NON-AUDITORY PHYSIOLOGICAL EFFECTS OF NOISE: FIVE YEAR REVIEW AND FUTURE DIRECTIONS

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Introduction There has been continuing research interest in the non-auditory physiological effects of noise over the last five years (1998-2003), since our previous review (Lercher, Stansfeld & Thompson, 1998), with particular recent emphasis on further studies of cardiovascular effects and research on children. More sophistication in choice of epidemiological designs and samples, in noise exposure measurements and outcome measures has helped progress in the field. However, there is still considerable uncertainty as to the nature and the size of the effects of environmental noise on human health and much further work that needs to be done.

Mental Health Child mental health has been examined in several studies. Child self-reported mental health on a standard scale and teacher ratings of classroom adjustment in response to highway, road and rail noise were studied in a large sample of 8-11 year old Austrian primary schoolchildren and a second stage sample of extreme noise exposed groups. Noise exposure was significantly associated with classroom adjustment scores but, intriguingly, child self reported mental ill health was only impaired in noisy settings in children of low birth weight and preterm birth (Lercher et al, 2002). In a London study of psychological distress in children exposed to chronic aircraft noise no differences were found attributable to noise exposure adjusting for socioeconomic status (Haines et al, 2001a). However, in a further, larger study, higher psychological distress scores were found in chronic noise exposed children (Haines et al, 2001b). High scores in noisy areas were significantly associated with the hyperactivity subscale; possibly susceptible to stimulating environmental stressors such as noise. More work is needed to examine confounding factors and confirm the possibility of especially vulnerable groups within children (eg children with developmental, learning problems, low birthweight, small for gestational age babies). Noise exposure and social disadvantage are highly correlated: the issue of separating the effects of noise from the broader effects of socioeconomic position on health still needs to be resolved (Haines et al, 2002). In adults, further Japanese results suggest that high levels of military aircraft noise may have effects on mental health; in a cross sectional study of 5,963 inhabitants around two air bases in Okinawa, those exposed to noise levels of Ldn 70 or above had higher rates of 'mental instability' and depressiveness (Hiramatsu, 2000). Those who were more annoyed showed higher risk of mental or somatic symptoms.

Indices of physiological arousal Studies of endocrine markers of noise exposure continue to demonstrate conflicting results. Short term exposure to intense industrial noise (98dB(A) in normal volunteers was associated with raised serum magnesium and calcium but had no effect on urinary adrenaline, noradrenaline and dopamine (Mocci et al, 2001). This is not in keeping with earlier studies that show persistent elevation of catecholamines in noisy industrial settings. It is clear that there are other factors that may interact with noise exposure such as chronicity of exposure, other workplace exposures and, perhaps, perceived control over noise exposure (Hatfield et al, 2002). Exposure to low frequency noise is being increasingly studied: male

students exposed to night time 40dB(A) low frequency noise, simulating a compressor, in the laboratory had an attenuated cortisol response on waking related to negative mood and tiredness (Persson Waye et al, 2003). Similarly, low frequency noise exposure during office performance tasks was related to a slowing of the usual circadian decline in salivary cortisol in noise sensitive subjects whose performance was also most impaired by noise (Persson Waye et al, 2002). Replication of these interesting findings in larger samples would be informative. Reviews of endocrine mechanisms and measurement have been carried out advancing the conceptual issues in this area (Maschke et al, 2000). In the last five years there has been increased focus on research on children as a potentially vulnerable group. Children exposed to longterm low frequency road traffic noise at night (on average one heavy vehicle Lmax > 80dB(A) every 2 minutes, indoor levels Lmax 33-52 dB(A)) had raised urinary free cortisol during the first but not the second half of the night (Ising, 2002). A study of 'everyday' Alpine traffic noise found that children exposed to road and rail noise levels of greater than 60dB(A) Ldn compared to less than 50dB(A) Ldn had raised urinary overnight cortisol levels, but no differences in urinary noradrenaline and adrenaline (Evans et al, 2001). Children were carefully matched on parental education, housing characteristics, family size, marital status and body mass index and pre-screened for auditory acuity. Children in noisier areas also had raised blood pressure and rated themselves higher on perceived stress symptoms. In contrast, children exposed to chronic aircraft noise around Heathrow Airport in London did not have raised salivary cortisol, measured in the morning pre- and post-cognitive testing. This might be because effects were masked by the extreme variability of morning cortisol. Nevertheless, in a further, larger, study in the same area, 12 hour urinary free cortisol and catecholamines were not elevated in relation to aircraft noise exposure (Haines et al, 2001a; Haines et al, 2001b). Hormonal studies are difficult to carry out in the field, and may be biased by unmeasured confounding factors; however, there does seem to be some growing evidence of road traffic noise, even if not aircraft noise, influencing cortisol levels in children.

Occupational noise and blood pressure continues to be studied. Longitudinal studies can help in overcoming some of the bias inherent in earlier cross-sectional studies where confounding by other workplace factors may be a problem. A recent pioneering longitudinal industrial noise study has shown that noise levels predicted raised systolic and diastolic pressure in those doing complex but not simple jobs (Melamed, 1999). This study also suggests a link between occupational noise exposure and mortality risk. A further advance has been the use of ambulatory blood pressure monitoring in industrial settings (Fogari et al, 2001). In 476 normotensive workers aged 20 to 50 years mean systolic blood pressure was 6mm higher and diastolic blood pressure was 3mm higher during noise exposure and for 2-3 hours after noise exposure compared to non-noise exposed subjects. As an added control measure ambulatory blood pressure was also measured at home and did not differ between noise exposed and non-exposed groups. By contrast, noise exposure was associated with an increase in heart rate not only at work (+3.7bpm) compared to non-exposed groups but also at home (2.8 bpm). It is instructive that clinic blood pressures did not differ between noise exposed and non-noise exposed groups suggesting ambulatory monitoring, measuring blood pressure during noise exposure, is a more sensitive method of identifying noise effects. These findings suggest transient blood pressure elevation in response to acute noise exposure. However, as the authors suggest, if this transient rise in blood pressure is sustained during long periods of noise exposure this may be a contributor to hypertension. A study of cardiovascular risk factors during day time sleep in shift workers found that blood pressure responses were more likely in response to sounds of sudden onset and spectral analysis of blood pressure variabilities was a sensitive measure of sympathetic vascular tone due to noise (Carter et al, 2002). A further extension of occupational blood pressure research that replicates earlier work is the finding that

the use of hearing protection is associated with a significant decrease in both systolic and diastolic blood pressure in 374 workers in an American car plant adjusting for gender, race and age using retrospective records of occupational noise exposure (Lusk et al, 2002). Cumulative noise exposure in industrial settings has also been related to diastolic blood pressure in two further studies, adjusting for various confounding factors (Talbott et al, 1999; Tomei et al, 2000). In combination with earlier studies, these additional studies of dynamic change in blood pressure add biological plausibility to the effects of noise exposure on blood pressure. They also suggest that homeostatic mechanisms return blood pressure to normal after acute noise exposure which is reassuring in terms of noise causing hypertension. What is needed is longitudinal studies of noise exposure and blood pressure to investigate whether prolonged noise exposure leads to sustained rises in blood pressure and hence, risk of hypertension.

Community studies of noise and blood pressure are more difficult than occupational studies in terms of controlling extraneous influences. A methodologically rigorous Austrian study of 572 people exposed to rail and road traffic noise measured blood pressure twice at home within a 10 day period (Lercher et al, 2000). In analysis with adjustment for sociodemographic, lifestyle, personality and environmental confounding factors the highest proportion of those with hypertension was found in those 'not at all annoyed'. There was however, a small consistent effect of distance of the dwelling from the local main road that may indicate noise level experienced within the dwelling. An analysis of 28,781 blood pressure records around Kadena airport in Okinawa revealed a clear dose-response relationship between aircraft noise exposure and systolic blood pressure, adjusting for age, sex and BMI, although a less clear effect for diastolic blood pressure (Matsui et al, 2001; Hiramatsu et al, 2002). It seems easier to detect small effects of noise on blood pressure in large samples such as this.

Some recent studies have specifically examined hypertension as an outcome. A recent Swedish study found that the prevalence of hypertension was higher among people exposed to time weighted energy averaged aircraft noise levels of at least 55dBA or maximum levels above 72dBA around Arlanda airport (Rosenlund et al, 2001). Maximum levels of noise may be more important in determining blood pressure risk than averaged noise levels- this as issue to consider in future studies. A study of self-reported hypertension in relation to road traffic noise found at more than 50 DB(A) the odds ratio for hypertension was 1.4 (95%CI 0.6-3.2) in men and 1.8 (95%CI 0.8-4.1) in women. A significant effect in women became non-significant after adjustment for age, smoking habits, education and type of residence with stronger effects in younger age groups and those with longer residence (Bluhm et al, 2001). There was a clear dose response relationship with increasing noise level. In summary, there is some evidence from community studies that environmental noise is related to hypertension. A meta-analysis of 43 epidemiological studies of occupational and community noise and blood pressure found small blood pressure changes associated with noise. For hypertension, relative risks of 1.14 (95%CI 1.01-1.29) were found for 5dB(A) rises in occupational noise and 1.26 (95%CI 1.14-1.30) for 5dB(A) rises in aircraft noise exposure.

Coronary Heart Disease The meta-analysis mentioned above also examined coronary heart disease (van Kempen et al, 2002): air traffic noise was associated with angina pectoris, consultation with a general practitioner or specialist, and use of cardiovascular medicines. In cross sectional studies road traffic noise also increased the risk of myocardial infarction and total cardiovascular disease. As the authors point out the associations here are still inconclusive because of the limitations in exposure measurement, consideration of all relevant confounding factors and the possibility of publication bias. A systematic review of available, largely prospective traffic noise studies, found evidence that environmental noise above 65-70dB(A)

may be a minor risk factor for coronary heart disease (Relative risk 1.1-1.5) (Babisch, 2000). Noise effects were larger when mediating factors such as years of residence, room orientation and window opening habits were also taken into account.

Noise and mortality Industrial noise exposure has been related to all-cause mortality. First, in an 8 year follow up of the CORDIS study in which a dose-response relationship was found between industrial noise exposure and all-cause mortality showing a significant effect in those exposed to noise levels greater or equal to 85dB(A) for 10 years or more. This effect (Hazard Ratio = 1.96 95%CI 1.01-3.83) remained after adjusting for age, body mass index, smoking, leisure time physical activity and use of hearing protectors. In further analysis at 12 year follow up of 2606 industrial workers (Melamed & Froom, 2002), this effect was found specifically in workers doing complex as opposed to simple jobs (OR=1.86, 95% CI 1.04-3.32). There was also a non-significant association in other subgroups; blue collar workers, less educated workers and those with longer tenure. These findings are interesting because they are consistent with performance studies suggesting that noise especially interferes with complex rather simple task performance (Smith & Broadbent, 1992). On the other hand they are at odds with research that finds that jobs with more variety and skill use are at less risk for coronary heart disease although these latter findings may be more relevant to white collar jobs. Some light may thrown on this by analyses of noise exposure, job complexity and blood pressure (Melamed et al, 2001). In low noise exposure conditions those with simple jobs had greater increases in blood pressure while those with complex jobs were relatively protected. However, under noisy conditions those with complex jobs had a threefold rise in blood pressure compared with those doing simple tasks. This interaction between task complexity, noise exposure and physiological effects may have wide implications for other health outcomes.

Mediating factors A number of recent studies have examined annoyance as a possible mediating factor for cardiovascular outcomes. In 3622 residents from Pancevo, Serbia, 'very much' or 'extremely' annoyed men had an increased risk of reporting hypertension (OR= 1.8 95% CI 1.0-2.4) and myocardial infarction (OR= 1.7 95% CI 1.0-2.9) than those 'not at all' annoyed, adjusting for age, body mass index and smoking (Belojevic & Saric-Tanaskovic, 2002). This effect was not found in women. These results are striking but a reliance on self-reported exposures and outcomes must raise the possibility of recall bias. Other possible explanations are either confounding by social disadvantage linked to noise exposure or reverse causation, that men with existing cardiovascular disease are more prone to develop annoyance in response to noise. Expectations about changes in noise exposure may also influence attribution of physiological symptoms to noise, as was found around Sydney airport, where expected change in noise level contributed to explaining the association between noise level and self-reported physiological symptoms (Hatfield et al, 2001). The association between annoyance to road traffic noise and incident coronary heart disease was examined in 3950 men from the Caerphilly and Speedwell cohort study (Babisch et al, 1999). Between 13 and 24% of men were either 'sometimes', 'often', or 'always' 'disturbed or annoyed' by traffic noise. Although objective traffic noise levels were not linked to disease prevalence, there was a higher prevalence of disease in those who were highly annoyed. In men with no pre-existing disease high levels of noise annoyance were predictive of incident disease. But this association was not found either in those with existing disease at baseline or for objective noise measurements. An effect of borderline significance of increased coronary risk was found for objective noise level in men with pre existing disease. This may reflect the increasing vulnerability of people with ill-health to further impairment of their health by noise. There is increasing evidence that those with existing illness are vulnerable to the effects of noise, especially in terms of increasing annoyance. The possibility of annoyance as an intervening

factor in the noise, coronary heart disease association deserves further work, as does, the possible health consequences of annoyance. The importance of noise sensitivity as a vulnerability factor has still not been settled. In a UK community study associations were examined between noise exposure, noise sensitivity, subjective symptoms and sleep disturbance in a random sample of 543 adults (Smith et al, 2000). Perceived noise exposure was related to subjective health but this association became non-significant after adjustment for negative affectivity. In a similar way, adjustment for negative affectivity eliminated the association between noise sensitivity and subjective health. Thus it was suggested that noise sensitivity was merely a proxy measure of negative affectivity or neuroticism. This explanation was also suggested from the earlier analyses of the Caerphilly Study, however, more recent analyses suggest that road traffic noise exposure shows a significant effect on psychological distress measured by the General Health Questionnaire in participants who report being sensitive to noise but not in those not sensitive to noise (Stansfeld et al, 2002).

Conclusions Research on children is beginning to establish a body of evidence on hormonal effects but child mental health effects need further definition. Occupational noise exposure does seem to show convincing elevation of blood pressure, but although there is also a striking link between noise and mortality, the longterm implications of raised blood pressure in these occupational studies needs to be demonstrated. Studies of environmental noise also seem to predict both hypertension and coronary heart disease, although self-report outcomes are probably insufficient in this area- the meta-analyses suggest small effects that may be important in view of the numbers of people exposed to environmental noise.

Future Directions for Research What has changed since the last 5 year review (Lercher & Stansfeld, 1998)? That review suggested a need for better characterization of noise measurement, concurrent measurement of other environmental stressors apart from noise, a focus on vulnerable groups, mediating and confounding factors, refinement of health outcomes, better assessment of the lag period from exposure to health outcome, use of multiple outcome measures in field studies and further consideration of the after effects of noise. Although some progress has been made in noise measurement, the assessment of vulnerable groups and more consideration of confounding factors and standardized health outcomes there remains much from the earlier review that is not yet achieved. There needs to be further consideration of other environmental stressors such as air pollution, which is a frequent accompaniment to noise pollution (Klaeboe et al, 2000). Air pollution may be a confounding factor or a moderator of noise effects on cardiovascular outcomes (Brunekreef & Holgate, 2002). The effects of noise and air pollution are difficult to separate because their exposures are often highly correlated emanating from the same source. One possibility is separation of their effects because of different dispersal patterns from the source. This requires collaborative research and a wider funding base than purely noise-related research. A systematic framework for these environmental studies that has developed recently is health impact assessment. This has been used to assess noise effects and may be a future way forward although its outcomes are usually broader than those of research studies (Franssen et al, 2002; Lercher, 2002). Both epidemiological studies and laboratory studies will continue to be important. Field studies, while giving valuable evidence on risk and population distribution of noise-related ill-health are prone to confounding and noise-related selection factors. Laboratory studies, although lacking in ecological validity can be helpful by providing controlled conditions and information on mechanisms. In terms of research needs for noise and coronary heart disease, Babisch (2002) has suggested a number of issues: do cardiovascular responses to noise observed in the laboratory habituate, and, if so, at what physiological cost, who are the sensitive groups for cardiovascular effects, are there coping mechanisms that might protect

against coronary risk, are some noise sources more pathogenic than others, how can we assess the impact of multiple sources, and how do sound level and annoyance interact to lead to health effects? Other intrinsic vulnerability factors like family history of hypertension or heart disease should be more frequently measured in noise studies and night as well as daytime noise exposure considered. A focus on dose response relationships is important but many of these more fundamental questions need to be answered first. There is a real need to involve measurement of other stressors as well as noise and to place noise effects in a total social and environmental context.

Keywords: noise, blood pressure, mental health, coronary heart disease, children

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