

Long-term exposure to traffic noise and development of obesity

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ABSTRACT

A few studies have shown associations between exposure to traffic noise and obesity markers but most of the evidence is cross-sectional or based on self-reported outcome information. We assessed individual long-term exposure to noise from road traffic, railways and aircraft based on residential histories in a cohort of 5151 men and women from Stockholm County. Noise levels were estimated at the most exposed façade of each dwelling based on a modification of the Nordic prediction method. Waist circumference, weight and height were measured by trained nurses at recruitment and after an average of 8.9 years of follow-up. Extensive information on potential confounders was available from repeated questionnaires and registers. There were clear trends in weight circumference gain as well as in incidence of central obesity in relation to exposure to noise from road traffic and aircraft ($p < 0.001$) but not from railways. Risks appeared particularly high in those with elevated exposure to all three traffic noise sources. Our results provide further evidence linking noise exposure to development of central obesity.

BACKGROUND

Several studies indicate increased risks of hypertension and myocardial infarction in relation to long-term exposure to road traffic noise [1]. We were the first to document a relation between long-term exposure to environmental noise and obesity markers [2], which has later been confirmed in other studies [3-6], but the evidence is not wholly consistent [7]. Only two of the studies, besides our first study, were longitudinal and these two studies used self-reported anthropometric data, which are prone to bias. Furthermore, associations have been reported between environmental noise exposure and type 2 diabetes, but only few studies are available [8]. Etiological mechanisms may be similar for noise induced cardiovascular and metabolic effects, including sleep disturbances chronic stress. There is a great need for further evidence from longitudinal studies on noise and overweight as well as diabetes, particularly on quantitative exposure-response relationships.

In a previous publication we reported results based on a cross-sectional analysis of traffic noise exposure and markers of obesity in a cohort from Stockholm County [4]. The present study is based on the same study population, but has a longitudinal design, and uses a newly developed methodology enabling more precise assessment of long-term exposure to traffic noise from different sources. The aim of this paper is to present some results regarding waist circumference development and risk of central obesity related to noise exposure from road traffic, railways and aircraft.

METHODS

Study outline and participants

The study was based on the Stockholm Diabetes Prevention Program cohort, which has been described in detail previously [9]. Participants were recruited between 1992 and 1998 from five municipalities in Stockholm. By an enrichment procedure approximately half of the participants were selected to have a family history of diabetes. A total of 3162 men and 4946 women participated in a baseline survey and medical examination. Eight to ten years later 5712 persons took part in a follow-up survey and medical examination, corresponding to 80% of those invited.

At both baseline and follow-up investigations, participants filled out questionnaires and trained nurses measured weight, height and waist circumference as well as performed an oral glucose tolerance test. The questionnaires covered health status and lifestyle habits such as smoking, alcohol intake and physical activity during leisure time, dietary habits, psychological distress, shiftwork, insomnia and job strain. Moreover, the follow-up questionnaire enquired about noise annoyance and noise sensitivity. We also obtained information on residential address history for the participants from the Swedish Population Register.

The study was conducted in accordance with the Helsinki Declaration and approved by the Regional Ethics Review Board at Karolinska Institutet.

Exposure assessment

To assess long-term individual road traffic noise exposure we have constructed a noise database for Stockholm County to represent the period from 1990 and onwards. The database contains information from several national, regional and local authorities and includes 3D terrain data as well as information on ground surface, road net, daily traffic flows, speed limits and percentage of heavy vehicles. To calculate noise levels we developed and used a modification of the Nordic prediction method for road traffic [10], where possible reflection and shielding are taken into account by a Ground Space Index based on building density [11]. Our method has been validated against the full Nordic prediction method modelled with SoundPlan and showed coherent estimates [12]. For each participant, the time-weighted average noise exposure during follow-up was calculated, taking into account all addresses in Stockholm County where the subject has lived, and considering the duration of residence at each address.

For railway noise, we used parts of the same database as for road traffic noise supplemented with relevant information on the railway net, such as speed limits, train counts and train types, as well as the exact 24h distribution for different parts of the railway net during follow-up. Annual aircraft noise contour data were available around Arlanda and Bromma airports from 1995 and onwards. Time-weighted exposure to railway and aircraft noise, respectively, was computed for each individual in the same way as for road traffic noise.

Assessment of exposure to locally emitted air pollution was based on a methodology developed by the Environment Department of Stockholm City, together with us, to estimate long-term source-specific exposure [13]. It entails geocoding of an individual's addresses and use of an emission inventory together with dispersion models to estimate outdoor levels of selected pollutants from different emission sources over time at relevant geographical locations. Based on the individual residential history we calculated time-weighted exposure to NO_x from road traffic for each participant during follow-up.

Questionnaires constituted the main source of information on important determinants for the health outcomes under study, such as life style factors and socioeconomic status (SES). In addition, information on SES variables was obtained for small socioeconomically homogeneous areas using data from Statistics Sweden, which enabled control of contextual confounding.

Outcome definitions

All measurements of weight and height as well as of waist circumference were performed by trained nurses according to a standard protocol during the baseline and follow-up investigations. Height and weight were measured standing without shoes and rounded to the nearest half centimetre or 100 g, respectively. Waist circumference was measured in lying position, midway between the lower costal margin and iliac crest. Anthropometric markers of obesity were defined according to the WHO criteria for the European population. BMI was calculated as the weight divided by the squared height (kg/m²) with a cut-off at ≥ 25 to define overweight. Sex-specific cut-off values for central obesity were applied for waist circumference: ≥ 88 cm for women and ≥ 102 cm for men [14].

Statistical methods

Linear regression models were used to analyze associations between traffic noise exposure and weight or waist circumference changes with estimation of regression coefficients and 95% confidence intervals (95% CI). Homoscedastic variance was checked by residual plots, and normality was assessed by normal probability plots of the residuals. The analyses were performed for continuous traffic noise exposures, and associations are presented for an increment of 10 dB L_{den}. To examine associations between traffic noise exposure and incidence of central obesity and overweight we used Poisson regression models estimating incidence-rate ratios (IRR) and 95% CI, excluding those with the outcome at baseline.

Additionally, we tested the assumption of linearity between traffic noise and weight or waist circumference changes. Firstly, we performed analyses with a categorical exposure variable (<45, 45-49, 50-54 and ≥ 55 dB L_{den}) by inserting it in the linear model. Secondly, we performed restricted cubic splines analyses with three knots. Furthermore, we assessed the effect of combined exposure to multiple noise sources by creating dummy variables, indicating subjects exposed to none, one, two or three traffic noise sources ≥ 45 dB L_{den}. We performed non-parametric trend tests for the ranks across exposure groups to estimate p-values for trend.

RESULTS

A total of 5151 subjects participated in the follow-up survey and had full information on all relevant exposure and outcome variables as well as covariates. The Table presents associations between traffic noise exposure from different sources and waist circumference increase during follow-up. In the fully adjusted model we observed an association between

road traffic noise and waist circumference increase of 0.03 cm/year (95%CI 0.02 to 0.05) per 10 dB L_{den} . For aircraft noise the waist circumference increase was 0.14 cm/year (95%CI 0.12 to 0.16) per 10 dB L_{den} . No clear association was observed between railway noise and waist circumference increase. Weight gain was associated with aircraft noise but not with road or railway traffic noise exposure (results not shown).

Table: Waist circumference increase (cm/year) in relation to noise exposure from different traffic noise sources during follow-up in a cohort from Stockholm County.

Exposure ^a	Road traffic noise		Railway noise		Aircraft noise	
	No.	β^b (95% CI)	No.	β^b (95% CI)	No.	β^b (95% CI)
Continuous						
per 10 dB L_{den}	5151	0.03 (0.02;0.05)	5151	0.00 (-0.02;0.03)	5151	0.14 (0.12;0.16)
Categorical, dB L_{den}						
<45	3435	0 (reference)	4757	0 (reference)	4235	0 (reference)
45-49	952	-0.05 (-0.10;0.004)	161	0.01 (-0.10;0.13)	126	0.31 (0.18;0.43)
50-54	561	0.12 (0.05;0.18)	124	0.07 (-0.06;0.19)	588	0.44 (0.38;0.50)
≥ 55	203	0.14 (0.04;0.24)	109	-0.06 (-0.20;0.07)	202	0.49 (0.39;0.59)
<i>trend p-value</i>		<i><0.001</i>		<i>0.901</i>		<i><0.001</i>

^a Time-weighted noise exposure expressed as L_{den} taking into account where the subject had lived during the follow-up period.

^b Results of linear regression model adjusted for sex, age, dietary habits, alcohol consumption, education level, physical activity, smoking status, psychological distress, job strain and shift work.

We observed an exposure-response relation between the number of traffic noise sources and risk of central obesity (Figure). The IRR increased from 1.22 (95%CI 1.07 to 1.38) among those exposed to only one source to 2.29 (95%CI 1.58 to 3.32) among those exposed to all three traffic noise sources (p-value for trend <0.001). For waist circumference a positive trend was observed with a change of 0.13 cm/year (95%CI 0.09 to 0.17) among those exposed to only one source and of 0.46 (95%CI 0.27 to 0.65) among those exposed to all three traffic noise sources (p-value for trend <0.001) (data not shown). On the other hand, no trend was seen in relation to weight gain.

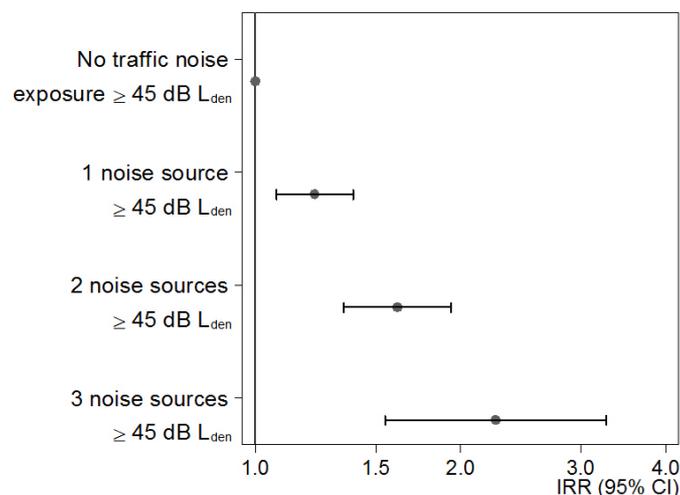


Figure: Incidence rate ratio (IRR) of central obesity in relation to noise exposure ≥ 45 dB L_{den} from road traffic, railways and/or aircraft during follow-up in a cohort from Stockholm County.

DISCUSSION

There is growing evidence that traffic noise affects adiposity markers. The first study to investigate this relationship focused on aircraft noise and showed an association particularly for waist circumference [2]. A Norwegian cross-sectional study found a positive association between road traffic noise and BMI only among some subgroups [7]. However, based on a longitudinal study a Danish group reported associations between residential exposure to road traffic or railway noise and waist circumference as well as weight changes in adults [3], confirming earlier cross-sectional evidence in the same cohort [6]. Our observed waist circumference increase related to road traffic (0.02-0.05 cm/year per 10 dB L_{den}) corresponds well to the findings in the Danish cohort (0.02-0.12 cm/year per 10 dB L_{den}) [3]. A limitation with the Danish study is that the anthropometric data at follow-up were based on self-reports. We already published results on traffic noise exposure and obesity markers based on cross-sectional analyses of the same cohort as in present study [4]. This new longitudinal analysis used a much more detailed methodology for assessment of noise exposure and showed associations primarily for waist circumference increase in relation to road traffic or aircraft noise exposure. Overall, the evidence is not fully consistent regarding a role for traffic noise in the development of central obesity (increased waist circumference or waist-hip ratio) or general adiposity (weight gain or BMI). Stress mechanisms as mediated by cortisol excretion would be expected to primarily result in central obesity, while noise-induced sleep disturbances and behavioral changes might also mediate effects on general adiposity [15]. Elucidation of noise effects on specific adiposity markers may provide evidence on causal mechanisms.

In our study, the waist circumference increase per unit of noise exposure was highest for aircraft noise. The effect appeared lower for road traffic noise, and there was no clear association for railway noise. This pattern is well in line with the effect of noise exposure on annoyance and sleep disturbances [16, 17], where aircraft noise causes more pronounced effects than road traffic noise at the same noise levels, while railway noise is less harmful than road traffic noise [18]. There is a great need for further longitudinal evidence on the association between traffic noise from different sources and obesity.

We saw a clear exposure-response relation related to number of noise sources for both risk of central obesity risks and waist circumference increase. This goes in line with previous studies of sleep effects and annoyance in relation to combined exposure to different noise sources [19, 20]. Our findings speak in favor of the multiple environmental stressors theory, where several stressors may enhance the effect of each other [21]. Moreover, interactions have been observed between traffic and occupational noise as well as job strain in relation to the risk of myocardial infarction [22]. It is important to further investigate interactions between different environmental stressors including noise for both cardiovascular and metabolic outcomes.

A limitation of our study is the relatively low traffic noise levels and the small number of highly exposed participants. Furthermore, in certain aspects, the data on noise exposures are imprecise. For example, we lack information on noise exposure other than from the three traffic sources, such as occupational exposure. Additionally, we do not have information on noise modifiers, such as façade and window insulation as well as bedroom location, open/closed windows, use of earplugs, etc. Moreover, by design, the study population was enriched with persons with a family history of diabetes and the results may not be generalizable to the population as a whole.

The strengths of the present study include the prospective design and anthropological data measured by trained nurses at recruitment as well as at follow-up. Additionally, detailed

information was available regarding potential individual confounders (socioeconomic position, diet, alcohol consumption, smoking and physical activity, etc.) as well as area-based confounders. Nevertheless, residual confounding cannot be excluded. Furthermore, we had a detailed residential history for all participants, allowing exposure assessment for the whole follow-up period. A particular feature of our study is that a sizable proportion of the study participants was exposed to several noise sources, allowing evaluation of health effects following exposure to multiple noise sources.

In conclusion, our study showed associations between exposure to noise from road traffic or aircraft and development of central obesity. The risk of central obesity appeared particularly high for aircraft noise and in those with concomitant exposure to different sources of traffic noise. These findings support the evidence linking traffic noise exposure to obesity.

Acknowledgements

The study was supported by a grant from the Swedish Research Council for Health, Working Life and Welfare.

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