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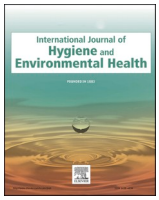

Depressive symptoms modify the association between noise and adiposity biomarkers: Evidence from a population study of Czech adults

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ABSTRACT

Objective: Environmental noise exposure is associated with adiposity. However, less is known about the individual vulnerability to environmental noise in abnormal adiposity development, particularly in relation to mental health. This study investigated the association between environmental noise exposure and four adiposity biomarkers and tested the moderation effect of depressive symptoms.

Methods: A cross-sectional population-based sample of 2031 participants aged 25–64 years (54.70% women) was drawn from the Kardiovize study in 2013. Global combined (road, railway, and airport) L_{den} (day-evening-night) noise exposures were obtained from the geographical prediction modelling for the 2nd report of Strategic noise mapping in the Czech Republic (2012). Four adiposity biomarkers (BMI, body fat percentage, waist circumference, and visceral fat area) were assessed. Depressive symptoms were measured by PHQ-9. Linear regression was used to estimate the separate effects of quartiles of noise exposure and depressive symptoms on adiposity biomarkers and to examine the interaction between noise exposure and depressive symptoms.

Results: The average noise exposure was 53.79 dB, ranging from 42.50 dB to 66.97 dB. All biomarkers were significantly elevated in the highest noise exposure quartile (>56 dB), compared to the lowest quartile (<51 dB) ($p < 0.05$). The association between noise and adiposity biomarkers was modified by presence of depressive symptoms; the increase in all adiposity biomarkers in the highest quartile of noise was significantly larger among subjects with moderate to severe depressive symptoms ($p < 0.005$).

Conclusion: The study confirmed the association between environmental noise exposure and several adiposity measures. The association was stronger in the presence of depressive symptoms.

1. Introduction

Abnormal adiposity is one of the largest challenges for contemporary public health systems worldwide. For example, in 2019, high body mass index was responsible for 15.0% of deaths in Central Europe (Roth et al., 2020). The strong body of evidence regarding the role of lifestyle factors in the aetiology of abnormal adiposity has resulted in numerous lifestyle interventions (Bendall et al., 2018; Castellana et al., 2020). However, individual lifestyle does not act in isolation. Instead, there is a complex network of stressors including social factors and environmental exposures (Juarez et al., 2020) affecting individual health. Environmental noise is one of the most important urban stressors impacting health (Hänninen et al., 2014), linked to the built environment including road traffic (WHO, 2018). In Europe, more than 20% of the population is exposed to harmful levels of environmental noise (the weighted 24-h noise levels $L_{den} \geq 55$ dB) (European Environment Agency, 2020) and this prevalence may be even underestimated, given that the Environmental Noise Directive (END) ("Directive, 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise," 2002) does not cover all urban areas across Europe (European Environment Agency, 2020). The 24-h noise limit represents a tolerable level of exposure from

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dose-response relationship studies below which most of the population will be protected from adverse health effects (Muralikrishna and Manickam, 2017).

Environmental noise has been consistently associated with cardiovascular diseases including elevated blood pressure (Münzel et al., 2018) and there is growing evidence about other cardiometabolic biomarkers. Some studies reported a significant association between type 2 diabetes and traffic noise exposure (Zare Sakhvidi et al., 2018). Traffic noise has also been positively associated with higher BMI and waist circumference, although the effects were generally small and less consistent (An et al., 2018; Christensen et al., 2015, 2016; Cramer et al., 2019; Dzhambov et al., 2017; Oftedal et al., 2015). Nighttime noise exposure also causes sleep deprivation which may lead to endocrine alterations and subsequently to abnormal adiposity (Bacaro et al., 2020; Chen et al., 2023). Among individuals with occupational noise exposure, higher blood cholesterol, triglyceride concentration and increased risk of dyslipidemia have been observed (Mehrdad et al., 2011). However, occupational exposures frequently exceed noise levels of 80 dB (Mehrdad et al., 2011), a level higher than experienced by the general population.

There are several underlying mechanisms of the relationship between noise and abnormal adiposity. Previous literature described two main pathways through which noise exposure affects health. The direct pathway encompasses the damage of sensory receptors of the auditory system caused by exposure to high-intensity noise, and sleep disruption. The indirect pathway operates through individual disturbance and annoyance leading to other mental health conditions such as depression or emotional response (Hahad et al., 2022).

Both pathways raise physiological stress response, particularly cortisol overproduction (Sivakumaran et al., 2022). The prolonged exposure to elevated cortisol levels has in turn been shown to increase reactive oxygen species at cellular levels, while increased oxidative stress may lead to lipid peroxidation, systematic inflammation (Wang et al., 2007), and autonomous nervous system alterations (Chen et al., 2023; Foraster et al., 2018). Previous evidence reported that individual ability to deal with chronic stress is, however, also influenced by individual mental health conditions. A meta-analysis conducted by Burke et al. (2005) reported that the presence of depressive symptoms leads to impaired stress recovery (Burke et al., 2005). Depressive symptoms might, therefore, enhance the effect of noise exposure on health. Given this evidence, we hypothesize that the relationship between environmental noise and adiposity may be affected by the presence of depressive symptoms. Thus, this study aimed to first, investigate the association between environmental noise exposure and four adiposity biomarkers (BMI, body fat percentage, waist circumference, and visceral fat area) and second, to test the effect modification by depressive symptoms.

2. Methods

2.1. Design and population

Data from the Kardiovize study (Movsisyan et al., 2018) were analysed. Kardiovize study is a cross-sectional population-based study, assessing the health of the adult population in Brno, the second-largest city in Czechia, with 373,327 residents. The study aimed to enroll 1% of the adult population of Brno randomly selected and stratified by sex and age. Eligibility criteria included age (25–64 years), permanent residence in Brno, and registration (required by the law) with any of the five health insurance companies operating in the Czech Republic. Survey sampling was performed in January 2013 with technical assistance from the health insurance companies. A random age- and sex-stratified sample of 2154 men and women were enrolled. Geocodes for residential locations were obtained at the street level for 2152 participants and at the municipal district level for 2 participants. For the current analysis, 2 participants with the geocodes at the municipal district level and 121 participants located outside the spatial extent of the environmental noise model were excluded. No information on non-respondents was available due to confidentiality.

2.2. Overview of data collection

In-person health interviews were performed by trained nurses and physicians at the International Clinical Research Centre of St Anne's University Hospital in Brno. The questionnaire included demographics, socioeconomic characteristics, cardiovascular risk behaviours, smoking status, medical history, and mental health. Anthropometric assessments were also performed, as outlined below.

2.3. Measures

2.3.1. Outcomes

The four outcome variables were measured as follows. (1) Body mass index was calculated based on height and weight measurements using a medical digital scale with a meter (SECA 799; SECA, GmbH and Co. KG, Germany). (2) Waist circumference was measured using manual tape. (3) Body fat percentage and (4) visceral fat area were measured using direct segmental multifrequency bioelectrical impedance analysis (InBody 370; BIOSPACE Co., Ltd., Korea).

2.3.2. Predictors

The environmental noise exposures were estimated using the prediction model developed for the 2nd report on Strategic noise mapping in the Czech Republic (2012), conducted in accordance with the Environmental Noise Directive (END)(2002) ("Directive, 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise," 2002) requirements and methods, further worked out in a position paper of the European Commission Working Group Assessment of Exposure to Noise (WG-AEN) Good Practice Guide for Strategic Noise Mapping and the Production of Associated Data on Noise Exposure (European Commission Working Group Assessment of Exposure to Noise, 2006). The recommended interim computation methods (paragraph 2.2 of Annex II of the END) were used for respective noise-emission types (ISO 9613-2 for industrial noise, ICAO for aircraft noise, RMR for railway noise, and NMPB method for road traffic noise) and were implemented in commercial software. A report, detailing the methods used in the Czech Strategic noise mapping was published for its first phase (Šlachtova et al., 2007). To estimate the individual exposures, we have used the global combined (road, railway, and airport) 24-h (day-evening-night (L_{den})) weighted average noise levels, penalizing evening and nighttime noise by 5 and 10 dB, respectively (END 2002("Directive, 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise," 2002)). The noise levels were obtained from Strategic noise maps, available as Web Map Service on the Ministry of Health's Geoportal (Ministerstvo zdravotnictví, 2012). The noise levels are represented as isoline curves, assessed at the height of 4 m in 5 dB ranges. The noise levels were obtained for each residential building at its centroid and mean, median, and standard deviation values were obtained for each street. Buildings with missing data in the noise prediction model within the borders of the modelled territory (the Brno agglomeration) – where the sound propagation is attenuated by buildings or topography below modelled threshold – were imputed with the lowest category of the noise level.

Depressive symptoms were measured by the Patient Health Questionnaire (Kroenke et al., 2001) (PHQ-9) instrument. Participants rated 9 items (e.g., "I feel down, depressed, hopeless.") on a four-point Likert-type scale ranging from never (0) to most of the time (3) with a recall period of two weeks before the visit. PHQ-9 score thus ranged between 0 (no depressive symptoms) and 27 (high score of depressive symptoms). Based on the previously defined cut-offs, we categorized the PHQ-9 score into three groups: <5 points; 5 to 9 points (mild depressive

symptoms; 10 points and higher (moderate to severe depressive symptoms) (Kroenke et al., 2001).

2.3.3. Covariates

Educational attainment was classified into three groups: "high", including subjects with higher professional or university education; "middle", defined as high school education; and "low", defined as elementary or vocational education without a final graduation exam.

Equalized household income was assessed to consider the differences in a household's size and composition. Household income was collected using categories defined by income ranges, and appropriate mid-value was then used in this calculation. The equivalent size is calculated by attributing a weight to all members of the household in the following way: 1.0 for the first person and 0.5 for each subsequent person in the household. The equivalent size is the sum of the weights of all the members of a given household (Eurostat, 2021). These weighted values of the household incomes were used as continuous variables in the analysis.

Dietary risk behaviours were assessed using a dietary risk score derived from the 43-item Food Frequency Questionnaire (FFQ). Participants were asked to indicate the frequency of consumption of specific food groups in the past week on a scale including 10 options from "almost never" to "six or more times a day". In total, six specific risky dietary patterns were identified based on the Global Burden of Disease (GBD) (Afshin et al., 2019) methodology (Supplementary Table 1). The occurrences of each risky dietary pattern were summed, so the total dietary risk score ranged from 0 to 6 points.

Smoking status was categorized as current smokers, ex-smokers, and non-smokers. We defined a current smoker as smoking either daily or less than daily or stopped smoking less than a year before the interview, an ex-smoker as having stopped smoking at least a year before the interview, and a non-smoker as having smoked fewer than 100 cigarettes in a lifetime.

Alcohol intake was estimated as the total amount of ethanol in grams consumed during the week before data collection. The data were obtained from a 7-day alcohol consumption recall, where a participant reported alcoholic beverages consumed 7 days before the visit (Fiala and Sochor, 2014).

Sedentary behaviors were evaluated by total sitting time in minutes per week before the examination, obtained from the long version of the International Questionnaire of Physical Activity(CRAIG et al., 2003) (IPAQ).

2.3.4. Data analysis

Data analyses were conducted using STATA (StataCorp, 2019) software (version 16.0, StataCorp, College Station, TX, USA) and Mplus 8.10 (Muthén and Muthén, 2017). Continuous variables were described using means and standard deviations. Differences between quartiles of noise exposure were tested using One-Way ANOVA and Chi-Square test. Furthermore, multiple linear regression was performed to examine the association between noise exposure categorized into quartiles and adiposity biomarkers. Second, we estimated the effect modification by depressive symptoms in the association between noise exposure and adiposity using the Wald test. Further, the stratum-specific analysis was performed to investigate the association between noise and adiposity separately at three levels of depressive symptoms. As a sensitivity analysis, we investigated the effect of depressive symptoms as a confounder in the association between noise exposure and adiposity biomarkers (Supplementary Table 2) as well as the effect modification by age and sex (Supplementary Table 3, Supplementary Table 4) in stratum specific analysis. All models estimated standardized β coefficients adjusted for sex, age in years, education (3-level variable: low, middle, high), income, smoking status (3-level variable: non-smoker, ex-smoker, smoker), alcohol intake, dietary risk behaviors, and sedentary behaviors as continuous variables. The covariates were selected based on previous research. P values less than 0.05 were considered statistically significant. A complete case analysis method was used for handling missing data.

3. Results

3.1. Subjects characteristics

The analytical sample consisted of 2031 (54.70% women) subjects with a mean age of 47.70 ± 11.18. The descriptive characteristics are provided in Table 1. The average noise exposure was L_{den} = 53.79 dB, ranging from 42.50 dB to 66.97 dB. In total, 34.9% of participants were exposed to environmental noise above 55 dB. We observed differences in the distribution of education among quartiles of noise exposure, showing that the highest proportion of subjects with low levels of education live in the areas with the highest quartile of noise exposure. The average values of all four assessed adiposity biomarkers (BMI, waist circumference, body fat percentage, visceral fat area) were higher in the highest quartile of noise exposure compared to the lowest quartile (Table 1).

3.2. Environmental noise exposure and adiposity

The results of linear regression models showed a positive association between noise and adiposity. In a fully adjusted model (model 1) we observed the associations between noise exposure and adiposity biomarkers: all four assessed outcomes were significantly worse in the highest quartile of noise exposure (>56 dB) compared to the lowest quartile of noise exposure (<51 dB) (Table 2). We found no evidence of the association between adiposity outcomes and the second and the third quartiles of noise exposure,

The effect modification by levels of depressive symptoms in the associations between noise and adiposity biomarkers was observed in the stratum specific analysis (Fig. 1). The results showed that the association between the highest quartile of noise exposure (>56 dB) and all four assessed biomarkers was larger in participants with the presence of 10 and more depressive symptoms compared to those with less than 5 depressive symptoms; however, the overall interaction was statistically significant only for BMI ($p = 0.042$), while not for waist circumference ($p = 0.104$), body fat percentage ($p = 0.131$), and visceral fat ($p = 0.259$).

Sensitivity analysis revealed statistically significant associations between experiencing depressive symptoms and increased risk to all adiposity biomarkers (Supplementary Table 2). There was no evidence of interactions between age and noise nor between sex and noise on adiposity (Supplementary Tables 3 and 4).

4. Discussion

The aim of this study was to investigate the relationship between environmental noise exposure and diverse adiposity biomarkers and to examine whether the association is modified by depressive symptoms. Within the range of the noise exposure (42–67 dB), the highest but not the medium levels of noise were associated with poorer measures of adiposity. Specifically, the values of BMI, waist circumference, body fat percentage, and visceral fat were higher in individuals falling into the highest quartile of estimated environmental noise (>56 dB), compared to those in the lowest quartile (<51 dB). Moreover, we observed the modifying effect of depressive symptoms, which exacerbated the association between the highest noise quartile exposure and adiposity biomarkers.

The association between environmental noise exposure and adiposity observed in the current study is consistent with previous studies performed in different populations. The systematic review and meta-analysis conducted by An et al., in 2018 investigated the effect of long-term noise exposure on adiposity biomarkers. According to that study, participants with long-term noise exposure at the level above

Table 1
Subjects characteristics in quartiles of environmental noise exposure, Kardiovize study, Brno, Czechia.

	Total	Quartiles of environmental noise exposure				p
		<51.0 dB	51.0–53.5 dB	53.6–56.0 dB	>56 dB	
n (% women)	2031 (54.70)	508 (54.13)	514 (55.06)	503 (57.46)	506 (52.17)	
	Mean (SD) or %	Mean (SD) or %	Mean (SD) or %	Mean (SD) or %	Mean (SD) or %	
Age	47.70 (11.18)	48.50 (11.23)	47.18 (11.34)	47.61 (11.23)	47.53 (10.91)	0.277
Adiposity biomarkers						
BMI (kg/m2)	26.2 (5.1)	25.7 (4.8)	26.0 (5.0)	26.1 (5.3)	26.9 (5.2)	<0.001
WC (cm)	90.2 (14.4)	89.1 (13.9)	89.6 (13.9)	89.8 (15.1)	92.3 (14.4)	0.002
Body fat %	26.7 (9.6)	26.0 (9.5)	26.5 (9.7)	26.6 (9.7)	27.6 (9.4)	0.056
VFA (cm^2)	89.8 (39.1)	86.5 (38.0)	88.8 (39.1)	88.4 (40.1)	95.6 (38.7)	0.002
Depressive symptoms						
No depressive symptoms	73.0	74.9	70.8	73.3	73.1	0.637
Mild depressive symptoms	21.4	20.8	23.0	21.5	20.4	
Moderate to severe depressive symptoms	5.6	4.4	6.3	5.2	6.6	
Education						
Low	20.3	15.8	21.2	20.7	23.5	0.014
Middle	38.3	43.1	34.2	37.6	38.3	
High	41.4	41.1	44.6	41.8	38.1	
Equalized household income (CZK)[a]	20724.2 (11721.3)	21523.1 (12616.2)	20970.9 (11371.6)	20460.5 (11894.8)	19978.0 (10930.1)	0.205
Dietary risk score[b]	4.7 (1.1)	4.6 (1.1)	4.7 (1.1)	4.6 (1.1)	4.8 (1.00)	0.006
Sedentary behaviours[c]	2882.8 (1206.9)	2823.5 (1183.9)	2983.9 (1224.5)	2838.6 (1195.4)	2883.6 (1219.9)	0.138
Alcohol consumption (g)[d]	77.5 (99.5)	80.4 (98.6)	78.0 (100.6)	74.3 (100.2)	77.4 (98.8)	0.812
Smoking						
Smokers	23.4	19.9	22.6	24.9	26.1	0.254
Ex-smokers	25.6	27.6	24.1	24.9	25.7	
Non-smokers	51.1	52.6	53.3	50.2	48.2	

BMI: Body mass index. VFA: Visceral fat area. WC: waist circumference. Differences using One-Way ANOVA or Chi-Square test.
[a] Household income values using weights based on the number of household members.
[b] Participants' engagement in risky dietary behaviors by summing the occurrences of specific identified risky dietary patterns, with a total score ranging from 0 to 6 points.
[c] Reported in total sitting time in minutes per week.
[d] Grams of ethanol consumed in the last 7 days.

Table 2
The associations between environmental noise exposure and adiposity biomarkers among adults aged 25–64 years from the Kardiovize study in Brno, Czechia.

	β	p
BMI		
Noise		
Q1 (<51.0 dB)	Ref.	
Q2 (51.0–53.5 dB)	0.032	0.220
Q3 (53.6–56.0 dB)	0.046	0.078
Q4 (>56 dB)	0.085	0.001
p for trend		0.001
Waist circumference		
Noise		
Q1 (<51.0 dB)	Ref.	
Q2 (51.0–53.5 dB)	0.018	0.409
Q3 (53.6–56.0 dB)	0.038	0.098
Q4 (>56 dB)	0.069	0.002
p for trend		0.002
Body fat percentage		
Noise		
Q1 (<51.0 dB)	Ref.	
Q2 (51.0–53.5 dB)	0.020	0.358
Q3 (53.6–56.0 dB)	0.022	0.315
Q4 (>56 dB)	0.066	0.003
p for trend		0.004
Visceral fat		
Noise		
Q1 (<51.0 dB)	Ref.	
Q2 (51.0–53.5 dB)	0.025	0.329
Q3 (53.6–56.0 dB)	0.022	0.384
Q4 (>56 dB)	0.084	0.001
p for trend		0.002

Results adjusted for age, sex, education, income, dietary risk behaviors, smoking status, alcohol intake, sedentary behaviors.

55–60 dB tend to increase their waist circumference by 7.5 mm per year; however, no significant association between noise exposure and BMI was found (An et al., 2018). Similarly, a systematic review and meta-analysis conducted by Gui et al., in 2022 showed that the highest versus the lowest level of noise exposure was associated with higher waist circumference ranging from 0.326 cm to 0.705 cm and higher odds of central obesity ranging from 5.5% to 16.7% (Gui et al., 2022). Another study from 2018 (Foraster et al., 2018) including 3796 participants from Switzerland reported the positive effect of road or railways noise on BMI and waist circumference (Foraster et al., 2018).

The mechanisms that underlie the relationship between noise exposure and adiposity include body stress response expressed in alteration of homeostasis and intensifying oxidative stress as well as systemic inflammation (Chen et al., 2023). These physiological changes may in the long run promote insulin resistance, and accumulation of adipose tissue (Björntorp and Rosmond, 2000; Recio et al., 2016). Our results showed that the presence of depressive symptoms modified the association between environmental noise and adiposity. Increased vulnerability to environmental noise in those with increased depressive symptoms is given by the impaired stress response. People experiencing depression tend to display a notably unresponsive and consistent cortisol secretion pattern (Burke et al., 2005). The individual ability to respond to environmental stressors involves the hypothalamic-pituitary-adrenal (HPA) axis and autonomic nervous systems which influence the cardiometabolic and immune systems to help the body respond appropriately to the environment (McEwen and Stellar, 1993). The results of previous studies suggested that depression may alter HPA axis functioning and therefore lead to blunted and exaggerated cortisol responses to and impaired recovery from stress(Fiksdal et al., 2019). In the long term, people with depression may face more significant effects of chronic stress, including the adiposity investigated in this study (Burke et al., 2005; Fiksdal et al., 2019).

In a broader explanation of underlying mechanisms, it is also important to consider the role of sleep disturbance. An experimental

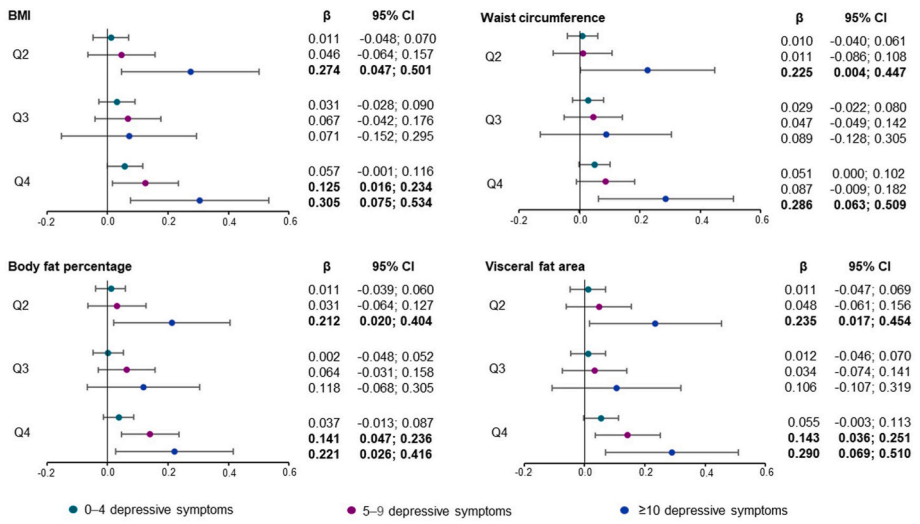

Fig. 1. The associations between environmental noise exposure (categorized into quartiles with Q1 as a reference) and adiposity biomarkers among Kardiovize study participants, stratified by depressive symptoms. Results adjusted for age, sex, education, income, dietary risk behaviours, smoking status, alcohol intake, and sedentary behaviours.

study conducted by Coborn et al., in 2019 reported a mediating role of sleep deprivation that explained the relationship between nighttime noise exposure and food intake and weight gain (Coborn et al., 2019). Similarly, sleep problems can be a consequence of depressive symptoms (Riemann et al., 2001). The depressive symptoms thus may deepen noise-related sleep deprivation even more. Sleep deprivation may influence the regulation of leptin and ghrelin, two hormones involved in metabolic processes, and therefore lead to abnormal adiposity (Bacaro et al., 2020; Van Cauter et al., 2008).

In addition, there are indirect behavioral pathways explaining the effect of noise exposure on abnormal adiposity risk. For instance, the study conducted by Foraster et al., in 2016 showed that long-term noise annoyance was associated with reduced physical activity, especially among women (Foraster et al., 2016). It is important to highlight that Foraster et al. investigated psychological noise appraisal, particularly noise annoyance, rather than objective measures of noise exposure. Noise annoyance represents the subjective evaluation of noise exposure which encompasses the unique individual susceptibility and vulnerability to noise, significantly influencing non-auditory health effects (Dratva et al., 2010). Noise annoyance, creating negative emotions and enhancing a pro-inflammatory state, was described by earlier studies as associated with depressive symptoms (Beutel et al., 2016; Eze et al., 2020; Hahad et al., 2022). Noise annoyance thus may be an element connecting the effect of noise exposure and mental condition in the effect on adiposity. The contribution of our study lies in the understanding of depression not only as the result of environmental noise exposure but rather as a moderator considering individual vulnerability to environmental noise. A similar idea was suggested in the study involving 15,501 female nurses aged 44 and older from Denmark that investigated the effect modification of job strain (as a substantial individual stressor) in the association between traffic noise exposure and adiposity biomarkers. They suggested a pronounced effect of noise exposure on BMI among those experiencing job strain (Cramer et al., 2019). Depressive symptoms and other mental health conditions undoubtedly play an important role in the health effects of noise. While our study provided support for consideration of effect modifications by depressive symptoms, there are also studies suggesting their mediation role (Hahad et al., 2022). The interconnection between diverse factors must be considered in public health strategies. Public health professionals should strive to reduce environmental noise exposure by interdisciplinary collaboration with urban planners and policymakers to develop and implement noise mitigation measures in cities. At the same time, we must still emphasize the role of mental health interventions which can help counteract the potential influence of noise on adiposity.

The main strength of our study was using objective anthropometric measures as well as clinically relevant outcomes of cardiometabolic health. Also, we included several potential confounding factors that were assessed using standardized methods. However, this study also has limitations. First, the cross-sectional design of the study does not allow us to evaluate causality, and reverse causation bias might occur as we are unable to establish the exact onset of outcomes. Although we investigated environmental noise exposure determined from environmental noise measurements conducted prior to the data collection where depressive symptoms were detected, broader data on the temporality of phenomena is not available. Thus, it is not possible to consider the onset of depressive symptoms and adiposity and define the continuity of events. We must therefore also admit the possibility of the opposite direction of the observed relationships when noise could act as a moderator of the effect of depressive symptoms on adiposity. Moreover, the lack of temporality in our data did not allow us to perform a more comprehensive analysis including an investigation of the mediation effect of depressive symptoms, as suggested in previous literature. Additionally, depressive symptoms were asked only in reference to the prior 2 weeks, thus it is unclear if the assessment represents an acute or chronic state. Second, the study sample only included a city-based population; thus, the study findings should not be generalized to other groups. Third, the environmental noise exposures were estimated only at the street (or district) level as a mean of noise exposure of each residential building, which lowers the accuracy of exposure estimates and may lead to misclassification, especially for streets or districts with higher geographical variation in noise levels. This may lead to less precise estimates of individual exposure to noise for the participants with a higher chance of personal exposure misclassification. The fourth limitation is not including the daytime (primarily occupational and social) noise and the indoor noise. Albeit especially for the nighttime noise exposure, the difference in indoor-outdoor noise estimates might not lead to a better model performance (Röösli et al., 2019), occupational/social noise is a major source of exposure, the effect of which may lead to nighttime sleep disturbance, regardless of the residential noise levels (Gitanjali and Ananth, 2003). Furthermore, additional environmental factors including air pollution and light at night exposures may modify the associations of noise exposure on adiposity biomarkers, but such data were not available. Additionally, the participants' residential history was not tracked, nor was the duration of time spent at their

current address recorded. Therefore, the long-term noise exposure assessment was based solely on one home address. Similarly, we were unable to estimate noise exposures from transportation or occupation which may lead to residual confounding. It is likely that the limitations mentioned above would lead to an underestimation of the strength of the association between noise and adiposity.

5. Conclusion

We found evidence of an association between environmental noise exposure and several adiposity measures indicating poorer health and this effect may be exacerbated by depressive symptoms. Future studies need to confirm this association and investigate deeper underlying mechanisms. Understanding the role of external environmental factors and their interactions with individual factors in abnormal adiposity development is essential for the effective targeting of public health strategies aimed at reducing the burden of abnormal adiposity.

CRediT authorship contribution statement

Anna Bartoskova Polcrova: Writing – original draft, Methodology, Formal analysis, Conceptualization. **Andrea Dalecka:** Writing – original draft. **Katarzyna Kordas:** Writing – review & editing, Supervision, Methodology. **Daniel Szabo:** Formal analysis. **Juan Pablo Gonzalez Rivas:** Writing – review & editing. **Martin Bobak:** Writing – review & editing, Supervision. **Hynek Pikhart:** Writing – review & editing, Supervision, Methodology, Conceptualization.

Data availability statement

The data that support the findings of this study are available from ICRC - FNUSA but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are however available from the authors upon reasonable request and with permission of ICRC-FNUSA.

Ethical statements

The study protocol complied with the Helsinki declaration and all participants signed the informed consent. The Kardiovize study was approved by the ethical committee of St Anne's University Hospital, Brno, Czech Republic on 13 June 2012 (reference number 2 G/2012).

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Declaration of competing interest

The authors declare that they have no competing interests.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijheh.2024.114481.

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