Noise and cardiovascular disease: A review of the literature 2008-2011

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ABSTRACT

A systematic literature review identified over 50 peer-reviewed English-language papers on noise and cardiovascular disease published in the period 2008-2011. With respect to environmental noise, we saw an emphasis on road traffic exposures and a significant focus on the issue of co-exposure to noise and traffic-related air pollution. Several studies examined the degree and determinants of these co-exposures, producing an important insight into correlations among these pollutants, which interestingly were not as high as had been expected. Other studies examined the joint effect on human health; the results of these studies were not entirely consistent but suggested independent cardiovascular health effects attributable to both noise and air pollution. Aircraft noise exposure also received considerable attention with the HYENA project maturing and producing several interesting results. Despite the very large potential public health problem associated with over-exposure to noise at work (i.e. given the ubiquity of exposure), occupation studies were still uncommon. Results showed a fairly consistent positive association between work-noise and both hypertension and ischemic heart disease. Finally, a handful of studies was reviewed that dealt with other issues such as policy, methodology and disease mechanisms. In this paper we summarize the progress of research in this field over the past three years, and propose research directions for the future.

METHODS

A literature review was undertaken to identify studies pertaining to the theme of the physiological effects of noise exposure. We searched PubMed using keywords pertaining to noise, transportation, traffic, aircraft, railway, proximity to road; and cardiovascular disease, coronary heart disease, ischemic heart disease, myocardial infarction, hypertension, and stroke. Literature was also hand-searched for additional studies. The search was limited to English language and the period 2008-2011.

INTRODUCTION

Over 50 peer-reviewed English-language papers were identified. This review is primarily bibliographic in nature and papers are presented by exposure, then disease sub- categories.

The association between noise and cardiovascular disease has been studied for several decades and the weight of evidence clearly supports a causal link between the two. Nevertheless many questions remain, such as the magnitude of the adverse effects of noise, whether there are thresholds for effects, how noise and other cardiotoxic pollutants (such as particulate matter) interact in disease causation, whether vulnerable populations exist (such as children or the elderly), or gender-based differences, and how epidemiological study in this area can be improved methodologically (Davies & van Kamp 2008; Babisch 2011). This review will focus on contributions to

literature over the past 3 years in the area of noise and CVD in general, but focus on these particular questions; the issue of vulnerable populations is taken up in more detail in a companion review by van Kamp & Davies (2011).

In addition, we refer the reader to several review papers published since 2008, especially those of Babisch (Babisch 2008, 2011; Babisch & van Kamp 2009), Kaltenbach et al. (2008), Pirrera et al. (2010) and, reflecting the European focus of research on the noise and cardiovascular disease, a collection of country-specific reviews published in *Noise and Health* in 2011 (Belojevic et al. 2011; Bluhm & Eriksson 2011; van Kempen 2011; Lercher et al. 2011; Maschke 2011; Stansfeld & Crombie 2011).

ROAD TRAFFIC NOISE

A key issue in the interpretation of studies of the association of road traffic noise with cardiovascular disease has been in understanding the role of air pollution, that have also been linked to cardiovascular disease (Brook et al. 2010). Noise and air pollution share a major common source – the motor vehicle. If co-exposures are strongly correlated we would anticipate confounding, or effect modification; there is also the possibility that one is simply a "marker" for the effect of the other. This is a particular problem for studies that rely on proximity to roadway as a surrogate for pollutant exposure, i.e. Gan et al. (2010).

Several recent studies examined the basic question of correlation between traffic-related air and noise pollution and their findings are summarized in Table 1.

Table 1: Correlation between traffic noise and air pollutants (pearson correlation coefficient unless otherwise stated)

	NO_X	NO	NO_2	Black smoke	UFP*
Noise	0.62[a] 0.64[b] 0.50[c]	0.41-0.60[d] 0.39[e]	0.53[b] 0.62[f] 0.16-0.62[d] 0.33[e]	0.24[i] 0.44**[e]	0.21-0.60[g] 0.22-0.41[d] 0.41-0.81[h]

* Ultrafine particulate; ** Black carbon; [a] (Sorensen et al. 2011); [b] (Davies et al. 2009a, b); [c] (Persson et al. 2007); [d] (Allen et al. 2009); [e] (Gan et al. 2011a - Spearman); [f] (Foraster et al. 2011); [g] (Boogaard et al. 2009); [h] (Weber 2009); [i] (Beelen et al. 2009)

Overall correlations were found to be fairly consistent, generally low to moderate. In addition to those shown in the table, correlations between $PM_{2.5}$ and noise were quite low, perhaps because road traffic is not an important generator of this size of particulate (Gan et al. 2011a; Boogaard et al. 2009). Huss et al. (2010) found low correlation between PM_{10} levels and aircraft noise, perhaps not unexpectedly due to the difference in source (Huss et al. 2010 - Spearman). Interestingly, correlations of modeled estimates (i.e. Gan et al. 2011a; Sorensen et al. 2011) were very similar to comparisons of measured values, even though the air and noise pollution models used a number of the same inputs (traffic volume etc.). The lower than expected correlations are likely due to differences in specific source (i.e. tyre noise vs engine exhaust pollution) and propagation paths; key determinants of variability seem to relate to traffic density, and meteorological conditions (Allen & Adar 2011; Davies et al. 2009a, b; Foraster et al. 2011).

The results of prior studies of road traffic noise and hypertension (HT) have been called "extremely heterogeneous" (Babisch 2006). Barregard et al. (2009) noted problems with study design as a possible cause of this heterogeneity (e.g. cross-sectional design, lack of control of confounders). Their own study examined both

prevalence of physician-diagnosed HT and incidence (using data on residential histories, and date of diagnosis) in a cohort of 1,953 adults in Lerum, Sweden. Information on potential confounders was collected by questionnaire; noise exposures were modeled. Increases in prevalence odds ratio were primarily evident in men; for HAT, prevalence odds ratio (POR) was 1.2, (95%CI 0.7-2.1) and for use of hypertensive medications, POR=1.5 (0.9-2.7). Men's POR increased greatly if analysis was restricted to >10 yrs in residence to 3.8 (1.6-9.0). Incidence rates of HT in males gave similar results, with a RR=1.3 (0.7-2.3) increasing to 2.9 (1.4-6.2) for >10 years in the same dwelling.

Belojevic et al. (2008) suggested that heterogeneity in findings may result in part from poor validity of exposure measures, suggesting the use of night-time road traffic noise exposure data may be less biased. They used measured night-time noise data from 70 downtown streets in Belgrade to assess exposure in a cohort of 2,503 adults, who had lived in the same residence for >10 years, and who slept on the street side of the house. Cases were those who were on HT medication or had measured BP >140/90 mmHg. HT prevalence was 19.2 %; the adjusted prevalence odds ratio was increased in men, but not women. For men exposed >45 dBA vs. <45 dBA the POR was 1.58 (1.03-2.42).

Bodin et al. (2009) focused on age as an interaction or effect modifier in the road traffic noise – HT relation. They used modeled exposure levels, and self-reported HT definition; risk of HT was "modestly" increased at levels <60 dBA ($L_{eq,24hr}$), and increased above 60 dBA. No gender differnces were noted. An age effect was seen with greatest risks seen in the "middle aged (40-59 years) compared to younger (18-39) or old-aged (60-80 years). Chang et al.'s (2010a, b) cross sectional study examined road traffic noise and HT in Taiwan, concerned about the generalizability of prior studies that had mostly been restricted to European cities. This was highly exposed population compared to prior studies, with a 90 % exposed above 75 dBA ($L_{eq,8hr}$ at residence). Overall adjusted prevalence odds ratio was 2.15 (95%CI 1.08-4.26) in those exposed over 80 dBA. In males only, a dose/response in prevalence was observed over 4 noise categories from <77 to ≥83 dBA.

The Hypertension and Exposure to Noise near Airports (HYENA) study provided interesting data in a series of papers on the effects of road traffic noise in their study of exposures around 6 major European airports (Floud et al. 2011; Haralabidis et al. 2008; Jarup et al. 2008; Selander et al. 2009a, b). Adjusted risk of HT was estimated at 1.097 (1.003-1.201) per 10 dB increase in road traffic noise (Jarup et al. 2008). A gender difference was apparent with a more pronounced dose-response relation for men, reaching OR=1.5 for those exposed ≥67.5 dBA. The gender difference was also apparent when hypertensive medication use was examined in this study group. Among a subset of 4,642 subjects, overall use of anti-hypertensive medications was not associated with road noise exposures, though an effect modification with age was apparent for men only (and not statistically significant) (Floud et al. 2011). Haralabidis et al. (2008) using 24-hour blood pressure profiles in a subset of 149 of the HYENA subjects, showed that exposure to road traffic noise at night was associated with an adverse reduction in the normal diastolic blood pressure "dipping". "Non-dipping" has been previously identified as a "...persisting consequence of major traumatic events.." and may be an independent risk factor for CVD (Haralabidis et al. 2008). Using similar methodology, Haralabidis et al. further showed that regardless of noise source, increased night time noise had an acute effect on both systolic and

diastolic BP, supporting the hypothesis that habituation to noise while sleeping is incomplete.

In other sleep studies, Griefahn et al. (2008) showed increased heart rate evoked by noise exposure in the range 45-77 dBA, with and without awakening; further, responses did not diminish during the night, further experimental evidence of incomplete habituation to noise during sleep. Similarly Tassi et al. (2010a, b), studying subjects who were chronically exposed to noise from railways, found that those chronically exposed at home to rail noise did have fewer awakenings, but they also found some degree of cardiovascular habituation that involved interactions with age and exposure duration. The cardiovascular effects of train noise at night seemed to be greatest for freight trains, and the effects greater among younger subjects (Tassi et al. 2010a, b). Graham et al. (2009) found night-time exposure at home to be negatively associated with parasympathetic tone, but found no association with sympathetic tone. Selander et al. (2009a, b) examined the relation between exposure to road traffic noise and morning cortisol levels in a subset of 439 of the HYENA cohort, but no obvious association was found.

Three studies examined simple proximity of residence to major roads and cardiovascular effects; this study design has an inherent limitation that complicates identification of the causal agent. Gan et al. (2010) showed that in a large cohort study in the Canadian city of Vancouver, compared to subjects who constantly lived further from traffic, those who constantly lived closer had an increased risk for coronary heart disease mortality (CHD; RR=1.29, 95%CI 1.18-1.41). During a 5-year window those who moved closer to traffic and those who moved away from traffic had intermediate levels of risk (1.20 and 1.14, respectively). Hoffman et al. (2009) in the Heinx-Nixdorf study showed that CHD risk was increased by 50-70 % in those living closer than 100 meters from a major road compared to those living over 200 m away. van Hee et al. (2009) showed that adults living very close to major roads had an increase in the left ventricular mass index (LVMI) of 1.4 g/m^2, equivalent to an increase in systolic blood pressure of 5.6 mmHg. This difference was not associated with $PM_{2.5}$ levels however, and all of these studies acknowledge the difficulty in attributing cause to specific agents and identify noise as one such potential agent, along with air pollutants.

Three studies looked specifically at road traffic noise and heart disease. All of these also examined joint effect of noise and air pollution; these latter findings are discussed below. Gan et al.'s (2011a) study of CHD mortality used a population-based cohort of 412,420 adults aged 45-85 yrs, and modeled noise exposures. They found a 9 % (95%CI 1 %-18 %) increase in CHD mortality associated with a 10 dB(A) increase in residential noise (all sources, L_{DEN}). There was little dose-response trend evident, but those exposed in the highest noise group [L_{DEN} > 70 dBA] had a RR of 1.30 (95%CI 1.12-1.51). Beelen et al. (2009) investigated several CVD mortality endpoints in the Netherlands Cohort Study on Diet and Cancer using modelled noise levels. The cohort size was 117,528 (with 6,137 cases). For the highest noise exposure category (>65 dBA L_{DEN}) relative risk (adjusted) increased for all CVD (1.25, 95%CI 1.01-1.53) and of heart failure (1.99, 1.05-3.79). Risk of ischemic heart disease and dysrhythmia were also elevated (1.15 and 1.23 respectively) but neither was statistically significant. Selander et al. (2009a, b) examined risk for acute myocardial infarction (MI) morbidity in the Stockholm Heart Epidemiology Program. Subjects were 5,452 adults 45-70 years; noise exposures ($L_{Aeq,24h}$) were modeled. For

subjects exposed over 50 dBA the RR was 1.12 (95%CI 0.95-1.33). When the study sample was restricted to reduce exposure misclassification, RR increased to 1.38 (1.11-1.71).

With respect to the joint effect of traffic-related air and noise pollution on CVD, the findings of these three studies were similar: that each pollutants seemed to have independent effects on CVD outcomes (Beelen et al. 2009; Gan et al. 2011a; Selander et al. 2009a, b). Selander et al.'s effect estimate (1.12, see previous paragraph) was already adjusted for NO_2 exposure level; unadjusted estimates were not reported. When they examined interaction through stratification, each exposure appeared to be independently capable of increasing risk but no interaction was evident. In Gan et al.'s models, adjusting for air pollution ($PM_{2.5}$, NO_2 and black carbon) did reduce the noise effect estimate (from 1.30 to 1.22) with black carbon having the greatest effect. However, in contrast to Selander et al. a simple additive interactive effect for noise and black carbon was seen (Gan et al. 2011a). Finally, Beelen et al. (2009) did not examine interactive effects; but after adjusting for air pollution (black smoke) in noise models risk estimates for noise effects were reduced, but not extinguished (i.e. all CVD was lowered to 1.17, IHD to 1.01). That of heart failure only dropped as far as 1.90, but became borderline significant (95%CI 0.96-3.78).

Stroke has not been widely examined with respect to noise exposure despite the fact that hypertension is an important risk factor. Sørensen et al. (2011) studied this relation in a Danish cohort of 57,053. They found road traffic noise (L_{DEN}) was positively associated with incidence of stroke, with a relative risk of 1.14 (95%CI 1.03-1.25). Risks were only found in those aged over 64.5 years, but in this group there was a clear dose-response relation with elevated risk beginning between 55 and 60 dBA.

Finally, Sobotova et al. (2010) showed that exposure to road traffic noise was associated with elevations in risk factor scores from predictive models such as the European Society of Cardiology's relative risk HeartScore®.

AIRCRAFT NOISE

Babisch & van Kamp summarized the literature on aircraft noise and hypertension in 2009. They concluded that there was sufficient evidence of association between aircraft noise and hypertension but that only a "best guess" quantitative effect estimate could be made (they recommended an odds ratio of 1.13 [95% CI 1.00-1.28]). In 2008 Jarup et al. published the first in a series of papers resulting from the HYENA study investigating the link between noise near airports and hypertension (also Eriksson et al. 2010; Floud et al. 2011; Haralabidis et al. 2008, 2011; Jarup et al. 2008). This study used modeled exposure data for aircraft noise, measured blood pressure levels and collected supplementary data on potential confounders and effect modifiers from 4,861 people living around six major European airports. A dose-response pattern for hypertension (using WHO definition SBP/DBP > 140/90 mmHg) was evident for the noise metric L_{night} but not $L_{Aeq,16hr}$. Odds ratios increased by 1.14 (95%CI 1.01-1.29) per 10 dB increase (Jarup et al. 2008). Floud et al. (2011) used the same study population to examine antihypertensive use related to aircraft noise exposure. Results varied by country; they were generally positive for both L_{night} and $L_{Aeq,16hr}$, but were only significant for the UK (both metrics) and the Netherlands (L_{night} only); estimates ranged up to OR=1.35. There was no apparent gender effect. The authors concluded "...results were more consistent for across countries for prescriptions for other stress-related conditions" including anaxiolytics. In Haralabidis et al.'s

two papers investigating 24-hour ambulatory blood pressure monitoring in a sub sample of 140 subjects, no effect was found of aircraft noise on night-time dipping (except in Athens, where the number of aircraft event and $L_{Aeq,\ night}$ was higher), (Haralabidis et al. 2011) but blood pressure increased - SBP by 6.2 mmHg, DBP by 7.4 mmHg - during 15-minute measurement intervals in which there was an aircraft event (Haralabidis et al. 2008). Finally, Selander et al. (2009a, b) found that in a subset of 439 HYENA subjects, morning cortisol levels were elevated in those exposed to aircraft noise at night, but in women only and that the increase was greatest among those women who were employed.

Most aircraft noise studies focus on civilian aircraft. Rhee et al. (2008) however examined the association between exposure to military aircraft noise (helicopters and fighter jets) in Korea. Exposed subjects lived with 5 km of either a helicopter base ($L_{Aeq,8hr}$ 71-72 dBA) or a fighter jet base (68-82 dBA), for a minimum of 10 years; they were compared to "non-exposed" control group. HT was defined as physician diagnosed HT, BP >140/90, or use of antihypertensives. Risk was elevated in both exposed groups (OR=1.62 and 1.23 for helicopter and fighter, respectively) but only that for helicopter exposure was statistically significant.

Gender specific effects were the focus of Eriksson et al. (2010) They examined cumulative incident HT (physician diagnosed HT or BP >140/90) in 4,721 subjects exposed to noise from Stockholm airport, and who were followed for 8-10 years. Elevated risk for HT was apparent in only males. Estimated risk estimates increased when the analysis was restricted to non-smokers.

Huss et al. (2010) examined the association of aircraft noise with Acute MI mortality, taking into account co-exposure to air pollutants. Four point six million subjects of the Swiss national cohort were followed for 5 years. Noise (L_{DN}) and PM_{10} levels were modelled. In their fully adjusted model containing both pollutants plus distance of residence from major road, they found a 50 % increase in AMI mortality in those exposed L_{DN} > 60 dBA. PM_{10} was not linked to increased risk, but residing with 100 meters of a major road increased risk approximately 18 %. Huss et al. found no link between aircraft noise exposure and stroke mortality in this study.

OCCUPATIONAL NOISE

Two cohort studies of occupational exposure and hypertension were reported in this period (Lee et al. 2009; Sbihi et al. 2008). Sbihi followed a cohort of over ten thousand sawmill workers for an 8-year period. Cases were identified from physician billing and hospital discharge records, and exposure based on work history data and a job-exposure matrix, described separately (Davies et al. 2009a, b). Eight hundred and twenty eight cases were identified; cumulative occupational noise exposure was a strong predictor of risk of hypertension, with a relative risk of 1.32 in the highest exposed population (>115 dBA*year) compared to controls (<95 dBA*year) and there was a significant dose response trend. Trends of duration of exposure at different thresholds were inconsistent. The authors attributed this to misclassification of exposure potentially due to the unmeasured effect of subjects wearing hearing protection (HPD, Friesen et al. 2008). This was addressed by later work by Sbihi et al. (2010a, b) examining methods for adjusting measured noise exposures for HPD use in retrospective studies. Lee et al. (2009) followed 530 male metal manufacturing workers for nine years, obtaining annual blood pressure measurements. They categorized exposure groups as (I) intermittent, unprotected, (II) <85 dBA (TWA), single HPD pro-

tection and (III) >85 dBA, double protection. A control group consisted of office workers (<60 dBA). Systolic, but not diastolic, blood pressure increased over time in all three exposed groups, in a dose-response fashion; 1.7, 2.0 and 3.8 mmHg for groups I, II, and III respectively.

Chang et al. (2009, 2010a, b) examined the role of workplace noise exposure and co-exposure to organic solvents (N,N-Dimethylformamide and Toluene) in a pair of studies. In the first, cross-sectional, study 59 workers in a synthetic leather plant were categorized as noise only, solvent only or co-exposed (using 'cut-points' of 80 dBA, and a solvent hazard index of 0.1). Subjects exposed to either noise or solvent showed greatly increased risk of hypertension, 7-8 fold when compared to a non-exposed control group, but co-exposure to both pollutants did not significantly increase this risk. In the second study, twenty subjects were divided into 4 similar exposure groups (none, noise-only, solvent only and combined) and undertook 24-hour ambulatory blood pressure monitoring. Only combined-exposure showed significantly elevated blood pressure (for diastolic only, both working hours [21mmHg] and 24-hours [16 mmHg]). Work time DBP were non-significantly elevated for noise only (13 mmHg) and solvent only (9 mmHg). Both studies were challenged by small numbers, cross-sectional study design and weakly controlled confounding.

Tomei et al. (2010) undertook a meta-analysis of studies of noise and hypertension, heart rate and ECG abnormalities. Fifteen studies yielded a total of 18,658 subjects. These were categorized as low (mean 62 dBA), intermediate (mean 85 dBA) or highly exposed (mean 92 dBA). Positive results were found for all hypothesized associations (systolic BP, diastolic BP, heart rate, and ECG abnormality). The odd ratio for overall prevalence of hypertension was 2.56 (95% CI 2.01-3.23).

Gan et al. (2011b) undertook a secondary data analysis of the US national Health and Nutrition Examination Survey (NHANES) to study noise-CVD links across a broad range of industries. Cross-sectional in design, data for 6,307 subjects aged 20 years and over were assessed for noise exposure at work (using self-report of having to raise voice to be heard). A variety of outcomes were examined including self-reports of physician-diagnosed cardiovascular disease, measurement of blood pressure and several biomarkers. Excess risk was observed for angina pectoris, myocardial infarction and coronary heart disease, with odds ratios of 2.9, 1.6 and 2.0, respectively. Dose-response trends were statistically significant for angina and CHD. Risks for CHD were stronger in younger age groups, current smokers, and in men. Noise was also associated with isolated diastolic BP (DBP>90 mmHg, SBP<140 mmHg), but not with any other BP measures or any biomarkers (including total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, circulatory inflammatory markers (i.e. C-reactive protein), homocysteine or plasma glucose.

SUMMARY

The last ICBEN congress review identified several areas for future research (Davies & van Kamp 2008). Several of these have been addressed in recent papers. Important progress was made on "untangling" the CVD effects of traffic co-exposures, noise and air pollution (TrAP). Several studies examined correlations of the two exposures, and determinants of variability. These showed us that the correlations were not as high as many researchers anticipated, and that epidemiologic studies of joint effects should be successfully pursued. Four large joint effects studies were reviewed here, and they were consistent in suggesting that both air pollution and noise are

likely independent risk factors for CVD. This is consistent with several other lines of evidence, such as that of animal and occupational studies - that are less susceptible to confounding - and with the fact that plausible biological mechanisms exist for both exposures (Allen & Adar 2011).

With respect to effects of gender on health associations, the majority of the studies found men to be at greater risk than women for noise-related cardiovascular disease irrespective of noise source (road vs aircraft) or outcome (HT or heart disease). Exceptions were the studies of Selander (cortisol response), Bodin (self-reported HT) and Sorenson (stroke) (Bodin et al. 2009; Selander et al. 2009a, b, 2011). Jarup et al. (2008) found men at greater risk in their main analysis, but only women, when the sample was restricted to those aged over 65 years. Other issues regarding vulnerable populations are reviewed in van Kamp and Davies in this volume (van Kamp & Davies 2011).

Results regarding effect levels (thresholds) were fairly consistent. For hypertension lowest observable effect levels (LOEL, for road taffic noise) were reported at between 50 dBA ($L_{Aeq,24hr}$ Jarup et al. 2008) and 60 dBA ($L_{Aeq,24hr}$ Barregard et al. 2009; Bodin et al. 2009); response to noise at night was seen at lower levels: 45 dBA ($L_{Aeq,night}$) Belojevic et al. 2008) and 40-44 dBA (Jarup et al. 2008). Heart disease responses were seen as low as 50 dBA (($L_{Aeq,24hr}$ Selander et al. 2009a, b) to 65 or 70 dBA (L_{DEN} Gan et al. 2011a; Beelen et al. 2009). Eriksson et al. 2010 and Huss et al. (2010) both identified LOEL for aircraft related noise for HT (L_{DEN}) and AMI (L_{DN}) respectively, at 60 dBA.

Generally speaking the methodological quality of the studies reviewed was high, especially compared to many of the early efforts in this field. The problem of misclassification of exposure has been addressed by the work on TrAP/noise correlation and in the occupational arena by the work of Sbihi et al.. It was encouraging to see other work on potential co-exposures such as organic solvents (Chang et al. 2009) Effects of joint-exposures are generally poorly studied in occupational epidemiology.

With respect to future research, reviewers are consistent in their call for more prospective studies to help elucidate underlying mechanisms of disease and the study of children, where results have been inconsistent (Basner et al. 2010; Babisch 2011; Bluhm & Eriksson 2011; Maschke 2011; Stansfeld & Crombie 2011).

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