Noise exposure and serum lipid levels when adjusted for established risk factors

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INTRODUCTION

Several studies have associated noise exposure with cardiovascular diseases (Melamed et al. 1999; Virtanen & Notkola 2002; Davies et al. 2005; Virkkunen et al. 2005; Gan et al. 2011) but van Kempen and colleagues concluded in a meta-analysis that the relation between noise exposure and ischemic heart disease is inconclusive (van Kempen et al. 2002). Serum lipids play a major role in the causation of cardiovascular diseases and may also be of causal importance for the association between noise exposure and cardiovascular disease (Nabel 2003). There is some empirical evidence that a noise exposure level above 80 dBA increases the level of cholesterol and triglycerides (Ortiz et al. 1974; Rai et al. 1981; Melamed et al. 1997; Vangelova & Deyanov 2007) but this effect has not been reported by others (Chang et al. 2003; Virkkunen et al , 2005, 2006). Serum lipid levels are influenced by sex, age, body mass index (BMI), waist width, statins, beta-blockers, and other medicines, diabetes, smoking, alcohol, physical activity and social status (Stone 1994; Kasiske et al. 1995; Hu et al. 2000; American Heart Association 2002; Virtanen & Notkola 2002; Carroll et al. 2005; Primatesta & Poulter 2006; Gossett et al. 2009), however, only Melamed et al. accounted for such competing risk factors (Melamed et al. 1997).

This study analysed if increasing levels of occupational noise exposure is associated with increasing levels of total cholesterol, low-density lipoprotein (LDL)-cholesterol, triglycerides and decreasing levels of high-density lipoprotein (HDL)-cholesterol when adjusted for well-documented risk factors.

METHODS

Subjects

In 2009-2010 we recruited 76 companies from manufacturing industries, construction and children day care with expected high noise exposure levels and finance intermediation as a reference. A total of 544 workers (396 male and 148 female workers) from these companies agreed to record noise levels and provide a blood sample and they comprised the study population. Triglycerides were not measured on 19 workers and therefore they have no estimates of LDL-cholesterol and triglycerides. Further details of recruitment procedures and measurements are described elsewhere (Kock et al. 2004). This cross-sectional study conformed to the Danish legal requirements and was approved by the local ethics committee.

Noise exposure assessment

Noise exposure levels were measured by portable dosimeters (*Bruel and Kjær 4443*). The dosimeters were calibrated, handed over and collected at the workplaces. The dosimeters were worn in a pouch attached to a belt at the participant's waist. Microphones were placed on the shoulder, right shoulder if right-handed and left shoulder if left-handed. The A-weighted equivalent sound level (L_{Aeq}) was recorded every

5 seconds for 24 hours. The dosimeters were set to a dynamic range of 50-120 dB. The display was dimmed during measurements to minimize noise dependent changes in the participant's behavior. On the day of the noise measurement, the participants registered the beginning and ending of working hours, transport time and leisure time, and we estimated the L_{Aeq} values for each of the three time periods.

Personal data

At the day of examination, we measured height, weight and waist width and calculated the BMI. We also collected a venous blood sample and analyzed the levels of total cholesterol, HDL-cholesterol and triglycerides by a chromogen catalytic method. We estimated LDL-cholesterol from the formula of Friedwald's: LDL-Cholesterol = total cholesterol – HDL-cholesterol – 0,45 x triglycerides (triglycerides < 4,5 mmol/l) (Danish Regions 2009).

Statistics

We classified participants into three full-shift occupational noise exposure groups: low (< 80 dBA), medium (80-85 dBA) and high (> 85 dBA). Potential confounders were tabulated by increasing noise levels. Lipid levels were normally distributed and arithmetic mean, standard deviation and 95% confidence intervals were tabulated by noise exposure levels. We analyzed serum lipids as a function of occupational noise exposure by linear regression. The models also included sex, age, BMI (<25, 25-35, >35) medicines (statins, beta-blockers, estrogens, retinoids, diuretics, levothyroxin or glucocorticoids; yes/no), diabetes, smoking, alcohol, physical activity, education, income, and noise exposure during leisure and transportation time. Trend test based on grouped and continuous data were performed in crude and adjusted models. Data processing and analysis were performed with STATA version 11 (STATA Corp., College Station, TX).

RESULTS

The median noise exposure level was 82 dBA (range: 57- 114 dBA). In all, 206 workers were exposed at a low, 198 at a median, and 140 at a high noise level. Increasing noise levels were associated with increasing BMI, waist width, and leisure and transportation time noise levels. Levels of total cholesterol, LDL-cholesterol, triglycerides and cholesterol/HDL ratio increased and HDL-cholesterol level decreased by increasing occupational noise exposure. The latter three findings were of statistical significance ($p<0,05$). However, when we adjusted for BMI no trend remained. Further adjustment by sex, age, medicines, diabetes, smoking, alcohol, physical activity, education, income, leisure time and transportation time noise levels confirmed this finding of no effect.

DISCUSSION

We observed no association between occupational noise exposure and serum lipid levels when account was taken for well-established risk factors. Thus, we could not confirm earlier suggested findings of such an effect. This discrepancy may be due to insufficient control for documented risk factors in earlier studies. Six studies (Ortiz et al. 1974; Rai et al. 1981; Melamed et al. 1997; Chang et al. 2003; Virkkunen et al. 2005, 2006; Vangelova & Deyanov 2007) have investigated the association between occupational noise exposure and lipid levels but only Melamed et al. adjusted for

such risk factors (sex, age, BMI, smoking, alcohol and physical activity). They found a significant association but only for a subpopulation (young men) and not for the total population. Four studies reported statistically significant crude associations between noise exposure and increasing lipid levels, but findings were not consistent across studies with respect to lipids affected (Ortiz et al. 1974; Rai et al. 1981; Melamed et al. 1997; Vangelova & Deyanov 2007). Our findings are, on the other hand, in line with those of Virkunen et al and Chang et al who observed no association between noise exposure level and serum lipid levels(Chang et al. 2003; Virkkunen et al. 2005, 2006).

This study has several strengths. Our extensive adjustment for risk factors has already been mentioned. Environmental noise exposure has also been suggested to affect cardiovascular health (Passchier-Vermeer & Passchier 2000; Babisch et al. 2005; Babisch 2011) and we were able to adjust for such potential confounding. Furthermore, each participant was equipped with personal dosimeters to ensure exact measurements of noise exposure in contradiction to stationary measurements. Selection bias is not expected to have affected findings since it is implausible that workers with high lipid levels and high noise exposure are more prone to enter or leave the noisy work sites recruited for this study. Differential recall is likewise unlikely to have biased findings because we analyzed objective measures of noise and lipids levels.

The study also has limitations. We did not take account of the use of hearing protectors. This might have contributed to the no effect seen since the higher exposed participants are expected to wear protective devices more often, thereby reducing the noise exposure contrast and the ability to detect a true effect of noise if existing. Further studies should account for the use of hearing protectors when assessing exposure at the ears of the individual worker.

To conclude, we observed no significant association between occupational noise exposure and serum lipid levels when adjusted for well-established risk factors. We could thus not support earlier findings suggesting that lipid levels may be of causal importance for the observed association between noise exposure and cardiovascular disease. Future studies should focus on improved exposure assessment at the ear in addition to established risk factors.

REFERENCES

American Heart Association (2002). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel Ø), Final Report. Circulation 106: 3143-3421.

Babisch W (2011). Cardiovascular effects of noise. Noise & Health 13: 201-204.

Babisch W, Beule B, Schust M et al. (2005). Traffic noise and risk of myocardial infarction. Epidemiology 16: 33-40.

Carroll MD, Lacher DA, Sorlie PD et al. (2005). Trends in serum lipids and lipoproteins of adults, 1960-2002. JAMA 294: 1773-1781.

Chang TY, Jain RM, Wang CS et al. (2003) Effects of occupational noise exposure on blood pressure. J Occup Environ Med 45: 1289-1296.

Danish regions (2009). Doctor's handbook.

Davies HW, Teschke K, Kennedy SM et al. (2005). Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology 16: 25-32.

Gan WQ, Davies HW, Demers PA (2011). Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999-2004. Occup Environ Med 68: 183-190.

Gossett LK, Johnson HM, Piper ME et al. (2009). Smoking intensity and lipoprotein abnormalities in active smokers. J Clin Lipidol 3: 372-378.

Hu D, Hannah J, Gray RS et al. (2000). Effects of obesity and body fat distribution on lipids and lipoproteins in nondiabetic American Indians: The Strong Heart Study. Obes Res 8: 411-421.

Kasiske BL, Ma JZ, Kalil RS et al. (1995). Effects of antihypertensive therapy on serum lipids. Ann Intern Med 122: 133-141.

Kock S, Andersen T, Kolstad HA et al. (2004). Surveillance of noise exposure in the Danish workplace: a baseline survey. Occup Environ Med 61: 838-843.

Melamed S, Froom P, Kristal-Boneh E et al. (1997). Industrial noise exposure, noise annoyance, and serum lipid levels in blue-collar workers--the CORDIS Study. Arch Environ Health 52: 292-298.

Melamed S, Kristal-Boneh E, Froom P (1999). Industrial noise exposure and risk factors for cardiovascular disease: findings from the CORDIS Study. Noise & Health 1: 49-56.

Nabel EG (2003). Cardiovascular disease. N Engl J Med 349: 60-72.

Ortiz GA, Arguelles AE, Crespin HA et al. (1974). Modifications of epinephrine, norepinephrine, blood lipid fractions and the cardiovascular system produced by noise in an industrial medium. Horm Res 5: 57-64.

Passchier-Vermeer W, Passchier WF (2000). Noise exposure and public health. Environ Health Perspect 108(Suppl 1): 123-131.

Primatesta P, Poulter NR (2006). Levels of dyslipidaemia and improvement in its management in England: results from the Health Survey for England 2003. Clin Endocrinol 64: 292-298.

Rai RM, Singh AP, Upadhyay TN et al. (1981). Biochemical effects of chronic exposure to noise in man. Int Arch Occup Environ Health 48: 331-337.

Stone NJ (1994). Secondary causes of hyperlipidemia. Med Clin North Am 78: 117-141.

van Kempen EE, Kruize H, Boshuizen HC et al. (2002). The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ Health Perspect 110: 307-317.

Vangelova KK, Deyanov CE (2007). Blood pressure and serum lipids in industrial workers under intense noise and a hot environment. Rev Environ Health 22: 303-311.

Virkkunen H, Kauppinen T, Tenkanen L (2005). Long-term effect of occupational noise on the risk of coronary heart disease. Scand J Work Environ Health 31: 291-299.

Virkkunen H, Harma M, Kauppinen T et al. (2006). The triad of shift work, occupational noise, and physical workload and risk of coronary heart disease. Occup Environ Med 63: 378-386.

Virtanen SV, Notkola V (2002). Socioeconomic inequalities in cardiovascular mortality and the role of work: a register study of Finnish men. Int J Epidemiol 31: 614-621.