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Long-term exposure to transportation noise and its association with adiposity markers and development of obesity

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ABSTRACT

The contribution of different transportation noise sources to metabolic disorders such as obesity remains understudied. We evaluated the associations of long-term exposure to road, railway and aircraft noise with measures of obesity and its subphenotypes using cross-sectional and longitudinal designs.

We assessed 3796 participants from the population-based Swiss Cohort Study on Air Pollution and Lung and Heart Diseases (SAPALDIA), who attended the visits in 2001 (SAP2) and 2010/2011 (SAP3) and who were aged 29–72 at SAP2. At SAP2 we measured body mass index (BMI, kg/m^2). At SAP3 we measured BMI, waist circumference (centimetres) and Kyle body Fat Index (%) and derived overweight, central and general obesity. Longitudinally for BMI, we derived change in BMI, incidence of overweight and obesity and a 3-category outcome combining the latter two. We assigned source-specific 5-year mean noise levels before visits and during follow-up at the most exposed dwelling façade (Lden, dB), using Swiss noise models for 2001 and 2011 and participants' residential history. Models were adjusted for relevant confounders, including traffic-related air pollution.

Exposure to road traffic noise was significantly associated with all adiposity subphenotypes, cross-sectionally (at SAP3) [e.g. beta (95% CI) per 10 dB, BMI: 0.39 (0.18; 0.59); waist circumference: 0.93 (0.37; 1.50)], and with increased risk of obesity, longitudinally (e.g. RR = 1.25, 95% CI: 1.04; 1.51, per 10 dB in 5-year mean). Railway noise was significantly related to increased risk of overweight. In cross-sectional analyses, we further identified a stronger association between road traffic noise and BMI among participants with cardiovascular disease and an association between railway noise and BMI among participants reporting bad sleep. Associations were independent of the other noise sources, air pollution and robust to all adjustment sets. No associations were observed for aircraft noise.

Long-term exposure to transportation noise, particularly road traffic noise, may increase the risk of obesity and could constitute a pathway towards cardiometabolic and other diseases.

1. Introduction

Road, railway, and aircraft traffic represent the most prevalent transportation noise sources in Europe (European Environment Agency, 2014). Epidemiological studies have mainly focused on road traffic noise, which constitutes the most prevalent of the three and one of the major environmental health hazards in Europe (Hänninen et al., 2014; Mueller et al., 2017; Vienneau et al., 2015). Among other ailments,

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long-term exposure to transportation noise has been consistently associated with sleep disturbance and cardiovascular disease, however evidence for its association with diabetes and particularly with obesity is still limited to few studies (Basner and McGuire, 2018; Pyko et al., 2017; van Kempen et al., 2018; Zare Sakhvidi et al., 2018). It is suggested that long-term exposure to noise may affect health through stress-related reactions and sleep disturbance (Münzel et al., 2016). These may lead to chronic endocrine and autonomous nervous system alterations, which may also enhance oxidative, inflammatory, or immune responses (Münzel et al., 2017; Recio et al., 2016; Schmidt et al., 2013) and finally contribute to cardiometabolic diseases (van Kempen et al., 2018; Münzel et al., 2016). Moreover, sleep models in rodents indicate that non-severe sleep disruption induced by environmental noise contributes to weight gain (Mavanji et al., 2013; Parrish and Teske, 2017).

Obesity represents a main public health issue worldwide due to its dramatic increase in the last decades (NCD Risk Factor Collaboration, 2016) and its importance as risk factor of morbidity and mortality particularly regarding cardiovascular disease, but also diabetes, cancer, respiratory diseases and overall mortality (The GBD 2015 Obesity Collaborators, 2017). Understanding the modifiable causal agents of obesity is crucial to decrease such a trend, however, little is known yet about the impact of environmental factors such as transportation noise. To our knowledge, six studies have analysed the cross-sectional (Christensen et al., 2016; Oftedal et al., 2015; Pyko et al., 2015) or longitudinal association (Christensen et al., 2015; Eriksson et al., 2014; Pyko et al., 2017) of transportation noise with obesity markers. Studies generally indicated positive associations and evidence was greater for the most commonly assessed exposure (road traffic noise). Out of the three cross-sectional studies, long-term exposure to road traffic noise was associated with waist circumference in two (Christensen et al., 2016; Pyko et al., 2015) and with BMI in one study (Christensen et al., 2016) in the entire population, whereas a Norwegian study observed associations with both adipose markers only in highly noise sensitive women (Oftedal et al., 2015). Two longitudinal studies observed associations between road traffic noise with change in waist circumference but found mixed evidence for change in weight (Christensen et al., 2015; Pyko et al., 2015). However, the available studies only represent three independent populations (in Stockholm, Denmark and Norway). In turn, reported associations between aircraft noise and adiposity markers all belonged to the same population in Stockholm (Eriksson et al., 2014; Pyko et al., 2015; Pyko et al., 2017), and results for railway noise were mixed (Christensen et al., 2015; Christensen et al., 2016; Pyko et al., 2015; Pyko et al., 2017). Moreover, the proportion of body fat (Christensen et al., 2016), change in BMI (Eriksson et al., 2014) and clinically relevant measured outcomes of obesity (categorical) (Pyko et al., 2017) were rarely addressed. Therefore, further studies from additional populations are needed, which assess the independent effects of different transportation noise sources with obesity and its different sub-phenotypes. Finally, as living near major roads or being exposed to air pollution has been associated with obesity (Jerrett et al., 2014; Li et al., 2016), traffic-related air pollution should further be considered as a potential confounder of the road traffic noise-obesity association.

In the current study of an adult population-based Swiss cohort, we evaluated the associations between long-term home-outdoor exposure to road, railway and aircraft noise with measures of general and central obesity and percent of body fat, using a cross-sectional design, and with incidence of overweight, obesity and change in BMI, using a longitudinal design. We also evaluated effect modification by personal characteristics such as sleepiness or comorbidities, and noise exposure modifiers such as sleeping with closed windows or bedroom orientation.

2. Methodology

2.1. Study population

We assessed 3796 adults from the population-based Swiss Cohort Study on Air Pollution and Lung and Heart Diseases (SAPALDIA), who had attended both the second and third examinations. The baseline recruitment was performed in 1991 (SAP1) and consisted of a random selection of 9651 adults aged 18–60 years from eight environmentally diverse areas in Switzerland (Martin et al., 1997). A total of 8047 participated in the first follow-up (SAP2, 2001–2003) (Ackermann-Liebrich et al., 2005) and 6088 participated in the second follow-up (SAP3, 2010–2011). Participants completed interviewer-administered questionnaires about lifestyles, socioeconomic status, environment, residential history, and health, underwent cardiorespiratory measurements, and provided blood. The latter was stored in a biobank for blood markers and genetics. The first (SAP2) and second (SAP3) follow-up represented the baseline and follow-up samples of the present study and a total of 5881 participated in both (follow-up attrition rate = 26.9%). Specifically, out of the latter, we assessed a subsample of 4552 participants (77.4%), who had answered the longer versions of the main questionnaire and underwent anthropometric measurements (See Flow chart in Fig. 1).

The ethics board of the eight SAPALDIA communities approved the study and all participants signed written informed consent.

2.2. Outcomes

Trained nurses in the study centres examined participants. We performed objective measures of height (SECA 206 body meter GmbH & Co. KG., Hamburg, Germany) and weight (SECA 877 GmbH & Co. KG., Hamburg, Germany) both in SAP2 and SAP3, following standard procedures. We derived *body mass index* (*BMI*) as the weight (in kg) divided by the height squared (m^2).

In SAP3 (not in SAP2), we also measured participants' abdominal fat and total body fat with waist circumference and percent body fat, respectively. *Waist circumference (cm)* was measured using a SECA 201 tape (SECA GmbH & Co. KG., Hamburg, Germany) at the end of passive expiration over the narrowest part of the trunk between the lowest rib and the iliac crest or, if not evident, at the level of the umbilicus. *Body fat (%)* was measured with bioelectric impedance analysis using a Helios device (Helios, Forana, Frankfurt, Germany).

To evaluate the clinical relevance and differential risk at SAP3, we further defined *overweight* (BMI ≥ 25), *obesity* (BMI ≥ 30), and *central obesity* (for Europeans) as waist circumference ≥ 94 (men) and ≥80 (women), respectively, following the standard classifications by World Health Organization (World Health Organization, 2011, 2000).

In longitudinal analyses, we derived the *change in BMI* defined as the difference between BMI at SAP3 and BMI at SAP2, and assessed the clinical relevance and severity of BMI change by calculating:

a) the *incidence of overweight* (BMI at SAP2 < 25 and BMI at SAP3 ≥ 25)
b) the *incidence of obesity* (BMI at SAP2 < 30 and BMI at SAP3 ≥ 30)
c) the *graded incidence* (*3-category outcome*):
   c.1. Reference (BMI at SAP2 and SAP 3 < 25)
   c.2. Incidence of overweight only between examinations (BMI at SAP2 < 25 and at SAP3 between 25 and 29.9)
   c.3. Incidence of obesity between examinations (BMI at SAP2 < 30 and BMI at SAP3 ≥ 30).

2.3. Noise exposure assessment

Outdoor exposure to transportation noise (road, railway and aircraft) was derived from detailed source-specific national Swiss noise exposure models for years 2001 (for SAP2) and 2011 (for SAP3),

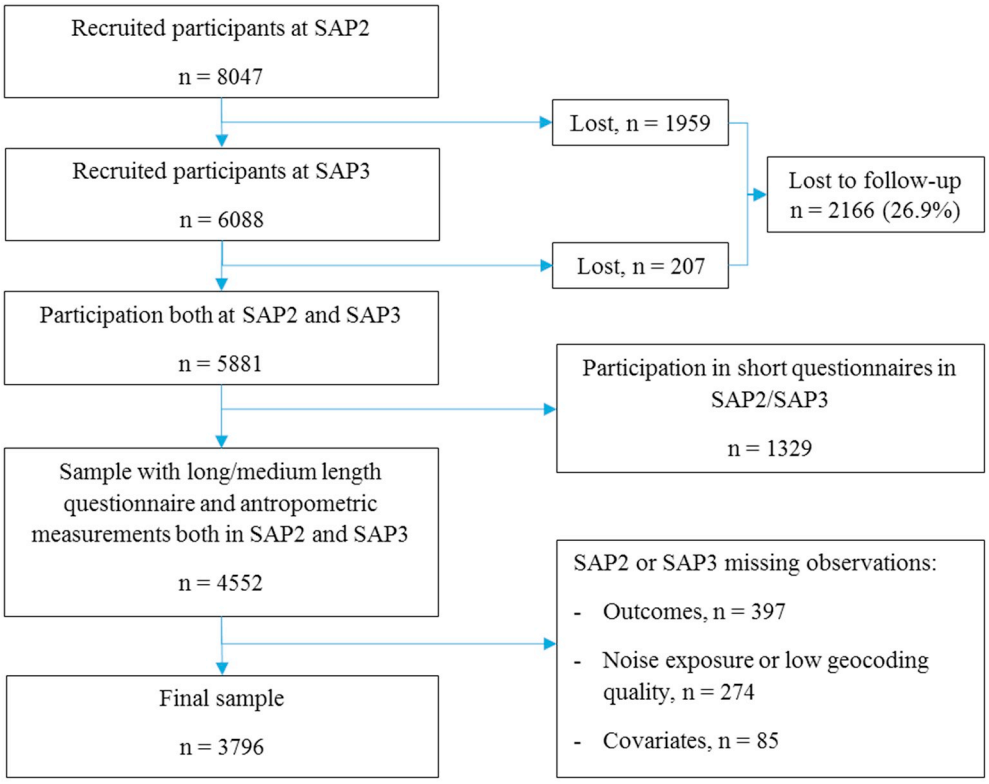

Fig. 1. Flow chart of sample selection.

developed in the context of the SiRENE project (Short and Long Term Effects of Transportation Noise Exposure) (Karipidis et al., 2014). Briefly, the aircraft noise modelling included the emissions of one military and the three major civil airports. Noise levels were calculated with FLULA2 software, and a combination of traffic statistics (Federal Office of Civil Aviation), radar data (Zürich airport), acoustical footprints (Basel, Geneva), and idealized number and time of flight paths for the military airport (Payerne). Railway noise emission levels were derived using the sonRAIL model and noise propagation was calculated with the Swiss railway noise model SEMIBEL. The calculation considered the railway tracks' geometry, location of switch points, noise barriers, train types, driving speed, and traffic statistics. Road noise levels were calculated using the sonROAD emission model and the StL-86 propagation model with input data from 3-D geometry, road slopes, type and width, speed limits, traffic statistics in an hourly resolution per each road type, and noise barriers location and height.

Noise models provided A-weighted annual average sound pressure levels for the day (Lday, 7–19 h), evening (Levening, 19–23 h) and night (Lnight, 23–7 h) (in dB). For this study, we averaged these time periods to obtain the standard 24 h-annual average EU indicator Lden (in dB), with 5 dB and 10 dB penalties for Levening and Lnight, respectively, according to Directive 2002/49/EC (European Parliament and Council of the European Union, 2002). To avoid unreliable estimated levels below the expected background noise level for each source, we further applied truncation on Lden, as done before (Eze et al., 2017a, 2017b; Foraster et al., 2017; Héritier et al., 2017). I.e. we applied a value of 35 dB for road and 30 dB for aircraft and railway noise levels when their levels were below such value (See Fig. S1).

To derive home-outdoor long-term noise exposure, we first geocoded all addresses from participants' residential history, which was self-reported and completed with population registers during the cleaning process and for unreached participants. Second we assigned Lden levels at the height of the residential floor from the most exposed dwelling façade. Third, we calculated the long-term averages as time-weighted Lden levels accounting for the time lived at each address during the 5-year period before examination at both SAP2 and SAP3 (main exposure), based on the 2001 and 2011 noise models, respectively. Additionally, to account for residential mobility between examinations, we also calculated average noise exposure to Lden levels during follow-up based on the 2011 noise model.

2.4. Air pollution exposure assessment

We assigned outdoor exposure to nitrogen dioxide concentrations (NO_2; $\mu g/m^3$), an indicator of traffic-related air pollution and potential confounder of road noise effects, at the geocoded participants' addresses. We used similar area-specific NO_2 models for years 2001 for SAP2 (Liu et al., 2012) and for 2010/2011 for SAP3 (Eeftens et al., 2016). These models combined land-use regression (LUR) (considering traffic variables, population, building density, etc.) with national Gaussian dispersion estimates at a resolution of 200 × 200 m (with traffic, agricultural and industrial emission inventories). The 2001 and 2010/2011 models provided similar NO_2 estimates (Spearman's rank correlation in non-movers = 0.8).

2.5. Other explanatory variables

We selected other potentially relevant variables from the interview-administered questionnaires in both surveys. For both SAP2 and SAP3, we considered age (years), sex (male/female), cumulative educational level (low/middle/high), smoking (Current/Former/Never), second-hand smoke exposure during the last 12 months (No/Yes), alcohol consumption [daily/weekly/rarely/never, recoded as Yes (any frequency) vs. No (never) for analyses]), consumption of raw vegetables (days/week), cooked vegetables (days/week), or fish (days/week), self-reported feeling of often being insufficiently rested after waking up early in the morning (No/Yes), the Epworth daytime sleepiness score (0: none, 24: maximal) (Johns, 1991), study area (municipality), and physical activity (≥150 min/week of at least moderate intensity, Yes/No) based on exercise duration and intensity according to guidelines

(World Health Organization, 2010). We considered a walkability index of 1 × 1 km around home, i.e. a function of the z-scores of residential density (i.e. proportion of area covered by buildings), intersection density and land use mix (i.e. diversity of land use types), which indicates how friendly an area is to walking (Frank et al., 2010). Transportation noise annoyance was assessed on an ICBEN (International Commission on the Biological Effects of Noise) 11-point scale from 0 (not at all annoyed) to 10 (extremely annoyed) (Fields et al., 2001). At SAP2, we also accounted for neighbourhood-level socio-economic index, based on the census of 2000 information for median household income, household occupancy, educational level, and occupation of the head of household (Panczak et al., 2012).

SAP3 variables also included self-reported doctor-diagnosed cardiovascular disease (No/Yes, defined as hypertension, angina pectoris, myocardial infarction, stroke, arrhythmia, or cardiac insufficiency), diabetes (No/Yes, defined as doctor-diagnosed diabetes or taking diabetes medication or HbA1c blood levels ≥6.5%, in the absence of clinical diabetes), self-reported sleep quality during the last month (Very Bad/Bad/Good/Very Good), bedroom orientation (street/backyard), closing windows at night (No/Yes), and the Weinstein's noise sensitivity score item “Are you sensitive to noise?” in a 6-point scale from 1 (strongly disagree) to 6 (strongly agree) (Weinstein, 1980, 1978). Mean greenness index was defined in a 1000 m buffer around home, as the Normalized Difference Vegetation Index (NDVI) in 2014, using satellite data with a 30 × 30 m resolution (U.S. Geological Survey, Earth Explorer, 2017; Vienneau et al., 2017).

2.6. Statistical analyses

We evaluated 3796 participants (i.e. 83.4% of the sample answering the longer surveys, See Flow chart in Fig. 1) who had complete data on the outcome, exposures, covariates and high quality geocoding (i.e. at street level) both at the first and second follow-up surveys.

We described the association between all independent variables and the outcomes, as well as their linearity, by using penalized smoothing splines, and transformed age to its cubic term to linearize its association with BMI.

We carried out cross-sectional analyses at SAP3 to evaluate the associations between the 5-year mean exposure to road, railway and aircraft noise levels (Lden) and the several obesity markers, which were only available in this visit (BMI, waist circumference, body fat, overweight, obesity, and central obesity). We used multivariable linear or logistic mixed regression models for continuous or binary outcomes, respectively, with a random intercept for study area. Models were adjusted for potential confounders according to previous literature (Christensen et al., 2015; Eriksson et al., 2014), following a progressive adjustment procedure to evaluate their confounding effect. We included age, age^2, age^3, sex, cumulative educational level, smoking, second-hand smoking, cooked vegetables, raw vegetables, and fish consumption, physical activity, and NO_2 at SAP3. We further adjusted for noise annoyance, NDVI and walkability, which could potentially affect the final noise exposure level and/or the physical activity level determining adiposity.

In longitudinal analyses between SAP2 and SAP3, we assessed the association of baseline exposure (5-year mean before SAP2) and follow-up exposure (follow-up time mean between SAP2 and SAP3) to road, railway and aircraft noise with: a) incidence of overweight, b) incidence of obesity c) graded incidence of overweight or obesity, and d) change in BMI. We used multivariable linear mixed regression models with a random effect by study area to analyse the association with change in BMI. To analyse the risk of overweight and obesity (binary incidence outcomes), we used multivariable Poisson regression with robust standard errors to control for overdispersion. Because the application of robust standard errors together with random effects is highly imprecise with few clusters, and the current analyses were based on only eight study areas, we first checked for the need to include random effects. Mixed effects Poisson regression with a random intercept by study area did not reveal clustering (alpha = 0, Likelihood ratio test p-value = 1). Therefore, we adjusted for study area as a fixed covariate in the model. Finally, to analyse the graded risk of overweight or obesity (3-category outcome) we used multinomial logistic regression also adjusting for study area as a fixed covariate. All models were adjusted for characteristics at SAP2 corresponding to the same variables described for cross-sectional analyses, and change in age (i.e. follow-up time), physical activity, alcohol consumption, and NO_2. We also considered further adjustment for change in diet, passive and active smoking, and neighbourhood deprivation index.

We studied potential effect modifiers of the association between the source-specific noise levels and BMI, introducing interaction terms between the noise variable and the respective potential modifying factor. The selected effect modifiers were: sex, daytime sleepiness (Normal < 10/High ≥ 10), insufficiently rested (No/Yes), sleep quality (Good/Bad), cardiovascular disease (No/Yes), Diabetes (No/Yes), bedroom facing a backyard (No/Yes), closing windows at night (No/Yes), noise annoyance (No/Yes), and noise sensitivity (< median / ≥ median). This analysis was carried out at SAP3 to maximize the availability of effect modifiers.

In sensitivity analyses, we assessed potential participation selection bias due to loss to follow-up from the baseline recruitment (SAP1, n = 9651) with inverse probability weighting, applying the inverse of the probability of participating in the present study to the main model. Probabilities were derived using variables from the baseline recruitment that predicted participation in the present study. We also evaluated the exposure-response function using penalized smoothing splines to assess if the studied associations were linear or if there was a threshold of effect.

Analyses were performed with Stata version 13.0 (StataCorp, College Station, TX, USA) and R version 3.1.3 (The R Foundation for Statistical Computing, Vienna, Austria). Statistical significance was considered at an alpha level of 0.05.

3. Results

The final sample had very similar characteristics to the samples from which it was derived both at SAP2 and SAP3 (See Table S1), except for including slightly higher educated participants than the original SAP2 sample (around 5%) and slightly less current smokers (around 4%). Part of such changes was due to lost-to-follow-up. According to Table 1, participants had an average (and median) age of 52 years [interquartile range (IQR): 16.8] at SAP2 and 50% were women. Time to follow-up was very similar between participants, with a mean (and median) of 8.3 years (standard deviation: 0.4; IQR: 0.6). The median (IQR) BMI at SAP2 was 25.1 (5.3) kg/m^2 and it exhibited a median increase of 0.6 (2.0) by SAP3. At SAP3, the median (IQR) waist circumference and percent body fat were 90.5 (18.8) cm and 30.6 (10.9) %, respectively. In turn, the percentages of overweight, obesity, and central obesity at SAP3 were 58.2%, 18% and 59.2%, respectively. The incidence of overweight and obesity at SAP3 were 21.3% and 7.4%, respectively, and the graded incidence led to 18.8% incident cases with overweight only and 11.6% with obesity (See Table S2).

Exposure to road traffic noise remained stable between SAP2 and SAP3, with medians (IQR) of 54.2 (10.1) and 54.0 (10.4) dB, respectively, and > 99% of participants were exposed above the truncation (background noise) value (See Table 1 and Table S2). In contrast, railway noise, aircraft noise and NO_2 levels changed slightly from 32.9 (11.2) to 30.0 (7.8) dB (% exposed: 61–46.7%), 30.0 (10.6) to 33.9 (8.3) dB (% exposed: 34.5–60.4%), and 21.0 (15.7) to 17.4 (10.2) μg/m^3, respectively. Each of the previous exposures exhibited a high temporal correlation between SAP2 and SAP3 (Spearman rank r range = 0.71–0.78) (See Table S3). We also observed a decrease in the percentage of current smokers and second-hand smokers, an increase in alcohol consumption, and in physical activity; and no change in diet

Table 1
Personal characteristics at the two study time periods (SAP2 and SAP3), (N = 3796).

Variables	Baseline (SAP2)[a]	Follow-up (SAP3)[a]
Body mass index (kg/m^2)	25.1 (5.3)	25.8 (5.7)
Waist circumference (cm)	n.a	90.5 (18.8)
Body fat (%)	n.a	30.6 (10.9)
5-y mean road traffic noise (Lden, in dB)	54.2 (10.1)	54.0 (10.4)
5-y mean railway noise (Lden, in dB)	32.9 (11.2)	30.0 (7.8)
5-y mean aircraft noise (Lden, in dB)	30.0 (10.6)	33.9 (8.3)
Follow-up mean road traffic noise (Lden, in dB)	n.a	53.9 (10.1)
Follow-up mean railway noise (Lden, in dB)	n.a	30.0 (8.0)
Follow-up mean aircraft noise (Lden, in dB)	n.a	34.0 (8.3)
Age (y)	52.2 (16.8)	60.5 (16.7)
Follow-up time (y)	n.a	8.3 (0.6)
Raw vegetables (days/week)	7.0 (3.0)	7.0 (3.0)
Cooked vegetables (days/week)	5.0 (4.0)	5.0 (4.0)
Fish (days/week)	1.0 (1.0)	1.0 (1.0)
2-y mean NO_2 (μg/m^3)	21.0 (15.7)	17.4 (10.2)
Sex, Women	1912 (50.4)	1912 (50.4)
Education, Low	161 (4.2)	161 (4.2)
Middle	2449 (64.5)	2449 (64.5)
High	1186 (31.2)	1186 (31.2)
Smoking, Never	1726 (45.5)	1692 (44.6)
Former	1235 (32.5)	1480 (39.0)
Current	835 (22.0)	624 (16.4)
Second-hand smoking in the last 12 months, Yes	889 (23.4)	448 (11.8)
Alcohol consumption[b], Yes	1511 (39.8)	1619 (42.7)
Moderate physical activity, Sufficient	1913 (50.4)	2184 (57.5)
Area, Basel	517 (13.6)	516 (13.6)
Wald	652 (17.2)	647 (17.0)
Davos	352 (9.3)	350 (9.2)
Lugano	495 (13.0)	497 (13.1)
Montana	353 (9.3)	353 (9.3)
Payerne	489 (12.9)	486 (12.8)
Aarau	618 (16.3)	625 (16.5)
Geneva	320 (8.4)	322 (8.5)
Bed faces backyard[c], Yes	n.a	2211 (58.6)
Close window at night[c], Yes	n.a	830 (22.0)
Noise annoyance[c], Yes	n.a	2152 (57.1)
Self-reported doctor-diagnosed CVD[c], Yes	n.a	1343 (35.4)
Epworth day sleepiness score[c], ≥10	n.a	3295 (87.0)
Insufficiently rested in the morning[d], Yes	n.a	950 (28.3)
Sleep quality[c], Bad or very bad	n.a	411 (10.9)
Diabetes[c], Yes	n.a	275 (7.3)
Noise sensitivity score[c]	n.a	3.0 (4.0)
Walkability (z-scores)	−0.2 (3.6)	−0.3 (3.3)
Green spaces in a 1000 m buffer (NDVI, range: −1 to 1)	n.a	0.6 (0.2)

[a]Data are median (interquartile range) for continuous variables and number (percent) for categorical variables. SAP2: Baseline time period of the present study; SAP3: Follow-up time period of the present study. [b]Any frequency; [c] < 1.1% missing observations; [d]11.5% missing observations.

patterns, educational level or the study area of examination.

Spearman rank correlations (See Table S3) further revealed strong relationships of BMI at SAP3 with BMI at SAP2 (r = 0.91), waist circumference (r = 0.82), and a moderate correlation with % body fat (r = 0.42). Percent body fat and waist circumference were less correlated with each other (r = 0.19). Source-specific noise levels were little correlated with each other and with NO_2 (r range = −0.05–0.22), except for a moderate correlation between road noise and NO_2 (r range = 0.35–0.43). From all noise sources, road traffic was the one showing a more consistent relationship with all adiposity markers and incidence outcomes (See Table S2 and Table S3).

3.1. Cross-sectional analysis at SAP3

In cross-sectional analysis at SAP3 (See Table 2), 5-year mean exposure to road traffic noise was significantly associated with all obesity markers in the crude and successive adjustment sets, including physical activity, walkability, exposure to NO_2 or green spaces, among others (See Table 2 and Supplementary Table S4). Specifically, in the fully adjusted model in Table 2, a 10 dB change in 5-year noise levels was associated with a statistically significant increase of 0.39 kg/m^2 (95% CI: 0.18; 0.59) in BMI, of 0.45% (95% CI: 0.17; 0.73) in body fat, and of 0.93 cm (95% CI: 0.37; 1.50) in waist circumference. A 10 dB change in the 5-year noise levels was also associated with a 17% increase in obesity (OR = 1.17, 95% CI: 1.03; 1.33), a 20% increase in overweight (OR = 1.20, 95% CI: 1.08; 1.33), and a 16% increase in central obesity (OR = 1.16, 95% CI: 1.04; 1.29). Exposure to railway or aircraft noise was not associated with any of the obesity markers at SAP3.

3.2. Longitudinal analysis

3.2.1. Road traffic noise

In longitudinal analysis (Fig. 2 and Table S5), exposure to road traffic noise was significantly associated with risk of obesity both for 5-year mean exposure before SAP2, i.e. baseline exposure in this study (RR = 1.25, 95% CI: 1.04; 1.51), and with exposure during the follow-up time between SAP2 and SAP3 (RR = 1.26, 95% CI: 1.05; 1.51). The estimated effect size between road traffic noise and risk of obesity was greater in the 3-category outcome, and statistically significant for both exposure windows (e.g. for 5-year mean before SAP2: RR = 1.40, 95% CI: 1.10; 1.76). No association was observed between exposure to road traffic noise and incidence of overweight (RR = 0.97, 95% CI: 0.84, 1.13, p-value = 0.724) or change in BMI (β = −0.04, 95% CI: -0.13, 0.06, p-value = 0.442), e.g. for 5-year mean before SAP2.

3.2.2. Railway noise

Furthermore (Fig. 2 and Table S5), 5-year mean exposure to railway noise before SAP2 was significantly associated with risk of overweight (BMI ≥ 25), (RR = 1.12, 95% CI: 1.001; 1.26), whereas it showed a positive non-significant association with exposure during the follow-up time. We observed the same pattern for the association between each of the exposure windows and incidence of overweight only (BMI between 25 and 29.9) in the 3-category outcome. However, we found no association between exposure to railway noise and incidence of obesity or change in BMI.

3.2.3. Aircraft noise

Exposure to aircraft noise was not associated with any of the incidence outcomes, if anything, there was an indication of a positive non-significant association between 5-year mean exposure before SAP2 and incidence of overweight (RR = 1.20, 95% CI: 0.89, 1.60, p = 0.233) and overweight only in the 3-category outcome (See Fig. 2 and Table S5). Effect estimates for aircraft noise exhibited wider confidence intervals than those for road and railway noise.

3.2.4. Adjustment sets

Estimated effects in longitudinal analysis changed < 10% for the different adjustment sets of confounders compared to the magnitude of the associations described above (See Supplemental Material Table S6).

3.3. Sensitivity analyses

We did not observe a threshold for the start of the effect of road traffic noise on the obesity markers. I.e. the magnitude of the association increased linearly already at low noise levels (See Supplemental Material, Figs. S2 and S3, p-values for non-linearity > 0.15). We only observed a departure in the linearity of the association between road traffic noise and percent body fat. However, this smooth term did not contribute to the model beyond the linear term (p-value = 0.09).

Finally, the application of inverse probability weighing on the studied associations to control for potential selection bias due to non-participation yielded similar results in cross-sectional analyses (See Supplemental Table S7, example for BMI) and similar or even greater effect estimates in longitudinal analyses (See Supplemental Table S8).

Table 2
Beta coefficients (β) or Odds Ratios (OR) and 95% confidence intervals (95% CI) for the association between 5-y mean noise (Lden) levels for road, railway, and aircraft (dB) and obesity outcomes at SAP3, per 10 dB, (N = 3796). Cross-sectional analyses.

Adjustment sets	Road traffic noise	Railway noise	Aircraft noise
BMI	β (95% CI)	β (95% CI)	β (95% CI)
Crude	0.33 (0.14, 0.52)**	0.07 (−0.15, 0.30)	0.03 (−0.31, 0.37)
+Noise sources	0.32 (0.13, 0.52)**	0.06 (−0.16, 0.29)	0.08 (−0.27, 0.43)
+Basic	0.29 (0.11, 0.47)**	0.03 (−0.18, 0.25)	0.09 (−0.24, 0.41)
+Lifestyles	0.30 (0.12, 0.48)**	0.04 (−0.18, 0.26)	0.07 (−0.26, 0.40)
+PA	0.30 (0.12, 0.48)**	0.03 (−0.18, 0.25)	0.05 (−0.27, 0.37)
+NO_2	0.39 (0.18, 0.59)**	0.04 (−0.18, 0.25)	0.04 (−0.26, 0.33)
% body fat	β (95% CI)	β (95% CI)	β (95% CI)
Crude	0.68 (0.35, 1.00)**	0.23 (−0.15, 0.62)	0.19 (−0.42, 0.80)
+Noise sources	0.62 (0.30, 0.95)**	0.24 (−0.14, 0.62)	0.29 (−0.32, 0.90)
+Basic	0.36 (0.11, 0.61)**	0.03 (−0.26, 0.32)	0.34 (−0.18, 0.87)
+Lifestyles	0.37 (0.12, 0.62)**	0.03 (−0.25, 0.32)	0.32 (−0.20, 0.84)
+PA	0.37 (0.13, 0.62)**	0.02 (−0.26, 0.31)	0.34 (−0.18, 0.86)
+NO_2	0.45 (0.17, 0.73)**	0.03 (−0.25, 0.32)	0.33 (−0.18, 0.85)
Waist circumference	β (95% CI)	β (95% CI)	β (95% CI)
Crude	0.72 (0.13, 1.30)**	0.10 (−0.58, 0.78)	−0.25 (−1.55, 1.04)
+Noise sources	0.68 (0.09, 1.27)**	0.07 (−0.61, 0.76)	−0.09 (−1.39, 1.22)
+Basic	0.67 (0.17, 1.18)**	0.03 (−0.55, 0.62)	0.23 (−0.88, 1.35)
+Lifestyles	0.66 (0.16, 1.16)**	0.02 (−0.56, 0.60)	0.19 (−0.92, 1.30)
+PA	0.66 (0.16, 1.17)**	0.01 (−0.57, 0.59)	0.23 (−0.87, 1.34)
+NO_2	0.93 (0.37, 1.50)**	0.05 (−0.53, 0.63)	0.21 (−0.89, 1.31)
Obesity	OR (95% CI)	OR (95% CI)	OR (95% CI)
Crude	1.14 (1.02, 1.27)**	1.07 (0.95, 1.22)	0.98 (0.83, 1.16)
+Noise sources	1.14 (1.02, 1.27)**	1.07 (0.95, 1.22)	0.99 (0.84, 1.18)
+Basic	1.12 (1.00, 1.25)**	1.06 (0.93, 1.20)	0.98 (0.82, 1.17)
+Lifestyles	1.13 (1.01, 1.27)**	1.06 (0.93, 1.21)	0.98 (0.82, 1.17)
+PA	1.13 (1.01, 1.27)**	1.06 (0.93, 1.20)	0.97 (0.81, 1.16)
+NO_2	1.17 (1.03, 1.33)**	1.06 (0.93, 1.21)	0.97 (0.81, 1.16)
Overweight	OR (95% CI)	OR (95% CI)	OR (95% CI)
Crude	1.15 (1.06, 1.26)**	1.06 (0.95, 1.17)	1.01 (0.88, 1.15)
+Noise sources	1.15 (1.05, 1.25)**	1.05 (0.95, 1.17)	1.02 (0.89, 1.18)
+Basic	1.16 (1.06, 1.26)**	1.05 (0.94, 1.17)	1.04 (0.90, 1.20)
+Lifestyles	1.15 (1.05, 1.26)**	1.05 (0.94, 1.17)	1.02 (0.88, 1.18)
+PA	1.15 (1.05, 1.26)**	1.05 (0.94, 1.17)	1.01 (0.88, 1.17)
+NO_2	1.20 (1.08, 1.33)**	1.05 (0.94, 1.17)	1.01 (0.88, 1.17)
Central obesity	OR (95% CI)	OR (95% CI)	OR (95% CI)
Crude	1.15 (1.05, 1.25)**	1.04 (0.93, 1.15)	0.90 (0.74, 1.10)
+Noise sources	1.13 (1.03, 1.24)**	1.03 (0.93, 1.15)	0.93 (0.76, 1.14)
+Basic	1.10 (1.00, 1.21)**	1.01 (0.90, 1.12)	0.99 (0.81, 1.22)
+Lifestyles	1.10 (1.00, 1.20)*	1.00 (0.90, 1.12)	0.98 (0.80, 1.21)
+PA	1.10 (1.00, 1.20)*	1.00 (0.90, 1.12)	0.99 (0.80, 1.22)
+NO_2	1.16 (1.04, 1.29)**	1.01 (0.90, 1.13)	0.98 (0.80, 1.21)

Mixed effects linear regression models (β: beta, 95% CI: 95% confidence intervals) and logistic regression models (OR: odds ratio, 95% CI) for continuous and binary outcomes, respectively.
Noise sources: adjustment for the other transportation noise sources, respectively. Basic: additionally adjusted for age, age^2, age^3, sex, education, random intercept by study area. Lifestyles: additionally adjusted for smoking, secondary-hand smoke, diet, alcohol intake. PA: additionally adjusted for physical activity. NO_2: Additionally adjusted for exposure to nitrogen dioxide. $^{**}p < 0.05$, $^{*}p$-value < 0.1.

3.4. Effect modification analysis

According to the interaction analyses (Fig. 3), the association between 5-year mean exposure to road traffic noise and BMI at SAP3 was stronger in participants who had reported doctor-diagnosed cardiovascular disease (β = 0.68, 95% CI: 0.37; 0.99) than those who did not (β = 0.24, 95% CI: 0.003; 0.47), *p*-value of interaction = 0.017. There was also an association between 5-year mean exposure to railway noise and BMI in those who reported bad sleep quality (β = 0.63, 95% CI: 0.11; 1.15) but not in those with good sleep quality (β = −0.01, 95% CI: −0.23; 0.22), p-value of interaction = 0.018. Such association was also suggestive for participants feeling often insufficiently rested when waking up, compared with well-rested participants, and for participants reporting noise sensitivity above the median, compared to the rest. These interactions, however, were not significant (*p*-values of interaction = 0.06 and 0.09, respectively). No interactions were detected for the association between exposure to 5-year mean exposure to aircraft noise and BMI.

4. Discussion

The present study contributed to the scant evidence available about

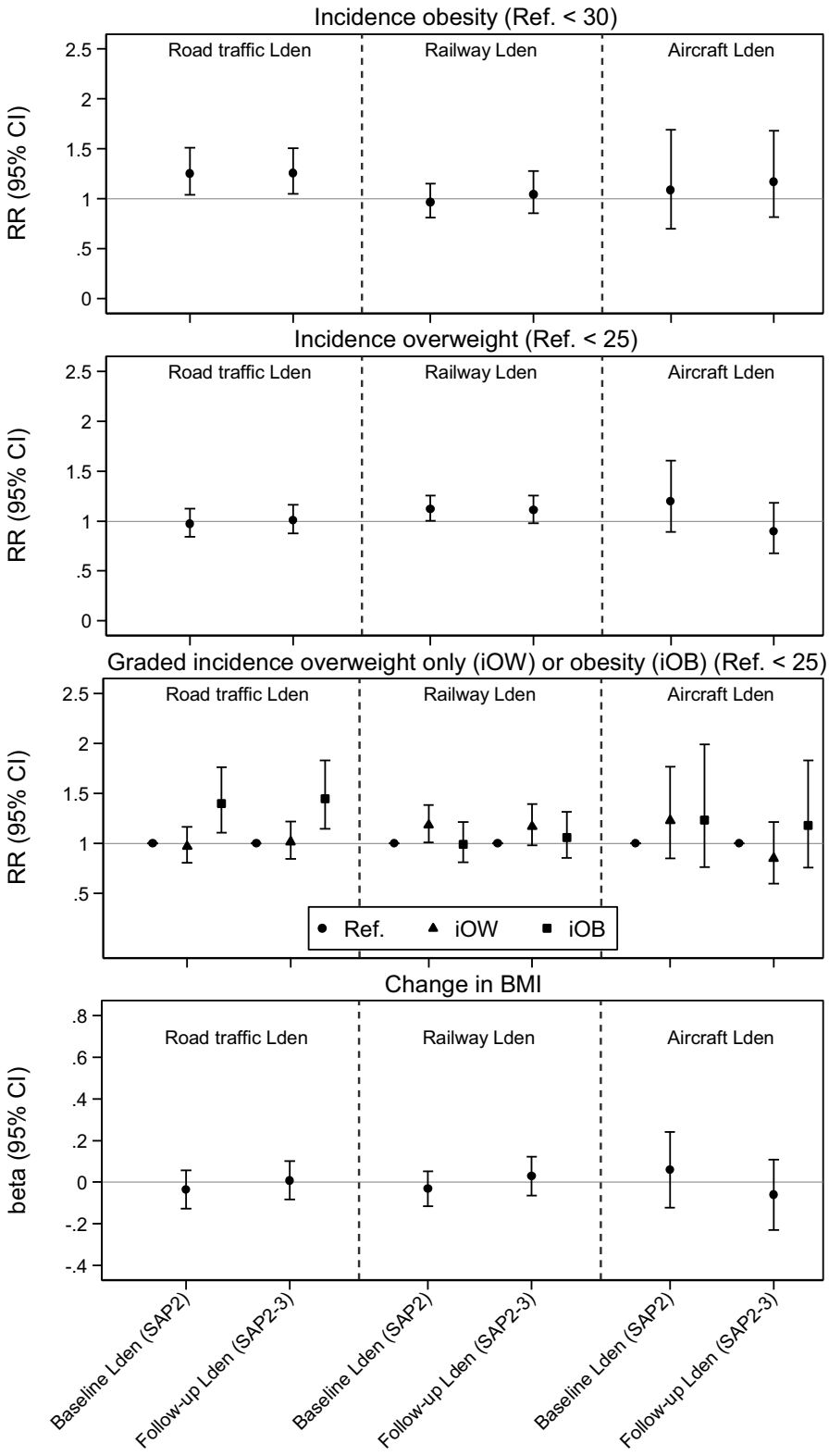

Fig. 2. Relative risk (RR, points) or beta coefficient (beta, points) and 95% confidence intervals (95% CI, spikes) for the associations between exposure to transportation noise at different time windows with incidence outcomes or change in BMI, respectively, per 10 dB, $N = 3268$ (incidence obesity), $N = 2089$ (incidence overweight only or obesity), $N = 1852$ (incidence overweight), N = 3796 (change in BMI). Multivariate Poisson regression models (binary incidence outcomes), multinomial regression models (graded incidence of overweight or obesity) and multivariate linear mixed models were adjusted for variables at SAP2: road, railway, aircraft noise levels (Lden, dB), age, age^2, age^3, sex, education, study area, smoking, secondary-hand smoke, diet, alcohol intake, being at least moderately physically active (PA), nitrogen dioxide levels; and changes between SAP2 and SAP3: age, alcohol, physical activity, and nitrogen dioxide levels. Linear mixed models included study area as a random intercept. Baseline: 5-year mean Lden before SAP2, Follow-up: Follow-up time mean Lden (between SAP2 and SAP3). iOW: overweight only (BMI at SAP2 $<$ 25 and at SAP3 between 25 and 29.9), iOB: BMI at SAP2 $<$ 30 and at SAP3 $\geq$ 30.

the association between long-term exposure to road, railway and aircraft noise and obesity. In particular, we were able to evaluate individual exposure to these most prevalent transportation noise sources, taking into account residential history and exposure to traffic-related air pollution, green spaces and walkability, among others. Furthermore, we analysed objective measures of clinically relevant outcomes, and importantly, we provided longitudinal evidence for the development of overweight and obesity. We observed a consistent association between long-term exposure to road traffic noise and all of the overweight and obesity indicators (related to general or central obesity and percent body fat) in cross-sectional analyses and with risk of obesity in longitudinal analyses. Exposure to railway noise was only associated with risk of overweight or overweight only. We observed no associations related to exposure to aircraft noise.

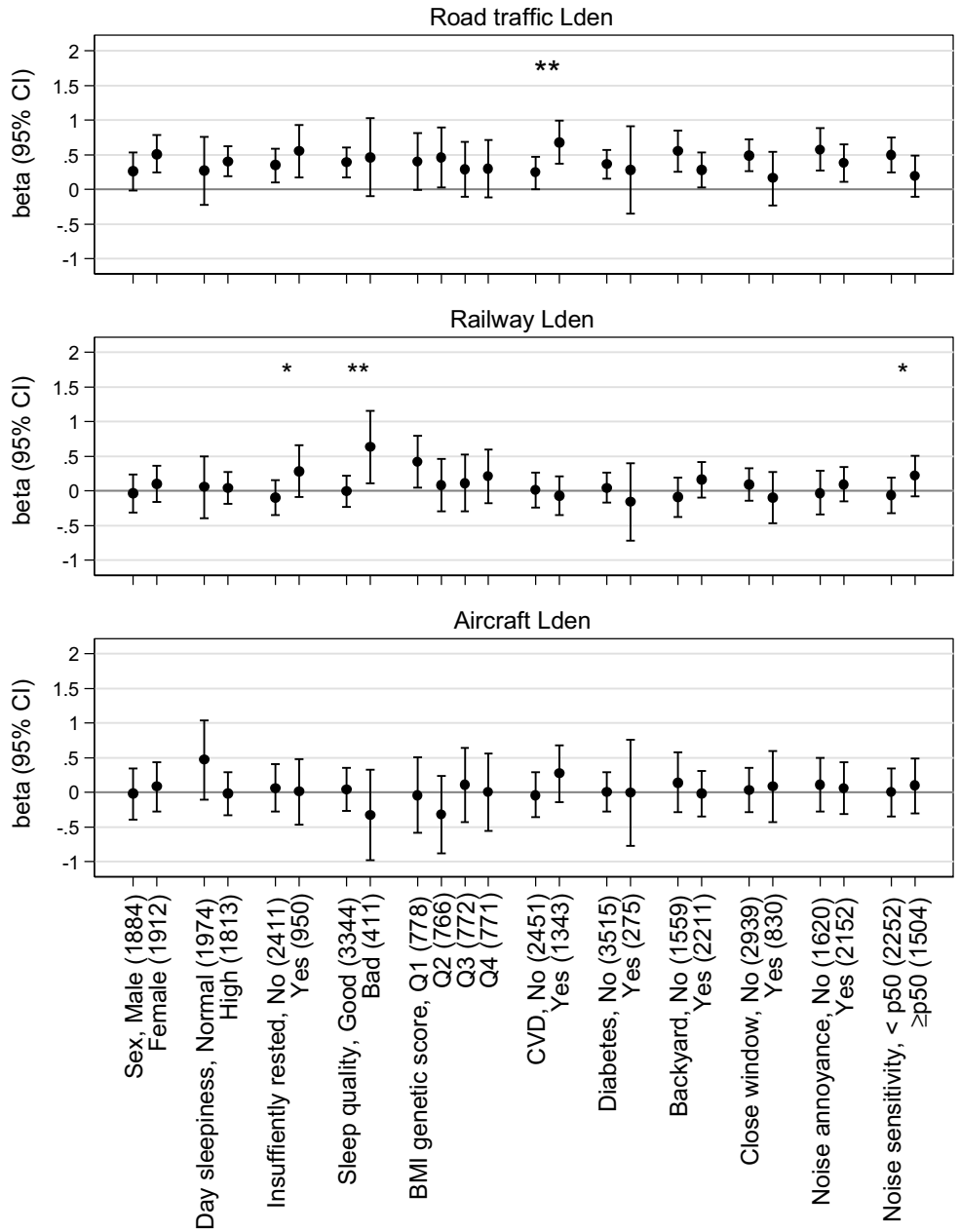

Fig. 3. Association between 5-year mean source-specific noise levels (Lden) and BMI at SAP3 across different personal characteristics, per 10 dB change of the respective noise indicator. Multivariate linear mixed models included an interaction term between Lden and the corresponding personal characteristic and were adjusted for the other transportation noise sources, age, age^2, age^3, sex, education, smoking, secondary-hand smoke, diet, alcohol intake, physical activity, NO_2, and a random intercept by study area. ***p*-value of interaction < 0.05, **p*-value of interaction < 0.100.

4.1. Comparison with cross-sectional studies

To our knowledge, there are six studies from three cohorts assessing the associations addressed in the present study: one in Denmark (two studies: one cross-sectional, one longitudinal), one in Norway (cross-sectional) and the third cohort in Stockholm (three studies: one cross-sectional and two longitudinal). Compared to the present study, only the cross-sectional study in Denmark (Christensen et al., 2016) also assessed BMI, waist circumference, and makers of body fat, and it reported associations of similar to slightly smaller magnitude between road traffic noise (Lden) and all the obesity markers. Oftedal et al., 2015 (Norway) only observed associations with waist circumference and BMI in highly noise sensitive women. Finally, Pyko et al., 2015 (Stockholm) detected associations with waist circumference and central obesity for road traffic noise (Lden) ≥ 45 dB, but not with BMI.

4.2. Comparison with longitudinal studies

4.2.1. Road traffic noise

Our findings indicate an association between exposure to road traffic noise and risk of obesity, but not with change in BMI. Actually, the two previous longitudinal studies also found a null association between road traffic noise exposure before baseline and change in weight. However, change in weight is a proxy for change in BMI, which does not take into account the influence of the person's height on body mass (Christensen et al., 2015; Pyko et al., 2017). Only with follow-up exposure did Christensen et al., 2015 observed a small positive association, though it was borderline significant after adjustment for lifestyles and based on self-reported weight at follow-up. In ancillary analyses for comparison, we also found no clear association with change in weight. This was either slightly negative with road traffic noise exposure before

baseline (β = −0.16 kg, 95% CI: −0.42, 0.11, *p*-value = 0.253) or null during follow-up (β = −0.01 kg, 95% CI: −0.27, 0.26, *p*-value = 0.941) per 10 dB. The previous two studies only observed more consistent positive associations between exposure to road traffic noise and change in waist circumference, information not available in our study.

In contrast, Christensen et al. (2015) observed an association between exposure to road traffic noise and a binary outcome defined as risk of weight gain of at least 5 kg between surveys (Yes/No). This association had a similar magnitude but was unsignificant in our ancillary analyses performed for comparison (RR = 1.07, 95% CI: 0.98–1.17 for baseline exposure). We speculate that the lack of association with change in BMI or weight may relate to the little contrast in these outcomes and their lower specificity regarding detrimental adipose increases. This might be easier to observe with waist circumference and with binary outcomes which provide information on a minimal relevant change and its severity, such as our finding for risk of obesity, a clinical binary outcome. In turn, although Pyko et al. (2017) did not evaluate risk of general obesity, they observed an association between exposure to road traffic noise and risk of central obesity, but not with general overweight. This would be in line with the present study, which found an association between road traffic noise and general obesity and no association with overweight or overweight only. Finally, in line with the minor changes generally observed in previous studies adjusting for air pollution (Christensen et al., 2015; Oftedal et al., 2015; Pyko et al., 2015), we found that associations between road traffic noise and adiposity markers or risk of obesity were robust to adjustment for traffic-related air pollution.

4.2.2. Railway noise

Fewer studies have analysed exposure to railway noise in association with obesity and results have been inconsistent. Findings point to positive associations related either to central obesity in one cross-sectional study (Pyko et al., 2015) and to waist circumference and BMI for Lden > 60 dB (Christensen et al., 2016), or to a risk of weight gain of at least 5 kg for Lden > 55 dB (Christensen et al., 2015). Only Pyko et al., 2017 analysed risk of overweight but found no relationship with railway noise, in contrast with the present study.

4.2.3. Aircraft noise

Of the transportation sources, exposure to aircraft noise was the least studied and it also led to null associations in the Danish cohort (Christensen et al., 2015). Previous positive findings with central obesity, cross-sectionally (Pyko et al., 2015) and longitudinally (Eriksson et al., 2014; Pyko et al., 2017) with change in BMI and waist circumference, were all related to the same study population in Stockholm.

4.3. Effect modification

Regarding effect modification, the current study indicated, based on cross-sectional analyses, that participants with CVD may be more susceptible to the impact of road traffic noise on BMI, a finding that should be replicated in future studies. The association between exposure to railway noise and BMI was positive in participants reporting noise sensitivity and those being insufficiently rested, although it was only statistically significant for participants reporting bad sleep quality. This could be a spurious finding, nevertheless the consistent effect modification in self-reported night-time sleep conditions and noise sensitivity might potentially relate to the evidence that suggests a greater subjective sleep disturbance response related to exposure to railway than to road traffic noise (Basner et al., 2011; Hong et al., 2010).

4.4. Public health relevance and biological interpretation

Our results suggest that long-term exposure to road traffic noise could be more detrimental than railway noise, which only increased the risk of overweight. However, such observation may be also influenced by the greater exposure to road than to railway or aircraft noise in the present sample. Interestingly, the severity of the effects of road traffic noise was reinforced by its association with direct measures of abdominal fat (waist circumference and central obesity) and adiposity (percent body fat), which predict disease risk beyond BMI (World Health Organization, 2011; Zeng et al., 2012). Both overweight and obesity contribute to morbidity and mortality, particularly to CVD but also to diabetes and cancer (The GBD 2015 Obesity Collaborators, 2017). The magnitude of the estimated effect of road traffic noise in the present study, combined with its prevalence (European Environment Agency, 2014), support that reducing exposure to road traffic noise could substantially reduce the obesity epidemic and decrease morbidity and mortality world-wide (NCD Risk Factor Collaboration, 2016). Environmental interventions could be particularly effective in preventing obesity, taking into account the difficulty of changing individual behaviours.

The current epidemiological findings may respond to the biological mechanisms of long-term exposure to noise. These suggest that repeated noise-related stress and sleep disturbance would lead to chronic endocrine and autonomous nervous system alterations (Münzel et al., 2016), and interrelated oxidative, inflammatory, or immune responses (Münzel et al., 2017; Recio et al., 2016; Schmidt et al., 2013), contributing to cardiometabolic diseases. The greater impact of railway noise exposure on BMI at SAP3 among those who reported sleep problems could potentially point to the noise-related sleep mechanisms and the subjective perception of railway noise (See Section 4.2). However, this observation should be confirmed in further studies, which also consider objective sleep indicators and formal mediation analysis for more mechanistic insights. Finally, two previous studies suggested an association between noise annoyance (Foraster et al., 2016) or the noise exposure level (Roswall et al., 2017) with physical activity, which could respond to change of behaviour due to noise perception or sleep disturbance, and ultimately impact obesity. However, in the present study, neither noise annoyance nor physical activity affected any of the associations between the noise exposure level and obesity markers.

4.5. Strengths and limitations

A major strength of the current study was the evaluation of a prospective population-based cohort, which allowed us to reduce reverse causality by studying exposure before the outcome happened. The consistency between longitudinal and cross-sectional results for exposure to road traffic noise further reinforced the findings and support associations for waist circumference and body fat, despite being only available at SAP3. We used objective and standard anthropometric measures, and BMI both at SAP2 and SAP3, as well as clinically relevant outcomes of overweight and obesity. This allowed us to assess the severity of the estimated effects of transportation noise on obesity in addition to its continuous change.

Another main strength was the high quality individual estimation of residential noise exposure for entire Switzerland with models representative of the different examination years. Moreover, we estimated long-term exposure to transportation noise based on residential history for each participant. This helped us to reduce exposure misclassification and selection bias, and to assess different exposure windows, which provided similar results. Moreover, we were able to assess exposure modification by closing windows at night or having the bedroom facing the backyard (Babisch et al., 2014; Foraster et al., 2014), which did not change findings. Besides, regular use of ear plugs at night was rare (3.9%), thus its omission is unlikely to affect findings. Non-differential exposure misclassification may remain from exposure to transportation and other noise sources at work, assuming day-time noise affected obesity similarly, which is unclear (Münzel et al., 2016). In such case, results would be biased towards the null and conservative.

Finally, we studied the independent effects of the main transportation noise sources, and controlled for many confounders, including traffic-related air pollution, not always available in previous studies. However, residual confounding can never be discarded.

One common limitation is selection bias due to loss of participants. Nevertheless, results did not seem to be affected (or were even slightly conservative) by lost-to-follow-up and missing observations, according to IPW and the similar characteristics of the final to the original samples. Therefore, results from this population-based cohort may be generalizable to the Swiss and other populations with similar noise exposure levels and sources. Regarding the small sample size in incidence analyses, it did not seem to influence the results for road traffic noise, which were consistent with cross-sectional analyses. However, the power to detect effects for railway or aircraft noise could have been limited due to lower prevalence and smaller contrast of exposure to these forms of transportation noise in the sample. Moreover, the small number of participants exposed to aircraft noise at night (12.6% exposed above 30 dB at night) due to restriction of aircraft traffic, might partly explain the null findings for this exposure, assuming the nighttime was the most relevant period of exposure to develop obesity (Münzel et al., 2016). Finally, although the residential address history was mainly self-reported, exposure misclassification seemed limited, according to the consistent findings across the 5-year time windows before each survey and the follow-up time window. This may be because the time to be remembered was rather short. Furthermore, register data was used at baseline, to complete the address history during the cleaning process, and for unreached participants in the next survey. Observations with low quality geocodes (i.e. not available at street level) were also excluded from analyses.

5. Conclusions

Long-term exposure to road traffic noise may increase the risk of obesity. According to cross-sectional results, this impact may relate to the alteration of all obesity parameters (i.e. high body mass index (BMI), abdominal fat and total adiposity). These results from the Swiss SAPALDIA cohort add to the initial evidence on the association between transportation noise and obesity provided by studies in Sweden, Denmark, and Norway. Obesity could represent one pathway through which transportation noise impacts cardiovascular disease and diabetes. These findings also shed light on prevention measures against the obesity epidemic.

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Disclosures

None

Appendix A. Supplemental Material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2018.09.057.

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