noise and modeled NO₂ are both determined by traffic volume ➔ over-adjustment (“NO₂ 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
- WHO, 1999
- WHO, 2009
- WHO, 2012
- EEA, 2010
- WHO, 2011
- WHO, 2012
- WHO, 2009
- WHO, 2012