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Associations of road traffic noise with adipose tissue depots and hepatic fat content – Results from the German National Cohort (NAKO)

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Abbreviations: AT, Adipose tissue; BMI, Body mass index; CI, Confidence Interval; CVD, Cardiovascular disease; DAG, Directed acyclic graph; DXA, Dual-energy x-ray absorptiometry; EIONET, European Environment Information and Observation Network; EEA, European Environment Agency; L_{den}, Day–evening–night noise level; END, Environmental Noise Directive; MASLD, Metabolic-dysfunction associated steatosis liver disease; MRI, Magnetic resonance imaging; NAKO, German National Cohort; NDVI, Normalized Difference Vegetation Index; NO_2, Nitrogen dioxide; OR, Odds ratio; PM, Particulate matter; VAT, Visceral adipose tissue; SCAAT, Subcutaneous abdominal adipose tissue; SCTAT, Subcutaneous thoracic adipose tissue; WHO, World Health Organization.

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ABSTRACT

Little is known about the relation between traffic noise exposure, an established environmental risk factor for cardiovascular disease, and early obesity-related risk markers such as adipose tissue (AT) and hepatic fat. Therefore, we aimed to assess associations of long-term road traffic noise exposure with AT depots measures from whole-body magnetic resonance imaging (MRI). We analyzed cross-sectional data from 11,343 participants from the population-based German National Cohort (NAKO) who underwent MRI examination between 2014 and 2016, considering visceral (VAT), subcutaneous abdominal (SCAAT), subcutaneous thoracic AT (SCTAT) and hepatic fat content as outcomes. Annual road traffic noise (L_{den}) data from the year 2017 (source: central EIONET data repository) was used to calculate weighted mean noise levels on a continuous scale within 10 and 100-meter buffers of participants' residencies. Among 11,101 participants with complete outcome data, 48.7 % were women, and the mean age was 51.9 years. Higher annual L_{den} was associated with increased AT depots and hepatic fat content in men (e.g., VAT: 1.72 %-change [95 % confidence interval: [0.14 %; 3.30 %]; SCAAT: 2.18 %-change [0.43 %; 3.93 %], hepatic fat content: 3.57 %-change [1.41 %; 5.78 %] per 10 dB(A) increase in L_{den} (10 m)) and women (e.g., VAT: 3.13 %-change [1.09 %; 5.18 %]; SCAAT: 2.38 %-change [0.55 %; 4.20 %], hepatic fat content: 3.08 %-change [1.00 %; 5.21 %] per 10 dB(A) increase in L_{den} (10 m)). Associations were robust with all outcomes after adjusting for air pollutants and surrounding greenness, and effect modification by obesity and hypertension was observed for SCAAT, SCTAT and hepatic fat content. Our findings indicate that annual exposure to road traffic noise is associated with increased adipose tissue depots and hepatic fat content, and thus present novel evidence for the cross-sectional association between noise and early MRI-derived metabolic health markers.

1. Introduction

The continuing rise in cardiometabolic diseases, such as cardiovascular disease (CVD), diabetes mellitus, hypertension, or dyslipidemia, is a severe threat to humans' health (Roth et al., 2020; Vos et al., 2020; Zhang et al., 2019; Cheng et al., 2022). Obesity is a major phenotype of cardiometabolic disease; an unfavorable distribution of body adipose tissue (AT) is associated with a clustering of adverse conditions (Tchernof and Despres, 2013; Huang et al., 2022; Munzel et al., 2024; Bechmann et al., 2012). Increased visceral adipose tissue (VAT) is also strongly associated with increased hepatic fat content and hepatic steatosis (Tchernof and Despres, 2013; Bechmann et al., 2012), resulting in metabolic dysfunction-associated steatotic liver disease (MASLD), whereas the role of other AT depots, such as subcutaneous adipose tissue, is less well-defined.

Not only behavioral but also non-behavioral risk factors such as the environment need to be considered in obesity prevention (Bluher, 2019). The WHO (Word Health Organization) has ranked noise exposure as the second most harmful environmental risk factor after air pollution (WHO, 2018). About one-fifth of the European population is exposed to harmful day-evening-night noise levels (L_{den}) above 55 dB(A), which are associated with various health outcomes including hypertension, CVD, cognitive and hearing impairments, and psychological well-being (WHO, 2018; Dzhambov and Lercher, 2019; Hao et al., 2022; Wang et al., 2020). Numerous studies have suggested direct pathways through which noise exposure affects health (Munzel et al., 2024; Sivakumaran et al., 2022). In particular, it is suggested that noise-induced chronic stress is associated with the promotion of cardiovascular risk factors, e.g., high blood pressure, and elevated blood glucose and lipids (Sivakumaran et al., 2022).

Recently, it has also been hypothesized that traffic noise is linked to metabolic disease and obesity (Wang et al., 2020; Gui et al., 2022; Thacher et al., Dec 2021; Altug et al., 2024). While previous studies conceptualized obesity by anthropometric measures such as body mass index (BMI) or waist circumference (Gui et al., 2022), a differentiation of AT depots and assessment of hepatic fat content is only possible by medical imaging such as dual-energy x-ray absorptiometry (DXA) or magnetic resonance imaging (MRI) (Altug et al., 2024; Schaapman et al., 2021; Bamberg et al., 2015). Contributing to this, defining obesity solely by BMI is currently reconsidered, as BMI may not provide reliable information on an individual disease risk (Rubino et al., 2025). As advocated by the Lancet Diabetes & Endocrinology commission (Rubino et al., 2025), excess adiposity determined by MRI-derived AT measures may more accurately inform about cardiometabolic disease risk (Tchernof and Despres, 2013; Kammerlander et al., 2021; Abraham et al., 2015). Studies using the Framingham Heart study showed that in particular VAT is an independent, strong risk factors for cardiometabolic diseases beyond overall adiposity, particularly in women (Kammerlander et al., 2021; Abraham et al., 2015). Consequently, studies of associations between road traffic noise and MRI-derived AT measures are essential to understand the relationship between environmental factors and cardiometabolic disease risk, but are currently lacking.

In addition, other environmental exposures may lead to increased measures of obesity and increased hepatic fat content and may therefore be potential confounders in the association between road traffic noise and obesity (Matthiessen et al., 2023; Wang et al., 2022; Cai et al., 2022). In order to assess unbiased associations between noise and health, it is crucial to take into account potential other environmental risk factors, particularly in urban areas, where air pollution and lack of greenness co-occur (Nieuwenhuijsen, 2020). Previous studies that adjusted for other environmental exposures indicated predominantly robust associations of noise exposure with various health outcomes, which need further confirmation (Thacher et al., Dec 2021; Vienneau et al., 2023; Sorensen et al., 2022; Foraster et al., 2018; Cai et al., 2020).

Previous studies in animals and humans also suggested that individuals with cardiometabolic diseases may be more susceptible to exposure to adverse noise levels (Foraster et al., 2018; Munzel et al., 2025), which may be attributable to stronger deterioration of endothelial dysfunction (Munzel et al., 2024; Munzel et al., 2025). Olbrich et al. (2023) showed that recurrence of CVD events was higher in patients exposed to higher levels of aircraft noise, with similar but insignificant trends for railway and road traffic noise (Olbrich et al., 2023). Therefore, we hypothesized that the association between noise and AT measures may be stronger in participants with existing cardiometabolic disease, providing important clues to potentially susceptible subgroups of the population.

Therefore, we aimed to assess the sex-specific associations of long-term road traffic noise exposure with AT depots and hepatic fat content measured by MRI in the German National Cohort (NAKO), testing the following hypotheses:

(1) Higher levels of annual road traffic noise are associated with increased AT depots, hepatic fat content, and increased frequency of MASLD.

(2) Adverse associations of long-term road traffic noise are independent of exposures to air pollution, and surrounding greenness.
(3) In participants with prevalent cardiometabolic disease, adverse associations of road traffic noise exposure are more pronounced than in participants without those conditions.

2. Methods

2.1. Study population

NAKO is a German-wide, population-based, multicenter cohort that assesses a range of chronic diseases, their risk factors, and etiology (Peters et al., 2022; German National Cohort, 2014). Between 2014 and 2019, 205,415 participants aged 20–74 years underwent a baseline examination across 18 NAKO study centers, where comprehensive medical and physical examinations were performed. A subgroup of 30,868 participants had a complete whole-body MRI using 3 T scanners (MAGNETOM Skyra, Siemens Healthineers, Erlangen, Germany) for neurologic, cardiovascular, thoracoabdominal, and musculoskeletal imaging at one of the five imaging sites. (Peters et al., 2022; Bamberg et al., 2024) NAKO was approved by the local ethics committees and all participants gave written informed consent before study enrollment. The current analysis includes 11,343 participants whose imaging data was collected between 2014 and 2016, and for whom image evaluation has been completed at the time of the study.

2.2. Outcome

The main outcome measures were VAT, subcutaneous abdominal AT (SCAAT), subcutaneous thoracic AT (SCTAT)), and hepatic fat content. For the assessment of AT depots and hepatic fat content, a T1-weighted 3D VIBE two-point Dixon technique was performed (Bamberg et al., 2015; Schlett et al., 2016; Haueise et al., 2023). Derivation of MRI-based outcomes used a deep learning approach (3D nnU-Net) that automatically segmented AT depots in the trunk, namely VAT (AT inside the abdominal cavity), SCAAT, SCTAT and the liver on MRI images (Haueise et al., 2023; Haueise et al., 2024; Isensee et al., 2021). AT quantification was standardized based on anatomical landmarks: mid-femoral head to cardiac apex for VAT and SCAAT, cardiac apex to mid-humeral head for SCTAT. AT volumes are reported as liters and hepatic content as percentages. Prior to hepatic fat segmentation, fat–water signal swap artifacts were detected and repaired in underlying images by an automatic approach based on nnU-Net. As a secondary outcome, we classified participants as having prevalent MASLD. Therefore, participants with hepatic fat content > 5.6 % and at least one of the following cardiometabolic conditions: BMI ≥ 25 kg/m^2, a diagnosis of diabetes, hypercholesterolemia, or hypertension were classified as having MASLD (alcohol consumption: < 30 g/day for men, < 20 g/day for women) or MetALD (alcohol consumption: ≥ 30 – < 60 g/day for men; ≥ 20 – < 50 g/day for women) (Rinella et al., 2023; American Association for the Study of Liver Diseases, Latin American Association for the Study of the Liver, European Association for the Study of the Liver, 2023). Participants with high hepatic fat content and excessive alcohol consumption (≥ 60 g/day for men and ≥ 50 g/day for women), classified as MetALD or with a known hepatitis B and C diagnosis were excluded from the analysis.

2.3. Exposure

Annual road traffic noise exposure data in Germany for the reference year 2017 is provided from the central European Environment Information and Observation Network (EIONET) data repository (https://cdr.eionet.europa.eu/). Separate datasets were available for each urban area (>100,000 inhabitants) or regions along major roads (>3 million vehicle passages annually). According to the Environmental Noise Directive (END) obligation 2002/49/EC Article 3 (European Commission, 2002), EU member countries are obliged to map harmful noise exposure levels ≥ 55 dB(A) in urban areas or in regions along major roads. Details on how these datasets were processed, harmonized and aggregated into a German-wide map (Fig. 1) can be found in Staab et al. (2025). Briefly, separate datasets in polygonal shapefile format were available for areas subject to noise mapping under the Directive, with road traffic noise exposure expressed as L_{den} noise classes in five decibel steps (55: 55–59 dB(A), 60: 60–64 dB(A), 65: 65–69 dB(A), 70: 70–74 dB(A), 75: ≥ 75 dB(A)) with a resolution of 10 m × 10 m (Staab et al., 2025; Wolf et al., 2025). We assigned the lower road traffic noise values of the 5 dB(A) band (e.g.: 55 dB(A) if 55–59 dB(A)) of the corresponding grid cells to the participants' geocoded addresses. In urban areas covered by the END obligation 2002/49/EC Article 3 section k (European Commission, 2002), grid cells with missing values were assigned a value of 40 dB(A) as the lower detection limit (Fig. 1), as missing data can be attributed to noise levels below the obligatory reporting thresholds of 55 dB(A). We then calculated mean noise levels in buffers of 10 m and 100 m around the geocoded address points to derive area-weighted levels on a continuous scale, which we used for our main analysis. Prior to this, grid cells without data (coded NA in Fig. 1; not covered by the END obligation as areas are not along major roads or urban areas, 2002/49/EC Article 3 section k and n (European Commission, 2002) were also set to 40 dB(A). In the following sections, we use the term noise for road traffic noise.

2.4. Covariates

Potential covariates were assessed by using standardized touchscreen-based questionnaires, face-to-face interview, or physical examinations conducted by trained staff at examination day. We considered age at examination, study center, various lifestyle factors and individual socioeconomic status. Age in years at examination was available and participants were grouped in eight study center regions (Augsburg, Berlin, Essen, Düsseldorf, Mannheim, Münster, Neubrandenburg, Saarbrücken). Lifestyle factors included physical activity, which was assessed in minutes per week using the standardized Global physical activity questionnaire (GPAQ) questionnaire and evaluated in accordance with the WHO protocol (Leitzmann et al., 2020; WHO, 2021).

Alcohol consumption was quantified in drinks per week and subsequently converted to grams per day. Participants were classified as never-smokers, ex-smokers, or current smokers based on their self-reported smoking behavior. Individual socioeconomic status was reflected by income given in euros. Body mass index (BMI) was calculated from measured height and weight by dividing weight by height in square meters (kg/m^2). Height and weight were measured using a seca stadiometer 274 and a scale mBCA 515 (seca GmbH & Co. KG, Hamburg, Germany) (Fischer et al., 2020). Information on physician-diagnosed diabetes mellitus and hypercholesterolemia is based on self-report by participants in face-to-face interviews. Hypertension was defined by measured systolic and diastolic blood pressure values (≥ 140 mmHg and ≥ 90 mmHg). The blood pressure values were taken with an Omron-Hem-705IT device in the sitting position after a five-minute rest period. Two measurements were conducted two minutes apart and the values of the second measurement were used (Schikowski et al., 2020; Moreno Velasquez et al., 2025). For diabetes, hypertension and hypercholesterolemia, information on antihypertensive, lipid-lowering or antidiabetic medication intake was not available during the time of the study and therefore, could not be considered. In addition, participants reported the extent to which noise annoys them, with options ranging from 1 = not annoyed at all to 5 = extremely annoyed (Wolf et al., 2020). A question about the living duration at place of residence was included in the touchscreen questionnaire.

Further spatial geocoded variables at residential addresses were available, including air pollution, surrounding greenness, unemployment rate and population density. A detailed description on the

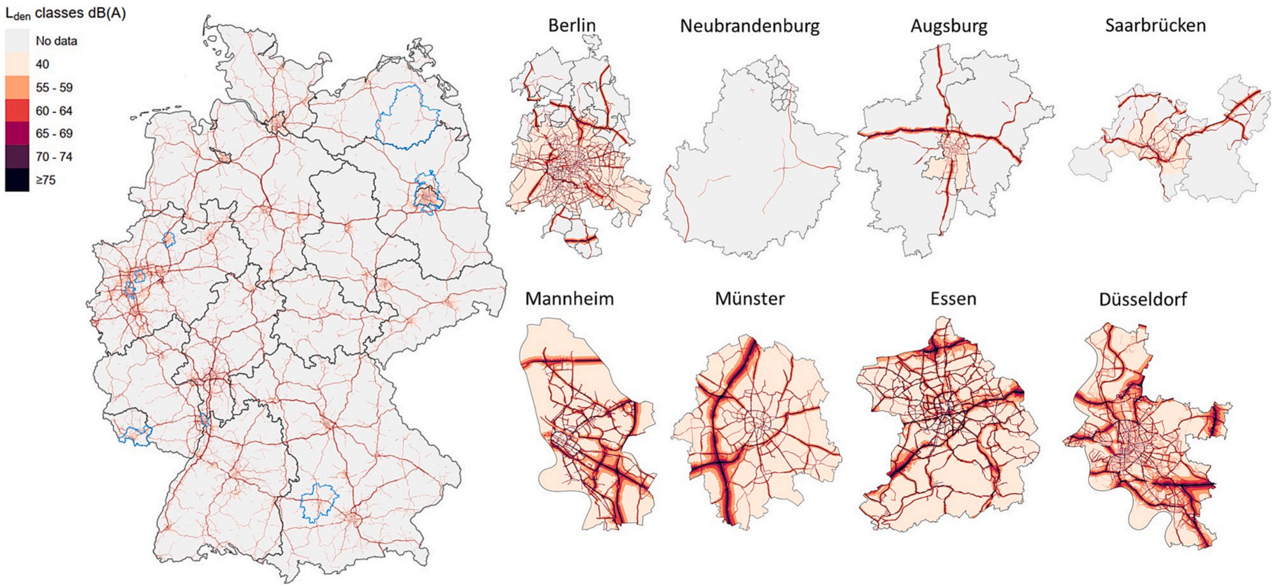

Fig. 1. Map of Germany showing spatial patterns of annual road traffic noise (L_{den}) in 2017. Legend: Zoom on NAKO study centers (in blue). 40 dB(A) is a lower detection limit applied to all grid cells in urban areas covered by Environmental Noise Directive (END) obligation 2002/49/EC Article 3 with levels < 55 dB(A). No data reflect areas where noise mapping is not required according to END. Abbreviations: L_{den} = day–evening–night road traffic noise level. NAKO = German National Cohort. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

assignment, data preparation, and resources can be found in Wolf et al. (2025). The ELAPSE (Effects of Low-Level Air Pollution: A Study in Europe) project provided high-resolution data on modeled nitrogen dioxide (NO_2) and particulate matter with a diameter below 2.5 μm ($PM_{2.5}$) on a 100 m × 100 m grid for the year 2010 (Wolf et al., 2025; de Hoogh et al., 2018). Land use regression models were developed to predict air pollution concentrations, incorporating measurements from ground-level monitoring stations, satellite observations, estimates from chemistry transport models, and further spatial predictors (de Hoogh et al., 2018). To quantify surrounding greenness, we used the Normalized Difference Vegetation Index (NDVI) on a 1 km × 1 km grid, extracted from monthly satellite images taken by the NASA Terra Moderate Resolution Imaging Spectroradiometer (MODIS) (Wolf et al., 2025). Briefly, the NDVI (= reflected radiation in the visible red minus in the near infrared divided by the sum of the two) takes values from −1 to 1. Negative NDVI values, reflecting mainly water, were set to missing, while values around 0 reflect areas with no vegetation and 1 reflects areas with intense green vegetation (Wolf et al., 2025). NDVI data covered the whole baseline period so we assigned each participant the annual NDVI of the respective grid cell averaged over the warm months (March to October) of their examination year. Area-level SES was represented by the unemployment rate at the district level for 2014 from the Federal Employment Agency (Bundesagentur für Arbeit [Federal Employment Agency]). Population density, given as inhabitants per 500 square meters, was available for the year 2018 from a private company (WiGeoGis GmbH).

All covariates were considered in directed acyclic graphs (DAG) to visualize interdependencies and to identify confounders, colliders, and mediators (Supplementary Fig. S1).

2.5. Statistical analysis

We performed all analyses with the statistical software RStudio (version 4.3.1) (R Core Team, 2021) and statistical significance was indicated by two-sided p-values < 0.05. Due to the high number of missing values in the covariates (Supplementary Fig. S2), which would have resulted in a reduction of the sample size by 15 % using a complete-case approach, we employed a random forest approach to impute the covariates using the function *missForest()* from the Rpackage missforest (Stekhoven and Buhlmann, 2012). In summary, the process starts with an initial estimation (mean for continuous variables and mode for categorical variables) and then employs a random forest to predict the missing values based on the observed data. These steps are repeated until a stopping criterion is met, or a maximum number of iterations has been reached (Stekhoven and Buhlmann, 2012). The imputation in our analysis achieved a low imputation error, as indicated by a normalized root mean square error (RMSE) of 2.3 % for continuous variables and a percentage falsely classified (PFC) of 8.8 % for categorical variables.

To assess the association of noise with our primary outcomes and prevalent MASLD, we performed linear and logistic regression models with covariate adjustment, respectively. We identified the following adjustment set with DAG: age, study region, alcohol consumption, physical activity, smoking behavior, and income (Supplementary Fig. S1). As visualized in the DAG (Supplementary Fig. S1), we hypothesized that unmeasured residual confounding would be explained by adjusting for study center region. The area-related variables available at the time of this study were either at a coarse resolution (e.g., district-level unemployment rate) or did not match years of baseline (population density from 2018). However, we extended the adjustment model for these area-level variables in a sensitivity analysis to see if the results changed substantially or if the study center regions captured unmeasured confounding related to contextual differences. We log-transformed hepatic fat content prior to linear regression analysis due to a left-skewed distribution of the regression residuals. All effect estimates are presented as a percentage change of the arithmetic or geometric outcome mean or as an odds ratio (OR) per 10 dB(A) increase in exposure, with the corresponding 95 % confidence interval (CI). The percentage change of the arithmetic outcome mean was calculated as follows: $\% - \text{change} = \frac{ß * \text{increment}}{\text{mean(outcome)}} * 100$, where the increment referred to the 10 dB(A) increase. As hepatic fat content was log-transformed, the calculation was as follows: $\% - \text{change} = (\exp(ß * \text{increment}) - 1) * 100$, which is interpreted as the percentage change of the geometric outcome mean. We visually inspected the exposure–response functions to detect non-linear associations between noise and outcomes by applying natural cubic splines with three degrees of freedom.

To assess whether the associations of noise were independent of other environmental exposures, we conducted two-exposure models adjusting for other environmental factors associated with exposure and outcome (An et al., 2018; Luo et al., 2020). We chose the air pollutants NO_2 and $PM_{2.5}$ because they have common sources with road traffic noise. Moreover, air pollution and noise exposure are more common in urban areas, which are known to have less greenness (Nieuwenhuijsen, 2020). To avoid multicollinearity in the two-exposure models, we performed Spearman correlation analysis *a priori* between all environmental exposures with a cut-off of ≤ 0.7. In addition, we calculated the variance inflation factor for the two-exposure models, assuming that if the variance inflation factor for each variable was < 5, there was no multicollinearity problem.

We assessed potential effect modification by cardiometabolic disease including a multiplicative interaction term between noise and the following indicators: obesity (BMI $\geq/< 30$ kg/m^2), hypercholesterolemia (yes/no), hypertension (yes/no), and "any metabolic disease", indicating either obesity, diabetes, or hypercholesterolemia. Due to the low prevalence of diabetes, a dedicated analysis on diabetes was not sensible. We also tested for potential interactions by age ($\geq/< 65$ years), smoking behavior (never smoker/ex and current smokers), and noise annoyance (no-low-moderate/high-extreme).

We conducted several sensitivity analyses to investigate the robustness of our findings.

(1) To account for potential misclassification of the road traffic noise exposure, we performed several sensitivity analyses. Firstly, those individuals who were assigned a value of 40 dB(A) in the continuous road traffic noise variable, but who lived outside the areas covered by the END obligation 2002/49/EC (European Commission, 2002) were excluded. Secondly, we examined associations using the categorical road traffic noise classes with the following dB(A) categories: 40, 55 – 59, 60 – 64, 65 – 70, $\geq$ 70. As this categorical variable gives the noise levels extracted at the address points, this sensitivity analysis addressed the potential bias introduced by averaging noise levels across cells in 10 m and 100 m buffers as used in our main analysis. Thirdly, we assessed the association with a binary road traffic noise variable ($</\geq$ 55 dB(A)) based on categorical L_{den} classes. Finally, a three-category road traffic noise variable was derived based on the continuous variables (40, $>$ 40 – $<$ 55 and $\geq$ 55 dB(A)). For all noise variables included in the sensitivity analysis, participants with missing data in the categorical L_{den} classes were excluded, as these individuals were not subject to the END obligation 2002/49/EG. An overview of the different noise variables used in sensitivity analyses with respective sample sizes can be seen in the Supplementary Table S1.

(2) We conducted a complete-case analysis excluding all cases with missing data in the covariates considered in regression models (age, center, physical activity, alcohol consumption, smoking behavior, income).

(3) We assessed the associations between road traffic noise and the outcome MASLD in combination with metabolic-associated alcoholic liver disease (MetALD).

(4) We adjusted for education as an additional indicator of individual socioeconomic status (SES) in addition to income. Moreover, we compared models where we dropped income or lifestyle factors to see whether lifestyle factors explained some SES-related unmeasured confounding not captured by income.

(5) We excluded all with less than five or less than ten years of living duration, hypothesizing that associations tend to be stronger in these subsamples.

3. Results

3.1. Baseline characteristics

After excluding all individuals with missing data in the main outcomes (Supplementary Fig. S2), the study sample comprised 11,101 participants (48.7 % women, Table 1). The mean age of participants was 51.9 years, and most participants had their MRI scan in 2016 (60 %). Furthermore, one-third of the participants lived in the study center Neubrandenburg, followed by Augsburg and Berlin. Mean levels of VAT and hepatic fat content were higher in men than in women (4.8 L and 8.7 % vs. 2.5 L and 6.4 %, respectively). Mean SCAAT and SCTAT were higher in women than in men (Table 1).

The mean L_{den} levels were 43.8 dB(A) and 44.1 dB(A) for the 10 m and 100 m buffer (Table 1). Based on the categorical noise variable, 43.8 % of the participants had missing data, 37.2 % were set to 40 dB(A), and the remaining 20 % were distributed over the L_{den} classes 55 dB(A) to $\geq$ 75 dB(A) (Supplementary Fig. S3 and Table S1). However, distributions varied widely between the study centers, with low proportions of missing data in Münster, Essen, Mannheim, and Düsseldorf and high proportions in Neubrandenburg. Noise indicators for sensitivity analysis are described in Supplementary Table S1.

Mean concentrations of NO_2, $PM_{2.5}$, and NDVI were 26.0 μg/m^3, 17.6 μg/m^3, and 0.5, respectively (Supplementary Table S2). Based on the Spearman correlation, NO_2 and $PM_{2.5}$ were positively (range r = 0.4 to 0.6) and NDVI negatively (r = -0.4 to -0.2) correlated with noise (Supplementary Fig. S4).

3.2. Associations of noise with outcomes

Increased noise was associated with increased AT depots and hepatic fat content in men and women after adjusting for age, study center, lifestyle factors and income (Table 2). We found the strongest association for hepatic fat content in men and women (men: 3.6 %-change (95 %-CI: 1.41; 5.78); women: 3.1 %-change (95 %-CI: 1.00; 5.21) per 10 dB (A) increase in L_{den} (10 m)). Accordingly, odds of MASLD were higher with increase of noise (Table 2). L_{den} (10 m and 100 m) were also associated with a 1.7 % to 1.9 %-change in VAT for men and a 3.1 % to 3.5 %-change in VAT for women, respectively. We found a positive association of 1.8 %-change in SCTAT and 2.2 %-change in SCAAT for men and 2.2 %-change in SCAAT and 2.7 %-change in SCTAT for women. Adjusting for area-level SES and population density did not alter the results substantially (Supplementary Table S3), although effect estimates tended to be higher, particularly after adjusting for population density.

The exposure–response curves showed some non-linear associations between noise variables and outcomes, in particular for SCAAT and SCTAT and L_{den} (10 m) levels above 60 dB(A) (Supplementary Fig. S5 and S6). As seen in Fig. 2, Supplementary Fig. S7 and S8, the effect estimates of noise with all outcomes were robust after adjustment for other environmental exposures. The variance inflation factor was < 5 for all two-exposure models, indicating no multicollinearity problem. Results of interaction analysis are presented in Fig. 3, and Supplementary Fig. S9 and S10. There was no effect modification by cardiometabolic disease on the association of noise with VAT. In men, obesity amplified the association of noise with SCAAT and SCTAT (Fig. 3), and hypertension amplified the association of noise with hepatic fat content (Fig. 3). In contrast, the association of L_{den} (10 m) with hepatic fat content was stronger in women without hypertension (Fig. 3). We found no significant interaction between noise and age, noise annoyance, or smoking, except for the association of L_{den} with hepatic fat content in men (Supplementary Fig. S9 and S10).

3.3. Sensitivity analysis

After excluding those with missing data (n = 4,864) in the categorical noise variable, the results for continuous variables were slightly stronger (Supplementary Table S4). Noise categories 40 to 54.9 dB(A) and $\geq$ 55 dB(A) were positively associated with all outcomes (Supplementary Table S4). We found similar trends for the original categorical noise variable but no clear dose response (Supplementary Table S4). Moreover, we observed robust associations in complete-case

Table 1
Subjects' characteristics of the final analytical German National Cohort sample stratified by sex.

	Overall (n = 11,101)	Men (n = 5,690)	Women (n = 5,411)
Sex, female n (%)	5,411 (48.7)	–	5,411 (100.0)
Age (years), mean (SD)	51.9 (11.4)	52.07 (11.5)	51.74 (11.3)
VAT (l), mean (SD)	3.7 (2.3)	4.8 (2.4)	2.5 (1.6)
SCAAT (l), mean (SD)	6.9 (3.6)	6.2 (3.1)	7.7 (3.9)
SCTAT (l), mean (SD)	3.3 (1.6)	2.8 (1.2)	3.8 (1.8)
Hepatic fat content (%), mean (SD)	7.6 (6.5)	8.7 (6.9)	6.4 (5.9)
MASLD, yes n (%)	3,639 (35.1)	2,333 (41.0)	1,306 (24.1)
L_{den} (10 m) (dB(A)), mean (SD)	43.8 (7.8)	43.8 (7.8)	43.8 (7.7)
L_{den} (100 m) (dB(A)), mean (SD)	44.1 (6.2)	44.1 (6.3)	44.0 (6.1)
Study center, n (%)			
Augsburg	2,569 (23.1)	1,410 (24.8)	1,159 (21.4)
Berlin	2,233 (20.1)	1,128 (19.8)	1,105 (20.4)
Düsseldorf	281 (2.5)	155 (2.7)	126 (2.3)
Essen	1,626 (14.6)	815 (14.3)	811 (15.0)
Mannheim	1,196 (10.8)	617 (10.8)	579 (10.7)
Münster	75 (0.7)	43 (0.8)	32 (0.6)
Neubrandenburg	3,023 (27.2)	1,473 (25.9)	1,550 (28.6)
Saarbrücken	98 (0.9)	49 (0.9)	49 (0.9)
Examination year, n (%)			
2014	541 (4.9)	277 (4.9)	264 (4.9)
2015	3,835 (34.5)	1,942 (34.1)	1,893 (35.0)
2016	6,725 (60.6)	3,471 (61.0)	3,254 (60.1)
Degree of urbanization, n (%)			
Urban	7,335 (66.1)	3,707 (65.1)	3,628 (67.0)
Suburban	1,615 (14.5)	884 (15.5)	731 (13.5)
Rural	2,151 (19.4)	1,099 (19.3)	1,052 (19.4)
Physical activity (min/week), mean (SD)	1,424 (1,569)	1,473 (1,622)	1,372 (1,510)
Alcohol consumption (g/day), mean (SD)	11.1 (16.5)	15.02 (19.8)	6.95 (10.8)
Income (Euros), mean (SD)	2,254 (1,426)	2,405 (1,585)	2,096 (1,218)
Smoking behavior, n (%)			
Never smoker	5,085 (45.8)	2,319 (40.8)	2,766 (51.1)
Ex-smoker	3,881 (35.0)	2,219 (39.0)	1,662 (30.7)
Smoker	2,135 (19.2)	1,152 (20.2)	983 (18.2)
Noise annoyance, high-extreme n (%)	1,727 (15.6)	839 (14.7)	888 (16.4)
Living duration (years), mean (SD)	15.6 (11.3)	15.4 (11.5)	15.8 (11.0)
Population density (n/500 m^2), mean (SD)	1,333 (1,296)	1,332 (1,305)	1,334 (1,287)
Unemployment rate (%) at district level, mean (SD)	9.5 (3.7)	9.4 (3.8)	9.7 (3.7)
Body-Mass-Index (kg/m^2), mean (SD)	26.8 (4.1)	27.4 (4.1)	26.2 (5.2)
Waist circumference (cm), mean (SD)	91.9 (13.7)	97.4 (12.0)	86.2 (13.0)
Obesity (BMI ≥ 30 kg/m^2), yes n (%)	2,378 (21.4)	1,265 (22.2)	1,113 (20.6)
Hypercholesterolemia, yes n (%)	2,957 (26.6)	1,572 (27.6)	1,385 (25.6)
Diabetes, yes n (%)	658 (5.9)	402 (7.1)	256 (4.7)
Any metabolic disease, yes n (%)	4,646 (41.9)	2,450 (43.1)	2,196 (40.6)
Hypertension, yes n (%)	3,092 (27.9)	1,923 (33.8)	1,169 (21.6)

Legend: Covariate information was imputed as described in the Methods section. Abbreviations: BMI = Body-Mass-Index, L_{den} = day-evening-night road traffic noise level, MASLD = metabolic dysfunction–associated steatotic liver disease, SCAAT = subcutaneous abdominal adipose, SD = standard deviation, SCTAT = subcutaneous thoracic adipose tissue, VAT = visceral adipose tissue.

Table 2
Associations of road traffic noise with adipose tissue depots, hepatic fat content and MASLD in the German National Cohort.

	Men (n = 5,690)		Women (n = 5,411)	
	%-change (95 %-CI)	p	%-change (95 %-CI)	p
VAT [l]				
L_{den} (10 m)	1.72 (0.14; 3.30)	0.033	3.13 (1.09; 5.18)	0.003
L_{den} (100 m)	1.85 (-0.15; 3.86)	0.070	3.47 (0.84; 6.09)	0.010
SCAAT [l]				
L_{den} (10 m)	2.18 (0.43; 3.93)	0.015	2.38 (0.55; 4.20)	0.011
L_{den} (100 m)	2.07 (-0.15; 4.29)	0.068	2.22 (-0.12; 4.57)	0.063
SCTAT [l]				
L_{den} (10 m)	1.79 (0.34; 3.25)	0.015	2.39 (0.75; 4.03)	0.004
L_{den} (100 m)	1.99 (0.15; 3.83)	0.034	2.68 (0.58; 4.79)	0.013
Hepatic fat content [%]				
L_{den} (10 m)	3.57 (1.41; 5.78)	0.001	3.08 (1.00; 5.21)	0.004
L_{den} (100 m)	3.87 (1.13; 6.69)	0.005	4.03 (1.33; 6.79)	0.003
MASLD[a]	**OR (95 %-CI)**		**OR (95 %-CI)**	
L_{den} (10 m)	1.10 (1.01; 1.19)	0.022	1.08 (0.99; 1.19)	0.098
L_{den} (100 m)	1.08 (0.98; 1.20)	0.135	1.10 (0.98; 1.24)	0.103

Legend: Effect estimates are given as percentage change of the arithmetic (or geometric for hepatic fat content) outcome mean per 10 dB(A) increase in exposure, with 95 % confidence intervals derived from linear and logistic regression models stratified by sex and adjusted for study center, age, alcohol consumption, physical activity, smoking behavior, income.

[a] Sample size deviate from full sample (n = 5,147 men and n = 5,258 women) due to MASLD definition. Abbreviations: CI = confidence interval, L_{den} = day-–evening–night road traffic noise level, MASLD = metabolic dysfunction–associated steatotic liver disease, OR = odds ratio, SCAAT = subcutaneous abdominal adipose tissue, SCTAT = subcutaneous thoracic adipose tissue, VAT = visceral adipose tissue.

analysis (Supplementary Tables S5 and S6). We observed similar associations of noise with MASLD and MetALD derived from logistic regression models (Supplementary Table S7). Dropping income or lifestyle factors did not change results substantially, however attenuated or stronger effect estimates could be observed, particularly for women (Supplementary Table S8). Furthermore, the associations were robust after additional adjustment for education (data not shown). The associations were stronger for participants who had the same residential address for at least five or ten years (Supplementary Table S9).

4. Discussion

4.1. Key findings and comparison to current literature

This cross-sectional analysis using data from a German-wide cohort study supports our hypothesis that annual exposure to road traffic noise is associated with increased AT depots and hepatic fat content, as derived by MRI, in adult men and women. Noise associations remained consistent after further adjustment for air pollutants and surrounding greenness, suggesting that long-term exposure to road traffic noise has an independent association with AT measures. Our findings indicated an effect modification by cardiometabolic disease, resulting in higher SCAAT and SCTAT with higher noise exposure in men with obesity and higher hepatic fat content in men with hypertension. All associations remained robust in multiple sensitivity analyses, including different adjustment sets.

Our findings are in line with previous literature on the association of noise with obesity markers. A recent meta-analysis identified 13 epidemiological studies from Europe that analyzed various noise

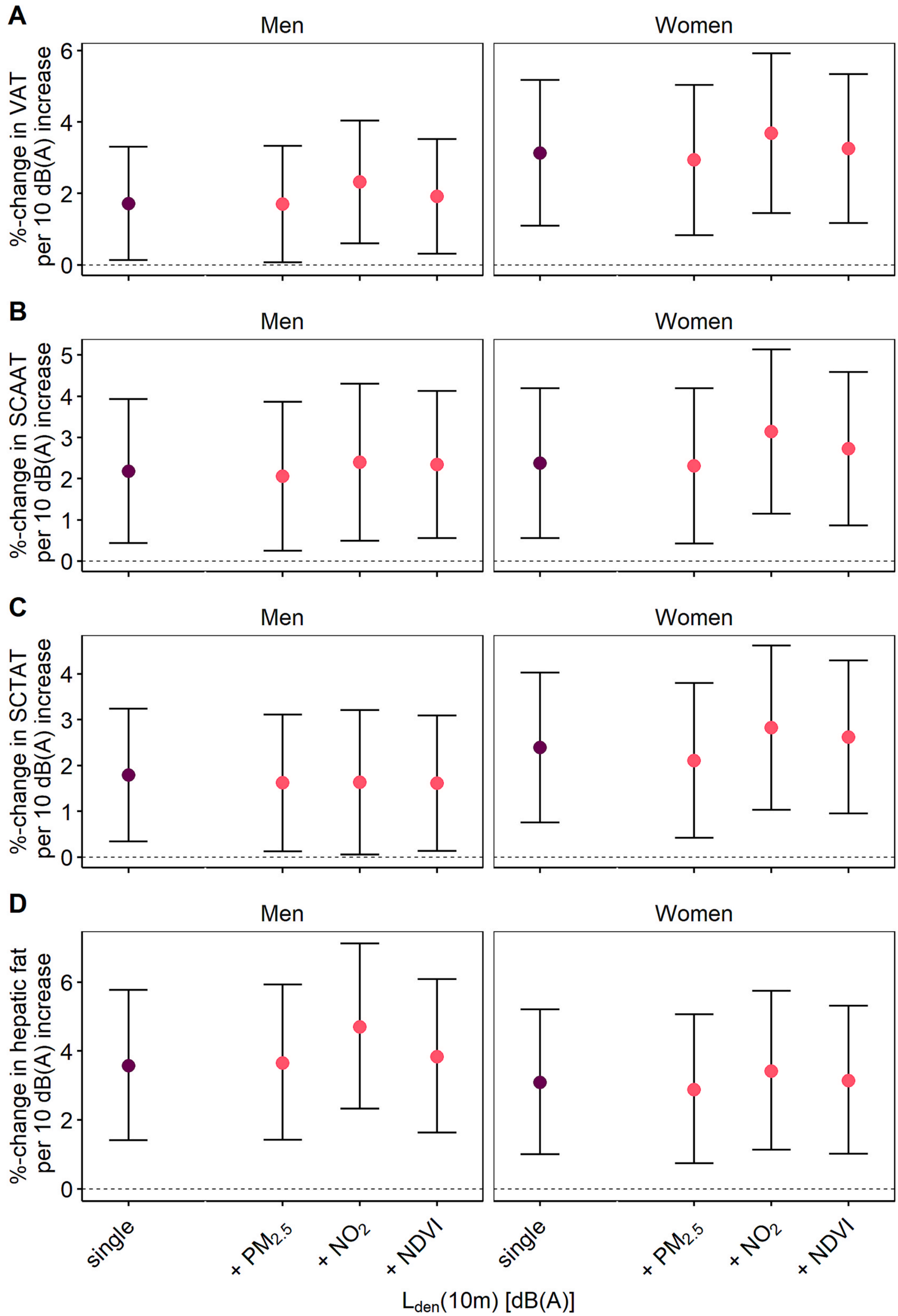

Fig. 2. Comparison of the estimates of road traffic noise (10 m buffer) in single and two exposure models adjusting for air pollutants and surrounding greenness in the German National Cohort. Legend: Estimates of single exposure models (purple) and two exposure models (pink) for outcomes (A) VAT, (B) SCAAT, (C) SCTAT, and (D) hepatic fat content. Effect estimates are given as percentage change of the arithmetic (or geometric for hepatic fat content) outcome mean per 10 dB(A) increase in exposure, with 95 % confidence intervals derived from linear and logistic regression models stratified by sex and adjusted for study center, age, alcohol consumption, physical activity, smoking behavior, income. Abbreviation: VAT = visceral adipose tissue, SCAAT = subcutaneous abdominal adipose tissue, SCTAT = subcutaneous thoracic adipose tissue, L_{den} = day–evening–night road traffic noise level, NO_2 = nitrogen dioxide, $PM_{2.5}$ = particulate matter with diameter < 2.5 µm, NDVI = normalized difference vegetation index. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

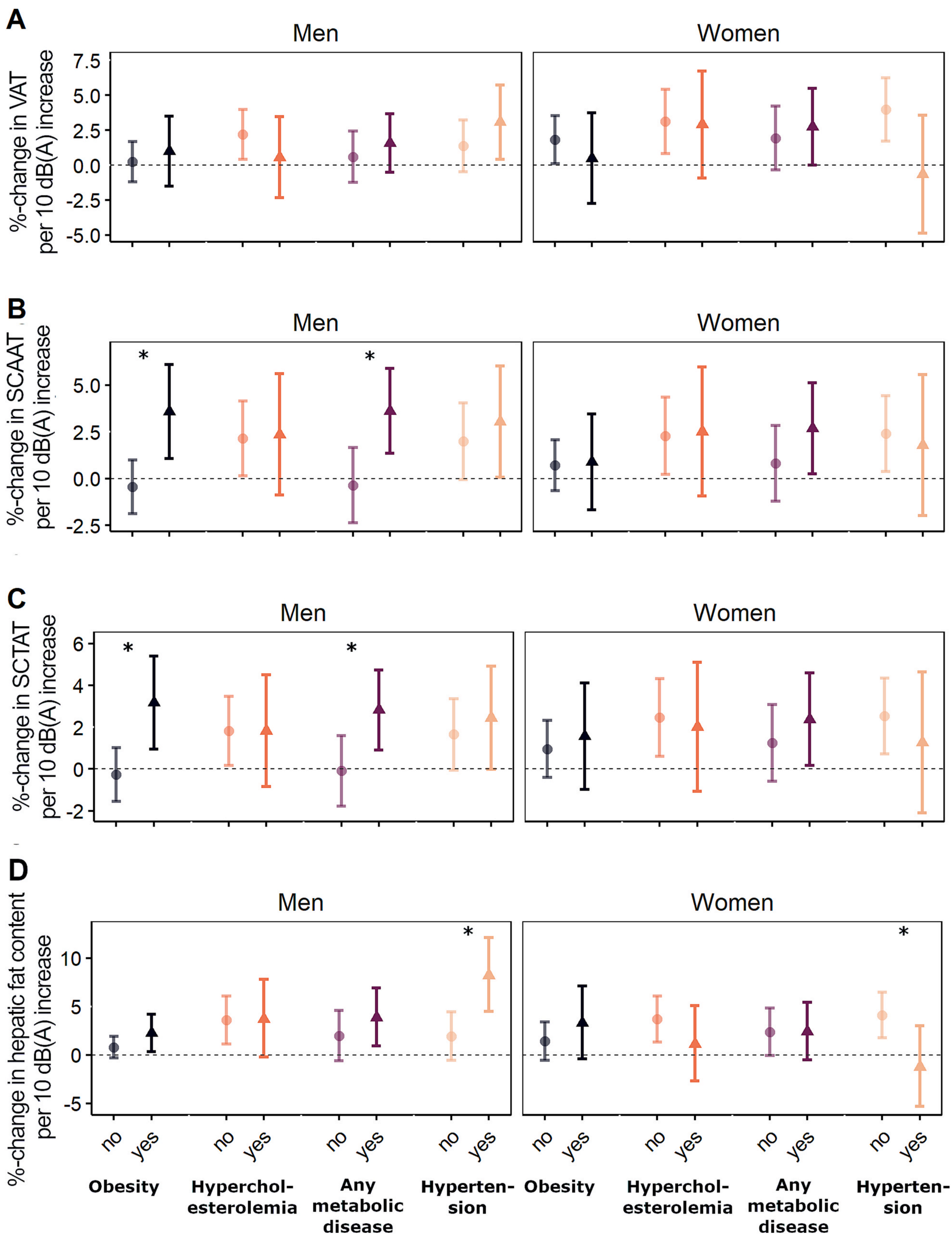

Fig. 3. Cardiometabolic disease-specific associations of L_{den} (10 m buffer) with VAT (A), SCAAT (B), SCTAT (C), and hepatic fat content (D) in the German National Cohort. Legend: Associations derived from linear regression models with a multiplicative interaction term between exposure and cardiometabolic disease indicator. Models were adjusted for study center, age, alcohol consumption, physical activity, smoking behavior, income. Effect estimates are given as percentage change of the arithmetic (or geometric for hepatic fat content) outcome mean outcome per 10 dB(A) increase in exposure, with 95 % confidence intervals. Asterix indicate significant interaction term with $p < 0.05$. Abbreviation: L_{den} = day–evening–night road traffic noise level, SCAAT = subcutaneous abdominal adipose tissue, SCTAT = subcutaneous thoracic adipose tissue, VAT = visceral adipose tissue.

exposures to obesity (Gui et al., 2022). Overall, studies showed that the increase in noise exposure is associated with higher waist circumference and higher odds of central obesity. However, waist circumference and BMI do not provide information on AT distribution, which can lead to misclassification of obesity in certain subgroups with low or high lean mass (Machann et al., 2013). To date, only a limited number of studies used alternative methods to quantify body fat mass with bioelectrical impedance measurements (Cai et al., 2020; Christensen et al., 2016), suggesting a positive association between road traffic noise and total body fat percentage. Findings from the LEAD study in Vienna did not find consistent associations between noise and body composition measures derived by DXA (Altug et al., 2024). Our study adds to current evidence by providing findings for metabolically relevant AT depots, including hepatic fat. In addition, our analysis showed that adjusting for population density may capture unmeasured residual confounding related to spatial variation, which may be particularly relevant for multi-center studies with varying urbanization degree. To summarize, our results suggest that noise may play a key role in the pathogenesis of (cardio-)metabolic diseases, as AT are known to be metabolically active, for example by secreting cytokines resulting in low-grade inflammation and altering glucose and lipid metabolism (Tchernof and Despres, 2013).

Investigating the role of environmental factors is necessary for obesity prevention, especially since visceral obesity and hepatic steatosis are reversible if treated early (Brunner et al., 2019). We found that higher annual noise exposure was associated with larger MRI-derived AT depots, independent of other environmental exposures including traffic-related air pollutants, which is partly in line with previous studies (Thacher et al., Dec 2021; Sorensen et al., 2022; Foraster et al., 2018; Cai et al., 2020). Reducing road traffic and its associated noise may therefore be a promising target to complement behavioral interventions to prevent the development and progression of cardiometabolic disease.

The health impacts of traffic noise may specifically affect susceptible subgroups of the population. Only a few studies investigated potential effect modifications by cardiometabolic disease, showing a stronger effect of noise in individuals with obesity and CVD diagnosis (Foraster et al., 2018; Munzel et al., 2025; Christensen et al., 2015). This is consistent with our findings although we only observed a stronger effect of noise on SCAAT and SCTAT in men with obesity. Although obesity defined as BMI $\geq$ 30 kg/m^2 and MRI-derived AT measures both reflect adiposity, they still capture distinct dimensions. BMI refers to overall adiposity whereas AT measures provide body fat quantification, which is more closely linked to cardiometabolic risk (Tchernof and Despres, 2013; Abraham et al., 2015). Thus, we aimed to assess whether the strength of the associations between noise and AT measures differed by overall obesity status, as it has been shown by Christensen et al. (2015). In this longitudinal Danish cohort study, associations between higher noise exposure with small increases in BMI and waist circumference were stronger in obese subjects (Christensen et al., 2015). Moreover, our findings indicated that the effect modification for hypertension was in opposite directions in men and women. We need to note that including cardiometabolic disease status into the model may induced bias from a DAG-point of view, as cardiometabolic disease are descendants of the MRI-derived AT measures. Therefore, the results on these analyses need to be interpreted with caution and future longitudinal analyses with multiple follow-ups, including repeated MRI measurements as provided in NAKO, are needed. This will allow to disentangle the temporal sequence of exposure, disease status and MRI-derived AT measures. In addition, we further noted that the contribution of noise to increased AT depots and hepatic fat content is rather small compared to estimates of cardiometabolic disease such as hypercholesterolemia, diabetes, and hypertension (data not shown).

To date, two main pathways are hypothesized for the effects of noise on human metabolism: sleep disturbance and stress response (Christensen et al., 2015). There is strong evidence for an association between (nighttime) noise exposure and sleep disturbance (Smith et al., 2022). The consequences of poor sleep can be increased energy intake, changes in appetite, and energy-controlling hormones such as leptin and ghrelin, all of which are associated with an increased risk of developing obesity and cardiometabolic disease (Cappuccio and Miller, 2017). Münzel and colleagues (Munzel et al., 2024) summarized how short- and long-term exposure to traffic noise affects endothelial function, hormone levels (e.g., cortisol and adrenaline), and inflammatory markers. These biomarkers are elevated in visceral obesity and are associated with increased cardiometabolic risk (Tchernof and Despres, 2013; Munzel et al., 2024). For example, chronic overactivation of the sympathetic nervous system via the hypothalamic–pituitary–adrenal axis may lead to increased cortisol release, which in turn may favor the accumulation of ectopic fat (Tchernof and Despres, 2013). However, a systematic review summarized and rated the evidence of the association between noise exposure and these mechanistic metabolic markers as inconsistent and low (Sivakumaran et al., 2022). Therefore, studies investigating the underlying mechanisms are needed to understand noise-induced metabolic dysfunction.

4.2. Outlook and implications

Several implications can be drawn from this study. First, road traffic noise reduction may be a promising target to complement behavioral interventions to prevent the development and progression of cardiometabolic disease. In addition, reducing road traffic would have co-beneficial effects by reducing air pollution emissions, another harmful environmental risk factor for humans health (Altug et al., 2024; Cai et al., 2022). To date, WHO has published noise guidelines with exposure limits, e.g., average noise levels from road traffic should not exceed 53 dB(A), which are only recommendations (WHO, 2018). Uniform, effective guidelines with step-by-step actions to be taken in communities where noise levels exceed these guidelines, similar to the European Union's Ambient Air Quality Directive (European Parliamentary Research Service, 2022), may be warranted. To justify such guidelines, improvements in the noise mapping are needed as our study also shows the shortcomings of the currently available data on noise exposure. These have been extensively discussed and outlined by Staab et al. (2025), including heterogenous mapping and potential biases introduced by the conversion from raster to polygons using different algorithms. At present, the END obligation is still vague, resulting in inconsistent reporting from EU member states which limits comparisons of noise exposure levels between years and countries (Staab et al., 2025; European Environment Agency, 2020). In addition, as END only requires noise mapping in urban areas and along major roads, we still lack accurate noise exposure levels in rural and suburban areas (European Commission, 2002). Moreover, the reporting threshold of 55 dB(A) may be too high, as the WHO provided evidence that even lower noise levels were associated with various adverse health outcomes (WHO, 2018). Therefore, a lowering of the current reporting threshold, small-scale assessment of noise levels and a fine-meshed network of monitoring stations combined with modelling techniques, e.g., using land use regressions as in Staab et al. (2021), is needed in future studies. This would provide nationwide noise exposure at high resolution and can improve the accuracy of health impact assessments of noise exposure.

4.3. Limitations

We need to address the following limitations. Mean annual noise exposure was only available for 2017, which is later in time than the baseline examination (2014–2016), but based on previous evidence we assumed that noise exposure was relatively stable over the years (Fecht et al., 2016; Babisch et al., 2014). In particular, substantial changes intra-urban contrasts would require major changes in road construction, engine technology or traffic density, which take several years. Align with this, a comparison of END data from 2012 and 2017 demonstrated a stable number of participants being exposed to harmful noise levels

(European Environment Agency, 2020). Therefore, we assume that noise exposure levels from 2017 can be used as a surrogate for long-term exposure prior to the outcome assessment. Furthermore, our exposure is limited to road traffic noise, while exposure to other noise sources, e.g., railway, aircraft or neighborhood, may also contribute to the development of obesity (Bozigar et al., 2024). Only one-fifth of the participants was exposed to noise exposure levels ≥ 55 dB(A). Additionally, 43.8 % of the participants lived in areas not covered by noise exposure mapping. It is therefore likely that misclassification of exposure occurred at the lower levels. However, so far it is reasonable to assume that this would have led to a bias towards the null, with the associations being stronger for more accurate noise exposure measurements. This is confirmed by our sensitivity analysis, where the associations tended to be stronger after excluding participants with missing categorical L_{den} classes. In addition, averaging noise levels within buffers may have introduced bias by altering the spatial scale. Consequently, noise levels within the 100 m buffer may reflect not only noise information, but also area-related factors or land use. Nevertheless, we assume that the 10 m buffer used in the main analyses was small enough to capture noise levels at buildings. Furthermore, sensitivity analyses using the categorical L_{den} classes, which reflected the exact noise levels at address points, revealed robust associations. A study by Vienneau et al. (Vienneau et al., 2019) demonstrated that different noise exposure assessment strategies are important, but changes in spatial scale led to attenuated estimates of the health effects. This further supports our assumption that improved noise exposure assessments would reveal stronger associations. Information on anti-diabetic, lipid-lowering or hypertensive medication was not available, which may have led to misclassification of participants. It is important to note that we used cross-sectional data, so longitudinal studies are needed to confirm our findings by considering the temporal occurrence of exposure and outcome and to conclude causal effects of noise on early obesity markers. Thereby, the NAKO is a unique and excellent database as multiple MRI re-examinations of NAKO participants will be available in the future (Peters et al., 2022; Bamberg et al., 2024).

5. Conclusion

We observed robust associations of annual mean exposure to road traffic noise with AT depots and hepatic fat content in men and women. Our data suggest that road traffic noise is associated with larger visceral AT depots and higher hepatic fat content, which could potentially mediate the risk of metabolic and cardiovascular diseases. Given that these associations were independent of air pollution, another prominent environmental risk factor in urban areas, preferably longitudinal studies should evaluate potential benefits of traffic noise reduction as an additional route for the prevention of cardiometabolic disease at the population level.

CRediT authorship contribution statement

Fiona Niedermayer: Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Data curation, Conceptualization. **Susanne Rospleszcz:** Writing – review & editing, Supervision, Methodology. **Clara Matthiessen:** Writing – review & editing, Resources. **Barbara Hoffmann:** Writing – review & editing, Supervision, Resources, Methodology. **Sophia Stoecklein:** Writing – review & editing, Methodology. **Tobias Haueise:** Writing – review & editing, Resources, Data curation. **Tobias Norajitra:** Writing – review & editing, Resources, Data curation. **Christopher L. Schlett:** Writing – review & editing, Resources, Data curation. **Johanna Nattenmüller:** Writing – review & editing, Resources, Data curation. **Fabian Bamberg:** Writing – review & editing, Resources, Data curation. **Jürgen Machann:** Writing – review & editing, Resources, Data curation. **Matthias Günther:** Writing – review & editing, Resources, Data curation. **Jochen Hirsch:** Writing – review & editing, Resources, Data curation. **Rajini Nagrani:** Writing – review & editing, Resources. **Henry Völzke:** Writing – review & editing, Resources. **Claudia Meinke-Franze:** Writing – review & editing, Resources. **Norbert Hosten:** Writing – review & editing, Resources. **Tobias Nonnenmacher:** Writing – review & editing, Resources, Data curation. **Bettina Katalin Budai:** Writing – review & editing, Resources. **Viktoria Palm:** Writing – review & editing, Resources. **Verena Katzke:** Writing – review & editing, Resources. **Karin Halina Greiser:** Writing – review & editing, Resources. **Jeanette Schulz-Menger:** Writing – review & editing, Resources. **Thoralf Niendorf:** Writing – review & editing, Resources. **Beate Endemann:** Writing – review & editing, Resources. **Tobias Pischon:** Writing – review & editing, Resources. **Jeroen Staab:** Writing – review & editing, Resources, Data curation. **Marco Dallavalle:** Writing – review & editing, Resources, Data curation. **Alexandra Schneider:** Writing – review & editing, Resources, Methodology. **Kathrin Wolf:** Writing – review & editing, Resources, Methodology, Data curation. **Annette Peters:** Writing – review & editing, Supervision, Resources, Methodology, Funding acquisition, Conceptualization.

Ethics approval and consent to participate

The German National Cohort (NAKO) was approved by the initial vote of the ethics committee of the Bavarian Medical Association ("Bayerische Landesärztekammer" (BLÄK), protocol code 13023), followed by all local on-site institutional review boards in charge of the five imaging sites, and written informed consent of all participants was obtained before study enrollment. The study was conducted in accordance with the Declaration of Helsinki of 1975 (in the current, revised version).

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data sharing

The datasets analyzed during the current study are not publicly available. Access to and use of NAKO data and biosamples can be obtained via an electronic application portal (https://transfer.nako.de). Analysis codes are available from the authors upon request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2025.109566.

Data availability

The authors do not have permission to share data.

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