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The relationship between transportation noise exposure and ischemic heart disease: A meta-analysis

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ABSTRACT

Background: There is a growing body of evidence that exposure to transportation related noise can adversely affect health and wellbeing. More recently, research on cardiovascular disease has specifically explored the hypothesis that exposure to transportation noise increases the risk for ischemic heart disease (IHD). Our objective was to review and conduct a meta-analysis to obtain an overall exposure–response association.

Methods and results: We conducted a systematic review and retained published studies on incident cases of IHD using sources of transportation noise as exposure. Study-specific results were transformed into risk estimates per 10 dB increase in exposure. Subsequently we conducted a random effects meta-analysis to pool the estimates. We identified 10 studies on road and aircraft noise exposure conducted since the mid-1990s, providing a total of 12 risk estimates. Pooled relative risk for IHD was 1.06 (1.03–1.09) per 10 dB increase in noise exposure with the linear exposure–response starting at 50 dB. Based on a small number of studies, subgroup analyses were suggestive of higher risk for IHD for males compared to females ($p=0.14$), and for persons over 65 years of age compared to under ($p=0.22$). Air pollution adjustment, explored only in a subset of four studies, did not substantially attenuate the association between noise exposure and IHD.

Conclusions: The evidence for an effect of transportation noise with IHD necessitates further research into the threshold and the shape of the exposure–response association, potential sources of heterogeneity and effect modification. Research in different cultural contexts is also important to derive regional and local estimates for the contribution of transportation noise to the global burden of disease.

1. Introduction

Noise exposure from transportation, 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 environmental noise and wellbeing. Basner et al. (2013) and Munzel et al. (2014) provide a concise review of the effects of noise, including environmental noise, on health. In addition to causing sleep disturbance and psychological effects such as annoyance, noise is postulated to induce biological stress on the cardiovascular system, leading to changes in blood pressure and to cause hypertension (EC, 2002; Laszlo et al., 2012; van Kempen and Babisch, 2012; Hume et al., 2012; van Kempen et al., 2002; Babisch, 2008). Most studies have investigated these and other non-auditory health effects of noise from road and aircraft traffic (e.g. cognitive impairment in children (Clark and Stansfeld, 2007) and diabetes in adults (Sørensen et al., 2013)), although noise from railways is also a concern. For example, Croy et al. (2013) demonstrated experimentally that night-time freight train noise and vibration can accelerate heart rate during sleep, which may in turn be linked to cardiovascular disease (CVD). Although less studied, recent research on the potential relationship between transportation noise and ischemic heart disease (IHD) has yielded inconsistent results (Selander et al., 2009; Beelen et al., 2009; Gan et al., 2012; Huss et al., 2010; Willich et al., 2006).

Babisch previously performed meta-analyses on studies of road traffic noise exposure and IHD. The first included five studies on incident myocardial infarction (MI) and reported a relative risk of 1.17 (95% CI 0.87–1.57) per 10 dB increase in daytime (Lday) noise (Babisch, 2008). Recently updated, the study by Babisch (Babisch, 2014) included 17 studies (incidence or prevalence) suggesting a

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relative risk of 1.08 (95% CI 1.04–1.13) per 10 dB increase (Ldn) in road noise. Other transportation sources, however, were not considered. In this paper, we also perform a meta-analysis on available studies on the association between exposure to any transportation noise and IHD. We expanded the previous meta-analysis (Babisch, 2014) to aircraft noise by giving both a main effect estimate and estimates by source. Further, we systematically evaluate the threshold and the shape of the exposure–response association, as well as potential sources of heterogeneity and effect modification. Recent noise exposure studies also include evaluation of co-exposure to air pollution, an important consideration given that both exposures derive from the same sources and are further both associated with CVD (Davies et al., 2009; Foraster et al., 2011; Tétreault et al., 2013).

2. Methods

2.1. Study selection and data extraction

We conducted a systematic review to identify papers using road, rail or aircraft noise as exposure and myocardial infarction (MI) or coronary heart disease (referred to here as ischemic heart disease [IHD]) as outcomes (ICD10 codes I20-I25). We included both non-fatal and fatal incident cases. Studies on prevalence (i.e. cross-sectional) were excluded. The search was conducted in PubMed and EMBASE, for the 20 year period prior to January 2014 and reference lists of relevant articles including the WHO burden of diseases report (WHO, 2011) were screened. No geographic constraints were defined, however the search was conducted in and limited to publications in the English language. The search strings are provided in Appendix A. Further inclusion criteria were: eligible studies had to quantify the association between modelled or measured exposure to the transportation noise source, and myocardial infarction (MI) or ischemic heart disease (IHD) had to be in the title and/or abstract. Studies which quantified this relationship in dB, either categorically or by a linear trend (i.e. increase in risk is constant per exposure interval), including a measure of precision (e.g. 95% confidence intervals), were retained. We conducted a double data extraction of retained studies. Data extraction included recording the risk estimates by noise exposure categories (including reference level value), noise metric (e.g. Lday, Lden – see Appendix B for description), noise source, study population by sex, study design, and whether the risk estimate was adjusted for air pollution. Where available, risk estimates for specific subsets of the study population were also extracted (e.g. age and sex stratified, and for continuous years at the same residential address [referred to as years in residence]).

2.2. Linear exposure–response (trend) estimation

Risk estimates from individual studies based on categorical noise exposures were transformed into a linear exposure–response (per 10 dB increase in Lden). For each study, a log-normal model in SAS was fitted to the data to estimate the mean level of exposure in each of the exposure intervals. Model fit was based on the proportion of person years or number of cases within different exposure intervals for cohort and case-control studies, respectively. If necessary, the study specific interval means were converted to Lden using approximations from the literature: L16h+2 dB; Ldn+0.3 dB; and LAeq,24+1.5 dB (EEA, 2010) .

Trend per 10 dB noise increment, zeroed for the study specific reference level, was estimated using generalised least squares (STATA glst). If the covariance matrix could not be specified, the variance-weighted least squares (STATA vwls) method was used (Orsini et al., 2006). This approach was tested in a sensitivity analysis with studies providing risk estimates both categorically and as a linear exposure–response. We further explored individual studies for departure of the exposure–response from linearity by including a quadratic term for exposure level, and also performed a meta-analysis of the respective estimates. As a final check, we performed a meta-regression of the original effect estimates on the study's mean exposure level since a non-linear exposure–response relationship might only be seen when comparing across studies. A positive (negative) association between study-specific estimates and mean exposure levels would be suggestive of an exposure–response relationship with positive (negative) curvature.

2.3. Meta-analysis

Random effects meta-analysis (using STATA metan (Harris et al., 2008)) was conducted based on the risk estimates per 10 dB increase in Lden noise for the individual studies. The percent total variance due to between-study heterogeneity was assessed with the I^2 statistic. Between strata heterogeneity was assessed on the basis of the p value of a Chi square test. To specify the starting point for the pooled linear exposure–response association, we pooled the study specific reference values using the derived meta-analysis weights of each study.

We used the following effect estimates in the main analysis: non-fatal IHD, for studies reporting separate estimates for non-fatal vs. fatal cases; both sexes combined, for studies reporting males, females and both. Subsequent stratified analyses were also conducted to explore potential sources of heterogeneity due to methodological considerations, including: outcome definition (non-fatal vs. fatal and MI specific vs. unspecified IHD); study date ($\leq$2005 vs. $>$2005) because studies after 2005 explored potential confounding due to air pollution; type of transportation noise source (road, rail, aircraft); study design (case control, cohort, small area study [i.e. individual health data aggregated on the level of census areas with exposure assigned on this group level]); method of linear trend estimation (estimated from categorical vs. linear model in the original study); adjustment for air pollution (no, yes); and noise reference level. Reference levels were defined on the basis of the computed mid-point of the reference exposure category. For studies providing linear exposure–response estimates, we used the reference value reported in the text. If not specified we assigned the category “no threshold.” We also explored potential effect modification using stratified analyses, due to: age ($<$65 years, $\geq$65 years); sex; and years in residence (not specified, $>$10 years).

The influence of individual risk estimates was assessed by performing repeated meta-analyses with one study left out each time (further referred to as leave-one-out meta-analysis). The effect of leaving out sets of studies on the basis of methodological features was also explored (i.e. small area studies, North American studies, those potentially over-adjusted, studies not adjusted for air pollution, and those not adjusted for smoking). Analyses were conducted in STATA 12.

3. Results

3.1. Selected studies

We have identified 10 studies, conducted mainly in Europe, focussing on road and aircraft noise and incident cases of IHD (Selander et al., 2009; Beelen et al., 2009; Gan et al., 2012; Huss et al., 2010; Sørensen et al., 2012; Hansell et al., 2013; Correia et al., 2013; Babisch et al., 1994, 1999, 2005). No studies specifically investigating effects of railway traffic on IHD were found.