Environmental noise and cardiovascular disease: Five year review and future directions

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INTRODUCTION

There continues to be steady research interest in the cardiovascular effects of noise. This summary of peer-reviewed English-language publications for the period of 2003 to 2007 shows a continuing trend of increasing sophistication in study design and analytical methods, and increased emphasis on research issues such as improved characterization of noise exposure, refinement of health outcomes, multiple outcome measures, dose-response relations and the joint effects of air pollution and noise, issues deemed important in the last review presented at an ICBEN meeting (Stansfeld & Lercher 2003). Several other reviews have been published in the intervening years, and we refer the readers to those reviews here (Babisch 2003, 2004, 2006; Babisch et al. 2005; Ising & Kruppa 2004; Rabinowitz 2005). The review is structured along dimensions of outcome and exposure, with a distinction made between short-term physiological response and long-term effects. Objective and subjective blood pressure changes are discussed alongside hypertension for efficiency reasons. Adult and child studies are discussed under the same headings, and mediating factors are discussed under the different headings, when measured.

Physiological Effects

Cortisol

In models of noise, stress and disease cortisol plays a key role in hypothalamic-pituitary-adrenal (HPA) axis activity and was examined in three recent studies of nighttime noise exposure. In the sole observational study, Ising obtained salivary cortisol samples from 68 children who had had recent physician contact for bronchitis (Ising et al. 2004). They found that nighttime noise levels above 53 dB(A) were associated with increased morning cortisol levels and perhaps led in the long term to aggravation of bronchitis in children. There were two laboratory sleep studies using salivary cortisol. In the first, low frequency noise (40 dB(A), ≤125 Hz) was associated with an attenuated cortisol response after wakening, such that cortisol levels had not peaked at 30 minutes post wakening as they did in controls (N_{TOT}=12) (Persson-Waye et al. 2003). In the second, exposure to simulated vehicle backup alarms (60-80 dB(A), 1000 Hz) failed to elicit change in cortisol concentration profiles the following day, but could not be directly compared with the first study because of the differences in timing of samples (Michaud et al. 2006). Interpretation of cortisol measurement data remains complex, as discussed in reviews of the use of stress hormone in noise research (Babisch 2003; Bigert et al. 2005). However there may be several factors that influence the variability seen in cortisol response to noise stimulation, including timing or measurement, type of stressor, controllability, individual response characteristic and individual psychiatric sequalae (Miller et al. 2007); future studies should consider these factors in analyses and interpretation. Finally, in an endeavor to use cortisol increase as a norm for aircraft noise exposure Spreng (2004) used a

simple physiological model to enumerate a table of "tolerable numbers of overflights per night" based on limiting cortisol accumulation.

Catecholamines

Sympathic-adrenal-medullary (SAM) pathway activation in response to stress is characterized by elevations in catecholamine responses. Serum noradrenaline levels (and HR, but not adrenaline nor cortisol) were shown to be elevated in conditions of high noise/high crowding compared to low noise/low crowding conditions in a small experimental study (Martimportugues-Goyenechea & Gomez-Jacinto 2005). While the use of salivary cortisol has largely replaced urinary measurement, salivary catecholamine measurement has proven difficult because of low levels and rapid degradation (Miyakawa et al. 2006). This has led to the investigation of novel markers such as alpha-amylase and chomogranin A that can be measured in saliva and whose correlation with catecholamine levels make them good potential substitutes for catecholamine measurement. Miyakawa looked at short-term salivary chomogranin A (CgA) changes in response to 15 minutes of 90 dB white noise. Among the 20 subjects, 3 distinct response type groups were observed: (a) cGA increased, and did not decrease after exposure; (b) cGA increased, and decreased after exposure; and (c) cGA did not increase during or after exposure. The differences were tentatively related to individual's noise sensitivity (Miyakawa et al. 2006). In the Michaud study, however, salivary alpha amylase level concentrations climbed steadily throughout the day from awakening to before sleep, but there was no apparent difference between noise exposed and non-exposed groups (Michaud et al. 2006).

Blood pressure/Hypertension

Occupational Studies

In what is probably the first major retrospective cohort study examining hypertension (HT), Sbihi followed 10,872 sawmill workers for 8 years, identifying 828 cases from physician-billing and hospital-discharge records. Noise exposure was estimated from predictive models based on 1,900 personal dosimetry measurements. The study reported a statistically significant exposure response for noise and HT reaching a RR_{ADJ} of 1.5 after 30 years exposure over 85 dB(A) (Sbihi et al. 2008). Lusk et al. (2004) examined ambulatory blood pressure (BP) and HR in 46 automobile engine assembly plant workers. Ambulatory BP is considered to provide better estimates of acute effects as it provides continuous monitoring of BP during normal work tasks, and perhaps reducing bias associated with traditional clinic measurement techniques. The study used mixed-effect modeling because of the repeated BP measures (taken at 10-minute intervals). Logged noise dosimetry allowed the calculation of short-term exposure metrics over the same intervals. After controlling for a large number of personal cardiovascular disease (CVD) risk factors, they found noise associated with 3 physiological measures (systolic and diastolic blood pressure [SBP, DBP], and heart rate [HR]), and showed a possible difference in mechanisms between BP (that they showed was correlated to average acute noise) and HR (which was correlated to peaks). As the authors had previously found chronic effects in same population, they suggested that acute effect may be "harbingers of future cardiac disease". Chang et al. (2003) used mixed-effect modeling in a small (N=20) cross-sectional study of 24-hour ambulatory BP data, with logging personal dosimetry collecting 5-minute readings (day) though only a single off-work nighttime measurement. Ambulatory BP was measured every 30 minutes during the day and every 60 minutes at night. SBP was increased in the exposed group (mean 85 dB(A)) compared to the unexposed (mean

59 dB(A)) during work and during sleep, demonstrating a sustained impact; diastolic differences were not as large, but examining effects "lagged" by 30 and 60 minutes strengthened the effect. The same group also examined arterial compliance or "stiffness" in this population (Chang et al. 2007). Arterial stiffness of large elastic arteries has been shown to be a major determinant of vascular function perhaps playing a role in left ventricular hypertrophy and arteriosclerosis. Several measures, including brachial artery compliance, brachial artery distensibility, systemic vascular compliance, and systemic vascular resistance showed sustained differences between exposed and unexposed groups, but the authors cautioned careful interpretation due to small study size and lack of complete control of potential confounders. Two further cross-sectional studies examined the noise-HT relation in different industrial settings. In the first, 77 turboprop pilots (L_{eq} = 93 dB(A)) were compared with 224 jet pilots (L_{eq}=79 dB(A)) with noise measured at ear inside helmet (Tomei et al. 2005). There were differences between groups in several hemodynamic parameters, including increased DBP and HR in the noisier group. However while the two groups were well matched for many risk factors it was not clear that adequate control could be made for the differences in environment and tasks for pilots flying turboprop transport aircraft versus jet fighter aircraft. Inoue et al. examined male pulp and paper workers who had held the same job since start of employment (Inoue et al. 2005). They compared 242 workers from paper manufacturing (mean 92 dB(A), and using hearing protection) versus 173 from chemical products (mean 75 dB(A)). They found a significant negative association for HT and noise in both crude and adjusted analyses. The authors suggested several possibilities for this unexpected result including exposure misclassification, that seems probable when plant mean levels are used, but a lack of control over potential confounders such as health promotion program participation.

Environmental studies - road traffic

Earlier reviews have noted a lack of adequate data to adequately assess the effect of road traffic on HT (van Kempen et al. 2002) but we have seen several recent studies addressing this issue and emphasizing nighttime noise exposures in the home. Belojevic et al. studied 328 children aged 3-7 years from 10 downtown Belgrade kindergartens, obtaining daytime noise levels at the kindergartens and nighttime noise measurements at the home (Belojevic et al. 2008). SBP was 5 mmHg higher in subjects from quiet-home/quiet-kindergarten versus noisy-home/noisy-kindergarten, and HT and HR were also significantly increased. In adjusted multivariable linear regression, only a significant difference for SBP remained. These findings differ from van Kempen et al; in this study (part of the multi-center RANCH study of cognitive and health outcomes (van Kempen et al. 2006)), BP was measured in 853 Children aged 9 – 10 years living around Heathrow (UK) and Schipol (NL) airports. Road noise was modeled at school in both countries, and for the home for Schipol only. Associations between road traffic noise at home and at school and BP measures were mostly negative, and in the combined UK/NL and NL sample negative estimates for SBP were statistically significant. This study had a large combined sample size, good contrast in exposure, and good control of confounders. There was a strong correlation between day (school) and night (home) noise levels, too high to disentangle school/home effects. The authors suggested that negative associations might have resulted in exposure misclassification (e.g. from measurements taken at building facades that might not reflect interior exposures). Bluhm et al. studied self-reported HT for 667 adults of a municipality near Stockholm (Bluhm et al. 2007). Road noise was modeled for major roads (55-65 db) and the rest (n=513) estimated by expert judg-

ment. Thirteen percent of subjects were diagnosed with HT. There was a linear exposure response relation between traffic noise and prevalence of HT with an OR_{ADJ} of 1.38 per 5 dB(A). The authors also showed interactions for time in residence, bedroom orientation, glazing, and older homes. In another study of self-reported HT, de Kluizenaar el al examined self-reported use of HT medications in 40,856 adults aged 28-75 years in Groningen, NL (de Kluizenaar et al. 2007). A subcohort of 8592 had follow-up screening (BP measurement, cholesterol, and pharmacy reports). Exposure measures (L_{DEN}) were from standard noise maps. There were significant associations in the unadjusted full group (OR=1.31) and subcohort (OR=1.35) per 10 dB(A). In adjusted models effects remained only for 45-55 year age group and for those exposed over 55 dB(A) (the latter in full cohort only). This study was perhaps the first to consider the joint-effects of air pollution. In the fully-adjusted noise models, further adjustment for PM_{10} did not affect noise-effect estimates; this was taken to mean that PM_{10} was not confounding the noise-HT relation, but the authors point out there was very limited contrast in PM_{10} exposure.

Environmental studies - aircraft traffic

Ericksson et al. examined HT in a cohort of 2,037 males who were participants in a 10-year follow up of a diabetes surveillance project. Subjects were from 4 Stockholm municipalities surrounding Arlanda airport (Eriksson et al. 2007). They utilized standard noise models to generate weighted $L_{A,eq,24}$ measures and L_{MAX} (highest level occurring > 3 times in 24 hours); these two metrics were highly correlated. Cases were defined as self-reported HT diagnosis in previous 10 years, or having SBP/DBP >140/90 mmHg at follow up. Adjusted relative risks were all statistically significant: 1.1 per 5 dB ($L_{A,eq,24}$) or per 3 dB (L_{MAX}), and 1.2 for > 50 dB ($L_{A,eq,24}$) or L_{MAX} > 70 dB(A). Effects were stronger in older subjects, and among non-smokers. The RANCH study also examined effects of aircraft noise (van Kempen et al. 2006). When UK and NL results were pooled for analysis, both aircraft noise at home and at school showed similar increases in SBP of 0.1 mmHg per dB(A) but after adjustment for cofactors the effect remained significant only for home exposure. No significant effects were evident in UK sample, while for the NL results were positive (between 0.13 and 0.20 mmHg/dB(A)) for SBP and DBP for both day and night noise exposure at home, and for both SBP and DBP during daytime exposure at school. The authors suggested that the country differences might be due to systematic biases due to differences in modeled exposures, difference in the insulation of homes and schools, differences in schooling system and/or teacher's attitude toward noise affecting children's attitude. Finally, Greiser et al. published a study that linked individual prescription data for 809,379 subjects around the Cologne-Bonn airport to individual residence through geo-coded addresses (Greiser et al. 2007). Following adjustment for a number of psychosocial factors, gender and other noise sources, they found evidence of increasing use of prescription drugs with increasing nighttime aircraft noise ($L_{EQ,A,0305}$) in all drug combinations reviewed, i.e. for HT-medications only, for CVD-medications, for combined HT/CVD medications, and for HT/CVD plus anxiolytic medications, but the exposure-response curves were not consistent.

Ischemic Heart Disease

Occupational Studies

Several large longitudinal studies of ischemic heart disease (IHD) in occupational settings were reported during the review period. Davies et al. examined acute myocardial infarction (AMI) mortality in a large (n=27,464) retrospective cohort study of

blue-collar workers (Davies et al. 2005). The study featured a quantitative exposure assessment using predictive models based on personal dosimetry and case ascertainment using linkage to national death registry. Increasing relative risks (to RR_{ADJ}=1.5) were associated with both increasing cumulative exposure and increasing duration of exposure. Risks were greatest during working years, and exposure-response trends strongest when analyses restricted to subjects who did not use HPD, suggesting that HPD use contributed to a misclassification of exposure measurement and results in attenuation of the dose-response relation. McNamee et al. conducted a nested case-control study in 2 UK nuclear power stations (McNamee et al. 2006a, b). Subjects comprised 1,101 male case-control pairs. Using comprehensive work histories, noise exposure was assessed using available noise surveys and expert judgment. The authors undertook comprehensive validation of their exposure assessment (McNamee et al. 2006a, b) by using their assessed exposures to predict changes in hearing thresholds obtained from subjects' audiometry data. Cases (1,220) had died from IHD aged 75 or under, and controls were matched on age, start date and site. Overall, and at one site ("A"), there was no effect; but at site "B", odds ratios were 1.15, 1.32 and 1.31 for low/medium/high exposure groups respectively. The authors concluded that the difference in findings between the two plants might have been related to exposure misclassification as validation was successful in plant "B" but not plant "A". In two, related, studies Virkunnen (Virkkunen et al. 2005, 2006) utilized extracts of industrially-employed participants from the Helsinki heart study; in both, health end-points were taken from national hospital discharge and death registries. In the first study (n=6,005), subjects were followed from 1982 to 1999; continuous and impulsive noise exposure was assigned from a national job-exposure matrix (FINJEM) linked on occupation, and combined into three exposure classes for analysis: (a) none, (b) continuous over 80 dB, and (c) both continuous and impulse noise. Relative risks for category (b) and (c) were increased, reaching 1.5 and remained significant when adjusted for potential confounders. One potential problem with this study was that 23 % of subjects were taking the lipid-lowering drug gemfibrozil. The authors suggested that a finding of reduced risk due to noise among those taking gemfibrozil may however have indicated specific HPA involvement in the pathophysiology of the disease, as gemfibrozil is known to lower risk of coronary heart disease (CHD), especially in those with metabolic syndrome. In the second study, the authors examined concomitant exposure to noise, shift work and physical activity in a population of 1,804 followed from 1987 until 1999. Shift work data was obtained by questionnaire, while noise and physical workload were ascertained using FINJEM. Only the joint effect for noise and physical workload (RR_{ADJ} 1.4) remained (borderline) significant after adjustment for potential confounders. Relative risk estimates were highly consistent among all four of these occupational studies. One major difference being that the effect persisted after retirement in Virkunnen's study, counter to the findings of Davies 2005 who found a decreased effect following retirement.

Heinonen-Guzejev et al. examined the role of noise sensitivity and noise exposure in CHD and CVD mortality in Finnish adults from the Finnish twin study (Heinonen-Guzejev et al. 2007). Among one thousand and five twin pairs noise sensitivity was assessed in 1988 and again in 2002 and found to be a highly stable trait. Lifetime exposure to noise was assessed as self-reported exposure duration of noise at residence, work and during recreation. Health outcome were CHD and CVD deaths from 1989 to 2003 obtained from the national death registry. No statistically significant increased risks were found in males for either noise or noise sensitivity. However in females elevated risks were found for noise sensitivity and CVD after adjusting for

potential confounders. Interaction effects were found in women only, both CHD and CVD (RR = 3.1, 2.9 respectively) in those with noise sensitivity who were also exposed, and 5.1 and 3.6 respectively for those with noise sensitivity and HT.

Environmental Studies

Babisch et al. examined incident myocardial infarction (MI) between 1998 and 2001, recruiting patients with confirmed MI's at 32 Berlin hospitals (Babisch et al. 2005). A sophisticated noise assessment was conducted utilizing noise maps for roads with volumes over 6,000 vehicles per day, with lower volume roads characterized as "quiet". This assumption was validated and subjects' addresses further checked and their exposures reassigned if they lived near but not on a main road that was noisier than their own road. In adjusted multivariate analyses there was a slight increase in risk for males only. This was strengthened when analysis was restricted to those who had lived in residence for > 10 years (RR_{ADJ}=1.3 > 65 dB(A), 1.8 > 70 dB(A)). There was no effect in females. Noise annoyance was linked to MI in males (for traffic noise at night, RR = 1.1) and females (for aircraft noise at night, RR = 1.3), and noise sensitivity was increased risk in males (RR = 1.14). The authors suggested that these gender differences might be due to difference in sex hormones, contraceptive use, different time/activity patterns or small sample size. Babisch *et al's* unexposed group was found (*a posteriori*) to have different relative risks for those with measured exposure versus those assumed to be "quiet" based on traffic volume. The reason for this was unknown, but as there were no grounds to distinguish the two subgroups on any acoustical basis, they were considered a valid reference group. A reanalysis was conducted however using only "measured" roads in the reference group (Willich et al. 2006a, b). In this analysis similar effects were seen in males, but strongly elevated levels found in females (RR=3.4); these authors also suggested different time/activity pattern might be influencing the results. A discussion of the two analyses followed Willich's paper (Babisch et al. 2006; Willich et al. 2006a, b). Babisch et al. had earlier examined the role of noise sensitivity and its interaction with pre-existing disease in IHD incidence (Babisch et al. 2003). Of a sample of 3,950, 1,519 were considered to have a pre-existing disease. Noise sensitivity was measured through several questions regarding general annoyance and specific questions regarding activity/relaxation disturbance or sleep problems. Health outcome was IHD death or non-fatal MI. Traffic noise levels were associated with IHD incidence (RR = 1.3 in those exposed 66-70 dB(A)) increasing to 1.8 in those with pre-existing disease, but neither estimate reached statistical significance. General annoyance, disturbance of relaxation or conversation, and feeling nervous or tense were all associated with increased risk for IHD, but in each case the risk was higher among those without pre-existing disease than for those with pre-existing disease, a fact the authors attributed to recall bias. Finally, Grazuleviciene examined males presenting with first-time MI's at 4 cardiology departments in Kaunas (Grazuleviciene et al. 2004). Subjects (N=518) were attributed an average noise exposure for their electoral district; analyses were stratified on age, but no personal risk factor information was available. The authors found elevated risk for older males, with greatest RR of 1.9 in males 55-64 exposed >70 dB (compared to "unexposed" group <60).

Conclusion, and future directions

The past five years has seen a number of high quality studies that addressed many of the past concerns of study design, power, analytical approach, exposure assessment and outcome classification. While these studies have largely supported the hypothesis that noise and mediating factors such as noise sensitivity are causally asso-

ciated with cardiovascular diseases including hypertension, there remain several areas that require future attention. First is the issue of the joint effects of air pollution and noise on CVD (Schwela et al. 2005). A number of studies have begun to examine this; one is reviewed in this paper (de Kluizenaar et al. 2007), and others (Jarup et al. 2005; Babisch et al. 2008) reported on the study design of HYENA and its early results, which will be the first large multi-center study to investigate modifying effects of air pollution on road and air traffic effects around 6 major EU airports. Other studies are in progress (Davies et al. 2008) and will be reported in the next few years. Other key issues that have surfaced or remain are the effects of noise on susceptible populations, the inconsistencies found in studies of noise and blood pressure/hypertension among children, and gender effects. This effort will require continued improvements in exposure assessment techniques, improvements in case definitions (eg objective measures of HT), analytic designs that better cope with the limitations of existing exposure assessment and systematic adjustment for confounding factors.

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