Association between Exposure to Noise and Air Pollution and Metabolic Syndrome

Mina Arabian (✉ m7arabian@gmail.com)

Yazd University of Medical Science: Shahid Sadoughi University of Medical Sciences and Health Services <https://orcid.org/0000-0003-1942-1975>

Masoud Mirzaei

Shahid Sadoughi University of Medical Sciences and Health Services

Mohammad Javad Zare Sakhvidi

Shahid Sadoughi University of Medical Sciences and Health Services

Sara Jambarsang

Shahid Sadoughi University of Medical Sciences and Health Services

Mohsen Mirzaei

Shahid Sadoughi University of Medical Sciences and Health Services

Research Article

Keywords: noise, noise exposure, air pollution, Particulate Matter, Metabolic Syndrome.

Posted Date: August 23rd, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-532984/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Background: In addition to the classical risk factors environmental pollution such as traffic noise and air pollution are suggested to be a risk factor for Metabolic Syndrome (MetS). In this study, we examined the between exposure to noise and air pollution (PM_{2.5}) and MetS.

Methods: This study was performed on 3513 participants in a prospective study on the health of the people of Yazd (YaHS). A wide range of demographic, anthropometric and blood biochemistry data were gathered during 2014-2015. Data on personal level noise and air pollution (PM_{2.5}) at residential address were collected through air pollution maps. Air and noise pollution modeling was performed using Kriging model. Using weighted logistic regression, we reported the odds ratio (95% confidence interval) of MetS for a unit increase in exposure to the pollutants.

Results: The prevalence of MetS in the total population was 43.7%. No association was found with MetS between noise exposures in the range (54.1-62.3) dB (A). There was a positive relationship between air pollution exposure in the range (32.82 - 16.38) micrograms per cubic meter and MetS in the raw model and after adjusting for the effect of age and sex, increased air pollution exposure, chance The incidence of MetS increases by 8% (95% CI = 1.06 - 1.10), while the fully adjusted model did not find a positive and significant relationship.

Conclusion: We did not find any association between noise and air pollution with MetS in the fully adjusted model.

Introduction

Urbanization is associated with increased exposure to air pollution and noise from traffic sources, which are suggested to be associated with increased risk of cardio metabolic disorders (Pyko et al., 2017, Hoi, 2020, Yu et al., 2020, Wang et al., 2020). Environmental pollution, including air pollution, is a significant risk factor for burden of disease and one of the most important environmental causes of death and disability (Ukaogo et al., 2020, Balakrishnan et al., 2019, Zarei Mahmoud Abadi, 2016, Jelokhani-Niaraki et al., 2018, Van Kempen et al., 2018, Vienneau et al., 2019, Münzel et al., 2017). However, environmental noise due to road traffic continues to increase, planned actions and appropriate decision-making by the government, such as the road traction priority index or quiet pavements, can be measures to deal with noise (Ruiz-Padillo et al., 2014, Ruiz-Padillo et al., 2016, Praticò and Anfosso-Lédée, 2012).

The effects of noise can be appreciated physiologically but also psychologically (annoyance And disturbed well-being such as stress) (Muzet, 2007, Erickson and Newman, 2017).

Known risk factors for Metabolic Syndrome (MetS) include age, sex, genetics, and race, ethnicity, and lifestyle factors such as physical activity, socioeconomic status. Recent studies suggested air and noise pollutants as the potential risk factors of MetS (Yu et al., 2020, Wallwork et al., 2017, Vos et al., 2017, Šoštarič et al., 2019, Saklayen, 2018).

Prolonged exposure to ambient air pollutants including particulate matter with less than 2.5 μm aerodynamic diameters could increase the risk of MetS (Hou et al., 2020). Exposure to PM with an aerodynamic diameter equal to or less than 2.5 μm (PM_{2.5}) was found to contribute to increased incidence and/or prevalence of diabetes mellitus (DM) and elevation of blood pressure (Auchincloss et al., 2008, Wu et al., 2013, Shamy et al., 2018).

Increased exposure to PM_{2.5} also causes an increase in systolic and diastolic blood pressure. On the other hand, inhalation of PM pollutants causes resistance to insulin, which is mechanically associated with high blood pressure (Clementi et al., 2019).

Mechanistically, the inhalation of air pollutants can trigger the following autonomic nervous system imbalance and insulin resistance (Rajagopalan and Brook, 2012) and epigenetic Modification (Peng et al., 2016, Wang et al., 2016, Yang et al., 2018b).

Some previous studies have shown an association between noise and diabetes, increased body mass index, hypertension, waist circumference and overweight (Sørensen et al., 2013, Christensen et al., 2015, Eriksson et al., 2014, Foraster et al., 2018, Pyko et al., 2017, Wallas et al., 2019, Cramer et al., 2019, Dratva et al., 2012).

Noise pollution can cause a stress response with activation of the hypothalamus-pituitary and adrenal glands, and ultimately increased levels of noradrenaline-adrenaline and corticosteroids, leading to sympathetic activation of the autonomic nervous system. In prolonged exposure time it can alter the metabolism, and leads to weight gain (Christensen et al., 2016, Erickson and Newman, 2017, Ising and Kruppa, 2004, Evans et al., 2001).

Previous studies reported that 99% of residential areas in Yazd are exposed to noise pollution. In addition, a combined map showed that 56% of Yazd lands are exposed to noise pollution (Gholami-Zarchi et al., 2014).

Material And Methods

2.1. Study population

The data of this study were extracted from the enrollment phase of Yazd Health Study (YaHS), the sample size of YaHS study was 9962 people aged 20 to 69 years in Yazd in 1394 – 1393, cluster random sampling was done, The profile and details of the YaHS methodology are described elsewhere (Mirzaei et al., 2018).

A standardized questionnaire was used to collect information from individuals including demographic characteristics (age, sex, level of education, income level), lifestyle habits (smoking status, physical activity) and family history of chronic diseases such as diabetes by trained people. Anthropometric indicators such as weight, height, waist circumference were measured by trained individuals For

biochemical tests, blood samples (to check fasting blood sugar levels, high-density cholesterol levels and triglyceride levels), a Pars kit calibrated by Ciba Corporation (Ciba Corp., Basel, Switzerland) were examined. Their blood pressure was measured three times with an interval of 5 minutes by trained individuals using Reister Electronic Sphygmomanometer (Champion model, Reister, Germany N- GMBH). To evaluate the levels of physical activity from the Metabolic Equivalent of Task (MET) with three categories of less than 3 (for low-intensity physical activity), 3 to 6 (for moderate-intensity physical activity), equal to and greater than 6 (for intense physical activity) was considered (Mendes et al., 2018). Body mass index was considered in three categories of less than 25 (including lean to normal people), between 25 and 30 (including overweight people), and above 30 (including obese people). Participants were weighed and recorded using an Omron BF511 digital and portable scale (Omron Inc. Nagoya, Japan) with an accuracy of 0.1 Kg. Waist size (according to the criteria of the adult treatment panel, 88 cm in women and 102 cm in men) was considered as the base number and above it was considered a risk factor. Socio-economic status (considering the level of education, employment status, marital status, housing ownership, health insurance status and the ratio of housing infrastructure to the household dimension was estimated and scored. The sum of socio-economic status scores was in the numerical range from 1 to 18, scores 1 to 9 indicated low socio-economic status, scores 10 to 14 were moderate socio-economic status, and scores 15 to 18 were considered high socio-economic status. In this study, tobacco consumption means the use of cigarettes and hookah, and was questioned and recorded in the interview. Stress index was assessed based on DASS questionnaire, participants' stress levels ranged from 0 to 7 levels were defined as normal or natural stress, ranged 8 to 9 levels as mild stress, ranged 10 to 12 levels as moderate stress, ranged 13 to 16 as people with severe stress levels, and in the range of up to 17 as people with very severe stress levels. Family history of diabetes in first-class relatives (father, mother, sister, brother, children) was also questioned by a questionnaire.

Among the participants in the YaHS project, after matching air and noise pollutant data, those who underwent blood biochemistry testing were selected for this analysis (n = 3513). The geographical location of all participants was recorded and determined using the Global Positioning System (longitude and latitude of the individual's residence in Yazd city using Garmin GPS device model MAP 78S).

2.2. Estimation of air pollution and noise

To measure noise and air pollution in different stations in the region, modeling of all points was performed from the Kriging model, a linear geo statistical interpolation method used to map the sound level (Huang et al., 2020).

Air pollutants in the present study mean particulate matter less than $2.5\mu\text{g}/\text{m}^3$. After determining the measuring points, according to data measurement program in 160 points of Yazd city in the period of 8:00 a.m. to 4:30 p.m. was performed daily in three-time intervals. Before the study, the rush hours of the city were identified and measurement was avoided during the rush hours And every day, 8 different points were measured in low traffic areas, high traffic areas, areas with air pollution, green areas (with trees), special urban areas and industrial areas.

The measuring instrument set was placed in a car and transported to the measuring points and the mass concentration of the suspended particles below $2.5 \mu\text{g}/\text{m}^3$ was estimated. The parameters related to noise pollution (in the same time period as the measurement of air pollution) were measured by the SVANTEK-POLAND sound level meter model 958 SVAN. Noise estimated as Lden.

2.3. Definition of MetS

MetS was defined by a combination of abdominal obesity, impaired fasting glucose, atherogenic dyslipidemia, and elevated blood pressure (Grundey et al., 2005, Alberti et al., 2009, Lee et al., 2020). MetS according to the criteria of the adult treatment panel (abdominal obesity $> 102 \text{ cm}$ for men and $> 88 \text{ cm}$ for women, high blood glucose level $\geq 110 \text{ mg/dL}$, high blood pressure $\geq 130/85 \text{ mmHg}$, hypertriglyceridemia $\geq 150 \text{ mg/dL}$ and low levels of High-Density Lipoprotein Cholesterol (HDL-C) $< 40 \text{ mg/dL}$ for men and $< 50 \text{ mg/dL}$ for women) were assessed. Those participants who had more than two out of five indicators were considered to have MetS (Samson and Garber, 2014).

2.4. Covariates

To collect information, a standard questionnaire was used about potential confounders, which were selected a priori. The following variables include: age group, gender (male / female), employment status (employed / housewife / unemployed), level of education (literate / illiterate), socio-economic status (low / medium / high), Body mass index (lean to normal / overweight / obese) and smoking status (smoker / non-smoker / hookah user / non-hookah user).

2.5. Statistical analysis

Associations between traffic exposures (noise and air pollution) and MetS were analyzed using binary logistic regression (into GWR4 software environment and geographical weight regression analysis was performed, (GWR) was applied to analyze the varying relationship between variables across the different parts of the study area. The technique allows to conduct local regressions that contain local parameter estimates as disaggregation of the ordinary/global regression that contain global parameter estimates. It is applied when the measurement of a relationship depends on the location where the

measurement is taken, and also GWR has been widely applied in urban planning (Fotheringham et al., 2003, Gao and Li, 2011, Pribadi and Pauleit, 2016)) in three adjustment models: In the crude model, the relationship between MetS and pollutants (for 1 dB increase of noise pollutants and 1 microgram per cubic meter of air pollutants) was investigated, This relationship was then examined by adjusting the effect of age and sex. Finally, the relationship with the adjusted of other confounding variables, which were socioeconomic status, marital status and employment, education level, insurance and housing status, blood pressure, physical activity, BMI, waist circumference, smoking, family history of diabetes and stress levels Checked out. Then in exchange for increased exposure to noise and air pollution by 5 dB and $10 \mu\text{g}/\text{m}^3$, respectively, the chance of developing MetS was evaluated and expressed with 95% confidence in the form of odds ratio

The data set related to the participants was entered into SPSS.20 software, the p-value less than 0.05 was considered statistically significant. This study has been approved by the ethics committee of Yazd University of Medical Sciences with the code IR.SSU.SPH.REC.1398.089.

Findings

Descriptive

Based on the main definition of MetS, the prevalence of MetS in the total population was 43.7%. The mean age of participants with MetS was higher than participants without MetS and among people with increase in BMI, rising unemployment rate and in people with moderate economic status. The basic characteristics of participants in the study, in terms of with and without MetS are given in Table 1.

Table 1
Distribution of Main characteristics of study participants with and without MetS.

Variable	Subgroup	N = 3513	MetS (n = 1538)	Non-MetS (n=	p-value
age category (Year N (%))	29 – 20	521(14.8)	(12.5%)65	(87.5%)456	< 0.0001
	39 – 30	(17.2)606	(25.8%)157	(74.2%)449	
	49 – 40	785(22.3)	(44.4%)349	(55.6%)436	
	59 – 50	)80623)	(56.1%)452	(43.9%)354	
	69 – 60	)79522.7)	(64.1%)515	(35.9%)280	
Gender N (%)	Male	(46.4)1555	(38.7%)602	(61.3%)953	< 0.0001
	Female	(53.6)1799	(48.1%)865	(51.9%)934	
Employment status N(%)	Employed	(35.7)1180	(34.7%)410	(65.3%)770	< 0.0001
	housewife	(43.8)1446	(45.7%)718	(54.3%)728	
	Unemployed	(20.5)676	(49.6%)309	(50.4%)367	
Education level N(%)	illiterate	(28.9)969	(61.7%)598	(38.3%)371	< 0.0001
	Literate	(71.1)2385	(36.4%)869	(63.6%)1516	
Socio-economic status N(%)	Down	(39.9)1236	(49.1%)607	(50.9%)629	< 0.0001
	medium	(55.1)1703	(40.4%)687	(59.6%)1016	
	Top	(5)155	(38.7%)60	(61.3%)95	
BMI N(%)	Slim to normal	(31.8)1060	(17.8%)189	(82.2%)871	< 0.0001
	Overweight	(40)1334	(40%)623	(53.3%)711	
	Fat	(28.2)942	(68.7%)648	(31.3%)294	
Smoking status	Yes	(6.4)213	(39.9%)85	(60.1%)128	< 0.244
Cigarettes	No	3141(93.6)	1382(%43.9)	1759(%56.1)	
Smoking status	Yes	102(3.1)	28(%1.9)	74(%3.9)	< 0.001
Hookah	No	3252(96.9)	1439(%98.1)	1813(%96.1)	
Fasting Blood Sugar Mean ± SD	mg/dL110≤	3513(100)	120.74 ± 46.65	96.16 ± 22.41	< 0.0001
Systolic blood pressure Mean ± SD	0mmHg13≤	3513(100)	138.43 ± 18.52	119.59 ± 14.05	< 0.0001

*: Mean ± SD

Variable	Subgroup	N = 3513	MetS (n = 1538)	Non-MetS (n=)	p-value
Diastolic blood pressure Mean ± SD	mmHg58≤	3513(100)	86.34 ± 12.16	76.55 ± 10.90	< 0.0001
High Density Lipoprotein Mean ± SD	40mg/dL(Femle) < Men))mg/dL50<	3513(100)	46.10 ± 12.83	51.55 ± 13.37	< 0.0001
Triglyceride Mean ± SD	mg/dL015≤	3513(100)	206.1 ± 112.55	125.07 ± 78.51	< 0.0001
Waist circumference Mean ± SD	88cm(Femle)< Men)) 102cm<	3513(100)	101.5 ± 11.31	89.68 ± 12.29	< 0.0001
Noise Pollution Mean ± SD	dB55< (54.10–62.30)	3513(100)	*57.24 ± 2.16	*57.19 ± 2.03	< 0.0001
Air pollution Mean ± SD	10 µg/m ³ < (16.32–38.82)	3513(100)	*25.73 ± 4.16	*25.66 ± 4.46	< 0.0001
*: Mean ± SD					

In this study, the exposure to noise pollution was in the range of 54.10 to 62.30 dB (A) and the average exposure to noise pollution in the whole population was 57.21 ± 2.08 dB (A). The exposure to air pollution was evaluated in the range of 16.32 to 38.82 µg/m³. The average exposure to air pollution in the whole population was 25.69 ± 4.33 (µg/m³). A higher percentage of patients with MetS was seen in women, the mean exposure to noise pollution in people with and without MetS was not significantly different between the two sexes and the average exposure to air pollution in people with and without MetS was estimated to be close to each other. In the stratified analysis by potential modifiers, the OR was MetS.

Association of Noise and Air pollution

No significant association with MetS was observed with an increase of 1dB noise pollutant. There was no significant relationship with MetS by adjusting the effect of age and sex variables for increase of 1dB noise pollution. Also after adjusting the effect of other confounding variables for increase of 1dB noise pollution, there was no significant relationship between MetS and noise pollution. With an increase of 1µg/m³ air pollution, the chance of developing MetS at the 5% level is 1.08 times. By adjusting the effect of age and sex, with an increase of 1µg/m³ air pollution, the chance of developing MetS at the level of 5% is 1.08 times. By adjusting the effect of other confounding variables for an increase of 1µg/m³ air pollution, no significant relationship was observed between MetS and air pollution, this could be due to the presence of over adjustment. It is worth mentioning that regarding noise pollution in Yazd city,

considering that most of the participants in the present study were in the range above 55 dB of noise intensity (base range for noise pollution index) and based on the subject, noise pollution is quantitative. In order to investigate the mentioned relationship, the relationship between total sound intensity and MetS was considered and therefore we did not have a division higher or lower than 55 dB in the analysis section.

Refer to Table 2 to investigate the relationship between MetS and exposure to noise and air pollution.

Table 2

Relationship between MetS for increasing 1 dB of noise and 1 $\mu\text{g}/\text{m}^3$ of air pollution (PM_{2.5} index).

Risk Factors (Dependent Variable / Independent variable)		Confidence Interval (Odds Ratio)
MetS		
Crude Model	Noise Pollution	1.01 (0.97: 1.05)
	Air Pollution	*1.08 (1.06: 1.10)
Model Adjusted to Age and Sex	Noise Pollution	1.01 (0.97: 1.05)
	Air Pollution	*1.08 (1.05: 1.11)
Adjusted Model with other Confounding Factors	Noise Pollution	0.99 (0.98: 1.02)
	Air Pollution	1.01 (0.97: 1.05)

- Odds ratio values estimated using the logistic model
- Other confounding factors include socioeconomic status, marital status and employment, education level, insurance and housing status, blood pressure, physical activity, BMI, waist circumference, smoking, family history of diabetes and stress levels.

Discussion

Regarding noise pollution with MetS, the findings show that the concentration of noise pollutants in Yazd is higher than those mentioned in the WHO guidelines (55dB [A]). The chance of developing MetS increases with age. The mean exposure to noise pollution in patients with and without MetS was estimated to be close to each other. In the present study, the relationship between noise and air pollution and MetS was investigated. At the level of exposure to noise pollution, there was no significant relationship with the prevalence of MetS. According to our findings, this is the first study to evaluate the relationship between noise pollution and MetS in Yazd. The results of some studies support our findings, in a cross-sectional study of 424 industrial workers with an average noise exposure of 79.9dB (A), exposure to workplace noise, has sharply associated with increased triglyceride levels, cholesterol ratio to

HDL, and lower HDL cholesterol levels. After modifying the effect of potential factors, there was no correlation between occupational noise exposure and serum fat levels (Arlie-Søborg et al., 2016); similarly, in a cohort study, there was a positive correlation between components of MetS (Hypertriglyceridemia) with noise exposure whilst this association was not seen with abdominal obesity (Yu et al., 2020). In another study, noise exposure was associated with significant increase in the risks of hypertriglyceridemia, abdominal obesity, and hyperglycemia while the risk ratio for noise on hypertension and low HDL cholesterol was lower and not statistically significant. Correlation was found to be periodically stronger among people exposed to moderate or high noise than in people exposed to low noise (Huang et al., 2020), in another cohort study, no increase was seen in the risk of exposure to the air traffic noise and the incidence of diabetes (Eriksson et al., 2014); similarly, in another cohort study associated with enrollment, no risk of hypertension was seen with exposure to noise in the lower half of range of 80–90 dB (A) (Stokholm et al., 2013), in several other studies no increased risk of hypertension related to exposure to traffic noise was found (Sørensen et al., 2011, Sørensen et al., 2012, Eriksson et al., 2010), also in a cross-sectional study of road traffic noise and fat markers in a Danish nurse group, no positive association was found between exposure to road traffic noise and BMI (Cramer et al., 2019).

On the other hand, there are a number of studies indicating opposite results showing a significant association between environmental noise exposure and lipid markers (Pyko et al., 2017, Oftedal et al., 2015, Eriksson et al., 2014, Pyko et al., 2015), and a number of studies have shown a significant association between noise exposure and hyperglycemia (Dendup et al., 2018, Wang et al., 2020, Dzhambov, 2015, Sørensen et al., 2013), also several other studies have found a positive association between exposure to noise and high blood pressure (Van Poll et al., 2014, Oftedal et al., 2015, Chang et al., 2014, Dratva et al., 2012, Liu et al., 2014); as well as, several cross-sectional studies have shown significant positive relationship between road traffic noise, waist circumference, and BMI. Road traffic noise was also associated with a higher prevalence of overweight and obesity (Christensen et al., 2016, Christensen et al., 2015, Pyko et al., 2015, Oftedal et al., 2015, Foraster et al., 2018, Pyko et al., 2017, Eriksson et al., 2014).

In our study, for every 5 dB increase in exposure to noise pollution, the chance of developing MetS was assessed and there was no significant change and no significant correlation was seen; similarly, no correlation was observed between road traffic noise in the fully adjusted model (in every 10 dB increase in a 1-year average of Lden) and BMI (Cramer et al., 2019). In another study, a positive correlation was found between rail noise exposure > 60 dB (vs. 0–20 dB) and BMI, waist circumference (Christensen et al., 2016). In another study, exposure to higher levels of noise per day was positively associated with higher triglycerides (Cai et al., 2017), also a cohort study showed that by increasing noise to 11.6 dB, the level of High-Density Lipoprotein Cholesterol (HDL-C) decreases (HR = 1.17, 95% CI: 1.01–1.35) and the risk of MetS increases by 17% (Yu et al., 2020); moreover, a cohort study showed that a 10-dB higher level of average road traffic noise at diagnosis and during the 5 years preceding diagnosis was associated with an increased risk of incident diabetes and also this association was slightly stronger with long-term exposure (5 years) compared to short-term exposure (1 year) (Sørensen et al., 2013). Similarly, in several other studies, exposure to higher levels of noise was associated with a higher risk of hyperglycemia (Eze

et al., 2017, Ohlwein et al., 2019, Chang et al., 2020, Clark et al., 2017, Sakhvidi et al., 2018, Van Poll et al., 2014). In another study, the strongest correlation between the effect of noise and hypertension at 4 kHz was observed. An increase in 20-dB exposure to noise at 4 kHz was associated with a 34% risk of hypertension (Liu et al., 2016). Similarly, in several other studies, the increased risk of hypertension increased with increasing exposure to noise pollution (Van Kempen and Babisch, 2012, Fuks et al., 2017, Lin et al., 2020).

We also examined the potential effects of some potential confounders, with no significant effect on correction for age, sex, socioeconomic status, marital and employment status, education, insurance and housing status, blood pressure, physical activity, BMI, waist circumference, smoking, family history of diabetes, and stress levels; similarly, in another study, there was no correlation between road traffic noise and BMI in correcting the effect of age (Christensen et al., 2016). In one study, the effects of road traffic noise on type 2 diabetes in the age group over 60 years or older showed more severe effects whereas no association was found between road traffic noise and diabetes and education (Sørensen et al., 2013). In some age-modifying studies, the association between noise exposure and hypertension is significant and is more likely in the age group under (Pyko et al., 2015). A Norwegian cross-sectional study showed no significant association between road traffic noise and obesity markers in the general population; however, there is a positive association between noise-sensitive women (Ofstedal et al., 2015). Also, in another study, the association of road noise with abdominal obesity was seen only among women (Pyko et al., 2015). In another study, the association between road traffic noise and diabetes was stronger among women than men (Sørensen et al., 2013); whilst in another study, there was association between noise and increased triglyceride levels, cholesterol to HDL ratio, decrease in HDL cholesterol with male gender, smoking, low level of education, low personal income, waist circumference, as well as BMI (Arlie-Søborg et al., 2016); several studies have also shown that exposure to road traffic noise may be significantly associated with BMI or waist circumference of individuals with specific allergies, such as those working under stress or living in urban areas (Cramer et al., 2019, Eriksson et al., 2014, Selander et al., 2013).

Regarding the relationship between air pollution and MetS, the findings showed that the concentration of air pollutants ($PM_{2.5}$ index) in Yazd is higher than those mentioned in the WHO guidelines ($10 \mu g/m^3$). The median exposure to air pollution in individuals with or without MetS was estimated to be close to each other. In the present study, no positive and significant relationship was observed between exposure to air pollution ($PM_{2.5}$ index) and the possibility of MetS. According to our findings, this is the first study to evaluate the relationship between air pollution and MetS in Yazd. Our study provides evidence that $PM_{2.5}$ is not associated with MetS or components of the MetS. Similarly the results of a cross-sectional study showed no significant relationship between air pollution ($PM_{2.5}$ and black carbon) with blood lipids or waist circumference (Rajkumar et al., 2019). One study in Taiwan reported no association between air pollution and triglycerides or HDL, while a positive association was found between total cholesterol and $PM_{2.5}$ (Chuang et al., 2011). Several other studies found no association between exposure to air pollution and incidence of diabetes (Coogan et al., 2012, Puett et al., 2011).

Whereas the results of several epidemiological studies have shown that prolonged exposure to ambient air pollutants increased the risk of MetS (Hou et al., 2020, Matthiessen et al., 2018, Wallwork et al., 2017, Yang et al., 2018a). Some studies have presented results related to MetS. In a study of life closer to a main road and exposure to the 1-year average level of particulate matter (PM_{2.5}) in the air was associated with an increased likelihood of overall obesity and abdominal obesity (increased BMI) (Li et al., 2016). Some experimental studies also provided evidence of a possible association of exposure to higher PM_{2.5} which leads to the induction of lipid metabolic disorders and an increased risk of obesity and MetS (Wei et al., 2016, Wu et al., 2019, Mendez et al., 2013, Sun et al., 2009). A Danish cross-sectional study showed with an increase in IQR in PM_{2.5} environment, increased 0.78 (0.22, 1.34) mg/dL in total cholesterol (Sørensen et al., 2015). A retrospective cohort indicated that higher LDL and triglycerides as well as lower HDL associated with higher PM_{2.5} concentrations (Yitshak Sade et al., 2016). Another study showed that short-term exposure to PM_{2.5} accelerated the progression of systemic insulin resistance and affected fat metabolism (Haberzettl et al., 2016). In some studies, short-term or long-term exposure to air pollution was associated with an increased risk of hypertension (Giorgini et al., 2016, Wu et al., 2013, Dong et al., 2013, Chen et al., 2014). Another study indicated exposure to air pollutants, especially traffic-related pollutants, may increase the risk of type 2 diabetes and hypertension (Coogan et al., 2012, Brook and Kousha, 2015, Brook et al., 2016, Eze et al., 2015, Liu et al., 2014, Ying et al., 2014, Fuks et al., 2017, Weinmayr et al., 2015, Thiering et al., 2013). In another study among non-diabetics, short- and medium-term exposure to PM_{2.5} was associated with increased blood sugar (Peng et al., 2016).

In our study, for an increase in 10 µg/m³ exposure to air pollution, the chance of developing MetS was assessed and no significant change and correlation was observed. Similarly, in another study after adjusting potential disruptors, an increase in 1-year mean of air pollutants (PM_{2.5}) led to an increase in systolic blood pressure, diastolic blood pressure, total cholesterol, triglyceride, HDL-C, fasting glucose, hemoglobin A1c (Chuang et al., 2011). Another study showed that increasing 10 µg/m³ of PM_{2.5}, significantly associated with the prevalence of MetS, hyperglycemia and hypertension (Shamy et al., 2018). An increase of 1 µg/m³ at an average annual concentration of PM_{2.5} was associated with a 27% increase in the risk of MetS in 587 elderly men (Wallwork et al., 2017). A cross-sectional study also showed that an increase of 10 µg/m³ of PM_{2.5}, was associated with a 9% increased risk for MetS in Chinese adults (Yang et al., 2018a). In a study with an increase of 10 µg/m³ of PM_{2.5}, the risk ratio of diabetes increased (Chen et al., 2013). In another study with an increase of 10 µg/m³ of PM_{2.5}, the risk ratio of diabetes and blood pressure increased (Coogan et al., 2012).). Similarly, in another study with an increase of 5 µg/m³ of PM_{2.5}, a 22% increased risk of self-report hypertension was observed (Fuks et al., 2017).

We also examined the potential effects of some potential confounders, with no significant effect on correction for age, sex, socioeconomic status, marital and employment status, education, insurance and housing status, blood pressure, physical activity, BMI, waist circumference, smoking, family history of diabetes, or stress levels. Similarly, in another study, long-term exposure to air pollution and diabetes was

not significantly associated with correction of gender and education (Eze et al., 2014), whilst in another study the association between air pollution and diabetes was stronger among women than men (Chen et al., 2013), in another study men residents of areas with PM_{2.5} or temperatures higher than the average level associated with an increased risk of metabolic disorders (Wallwork et al., 2017). In another study, there was evidence of a stronger effect in women ≥ 40 years old than women < 40 years old for association of pollution of PM_{2.5} with MetS (Rajkumar et al., 2019), while the findings of a study showed that in men, the elderly, and those who had an unhealthy lifestyle (smoking, drinking, not exercising regularly, as well as consuming more sweet soft drinks, high-calorie, and high-fat foods), and low-income people, the relation between ambient air pollution and MetS was very high and they may become more vulnerable (Yang et al., 2018a). Another study showed that physical activity reduced the effects of ambient air pollutants in increasing the risk of MetS (Hou et al., 2020); similarly, Kubesch et al. indicated that physical activity has potentially a preventive effect against the negative impact of air pollution on systolic blood pressure (Kubesch et al., 2015).

Limitations

In this study, only one serum sample from each participant was collected, and serum lipids as well as blood sugar levels may vary between days (Arlie-Søborg et al., 2016). Also, measurements were taken over a relatively short period of time, and various measurement factors included and may cause defects in the assessment.

Conclusion

We did not find any association between noise and air pollution and MetS in the fully adjusted model. In the present study, the average air pollution (PM_{2.5} index) and noise pollution at most sites measured is higher than the annual level recommended by the World Health Organization (Who, 2006), but generally in all cases of exposure to pollution the sound was less than 65 dBs. According to other studies, more amounts of pollutants may be needed as a risk factor for MetS compared to amounts of pollutants measured in the current study.

Declarations

Ethical Approval :Ethics code IR.SSU.SPH.REC.1398.089 is related to the article " Association between exposure to noise and air pollution and Metabolic Syndrome "of the dissertation approved in the Department of Statistics and Epidemiology of the Faculty of Health, related to Shahid Sadoughi University of Medical Sciences. The manuscript should not be submitted to more than one journal for simultaneous consideration. This manuscript is original and has not been published elsewhere in any form or language (partially or in full).

Consent to Participate: The data of this article are taken from the study of YaHS cohort and this study has a code of ethics From the ethics committee of Shahid Sadoughi University of Medical Sciences, No. 17/1/73941.

Consent to Publish: All authors agree to publish this article in your journal (Environmental Science and Pollution Research).

Authors Contributions : All of the authors have read and approved the paper and it has not been published previously nor is it being considered by any other peer-reviewed journal.

Funding: We did not use financial resources.

Competing Interests: 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.

Availability of data and materials: All information is ready to be sent.

Contributions: Mina Arabian, first author. Masoud Mirzaei, correspond author. Mohammad Javad Zare Sakhvidi, data collector. Sara Jambarsang, data analyzer. Mohsen Mirzaei editor and reviewer.

References

1. ALBERTI, K., ECKEL, R. H., GRUNDY, S. M., ZIMMET, P. Z., CLEEMAN, J. I., DONATO, K. A., FRUCHART, J.-C., JAMES, W. P. T., LORIA, C. M. & SMITH JR, S. C. 2009. Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. *Circulation*, 120, 1640-1645.
2. ARLIEN-SØBORG, M. C., SCHMEDES, A. S., STOKHOLM, Z., GRYNDERUP, M., BONDE, J., JENSEN, C., HANSEN, Å., FREDERIKSEN, T., KRISTIANSEN, J. & CHRISTENSEN, K. 2016. Ambient and at-the-ear occupational noise exposure and serum lipid levels. *International archives of occupational and environmental health*, 89, 1087-1093.
3. AUCHINCLOSS, A. H., DIEZ ROUX, A. V., DVONCH, J. T., BROWN, P. L., BARR, R. G., DAVIGLUS, M. L., GOFF JR, D. C., KAUFMAN, J. D. & O'NEILL, M. S. 2008. Associations between recent exposure to ambient fine particulate matter and blood pressure in the Multi-Ethnic Study of Atherosclerosis (MESA). *Environmental health perspectives*, 116, 486-491.
4. BALAKRISHNAN, K., DEY, S., GUPTA, T., DHALIWAL, R., BRAUER, M., COHEN, A. J., STANAWAY, J. D., BEIG, G., JOSHI, T. K. & AGGARWAL, A. N. 2019. The impact of air pollution on deaths, disease burden, and life expectancy across the states of India: the Global Burden of Disease Study 2017. *The Lancet Planetary Health*, 3, e26-e39.

5. BROOK, R. D. & KOUSHA, T. 2015. Air pollution and emergency department visits for hypertension in Edmonton and Calgary, Canada: a case-crossover study. *American journal of hypertension*, 28, 1121-1126.
6. BROOK, R. D., SUN, Z., BROOK, J. R., ZHAO, X., RUAN, Y., YAN, J., MUKHERJEE, B., RAO, X., DUAN, F. & SUN, L. 2016. Extreme air pollution conditions adversely affect blood pressure and insulin resistance: the air pollution and cardiometabolic disease study. *Hypertension*, 67, 77-85.
7. CAI, Y., HANSELL, A. L., BLANGIARDO, M., BURTON, P. R., BIOSHARE, DE HOOGH, K., DOIRON, D., FORTIER, I., GULLIVER, J. & HVEEM, K. 2017. Long-term exposure to road traffic noise, ambient air pollution, and cardiovascular risk factors in the HUNT and lifelines cohorts. *European heart journal*, 38, 2290-2296.
8. CHANG, T.-Y., BEELEN, R., LI, S.-F., CHEN, T.-I., LIN, Y.-J., BAO, B.-Y. & LIU, C.-S. 2014. Road traffic noise frequency and prevalent hypertension in Taichung, Taiwan: a cross-sectional study. *Environmental Health*, 13, 37.
9. CHANG, T.-Y., YU, T.-Y., LIU, C.-S., YOUNG, L.-H. & BAO, B.-Y. 2020. occupational noise exposure and its association with incident hyperglycaemia: a retrospective cohort study. *Scientific Reports*, 10, 1-10.
10. CHEN, H., BURNETT, R. T., KWONG, J. C., VILLENEUVE, P. J., GOLDBERG, M. S., BROOK, R. D., VAN DONKELAAR, A., JERRETT, M., MARTIN, R. V. & BROOK, J. R. 2013. Risk of incident diabetes in relation to long-term exposure to fine particulate matter in Ontario, Canada. *Environmental health perspectives*, 121, 804-810.
11. CHEN, H., BURNETT, R. T., KWONG, J. C., VILLENEUVE, P. J., GOLDBERG, M. S., BROOK, R. D., VAN DONKELAAR, A., JERRETT, M., MARTIN, R. V. & KOPP, A. 2014. Spatial association between ambient fine particulate matter and incident hypertension. *Circulation*, 129, 562-569.
12. CHRISTENSEN, J. S., RAASCHOU-NIELSEN, O., TJØNNELAND, A., NORDSBORG, R. B., JENSEN, S. S., SØRENSEN, T. I. & SØRENSEN, M. 2015. Long-term exposure to residential traffic noise and changes in body weight and waist circumference: a cohort study. *Environmental research*, 143, 154-161.
13. CHRISTENSEN, J. S., RAASCHOU-NIELSEN, O., TJØNNELAND, A., OVERVAD, K., NORDSBORG, R. B., KETZEL, M., SØRENSEN, T. I. & SØRENSEN, M. 2016. Road traffic and railway noise exposures and adiposity in adults: a cross-sectional analysis of the danish diet, cancer, and health cohort. *Environmental health perspectives*, 124, 329-335.
14. CHUANG, K.-J., YAN, Y.-H., CHIU, S.-Y. & CHENG, T.-J. 2011. Long-term air pollution exposure and risk factors for cardiovascular diseases among the elderly in Taiwan. *Occupational and environmental medicine*, 68, 64-68.
15. CLARK, C., SBIHI, H., TAMBURIC, L., BRAUER, M., FRANK, L. D. & DAVIES, H. W. 2017. Association of long-term exposure to transportation noise and traffic-related air pollution with the incidence of diabetes: a prospective cohort study. *Environmental health perspectives*, 125, 087025.
16. CLEMENTI, E. A., TALUSAN, A., VAIDYANATHAN, S., VEERAPPAN, A., MIKHAIL, M., OSTROFSKY, D., CROWLEY, G., KIM, J. S., KWON, S. & NOLAN, A. 2019. Metabolic syndrome and air pollution: a narrative review of their cardiopulmonary effects. *Toxics*, 7, 6.

17. COOGAN, P. F., WHITE, L. F., JERRETT, M., BROOK, R. D., SU, J. G., SETO, E., BURNETT, R., PALMER, J. R. & ROSENBERG, L. 2012. Air pollution and incidence of hypertension and diabetes mellitus in black women living in Los Angeles. *Circulation*, 125, 767-772.
18. CRAMER, J., JØRGENSEN, J. T., SØRENSEN, M., BACKALARZ, C., LAURSEN, J. E., KETZEL, M., HERTEL, O., JENSEN, S. S., SIMONSEN, M. K. & BRÄUNER, E. V. 2019. Road traffic noise and markers of adiposity in the Danish Nurse Cohort: A cross-sectional study. *Environmental research*, 172, 502-510.
19. DENDUP, T., FENG, X., CLINGAN, S. & ASTELL-BURT, T. 2018. Environmental risk factors for developing type 2 diabetes mellitus: a systematic review. *International journal of environmental research and public health*, 15, 78.
20. DONG, G.-H., QIAN, Z., XAVERIUS, P. K., TREVATHAN, E., MAALOUF, S., PARKER, J., YANG, L., LIU, M.-M., WANG, D. & REN, W.-H. 2013. Association between long-term air pollution and increased blood pressure and hypertension in China. *Hypertension*, 61, 578-584.
21. DRATVA, J., PHULERIA, H. C., FORASTER, M., GASPOZ, J.-M., KEIDEL, D., KÜNZLI, N., LIU, L.-J. S., PONS, M., ZEMP, E. & GERBASE, M. W. 2012. Transportation noise and blood pressure in a population-based sample of adults. *Environmental health perspectives*, 120, 50-55.
22. DZHAMBOV, A. M. 2015. Long-term noise exposure and the risk for type 2 diabetes: a meta-analysis. *Noise & health*, 17, 23.
23. ERICKSON, L. C. & NEWMAN, R. S. 2017. Influences of background noise on infants and children. *Current directions in psychological science*, 26, 451-457.
24. ERIKSSON, C., BLUHM, G., HILDING, A., ÖSTENSON, C.-G. & PERSHAGEN, G. 2010. Aircraft noise and incidence of hypertension—gender specific effects. *Environmental research*, 110, 764-772.
25. ERIKSSON, C., HILDING, A., PYKO, A., BLUHM, G., PERSHAGEN, G. & ÖSTENSON, C.-G. 2014. Long-term aircraft noise exposure and body mass index, waist circumference, and type 2 diabetes: a prospective study. *Environmental health perspectives*, 122, 687-694.
26. EVANS, G. W., LERCHER, P., MEIS, M., ISING, H. & KOFLER, W. W. 2001. Community noise exposure and stress in children. *The Journal of the Acoustical Society of America*, 109, 1023-1027.
27. EZE, I. C., FORASTER, M., SCHAFFNER, E., VIENNEAU, D., HÉRITIER, H., RUDZIK, F., THIESSE, L., PIEREN, R., IMBODEN, M. & VON ECKARDSTEIN, A. 2017. Long-term exposure to transportation noise and air pollution in relation to incident diabetes in the SAPALDIA study. *International journal of epidemiology*, 46, 1115-1125.
28. EZE, I. C., SCHAFFNER, E., FISCHER, E., SCHIKOWSKI, T., ADAM, M., IMBODEN, M., TSAI, M., CARBALLO, D., VON ECKARDSTEIN, A. & KÜNZLI, N. 2014. Long-term air pollution exposure and diabetes in a population-based Swiss cohort. *Environment international*, 70, 95-105.
29. EZE, I. C., SCHAFFNER, E., FORASTER, M., IMBODEN, M., VON ECKARDSTEIN, A., GERBASE, M. W., ROTHE, T., ROCHAT, T., KÜNZLI, N. & SCHINDLER, C. 2015. Long-term exposure to ambient air pollution and metabolic syndrome in adults. *PloS one*, 10, e0130337.

30. FORASTER, M., EZE, I. C., VIENNEAU, D., SCHAFFNER, E., JEONG, A., HÉRITIER, H., RUDZIK, F., THIESSE, L., PIEREN, R. & BRINK, M. 2018. Long-term exposure to transportation noise and its association with adiposity markers and development of obesity. *Environment international*, 121, 879-889.
31. FOTHERINGHAM, A. S., BRUNSDON, C. & CHARLTON, M. 2003. *Geographically weighted regression: the analysis of spatially varying relationships*, John Wiley & Sons.
32. FUKS, K. B., WEINMAYR, G., BASAGAÑA, X., GRUZIEVA, O., HAMPEL, R., OFTEDAL, B., SØRENSEN, M., WOLF, K., AAMODT, G. & AASVANG, G. M. 2017. Long-term exposure to ambient air pollution and traffic noise and incident hypertension in seven cohorts of the European study of cohorts for air pollution effects (ESCAPE). *European heart journal*, 38, 983-990.
33. GAO, J. & LI, S. 2011. Detecting spatially non-stationary and scale-dependent relationships between urban landscape fragmentation and related factors using Geographically Weighted Regression. *Applied Geography*, 31, 292-302.
34. GHOLAMI-ZARCHI, M., KHOSRAVANI, L. & SHAHMORADI, B. 2014. Noise pollution and its spatial distribution in urban environments (case study: Yazd city, Iran). *Journal of Advances in Environmental Health Research*, 2, 135-141.
35. GIORGINI, P., DI GIOSIA, P., GRASSI, D., RUBENFIRE, M., D BROOK, R. & FERRI, C. 2016. Air pollution exposure and blood pressure: an updated review of the literature. *Current pharmaceutical design*, 22, 28-51.
36. GRUNDY, S. M., CLEEMAN, J. I., DANIELS, S. R., DONATO, K. A., ECKEL, R. H., FRANKLIN, B. A., GORDON, D. J., KRAUSS, R. M., SAVAGE, P. J. & SMITH JR, S. C. 2005. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*, 112, 2735-2752.
37. HABERZETTL, P., O'TOOLE, T. E., BHATNAGAR, A. & CONKLIN, D. J. 2016. Exposure to fine particulate air pollution causes vascular insulin resistance by inducing pulmonary oxidative stress. *Environmental health perspectives*, 124, 1830-1839.
38. HOI, H. T. Impacts of Urbanization on the Environment of Ho Chi Minh City. IOP Conference Series: Earth and Environmental Science, 2020. IOP Publishing, 012035.
39. HOU, J., LIU, X., TU, R., DONG, X., ZHAI, Z., MAO, Z., HUO, W., CHEN, G., XIANG, H. & GUO, Y. 2020. Long-term exposure to ambient air pollution attenuated the association of physical activity with metabolic syndrome in rural Chinese adults: A cross-sectional study. *Environment International*, 136, 105459.
40. HUANG, T., CHAN, T.-C., HUANG, Y.-J. & PAN, W.-C. 2020. The Association between Noise Exposure and Metabolic Syndrome: A Longitudinal Cohort Study in Taiwan. *International journal of environmental research and public health*, 17, 4236.
41. ISING, H. & KRUPPA, B. 2004. Health effects caused by noise: evidence in the literature from the past 25 years. *Noise and Health*, 6, 5.

42. JELOKHANI-NIARAKI, M., HAJILOO, F. & HASANZADE, L. 2018. The use of geographic information systems (GIS) for assessing noise pollution in industrial workplaces: a case study of knitting industry. *Iranian Journal of Health and Environment*, 11, 377-390.
43. KUBESCH, N., DE NAZELLE, A., GUERRA, S., WESTERDAHL, D., MARTÍNEZ, D., BOUSO, L., CARRASCO-TURIGAS, G., HOFFMANN, B. & NIEUWENHUIJSEN, M. 2015. Arterial blood pressure responses to short-term exposure to low and high traffic-related air pollution with and without moderate physical activity. *European Journal of Preventive Cardiology*, 22, 548-557.
44. LEE, M.-K., HAN, K., KIM, M. K., KOH, E. S., KIM, E. S., NAM, G. E. & KWON, H.-S. 2020. Changes in metabolic syndrome and its components and the risk of type 2 diabetes: a nationwide cohort study. *Scientific reports*, 10, 1-8.
45. LI, W., DORANS, K. S., WILKER, E. H., RICE, M. B., SCHWARTZ, J., COULL, B. A., KOUTRAKIS, P., GOLD, D. R., FOX, C. S. & MITTLEMAN, M. A. 2016. Residential proximity to major roadways, fine particulate matter, and adiposity: The framingham heart study. *Obesity*, 24, 2593-2599.
46. LIN, Y.-T., CHEN, T.-W., CHANG, Y.-C., CHEN, M.-L. & HWANG, B.-F. 2020. Relationship between time-varying exposure to occupational noise and incident hypertension: A prospective cohort study. *International Journal of Hygiene and Environmental Health*, 226, 113487.
47. LIU, C., FONKEN, L. K., WANG, A., MAISEYEU, A., BAI, Y., WANG, T.-Y., MAURYA, S., KO, Y.-A., PERIASAMY, M. & DVONCH, T. 2014. Central IKK β inhibition prevents air pollution mediated peripheral inflammation and exaggeration of type II diabetes. *Particle and fibre toxicology*, 11, 53.
48. MATTHIESSEN, C., LUCHT, S., HENNIG, F., OHLWEIN, S., JAKOBS, H., JÖCKEL, K.-H., MOEBUS, S., HOFFMANN, B. & GROUP, H. N. R. S. I. 2018. Long-term exposure to airborne particulate matter and NO₂ and prevalent and incident metabolic syndrome—Results from the Heinz Nixdorf Recall Study. *Environment international*, 116, 74-82.
49. MENDES, M. D. A., DA SILVA, I., RAMIRES, V., REICHERT, F., MARTINS, R., FERREIRA, R. & TOMASI, E. 2018. Metabolic equivalent of task (METs) thresholds as an indicator of physical activity intensity. *PloS one*, 13, e0200701.
50. MENDEZ, R., ZHENG, Z., FAN, Z., RAJAGOPALAN, S., SUN, Q. & ZHANG, K. 2013. Exposure to fine airborne particulate matter induces macrophage infiltration, unfolded protein response, and lipid deposition in white adipose tissue. *American journal of translational research*, 5, 224.
51. MIRZAEI, M., SALEHI-ABARGOUEI, A., MIRZAEI, M. & MOHSENPOUR, M. A. 2018. Cohort Profile: The Yazd Health Study (YaHS): a population-based study of adults aged 20–70 years (study design and baseline population data). *International journal of epidemiology*, 47, 697-698h.
52. MÜNZEL, T., SØRENSEN, M., GORI, T., SCHMIDT, F. P., RAO, X., BROOK, J., CHEN, L. C., BROOK, R. D. & RAJAGOPALAN, S. 2017. Environmental stressors and cardio-metabolic disease: part I—epidemiologic evidence supporting a role for noise and air pollution and effects of mitigation strategies. *European heart journal*, 38, 550-556.
53. MUZET, A. 2007. Environmental noise, sleep and health. *Sleep medicine reviews*, 11, 135-142.

54. OFTEDAL, B., KROG, N. H., PYKO, A., ERIKSSON, C., GRAFF-IVERSEN, S., HAUGEN, M., SCHWARZE, P., PERSHAGEN, G. & AASVANG, G. M. 2015. Road traffic noise and markers of obesity—a population-based study. *Environmental research*, 138, 144-153.
55. OHLWEIN, S., HENNIG, F., LUCHT, S., MATTHIESSEN, C., PUNDT, N., MOEBUS, S., JÖCKEL, K.-H. & HOFFMANN, B. 2019. Indoor and outdoor road traffic noise and incident diabetes mellitus: Results from a longitudinal German cohort study. *Environmental Epidemiology*, 3, e037.
56. PENG, C., BIND, M.-A. C., COLICINO, E., KLOOG, I., BYUN, H.-M., CANTONE, L., TREVISI, L., ZHONG, J., BRENNAN, K. & DEREIX, A. E. 2016. Particulate air pollution and fasting blood glucose in nondiabetic individuals: associations and epigenetic mediation in the normative aging study, 2000–2011. *Environmental health perspectives*, 124, 1715-1721.
57. PRATICÒ, F. G. & ANFOSSO-LÉDÉE, F. 2012. Trends and issues in mitigating traffic noise through quiet pavements. *Procedia-Social and Behavioral Sciences*, 53, 203-212.
58. PRIBADI, D. O. & PAULEIT, S. 2016. Peri-urban agriculture in Jabodetabek Metropolitan Area and its relationship with the urban socioeconomic system. *Land Use Policy*, 55, 265-274.
59. PUETT, R. C., HART, J. E., SCHWARTZ, J., HU, F. B., LIESE, A. D. & LADEN, F. 2011. Are particulate matter exposures associated with risk of type 2 diabetes? *Environmental health perspectives*, 119, 384-389.
60. PYKO, A., ERIKSSON, C., LIND, T., MITKOVSKAYA, N., WALLAS, A., ÖGREN, M., ÖSTENSON, C.-G. & PERSHAGEN, G. 2017. Long-term exposure to transportation noise in relation to development of obesity—a cohort study. *Environmental health perspectives*, 125, 117005.
61. PYKO, A., ERIKSSON, C., OFTEDAL, B., HILDING, A., ÖSTENSON, C.-G., KROG, N. H., JULIN, B., AASVANG, G. M. & PERSHAGEN, G. 2015. Exposure to traffic noise and markers of obesity. *Occupational and environmental medicine*, 72, 594-601.
62. RAJAGOPALAN, S. & BROOK, R. D. 2012. Air pollution and type 2 diabetes: mechanistic insights. *Diabetes*, 61, 3037-3045.
63. RAJKUMAR, S., YOUNG, B. N., CLARK, M. L., BENKA-COKER, M. L., BACHAND, A. M., BROOK, R. D., NELSON, T. L., VOLCKENS, J., REYNOLDS, S. J. & L'ORANGE, C. 2019. Household air pollution from biomass-burning cookstoves and metabolic syndrome, blood lipid concentrations, and waist circumference in Honduran women: a cross-sectional study. *Environmental research*, 170, 46-55.
64. RUIZ-PADILLO, A., RUIZ, D. P., TORIJA, A. J. & RAMOS-RIDAO, Á. 2016. Selection of suitable alternatives to reduce the environmental impact of road traffic noise using a fuzzy multi-criteria decision model. *Environmental Impact Assessment Review*, 61, 8-18.
65. RUIZ-PADILLO, A., TORIJA, A. J., RAMOS-RIDAO, Á. & RUIZ, D. P. 2014. A methodology for classification by priority for action: Selecting road stretches for network noise action plans. *Transportation Research Part D: Transport and Environment*, 29, 66-78.
66. SAKHVIDI, M. J. Z., SAKHVIDI, F. Z., MEHRPARVAR, A. H., FORASTER, M. & DADVAND, P. 2018. Association between noise exposure and diabetes: A systematic review and meta-analysis. *Environmental research*, 166, 647-657.

67. SAKLAYEN, M. G. 2018. The global epidemic of the metabolic syndrome. *Current hypertension reports*, 20, 12.
68. SAMSON, S. L. & GARBER, A. J. 2014. Metabolic syndrome. *Endocrinology and Metabolism Clinics*, 43, 1-23.
69. SELANDER, J., BLUHM, G., NILSSON, M., HALLQVIST, J., THEORELL, T., WILLIX, P. & PERSHAGEN, G. 2013. Joint effects of job strain and road-traffic and occupational noise on myocardial infarction. *Scandinavian journal of work, environment & health*, 195-203.
70. SHAMY, M., ALGHAMDI, M., KHODER, M. I., MOHORJY, A. M., ALKHATIM, A. A., ALKHALAF, A. K., BROCATO, J., CHEN, L. C., THURSTON, G. D. & LIM, C. C. 2018. Association between exposure to ambient air particulates and metabolic syndrome components in a Saudi Arabian population. *International journal of environmental research and public health*, 15, 27.
71. SØRENSEN, M., ANDERSEN, Z. J., NORDSBORG, R. B., BECKER, T., TJØNNELAND, A., OVERVAD, K. & RAASCHOU-NIELSEN, O. 2013. Long-term exposure to road traffic noise and incident diabetes: a cohort study. *Environmental health perspectives*, 121, 217-222.
72. SØRENSEN, M., HJORTEBJERG, D., ERIKSEN, K. T., KETZEL, M., TJØNNELAND, A., OVERVAD, K. & RAASCHOU-NIELSEN, O. 2015. Exposure to long-term air pollution and road traffic noise in relation to cholesterol: a cross-sectional study. *Environment international*, 85, 238-243.
73. SØRENSEN, M., HOFFMANN, B., HVIDBERG, M., KETZEL, M., JENSEN, S. S., ANDERSEN, Z. J., TJØNNELAND, A., OVERVAD, K. & RAASCHOU-NIELSEN, O. 2012. Long-term exposure to traffic-related air pollution associated with blood pressure and self-reported hypertension in a Danish cohort. *Environmental health perspectives*, 120, 418-424.
74. SØRENSEN, M., HVIDBERG, M., HOFFMANN, B., ANDERSEN, Z. J., NORDSBORG, R. B., LILLELUND, K. G., JAKOBSEN, J., TJØNNELAND, A., OVERVAD, K. & RAASCHOU-NIELSEN, O. 2011. Exposure to road traffic and railway noise and associations with blood pressure and self-reported hypertension: a cohort study. *Environmental health*, 10, 1-11.
75. ŠOŠTARIČ, A., JENKO, B., KOZJEK, N. R., OVIJAČ, D., ŠUPUT, D., MILISAV, I. & DOLŽAN, V. 2019. Detection of metabolic syndrome burden in healthy young adults may enable timely introduction of disease prevention. *Archives of medical science: AMS*, 15, 1184.
76. STOKHOLM, Z. A., BONDE, J. P., CHRISTENSEN, K. L., HANSEN, Å. M. & KOLSTAD, H. A. 2013. Occupational noise exposure and the risk of hypertension. *Epidemiology*, 135-142.
77. SUN, Q., YUE, P., DEIULIIS, J. A., LUMENG, C. N., KAMPFRATH, T., MIKOLAJ, M. B., CAI, Y., OSTROWSKI, M. C., LU, B. & PARTHASARATHY, S. 2009. Ambient air pollution exaggerates adipose inflammation and insulin resistance in a mouse model of diet-induced obesity. *Circulation*, 119.
78. THIERING, E., CYRYS, J., KRATZSCH, J., MEISINGER, C., HOFFMANN, B., BERDEL, D., VON BERG, A., KOLETZKO, S., BAUER, C.-P. & HEINRICH, J. 2013. Long-term exposure to traffic-related air pollution and insulin resistance in children: results from the GINIplus and LISAplus birth cohorts. *Diabetologia*, 56, 1696-1704.

79. UKAOGO, P. O., EWUZIE, U. & ONWUKA, C. V. 2020. Environmental pollution: causes, effects, and the remedies. *Microorganisms for Sustainable Environment and Health*, 419.
80. VAN KEMPEN, E. & BABISCH, W. 2012. The quantitative relationship between road traffic noise and hypertension: a meta-analysis. *Journal of hypertension*, 30, 1075-1086.
81. VAN KEMPEN, E., CASAS, M., PERSHAGEN, G. & FORASTER, M. 2018. WHO environmental noise guidelines for the European region: a systematic review on environmental noise and cardiovascular and metabolic effects: a summary. *International journal of environmental research and public health*, 15, 379.
82. VAN POLL, R., AMELING, C., BREUGELMANS, O., HOUTHUIJS, D., VAN KEMPEN, E., MARRA, M. & SWART, W. 2014. Gezondheidsonderzoek Vliegbasis Geilenkirchen (Desk research) I: Hoofdrapportage: samenvatting, conclusies en aanbevelingen Gezondheidsonderzoek Vliegbasis Geilenkirchen.
83. VIENNEAU, D., EZE, I. C., PROBST-HENSCH, N. & RÖÖSLI, M. 2019. Association between transportation noise and cardio-metabolic diseases: an update of the WHO meta-analysis.
84. VOS, T., ABAJOBIR, A. A., ABATE, K. H., ABBAFATI, C., ABBAS, K. M., ABD-ALLAH, F., ABDULKADER, R. S., ABDULLE, A. M., ABEBO, T. A. & ABERA, S. F. 2017. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *The Lancet*, 390, 1211-1259.
85. WALLAS, A., EKSTRÖM, S., BERGSTRÖM, A., ERIKSSON, C., GRUZIEVA, O., SJÖSTRÖM, M., PYKO, A., ÖGREN, M., BOTTAI, M. & PERSHAGEN, G. 2019. Traffic noise exposure in relation to adverse birth outcomes and body mass between birth and adolescence. *Environmental research*, 169, 362-367.
86. WALLWORK, R. S., COLICINO, E., ZHONG, J., KLOOG, I., COULL, B. A., VOKONAS, P., SCHWARTZ, J. D. & BACCARELLI, A. A. 2017. Ambient fine particulate matter, outdoor temperature, and risk of metabolic syndrome. *American journal of epidemiology*, 185, 30-39.
87. WANG, C., CHEN, R., CAI, J., SHI, J., YANG, C., TSE, L. A., LI, H., LIN, Z., MENG, X. & LIU, C. 2016. Personal exposure to fine particulate matter and blood pressure: A role of angiotensin converting enzyme and its DNA methylation. *Environment international*, 94, 661-666.
88. WANG, S., GAO, S., LI, S. & FENG, K. 2020. Strategizing the relation between urbanization and air pollution: Empirical evidence from global countries. *Journal of Cleaner Production*, 243, 118615.
89. WEI, Y., ZHANG, J., LI, Z., GOW, A., CHUNG, K. F., HU, M., SUN, Z., ZENG, L., ZHU, T. & JIA, G. 2016. Chronic exposure to air pollution particles increases the risk of obesity and metabolic syndrome: findings from a natural experiment in Beijing. *The FASEB Journal*, 30, 2115-2122.
90. WEINMAYR, G., HENNIG, F., FUKS, K., NONNEMACHER, M., JAKOBS, H., MÖHLENKAMP, S., ERBEL, R., JÖCKEL, K.-H., HOFFMANN, B. & MOEBUS, S. 2015. Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution. *Environmental Health*, 14, 53.

91. WHO 2006. *Air Quality Guidelines: Particulate Matter, Ozone, Nitrogen Dioxide and Sulfur Dioxide*, World Health Organization.
92. WU, G., BROWN, J., ZAMORA, M. L., MILLER, A., SATTERFIELD, M. C., MEININGER, C. J., STEINHAUSER, C. B., JOHNSON, G. A., BURGHARDT, R. C. & BAZER, F. W. 2019. Adverse organogenesis and predisposed long-term metabolic syndrome from prenatal exposure to fine particulate matter. *Proceedings of the national academy of sciences*, 116, 11590-11595.
93. WU, S., DENG, F., HUANG, J., WANG, H., SHIMA, M., WANG, X., QIN, Y., ZHENG, C., WEI, H. & HAO, Y. 2013. Blood pressure changes and chemical constituents of particulate air pollution: results from the healthy volunteer natural relocation (HVNR) study. *Environmental health perspectives*, 121, 66-72.
94. YANG, B.-Y., QIAN, Z., HOWARD, S. W., VAUGHN, M. G., FAN, S.-J., LIU, K.-K. & DONG, G.-H. 2018a. Global association between ambient air pollution and blood pressure: a systematic review and meta-analysis. *Environmental pollution*, 235, 576-588.
95. YANG, B.-Y., QIAN, Z. M., LI, S., FAN, S., CHEN, G., SYBERG, K. M., XIAN, H., WANG, S.-Q., MA, H. & CHEN, D.-H. 2018b. Long-term exposure to ambient air pollution (including PM₁) and metabolic syndrome: The 33 Communities Chinese Health Study (33CCHS). *Environmental research*, 164, 204-211.
96. YING, Z., XU, X., BAI, Y., ZHONG, J., CHEN, M., LIANG, Y., ZHAO, J., LIU, D., MORISHITA, M. & SUN, Q. 2014. Long-term exposure to concentrated ambient PM_{2.5} increases mouse blood pressure through abnormal activation of the sympathetic nervous system: a role for hypothalamic inflammation. *Environmental health perspectives*, 122, 79-86.
97. YITSHAK SADE, M., KLOOG, I., LIBERTY, I. F., SCHWARTZ, J. & NOVACK, V. 2016. The association between air pollution exposure and glucose and lipids levels. *The Journal of Clinical Endocrinology & Metabolism*, 101, 2460-2467.
98. YU, Y., PAUL, K., ARAH, O. A., MAYEDA, E. R., WU, J., LEE, E., SHIH, I.-F., SU, J., JERRETT, M. & HAAN, M. 2020. Air pollution, noise exposure, and metabolic syndrome—A cohort study in elderly Mexican-Americans in Sacramento area. *Environment international*, 134, 105269.
99. ZAREI MAHMOUD ABADI, H. 2016. The Distribution and Environmental Impacts of Noise Pollution Sadouqi Martyr Yazd International Airport Using GIS. *Tolooebehdasht*, 14, 183-199.

Figures

Figure 1

shows a flowchart of study data collection: Using geographical weight regression, the odds ratio of MetS in exposure to noise and air pollution (PM2.5 index) was assessed with 95% confidence.