NOISE CONTROL FOR QUALITY OF LIFE

# The relationship between traffic noise exposure and ischemic heart disease: a meta-analysis

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## ABSTRACT

Since publication of the World Health Organization's report Burden of disease for environmental noise in 2011, several new studies on traffic noise and ischemic heart disease (IHD) have been published. There is thus a need for a meta-analytic update of risk estimates for these outcomes. We conducted a systematic review and retained published cohort and case-control studies using road, rail or aircraft noise as exposure. Study-specific results were transformed into risk estimates per 10dB using generalised least squares for trend estimation of summarised dose–response data. Subsequently a random effects meta-analysis was conducted. We identified 7 studies for IHD incidence and 4 for mortality. Per 10dB increase in noise, the risk estimate was 1.08 (95%CI: 1.03-1.14) for IHD incidence (total of 6,000 new cases). The risk estimate for IHD or myocardial infarction mortality accounting for a total of 22,000 deaths was 1.04 (0.98-1.09). Combined incidence and mortality risk for IHD was 1.05 (1.02-1.09). Preliminary results confirm an increase risk in IHD incidence with traffic noise exposure, and subgroup analyses suggest higher risk for MI compared to all IHD combined, for males compared to females, for road traffic noise compared to aircraft noise and for studies without air pollution adjustment compared to those with adjustment. Future analysis will address the form of the exposure-response curve before the risk estimates will be used in an assessment of the external health costs of traffic in the Swiss population.
Keywords: Noise, Exposure, Ischemic heart disease (IHD)

## 1. INTRODUCTION

Noise exposure from road, rail and aircraft traffic, especially in urban areas, is one of the most widespread sources of environmental stress in the daily lives. There is much evidence supporting the relationship between exposure to traffic related noise and wellbeing. In addition to causing psychological effects and stresses, such as annoyance and sleep disturbance, noise is postulated to induce biological stress on the body such as changes in blood pressure and to cause hypertension [1-6]. Most studies have investigated noise from road traffic and aircraft, although noise from railway traffic is also a concern.

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As indicated by this association with hypertension, the proposed biological mechanism by which noise exerts health effects is through stress on the cardiovascular system. In general, biological reaction models have shown noise to be a nonspecific stressor that arouses the autonomous nervous system and endocrine system [7]. More recently, therefore, research on cardiovascular disease (CV) disease has specifically addressed the hypothesis that exposure to traffic noise increases the risk for ischemic heart disease (IHD) [8-10].

An early study [6] compiled and meta-analysed studies on myocardial infarction (MI) and road traffic noise [9, 11, 12] to derive exposure-response curves, which were reported in the 2011 WHO Burden of Disease (BOD) from environmental noise report [7]. Although the WHO report is relatively recent, including studies conducted from the mid-1990s to 2005, it does not include estimates from the latest epidemiological noise research. The new studies included in this meta-analysis were sought, and selected on the basis of having also adjusted for air pollution levels. This is important given that noise and air pollution exposures are often highly correlated, both deriving from traffic sources, and are further both associated with CV disease [13, 14].

This paper thus updates the meta-analysis on exposure to traffic noise and IHD, including studies where co-exposure to air pollution is evaluated offering the opportunity to evaluate potential heterogeneity in the combined risk estimates.

## 2. METHODS

### 2.1 Study Selection and Data Extraction

We conducted a systematic review of recent cohort and case-control studies, retaining papers using road, rail or aircraft noise as exposure and myocardial infarction (MI) or ischemic heart disease (IHD) as outcomes. The search was conducted in PubMed for the period 2005 to present. No geographic constraints were defined, however the search was conducted in and limited to publications in the English language.

In addition to new studies revealed by the search, original publications for studies pre-2005 as provided in the 2011 WHO report [7] were also obtained. Individual studies were evaluated for relevance based on mention of exposure to traffic noise (road, rail or aircraft) in relation to MI or IHD in the title and/or abstract. Studies which quantified this relationship in dB, either categorically or by a linear trend, were retained. Data extraction included recording the risk estimates by noise exposure categories (including reference level), noise metric, noise source, study population by sex, study design, and whether the risk estimate was adjusted for air pollution.

### 2.2 Linear Dose Response Estimation

Risk estimates from individual studies based on categorical noise exposures were transformed into a linear risk estimate (per 10dB increase) using general least square estimation accounting for trend (using the STATA glst command [15]). Where this was not possible, due to missing values in the matrix, variance-weighted least squares (STATA vwls command) was used instead.

A log-normal model in SAS was first fitted to the data to estimate the mean level of exposure in each of the exposure intervals. This was done on the basis of the proportion of person years or number of cases for cohort and case-control studies, respectively. In order to be able to deal with open exposure intervals, the exposure range was assumed to be limited by 0 and 100dB, respectively.

If necessary the mean noise levels for each category were next converted to Lden based on the following approximations:
- Lden=L16h+2dB for road traffic in urban environments [7]
- Lden=Ldn+0.3dB [16]
- Lden=LAeq,24 + 1.5dB [17]

Finally, the mean noise level for each category was translated into a noise increment by zeroing on the respective reference level.

### 2.3 Meta-analysis

Random effects meta-analysis (using STATA metan [18]) was conducted based on the risk estimates per 10dB increase in Lden noise for the individual studies. Where, in individual studies, effect estimates were reported for males, females and both combined, the sex-specific estimates were used in the main analysis.

Subsequent stratified analyses were also conducted to explore potential sources of heterogeneity including: disease state (morbidity vs. mortality); disease outcome (MI specific vs. unspecified IHD); sex; noise reference level; type of noise source (road, rail, aircraft); and adjustment for air pollution.

Analysis was conducted in STATA 12.

## 3. RESULTS

### 3.1 Selected Studies

The recent meta-analysis for MI in the 2011 WHO report [7] contains 4 risk estimates from analytical studies pertaining to road traffic noise conducted prior to 2005 [9, 11, 12]. To date we have identified five additional epidemiological studies, conducted mainly in Europe, focusing on road and aircraft noise and IHD incidence or mortality [19-23]. Four of the new studies [19-22] relate to road traffic noise while one large Swiss National Cohort (SNC) by Huss et al. [23] focused on aircraft noise. No studies were found specifically for noise due to railway traffic. Unlike the studies conducted prior to 2005, all of the recent studies included here have adjusted for air pollution.

Conversion of risk estimates to a 10dB increment was required for all studies except the two studies conducted in 2012 [19, 22].

### 3.2 Main Effect Estimate

All studies combined, regardless of disease state, CV outcome, and noise source, resulted in overall significant risk estimate of 1.05 (95%CI: 1.02-1.09) per 10dB increase in noise exposure. The main result is presented in the Forest plot (Figure 1), showing the effect estimates per 10dB increase in traffic noise (Lden) and association with IHD incidence and mortality (also shown in Table 1).

For incident IHD, based on a total of 6,000 new cases, the risk estimate was 1.08 (1.03-1.14) per 10dB increase in road traffic noise. There was no heterogeneity at all (i.e., I2 = 0% and tau = 0). This risk estimate is largely based on studies specifically focusing on MI, all exploring associations with road traffic noise. The two recent studies combined both sexes [19, 20] while those conducted pre-2005 were stratified by sex. Most of the pre-2005 studies looked only at IHD in men while only one also considered women [11].

The risk estimate for IHD or specifically MI mortality accounting for a total of 22,000 deaths was not significant with a risk estimate of 1.04 (0.98-1.09). Three of the 4 studies were for road noise exposure [20-22] while one, Huss et al. [23], the large SNC study mentioned above, related to aircraft noise. Some heterogeneity among the studies was detected (I2 = 44.9%).
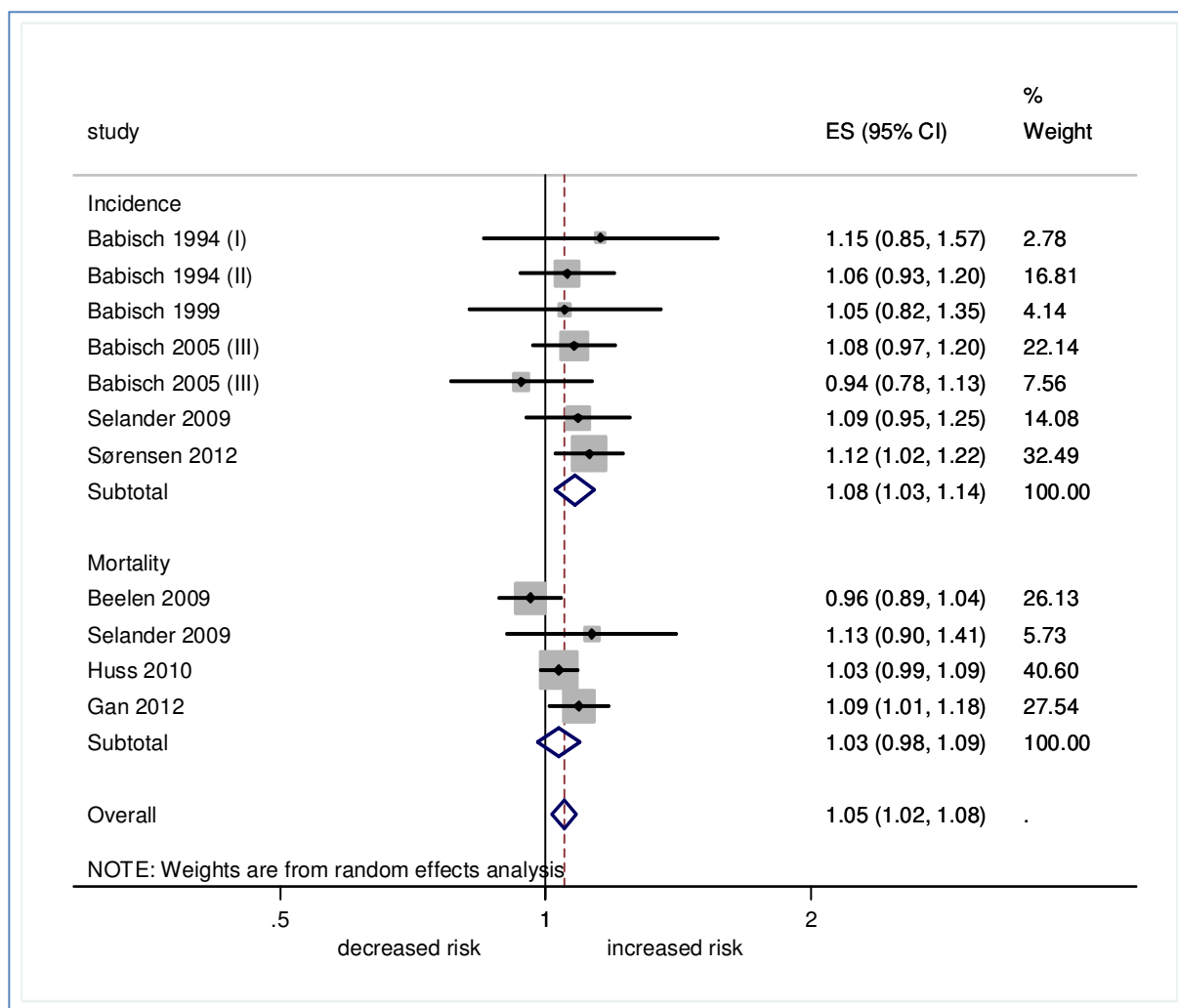
Figure 1 – Forest plot of effect estimates per 10dB increase in traffic noise (Lden) and association with IHD incidence and mortality

### 3.3   Subgroup Analysis

We further explored potential changes in the effect estimates related to relevant factors including source of traffic noise (road vs. aircraft), age, sex, and whether air pollution exposure was considered in the original studies. The results of these subgroup analyses are presented in Table 1. Most subgroup analyses showed moderate, though not always statistically significant, heterogeneity with $I2 > 40\%$ in at least one strata.

Regarding disease outcome, the risk estimates were greater for MI specific [1.06 (1.02-1.10)] compared to all IHD combined (i.e. the main effect model: 1.05 (1.02-1.09)). Some heterogeneity was observed among studies on unspecified IHD, though there were few studies in this category and the association was not significant [1.03 (0.93-1.13)].

Risk estimates for males [1.10 (1.03-1.16)] were greater than for females [1.06 (0.92-1.16)].

Where the noise reference level for individual studies was >=50dB, the risk estimate per 10dB was elevated [1.08 (1.03-1.13)] compared to the main effect model.

Including the one study for aircraft noise, with a large population, does not substantially alter the risk estimate for noise and IHD: 1.05 (1.02-1.09) all studies compared to 1.06 (1.01-1.10) for road traffic noise only.

Stratification of studies on the basis of air pollution adjustment introduced significant heterogeneity into both subgroups. Risk estimates for studies not adjusting for air pollution [1.07 (1.01-1.14)] were elevated compared to the main effect model. Stratification by study date, however, does not alter the risk estimate compared to the main effect model.

Table 1 – Subgroup meta-analyses

| Subgroup | Number of estimates | Effect Estimate (per 10dBA) | 95% CI | Heterogeneity | | |
|---|---|---|---|---|---|---|
| | | | | p* | I2 | tau2 |
| None | 10 | 1.05 | 1.02-1.09 | 0.34 | 11.1% | 0.0003 |
| Disease state[1] | | | | | | |
|   Morbidity (incidence) | 7 | 1.08 | 1.03-1.14 | 0.78 | 0.0% | 0.0000 |
|   Mortality | 4 | 1.04 | 0.98-1.09 | 0.14 | 44.9% | 0.0014 |
| Disease outcome | | | | | | |
|   MI specific | 7 | 1.06 | 1.02-1.10 | 0.59 | 0.0% | 0.0000 |
|   Unspecified IHD | 3 | 1.03 | 0.93-1.13 | 0.09 | 58.5% | 0.0041 |
| Sex[2] | | | | | | |
|   Males | 3 | 1.10 | 1.03-1.16 | 0.63 | 0.0% | 0.0000 |
|   Females | 3 | 1.06 | 0.92-1.16 | 0.30 | 17.3% | 0.0013 |
| Noise reference level | | | | | | |
|   >=50 dBA | 7 | 1.08 | 1.03-1.13 | 0.78 | 0.0% | 0.0000 |
|   <50 dBA | 3 | 1.02 | 0.96-1.09 | 0.17 | 43.3% | 0.0013 |
| Type of Noise | | | | | | |
|   Road traffic | 9 | 1.06 | 1.01-1.10 | 0.29 | 17.8% | 0.0008 |
|   Aircraft | 1 | 1.04 | 0.99-1.09 | - | - | - |
| Air pollution[2] | | | | | | |
|   No | 4 | 1.07 | 1.01-1.14 | 0.01 | 74.6% | 0.0028 |
|   Yes | 4 | 1.05 | 0.99-1.11 | 0.06 | 60.6% | 0.0021 |
| Study date | | | | | | |
|   Pre-2005 | 5 | 1.05 | 0.98-1.13 | 0.73 | 0.0% | 0.0000 |
|   Post-2005 | 5 | 1.05 | 1.00-1.11 | 0.09 | 50.6% | 0.0016 |

* p-value of Chi-square
1 Study N exceeds 10 because cause-specific estimates used where available
2 Limited to studies with estimates for both strata

## 4. DISCUSSION

In an attempt to update the recent meta-analysis for MI in the 2011 WHO report [7], five additional studies, mainly conducted in Europe, have been identified for IHD incidence or mortality and traffic noise exposure. Three of these relate to MI incidence [19, 20, 23], with one based on the Swiss population in the SNC [23]. IHD mortality has been addressed in a Dutch cohort [21] as well as in Stockholm [20] and Vancouver [22]. In total these new studies contribute ca. 28,000 deaths or incident cases of IHD and have a combined total population of ca. 5.2 million (4.6 of which is from the SNC study). The new studies have used modelled traffic noise, rather than measurements, to assess exposure at the home address. All focus on road traffic noise except the SNC which explores aircraft noise as the main exposure. Additionally, all adjust for air pollution which was not the case in older studies.

Our risk estimates in general were found to be lower than that derived in the previous meta-analysis by Babisch [6, 7]. Based on four studies on MI and road traffic exposure, they found an OR of 1.17 (0.87-1.57) whereas we found a lower yet significant result of 1.05 (1.02-1.09) when

including a further 4 studies, one of which was for aircraft noise. Babisch [6], however, used a different methodological approach to pool the risk estimates, which strikingly resulted in considerably wider confidence intervals. Furthermore, none of the studies in the meta-analysis by Babisch [6] included adjustment for air pollution which seems to attenuate the risk estimates.

One source of uncertainty in our meta-analysis is the conversion of categorical risk estimates to a risk increase per 10dB, since this conversion implies the absence of a threshold below which no effects occur. Whether this reflects the true exposure-response association is, at present, not known. A graphical non-parametric analysis in Sørensen et al. [19] does suggest a rather linear exposure-response function for MI in the Lden range of 40 to 80dB, which is also relevant for this meta-analysis. On the other hand, recent health impact assessment applied either a threshold of 55 dB [7] or 60dB [16] for Lden.

Our subgroup analyses suggest higher risk for MI compared to all IHD combined, for males compared to females, for studies with a higher reference threshold, for road traffic noise compared to aircraft noise and for studies without air pollution adjustment compared to those with adjustment. The number of studies, however, is small and tended to be clustered, which prevents an independent evaluation of these factors. The early studies (pre-2005) tended to look mainly at IHD incidence in males without air pollution adjustment and applying a high reference noise exposure level. Should more studies be available on this topic in the future meta-regression will enable independent evaluation of the relevance of these factors.

## 5. CONCLUSIONS

Noise exposure from road, rail and aircraft traffic, especially in urban areas, is one of the most widespread sources of environmental stress in the daily lives. Preliminary results confirm an increased risk of 1.08 and 1.04 in IHD incidence and mortality, respectively, with traffic noise exposure. Future analysis will include further subgroup analysis, by age and years in residence, as well as investigating the form of the exposure-response curve before the risk estimates will be used in an assessment of the external health costs of traffic in the Swiss population.

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