Exposure to traffic noise and central obesity

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ABSTRACT

Background: Exposure to traffic noise has been linked to health effects which are partly stress related, e. g. hypertension, type 2 diabetes and obesity. The evidence is very limited for obesity, although the public health implications may be important. This study aimed to elucidate associations between exposure to noise from road traffic, railways or aircraft and markers of obesity.

Methods: The study included 5075 men and women living in Stockholm County during 2002-2006. Exposure to traffic noise from different sources at residential addresses was estimated based on geographical coordinates and digital noise maps. Questionnaires and clinical examinations provided information on risk factors and markers of obesity, primarily waist circumference, waist-hip ratio and body mass index (BMI). Linear and logistic regression was used to assess associations between noise exposure from the different traffic sources and obesity markers, with adjustments for potential confounding factors.

Results: There were statistically significant associations between traffic noise and waist circumference, with a 0.3 cm increase (95 % CI 0.1-0.5) per 5 dB(A) L_{den} for road traffic, 0.6 cm (0.2-1.0) for railway and 1.0 cm (0.7-1.4) for aircraft noise. Combined exposure to all three sources exceeding 45 dB(A) was associated with an odds ratio for obesity of 1.9 (1.2-3.0). Similar patterns were seen for waist-hip ratio while no clear associations were found for BMI.

Conclusions: Traffic noise exposure appears to increase the risk of central obesity, particularly in relation to combined exposure from different sources.

INTRODUCTION

Increasing evidence points to traffic noise as a risk factor for cardiovascular diseases such as hypertension, myocardial infarction and stroke (Babisch 2008; Selander et al. 2009b; Eriksson et al. 2010; Sörensen et al. 2011; Dratva et al. 2012; Hansell et al. 2013). Recent findings suggest that traffic noise may also affect the metabolic system, for example inducing Type 2 diabetes and central obesity (Sörensen et al. 2013; Eriksson et al. 2014). Traffic noise can influence cardiovascular and metabolic

functions through sleep disturbances and chronic stress. For example, short sleep duration is associated with a reduction of serum leptin and elevation of ghrelin, leading to an increased appetite and reduced energy expenditure (Taheri et al. 2004; Chaput et al. 2007). Furthermore, an activation of the Hypothalamic-Pituitary-Adrenal axis results in elevated levels of cortisol, which promotes central fat deposition and impaired glucose regulation (Björntorp 1997; Spiegel et al. 1999; Rosmond 2003). Recent evidence indicates that combined exposure to several environmental stressors, including community noise, is particularly harmful (Selander et al. 2013). The aim of the present was to investigate exposure to multiple traffic noise sources, i.e. road, railway and aircraft traffic, in relation to markers of obesity. In particular, the role of combined exposure to different noise sources was assessed.

METHODS

Study population

This cross-sectional study is based on a follow-up survey performed between 2002 and 2006 within the Stockholm Diabetes Prevention Program (Eriksson et al. 2008). Briefly, the aim of the program was to study risk factors for Type 2 diabetes as well as to implement and evaluate methods for prevention. Participants in the original study were recruited between 1992 and 1998 from five municipalities in Stockholm including suburban or semirural areas. Eight to ten years later, the participants were invited to the follow-up survey. In total, 5 712 persons participated in the follow-up survey (71% of those at baseline), including 2 383 men and 3 329 women aged 47 to 64 years. At the follow-up survey, the participants completed a detailed questionnaire covering lifestyle habits, such as food behavior, smoking, alcohol intake and physical activity, as well as health status, psychological distress, insomnia and job strain. In addition, questions were asked on noise annoyance and noise sensitivity. The survey also included measurements of height, weight, waist and hip circumference as well as a medical examination with blood pressure measurements and an oral glucose tolerance test.

Exposure assessment

Information on the complete residential history from 1991 to follow-up was used to identify participants who had lived at the same address for more than 10 years. The geocoded address points were combined with digital maps of road traffic, railway and aircraft noise. Noise levels were modeled using the Nordic Prediction Method for road traffic and took into account terrain data, ground surface, building height and traffic flow, including 24 h yearly average, diurnal distribution, speed limits as well as information on noise barriers. Railway noise was assessed for the year 2007 according to the European Environmental Noise Directive and recalculated to L_{den}. Information on 3D terrain data, ground surface, building height, noise screens, type and number of trains per hour, average speed and length of the trains was considered. The assessment of aircraft noise around Stockholm Arlanda Airport was assessed using 1 dB resolution contours ranging from 50 to 65 dB L_{den} averaged during 1997 to 2002. The noise levels were modelled from radar tracks by the Swedish Airports and Air Navigation Service.

Definition of outcome

Anthropometric markers of obesity were defined according to the WHO criteria for the European population. Body mass index (BMI) was calculated as the weight divided by the squared height [kg/m^2] with a cut-offs at 30 to define obesity. Gender specific cut-off values for central obesity were applied for waist circumference: ≥88 cm for women and ≥ 102 cm for men. Waist-hip ratio was calculated as the waist circumference divided by the hip circumference. Cut-offs of 0.85 and 0.90 were applied to women and men, respectively, as definition of central obesity.

Statistical analysis

The association between road traffic noise and each of the outcomes was analyzed using linear regression models, estimating regression coefficients and 95% confidence intervals (CI). Associations between road traffic noise and prevalence of overweight as well as central obesity, defined as binary variables according to WHO cutoffs, were analyzed using logistic regression models, estimating odds ratios (OR) and 95% CI. Final models included age, gender, diet, alcohol consumption, education, physical activity during leisure time, smoking status, psychological distress, job strain, shift work and noise sensitivity.

In sensitivity analyses, we investigated how the effects of road traffic noise on obesity markers were altered by adjustment for contextual confounding, other sources of noise as well as air pollution. The analyses of contextual confounding were performed based on mean income in small areas considered homogenous with respect to socio-economic characteristics. Furthermore, we restricted the population to those who had lived at the same address for at least 10 years, were exposed to road traffic noise only or had no family history of diabetes. To investigate interactions, we performed stratified analyses based on gender, age group, occupational status, physical activity, smoking**,** education level, diabetes heredity, noise sensitivity, noise annoyance, job strain as well as sleep disturbances.

In total, 637 (11%) out of the 5 712 participants were excluded from further analyses because they had moved out of the municipalities under study before follow-up or due to missing information on exposure, outcome or confounding variables. Thus the final sample for analyses included 5 075 participants, 2 040 men and 3 035 women.

RESULTS

We observed an association between road traffic noise and waist circumference with a 0.3 cm (95% CI 0.1-0.5) increase per 5 dB rise in L_{den}. An association was also seen for waist-hip ratio. On the other hand, BMI was not clearly related to road traffic noise. Increments per 5 dB were 0.6 cm (95% CI 0.2-1.0) and 1.0 cm (95% CI 0.7-1.4) for railway and aircraft noise, respectively. There were no clear exposure-response relations, except for aircraft noise, thus the results based on linear increments should be interpreted with some caution.

The OR of central obesity among those who were exposed to road traffic noise ≥45 dB was 1.2 (95% CI 1.0-1.3). In sensitivity analyses, this estimate was not affected by adding area-based mean income to the model or by adjustment for air pollution. Furthermore, there were no strong interactions in relation to road traffic noise exposure and waist circumference except age, where associations were confined to those below 60 years of age. We observed a clear exposure-response association between the number of traffic noise sources ≥45 dB and risk of abdominal obesity. The OR increased from 1.3 (95% CI 1.1-1.5) among those exposed to one source only to 1.9 (95% CI 1.2-3.0) among those exposed to all three traffic noise sources.

DISCUSSION

Our findings show relationships between traffic noise exposure and markers of central obesity. Associations were indicated for noise from different sources, including roads, railways and aircraft, but appeared strongest for aircraft noise. Road traffic was the most common source of noise and the association with central obesity was not affected by contextual socio-economic indicators or exposure to air pollution from road traffic. Strong interactions between road traffic noise and other risk factors were not indicated with exception of age, where associations were only seen in those below 60 years of age. Furthermore, the risks appeared particularly elevated in those concomitantly exposed to traffic noise from all three noise sources.

There is only limited evidence from epidemiologic studies linking noise exposure to overweight or obesity. However, noise may be an important stressor, inducing cortisol production and other stress reactions (Ising & Braun 2000; Spreng 2000; Babish et al. 2001; Persson Waye et al. 2003; Ising & Kruppa 2004; Selander et al. 2009a). Elevated levels of cortisol can lead to storage of fat in visceral depots, contributing to central rather than generalized obesity (Björntorp 1997; Kyrou et al. 2006). This may explain why the effects of noise are mainly seen for markers of central obesity, such as waist circumference and waist-hip ratio. Furthermore, traffic noise may affect metabolic as well as cardiovascular functions through sleep disturbances (Zaharna & Guilleminault 2010), which may influence the central control of appetite and energy expenditure (Spiegel et al. 1999; van Cauter et al. 2008).

Recent studies indicate health effects of residential road traffic noise exposure and myocardial infarction (Babisch 2008; Selander et al. 2009b), stroke (Sörensen et al. 2011), hypertension (Dratva et al. 2011) as well as diabetes (Sörensen et al. 2013). The results of the present study suggest that exposure to road traffic noise may be associated also with central obesity, in particular waist circumference. These results are in line with the general stress concept for noise induced health effects (Babisch 2003) and identify central obesity as a potential mediator of noise effects on development of cardiovascular diseases as well as diabetes.

There appeared to be stronger effects by air craft noise on central obesity than by traffic noise from other sources. This is consistent with results in studies on annoyance and sleep disturbances (Miedema 2004; Griefahn et al. 2006). Furthermore, we observed a clear exposure-response association between the

number of traffic noise sources and risk of abdominal obesity, which is also supported by data on annoyance and sleep disturbances. However, our study is the first to assess combined effects from different noise sources on metabolic outcomes.

In conclusion, our study showed an association between traffic noise exposure and waist circumference or central obesity. The relations appeared particularly strong for aircraft noise and in those with concomitant exposure to different sources of traffic noise. Our findings may be of importance for the understanding of noise induced cardiovascular effects and open up the possibility of a wide variety of other types of adverse health effects.

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