Associations between metabolic syndrome and anthropogenic heat emissions in northeastern China

Abstract

Background: Recent research attention has been paid to anthropogenic heat emissions (AE), temperature increase generated by human activity such as lighting, transportation, manufacturing, construction, and building climate controls. However, there is no epidemiological data available to investigate the association between anthropogenic heat emissions and metabolic syndrome (MetS), a cluster of conditions that increase risk of stroke, heart disease and diabetes.
Objective: To explore the relationships between AE and MetS in China.
Methods: We recruited 15,477 adults from the 33 Communities Chinese Health Study, a cross-sectional study in northeastern China. We retrieved anthropogenic heat flux by collecting socio-economic and energy consumption data as well as satellite-based nighttime light and Normalized Difference Vegetation Index datasets, including emissions from buildings, transportation, human metabolism, and industries. We also measured MetS components consisting of triglycerides, high density lipoprotein cholesterol, fasting glucose, systolic blood pressure, and diastolic blood pressure, and waist circumference. Restricted cubic spline models were applied to assess the associations between AE and MetS.
Results: The median flux of total AE was 30.98 W/m2 and industrial AE was the dominant contributor (87.64%). The adjusted odds ratio and 95% confidence interval (CI) of MetS for the 75th and 95th percentiles of the total AE against the threshold were 1.29 (95% CI: 1.21, 1.38) and 1.65 (95% CI: 1.47, 1.85). Greater AE was associated with higher odds of MetS in a dose-response pattern, and the lowest point of U-shape curve indicated the threshold effect. Participants who are young and middle-aged exhibited stronger associations between AE and MetS.
Conclusions: Our novel findings reveal that AE are positively associated with MetS and that associations are modified by age. Further investigations into the mechanisms of the effects are needed.

Keywords: Anthropogenic heat emissions; Metabolic syndrome; Dose-response relationship; Threshold effect

1. Introduction

Anthropogenic heat is generated by human activities and released into the air. Anthropogenic heat emissions (AE) are commonly generated from transportation, industries, human metabolism, and buildings (Chen et al., 2020). With rapid economic development, the emission of anthropogenic heat shows unprecedented growth (Jin et al., 2019). Therefore, accurately assessing anthropogenic heat is important for environmental monitoring (Cao et al., 2021). At present, there are several approaches to estimate AE, such as using the bottom-up and top-down inventory-based methods (Cao et al., 2019; Sailor, 2011), past and future global gridded anthropogenic heat flux (AHF) (Jin et al., 2019), and multisource remote sensing (Chen et al., 2019, 2020). Furthermore, AE are tremendous influences on thermal environments and play a key role in the effects of urban heat islands (UHI) (Crutzen, 2004). Several epidemiological studies have indicated that UHIs are closely related to impairment of human metabolism-related functions such as cardiovascular diseases and hypertension (Laverdière et al., 2015; Méndez-Lázaro et al., 2018). Therefore, estimation of anthropogenic heat emissions is essential for human health research.

Metabolic syndrome (MetS) is a group of common metabolic disorders. MetS is one of the major risk factors for type 2 diabetes and cardiovascular diseases (Eckel et al., 2005; Lee et al., 2017; Mongraw-Chaffin et al., 2018). MetS are usually characterized by elevated blood pressure, elevated blood glucose, abdominal obesity, and dyslipidemia (Cornier et al., 2008; Eckel et al., 2005; Kassi et al., 2011). The prevalence of metabolic syndrome is as high as 34.3% in USA adults, and 33.9% in Chinese adults and is gradually increasing worldwide (Lu et al., 2017; Shin et al., 2018). The major contributors to this high prevalence is not only unhealthy lifestyles (smoking, drinking, lack of exercise) and genetic factors, but also various environmental pollution exposures, such as to nitrogen oxides and organochlorine pesticide particulate matter (PM1, PM2.5, PM10) (Hou et al., 2020; Rosenbaum et al., 2017; Yu et al., 2020). However, to the best of our knowledge, association between exposure to anthropogenic heat and MetS has not been reported.

The present study sought to address these gaps in the literature. Specifically, we estimated anthropogenic heat emission by collecting socio-economic and energy consumption data as well as satellite-based nighttime light and Normalized Difference Vegetation Index datasets. We analyzed the associations between anthropogenic heat emissions and metabolic syndrome and explored their dose-response relationships using data from the 33 Communities Chinese Health Study (33CCHS). We hypothesized that anthropogenic heat emissions will be related

to metabolic syndrome, including its components, and that greater AE is associated with a higher prevalence rate of MetS.

2. Materials and methods

2.1. Study participants

The 33CCHS was a cross-sectional study conducted in Liaoning province, located in northeastern China, from April 1 to December 31, 2009 (Yang et al., 2020). With a booming economy and rapid urbanization in recent years, China is also facing severe environmental pollution caused by increased coal consumption and power plant and traffic emissions, especially in the northern China (Ren et al., 2020; Song et al., 2017). Liaoning is an important, heavily industrial province in northern China and has developed rapidly in the past several decades. Previous studies also observed that Liaoning province had a significant, high prevalence of MetS (Yu et al., 2014). The 33CCHS aimed to assess the associations between air pollution and human health. Details surrounding the study population has been published previously (Yang et al., 2020). Briefly, we adopted a four-stage, randomly stratified, cluster sampling scheme. The study participants were randomly selected from 33 communities in 11 districts in Liaoning province (Shenyang, Anshan, and Jinzhou). Based on the sampling frame, a total of 28,830 eligible participants were invited to attend the study. Then, 24,845 completed the questionnaire survey, and among them, 15,477 participants (53.7%) who provided fasting venous blood samples were ultimately included in this study. The study protocol was approved by the Human Studies Committee of Sun Yat-sen University. All participants were informed the purpose, benefits, and personal information confidentiality agreement of this study and required written informed consent before collecting data.

2.2. Anthropogenic heat emissions estimation

Anthropogenic heat emission was estimated based on the methods noted in Wang et al. (2020). This was an extension of previous studies for anthropogenic heat mapping across China for different regions and time-intervals (Chen and Hu, 2017; Chen et al., 2019) with a finer spatial resolution (500-m), which is superior to (1-km) spatial resolution of the results retrieved from global studies (Jin et al., 2019; Yang et al., 2017). Moreover, socio-economic and energy consumption data as well as satellite-based nighttime light and Normalized Difference Vegetation Index (NDVI) datasets were used to retrieve anthropogenic heat flux

across China from 2000 through 2016. This detailed inventory method has been described in the Supplemental Materials (Wang et al., 2020). Total emission of anthropogenic heat as well as emissions from different sources (industry, transportation, building, and human metabolism) were estimated separately. The sources of industrial AE were mainly obtained coal, oil, gas, electricity, etc. The AE from transportation was calculated according to vehicle driving distance and the combustion efficiency of fuel. Buildings were divided into commercial buildings and residential buildings. Heat emissions from human metabolism were mainly calculated based on period of the active state and metabolic rate.

2.3. Clinical measurements

Before starting to collect data, all researchers and staff completed a training program developed by the American Heart Association (AHA) (Grundy et al., 2004). According to the AHA guidelines, we measured blood pressure (BP) three times with 2-min intervals after at least 5 min of rest using a standardized mercuric-column sphygmomanometer. Before the measurement, the investigators generally advised participants to avoid drinking coffee, tea and taking exercise for at least 30 min. When measured, the participants were seated in a quiet and comfortable room. We carefully recorded the average systolic blood pressure (SBP) and diastolic blood pressure (DBP) for the subsequent data analyses. Body mass index (BMI) was calculated as weight divided by the square of height. The heights, weights and waist circumferences were measured based on the standardized protocol developed by WHO (World Health Organization, 1995).

The basic biochemical components of metabolic syndrome include fasting glucose, triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C). We collected fasting peripheral venous blood samples drawn from the study participants, separated serum from red blood cells, and determined above-mentioned biochemical indicators measured in mmol/L using the Hitachi Autoanalyzer (Type 7170A; Hitachi Ltd.; Tokyo, Japan) based on the manufacturer's instructions.

2.4. Definition of MetS

Our study followed the Joint Interim Statement (JIS) of the definition of metabolic syndrome (Alberti et al., 2009). MetS was defined as yes when a participant met three or more of the following criteria: (1) waist circumference ≥85 cm for males or 80 cm for females; (2) fasting glucose level ≥5.6 mmol/L or pharmaceutical treatment of elevated fasting glucose; (3) HDL-

C < 1.03 mmol/L for males or 1.30 mmol/L for females or medical treatment of reduced HDL-C; (4) either systolic blood pressure (SBP) ≥ 130 mmHg or diastolic blood pressure (DBP) ≥ 85 mmHg or medical treatment of hypertension; (5) TG ≥ 1.70 mmol/L or medical treatment of hyperlipidemia (Yang et al., 2018; Ye et al., 2020). We also used other definitions of MetS including the National Cholesterol Education Program/Adult Treatment Panel III Criteria (NCEP/ATP III) (Grundy et al., 2004) and Chinese Diabetes Society (CDS) (Zhang et al., 2011). The details are shown in supplemental material Table S1.

2.5. Covariates

We considered previous research studies in terms of air pollution and MetS (Yang et al., 2018; Ye et al., 2020; Zhang et al., 2021) and used directed acyclic graphs (DAGs) (www.dagitty.net) when constructing covariates. These covariates could affect both anthropogenic heat emissions and metabolic syndrome including its components. According to previous studies and based on our DAGs (Fig. S1), the following covariates were included: age (years, continuous), sex (male or female), ethnicity [Han (a majority ethnicity in China) or others], education (≤9 years or > 9 years), annual household income in Chinese Yuan (≤10,000 CNY; 10,001–29,999 CNY; ≥ 30,000 CNY), career (officer, worker, farmer, or other), smoking (yes or no), alcohol consumption (yes or no), and regular exercise (yes or no). We classified education as > 9 years (the highest educational attainment was high school, junior college or higher) or ≤ 9 years (no school, primary school and middle school). We also classified annual household income as low income ≤10,000 CNY, middle income 10,001–29,999 CNY, and high income ≥30,000 CNY). In our study, “smoking” was defined as quitting smoking less than six months ago or persisting in smoking in the past year; “alcohol consumption” was defined as any alcohol consumption more than two times per week for males and once per week for females; “regular exercise” was defined as engaging in more than 180 min of exercise per week for the following exercises, including running, walking, bicycling, dancing, or swimming (Yang et al., 2018).

2.6. Statistical analysis

2.6.1. Descriptive statistics

We employed Q-Q plots and Bartlett's test to evaluate normality and homogeneity. For normally distributed continuous variables, we reported the mean ± standard deviation (SD); and for skewed distributed continuous variables, we reported mean ± SD, minimum,

maximum and 5th, 25th, 50th, 75th and 95th percentiles. For categorical variables, we reported numbers and percentages. We compared the characteristics of MetS and non-MetS participants using t-test for continuous variables and chi-square test for nominal variables.

2.6.2. Main analyses

Restricted cubic spline (RCS) models were applied to assess the associations between AE and MetS, including their dose-response relationships after adjustment for several covariates (age, sex, ethnicity, career, education, smoking, alcohol drinking, annual household income, and regular exercise). We also assessed the relationship of AE with MetS in crude models. We used 3 knots (5th, 50th and 95th percentiles) in RCS function with a best-fit Akaike Information Criterion (AIC) value (Desquilbet and Mariotti, 2010). We calculated the odds ratios (OR) and the 95% confidence intervals (CI) of MetS at 75th and 95th percentiles of AE against the threshold, namely the flux of AE corresponding to minimum OR of MetS in all following analyses. The 75th and 95th percentiles of the total AE generally represents the slight high exposure level and the higher exposure level, respectively.

In order to determine the threshold of AE, we used the same method used in previous literature (Chung et al., 2009; Li et al., 2016). Specifically, we first drew a curve of the adjusted OR (95% CI) of MetS associated with AE using RCS models, and initially observed the possible range of the threshold. Then, in the above-mentioned possible range, we estimated each OR values of MetS by 0.1-unit increments in AE using the RCS function. Thereafter, the anthropogenic heat flux corresponding to the lowest values for OR of MetS was used as the threshold.

In addition, we also used RCS models to assess the associations between each AE component and MetS. To compare the analysis with MetS, the components of MetS were treated as binary variables in the models according to the Joint Interim Statement on MetS definition. Previous studies reported that sex and age were important modifying factors for thermal environment and health (Alahmad et al., 2020; Chen et al., 2018; Yu et al., 2010). Therefore, we assessed the stratified analyses of the associations between AE and MetS by sex (male or female) and age (<60 years or ≥ 60 years), respectively. We assessed the interaction by adding a multiplicative term for the spline term of AE × sex/age and comparing the model fit using likelihood ratio test (Chambers, 1992).

2.6.3. Sensitivity analyses

We performed several sensitivity analyses. First, we assessed the main relationship between AE and MetS using the other two definitions of MetS from NCEP/ATP III (Grundy et al., 2004) and CDS (Zhang et al., 2011). Moreover, taking into account that family history, medicine use, or unhealthy lifestyle such as smoking or alcohol consumption could affect metabolic syndrome, we excluded participants with family history (hypertension, diabetes, hyperlipidemia, and obesity) (n = 7985), those taking medicine (antihypertensive, hypoglycemic, or lipid-lowering drugs) (n = 520), smokers (n = 4640), and alcohol drinkers (n = 3809) from the regression models, respectively. In addition, given PM2.5 may affect thermal environment and Mets (Yang et al., 2018), we assessed OR (95% CI) for MetS with 75th and 95th percentiles of AE against the threshold confounding by atmospheric particulate matter 2.5 (PM2.5). The average concentrations of PM2.5 for the 33 communities are 82.02 μg/m^3 (Yang et al., 2018).

Significant levels for associations between AE and MetS were $P < 0.05$. SAS were used for all the above statistical analyses (version 9.4, SAS Institute Inc., Cary, NC, USA).

3. Results

3.1. Descriptive statistics

The average age of the 15,477 participants was 44.97 ± 13.45 years, and 47.30 percent were females (Table 1). The prevalence of MetS was 30.37% among this population. Significant correlations were found among each characteristic between MetS and non-MetS subgroups. We present detailed characteristics of the participants in Table S2. Table 2 summarizes the statistics of anthropogenic heat emissions in Liaoning, China from April 1 to December 31, 2009. The median flux of total anthropogenic heat emissions was 30.98 W/m2. Industrial anthropogenic heat emission was the dominant anthropogenic heat in this region, which was 27.15 W/m^2.

Table 1. Characteristics of the study population.

Characteristics	Total (n = 15,477)	MetS (n = 4701)	Non-MetS (n = 10,776)	*P* - Value [d]
Demographics				
Age (years)[a]				
< 60	13,132 (84.85)	3735 (79.45)	9397 (87.20)	< **0.001**
≥60	2345 (15.15)	966 (20.55)	1379 (12.80)	

Sex[b]				
Male	8156 (52.70)	2738 (58.24)	5418 (50.28)	< **0.001**
Female	7321 (47.30)	1963 (41.76)	5358 (49.72)	
BMI[a]				
<18.5 kg/m^2	527 (3.41)	11 (0.23)	516 (4.79)	< **0.001**
18.5–24.9 kg/m^2	8679 (56.08)	1292 (27.48)	7387 (68.55)	
25–29.9 kg/m^2	5432 (35.10)	2844 (60.50)	2588 (24.02)	
≥30 kg/m^2	839 (5.42)	554 (11.78)	285 (2.64)	
Ethnicity[b]				
Han	14,554 (94.04)	4482 (95.34)	10,072 (93.47)	< **0.001**
Others	923 (5.96)	219 (4.66)	704 (6.53)	
Education (years)[b]				
≤9	11,898 (76.88)	3761 (80.00)	8137 (75.51)	< **0.001**
>9	3579 (23.12)	940 (20.00)	2639 (24.49)	
Annual household income[b,c]				
≤10,000	3144 (20.31)	1047 (22.27)	2097 (1946)	< **0.001**
10,001–29,999	7869 (50.84)	2288 (48.67)	5581 (51.79)	
≥30,000	4464 (28.84)	1366 (29.06)	3098 (28.75)	
Career[b]				
Officer	2900 (18.74)	945 (20.10)	1955 (18.14)	**0.003**
Worker	4996 (32.28)	1484 (31.57)	3512 (32.59)	
Farmer	2210 (14.28)	701 (14.91)	1509 (14.00)	
Others	5371 (34.71)	1571 (33.42)	3800 (35.26)	
Smoking[b]	4640 (29.98)	1531 (32.57)	3109 (28.85)	< **0.001**
Alcohol drinking[b]	3809 (24.61)	1370 (29.14)	2439 (22.63)	< **0.001**
Regular exercise[b]	4932 (31.87)	1625 (34.57)	3307 (30.69)	< **0.001**
MetS components[a]				
Waist circumference (cm)	82.95 ± 10.60	90.89 ± 8.06	79.49 ± 9.68	< **0.001**
Fasting glucose (mmol/l)	5.44 ± 1.46	6.08 ± 1.97	5.16 ± 1.06	< **0.001**
Triglyceride (mmol/l)	1.72 ± 1.43	2.67 ± 1.94	1.31 ± 0.85	< **0.001**
HDL-C (mmol/l)	1.37 ± 0.34	1.25 ± 0.36	1.42 ± 0.32	< **0.001**
SBP (mmHg)	128.11 ± 21.39	142.38 ± 21.65	121.89 ± 18.04	< **0.001**
DBP (mmHg)	81.90 ± 12.51	90.00 ± 12.29	78.37 ± 10.85	< **0.001**

Abbreviations: MetS, metabolic syndrome; HDL-C, high density lipoprotein-cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Note: Bolding indicates significant ($P < 0.05$).

[a] Values are mean ± SD.

[b] Values are n (%).

[c] RMB, Chinese Yuan.

[d] *P*- values for χ^2 test for nominal variables or *t*-test for continuous variables.

Table 2. Summary statistics of anthropogenic heat emissions in 2009 in Liaoning, China (W/m^2).

Pollutant	Mean ± SD	Minimum	Maximum	Percentile				
				5th	25th	50th	75th	95th
Total AE	29.50 ± 6.02	11.92	37.99	14.63	25.69	30.98	33.36	37.73
AE industries	25.36 ± 5.67	10.66	33.98	11.41	21.99	27.15	29.41	33.74
AE buildings	2.29 ± 0.89	0.74	4.57	1.45	1.79	1.98	2.42	4.18
AE transportion	1.85 ± 0.57	0.51	3.26	1.07	1.47	1.82	1.99	2.98
AE human metabolism	.003 ± .001	.001	.006	.002	.002	.002	.003	.006

Abbreviations: AE, Anthropogenic heat emissions; SD, standard deviation.

3.2. Association between AE and MetS

Table 3 and Fig. 1 shows the significantly positive associations between anthropogenic heat emissions and metabolic syndrome. We found that the dose-response curve between the total AE and MetS was generally U-shaped, and the lowest point of the curve indicated the threshold effect. The threshold flux of the total AE was 23.00 W/m2, and the greater flux of total AE were statistically associated with the greater odds of MetS above threshold in covariate-adjusted models. The OR (95% CI) of MetS for the 75th and 95th percentiles of the total AE against the threshold were 1.29 (95% CI: 1.21, 1.38) and 1.65 (95% CI: 1.47, 1.85). The associations between AE and MetS in crude models were consistent with our main findings (Table S3). The trend was approximately similar for associations between AE and each MetS components to our original results (Table S4).

Table 3. Adjusted ORs (95% confidence intervals) for MetS with 75th and 95thpercentiles of AE against the threshold. [a]

Pollutant	Threshold (W/m^2)	75th (W/m^2)	95th (W/m^2)	OR (95% CI)	
				75th vs. threshold	95th vs. threshold
Total AE	23.00	33.36	37.73	**1.29 (1.21, 1.38)**	**1.65 (1.47, 1.85)**
AE industries	10.67	29.41	33.74	**1.24 (1.06, 1.45)**	**1.46 (1.25, 1.71)**
AE buildings	0.75	2.42	4.18	**1.47 (1.22, 1.78)**	**1.76 (1.49, 2.09)**
AE transportion	0.52	1.99	2.98	1.07 (0.88, 1.29)	**1.42 (1.19, 1.69)**
AE human metabolism	.001	.003	.006	**1.48 (1.23, 1.78)**	**1.70 (1.45, 1.99)**

Abbreviations: MetS, metabolic syndrome; OR, odds ratio; CI, confidence interval. AE, Anthropogenic heat emissions.

Note: Bolding indicates significant ($P < 0.05$).

[a] Adjusted for age, sex, ethnicity, career, education, smoking, alcohol drinking, annual household income, regular exercise.

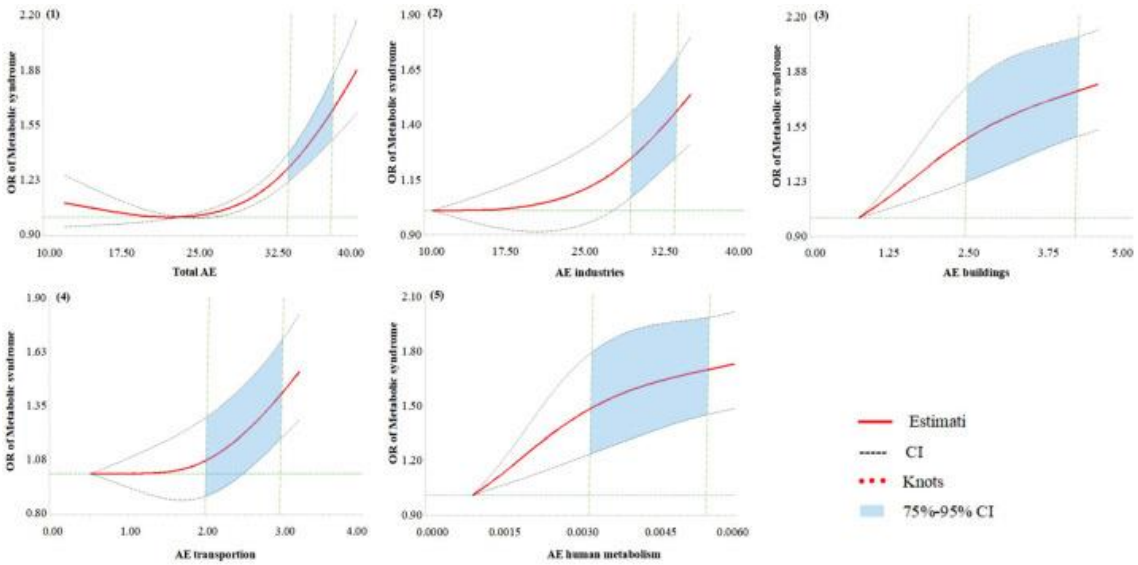

Fig. 1. Adjusted ORs and 95% CIs of MetS in relation to AE using RCS models.

Abbreviations: MetS, metabolic syndrome; OR, odds ratio; CI, confidence interval; RCS, restricted cubic spline; AE, Anthropogenic heat emissions. Models are adjusted for age, sex,

ethnicity, career, education, smoking, alcohol drinking, annual household income, regular exercise. NOTE: The red solid line represents the estimated ORs, red dots represent the 3 knots (5th, 50th, 95th) used in RCS models, the black dotted line represents the 95% CIs, and green dotted line represents the reference where OR of Metabolic Syndrome is 1, the blue area between two vertical black dotted line represents 75th and 95th percentiles of AE against the threshold. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

In Fig. 1, we also observed that the curves of associations of industrial AE and traffic AE with MetS were nearly U-shaped. Increased flux of industrial AE and traffic AE were positively associated with increased odds of MetS with the rapidly increment change. In contrast, the curves of relationships between AE from buildings and human metabolism with MetS were inverted J-shaped. These AE fluxes were also positively related to greater odds of MetS, while the increment became slowly at higher flux levels.

Table 4 shows the associations between AE and MetS stratified by sex and age in RCS models after adjusted covariates. The interaction between total AE and sex on risk of MetS did not reach statistical significance. When stratified by age, the associations between AE and MetS were stronger in the age <60 years group than in the age ≥60 years group.

Table 4. Adjusted ORs (95% confidence intervals) for MetS with 75th and 95th percentiles of AE against the threshold stratified by sex, age.

OR (95% CIs)										
Pollutant	**Sex**					**Age**				
	Male (n = 8156)[a]		**Female (n = 7321)[a]**		***P*[c]**	**Age <60 (n = 13,132)[b]**		**Age ≥60 (n = 2345)[b]**		***P*[c]**
	75th vs. threshold	**95th vs. threshold**	**75th vs. threshold**	**95th vs. threshold**		**75th vs. threshold**	**95th vs. threshold**	**75th vs. threshold**	**95th vs. threshold**	
Total AE	**1.16 (1.06, 1.28)**	**1.36 (1.16, 1.59)**	**1.36 (1.23, 1.50)**	**1.76 (1.48, 2.10)**	0.348	**1.29 (1.20, 1.38)**	**1.65 (1.46, 1.86)**	1.15 (0.99, 1.33)	**1.35 (1.02, 1.80)**	<0.001
AE industries	1.17 (0.93, 1.46)	1.20 (0.96, 1.50)	**1.36 (1.23, 1.50)**	**1.78 (1.49, 2.13)**	0.285	**1.24 (1.11, 1.39)**	**1.47 (1.29, 1.69)**	1.15 (0.99, 1.33)	**1.36 (1.01, 1.81)**	0.002
AE buildings	1.02 (0.96, 1.09)	**1.50 (1.30, 1.72)**	**1.57 (1.23, 2.01)**	**1.35 (1.07, 1.70)**	<0.001	**1.57 (1.28, 1.94)**	**1.80 (1.50, 2.16)**	1.48 (0.91, 2.41)	1.34 (0.88, 2.06)	<0.001

OR (95% CIs)										
Pollutant	**Sex**					**Age**				
	Male (n = 8156)[a]		**Female (n = 7321)[a]**		***P*[c]**	**Age <60 (n = 13,132)[b]**		**Age ≥60 (n = 2345)[b]**		***P*[c]**
	75th vs. threshold	**95th vs. threshold**	**75th vs. threshold**	**95th vs. threshold**		**75th vs. threshold**	**95th vs. threshold**	**75th vs. threshold**	**95th vs. threshold**	
AE transportation	1.03 (0.99, 1.07)	**1.59 (1.39, 1.83)**	1.21 (0.95, 1.54)	1.12 (0.88, 1.42)	<0.001	1.07 (0.87, 1.31)	**1.38 (1.14, 1.67)**	1.16 (0.72, 1.88)	1.13 (0.72, 1.76)	<0.001
AE human metabolism	1.03 (0.97, 1.09)	**1.49 (1.29, 1.71)**	**1.59 (1.24, 2.02)**	**1.31 (1.05, 1.62)**	<0.001	**1.58 (1.29, 1.94)**	**1.73 (1.46, 2.06)**	1.48 (0.92, 2.38)	1.30 (0.88, 1.94)	<0.001

Abbreviations: MetS, metabolic syndrome; AE, Anthropogenic heat emissions; OR, odds ratio; CI, confidence interval.

Note: Bolding indicates significant ($P < 0.05$).

[a] Adjusted for age, ethnicity, career, education, smoking, alcohol drinking, annual household income, regular exercise.

[b] Adjusted for sex, ethnicity, career, education, smoking, alcohol drinking, annual household income, regular exercise.

[c] *P* for interaction by adding a multiplicative term for the spline term of AE × sex/age.

3.3. Sensitivity analyses

When we employed other two definitions of MetS, the associations between AE and MetS were consistent with the main results (Table S5). Moreover, Table S6 shows that the associations between AE and MetS were similar to the main findings after excluding participants with family history, medicine usage, smokers, and alcohol drinkers, respectively. Furthermore, Table S7 shows that the results were consistent after adding confounding by PM2.5.

4. Discussion

Estimation of anthropogenic heat emissions and their effects is essential for human health research. In the present investigation, we observed significant positive associations between anthropogenic heat emissions and metabolic syndrome. To our knowledge, we are among the first to examine the dose-response relationships and identify a much-needed threshold for the association between AE and MetS.

In our study, we retrieved total fluxes of anthropogenic heat emissions by collecting socio-economic and energy consumption data as well as satellite-based nighttime light and NDVI datasets, which in Liaoning were 30.98 W/m2, and industrial AE was the major contributor (accounting for 87.64%). Several previous studies had results similar to ours (Chakraborty et al., 2015; Koralegedara et al., 2016; Lu et al., 2016). For example, Lu et al. (2016) reported that the anthropogenic heat flux ranged from 20 to 100 W/m2 in urban China, and industrial AE accounted for 73.00%; this estimate was based on the top-down inventory approach. However, Chen et al. (2020) analyzed AE in China using cubist with points-of-interest and multisource remote sensing data and found that the heat flux of total AE was 8.5 W/m2 in Shanghai, China. This relatively low emission flux differed from our results and may be due in part to the different estimation methods used and their survey region, which was not heavily industrial and also not in a small spatial area. Further investigations are warranted to gain a clearer understanding of this phenomenon.

To date, there are no epidemiological data available to assess the associations between anthropogenic heat emissions and metabolic syndrome. Our results suggest that the flux of AE is statistically related to MetS. Although we could not easily compare these relationships with other studies due to the limitation of published data, several studies have observed the associations of thermal environment and urban heat island effect with human health issues, such as cardiovascular disease (CVD) (Huang et al., 2020; Méndez-Lázaro et al., 2018; Urban et al., 2014; Wilker et al., 2012). For instance, Huang et al. (2020) used the Landsat remote-sensing images' data from 96 meteorological stations in Beijing, China and found that increased temperature due to heat island effect was associated with increased mortality from CVD. Also consistent with our results, a recent animal study showed urban daphnia living in city ponds exposed to the urban heat island effect had significantly higher body fat (Brans and Stoks, 2018). This area of empirical research, however, is still in its embryonic stage and a greater number of investigations of anthropogenic heat emissions and human health in other populations, especially using longitudinal designs, are critically needed. We encourage future research to build on these findings.

Our results showed that the curve shape of the dose-response relationship of AE with MetS was generally U-shaped and indicated a threshold effect, which is a major concern in ambient air pollution research. Our findings are consistent with some previously reported literature (Alahmad et al., 2020; Chen et al., 2018; Li et al., 2016). For example, Chen et al. (2018) assessed the associations between ambient temperature and various cardiopulmonary

disease mortality in 272 cities in China from 2013 to 2015. They found that the curve of the above associations was nearly U-shaped and increased mortality risks from cardiopulmonary diseases for higher temperatures above threshold. Interestingly, we also observed that the shapes of relationships between industrial AE and traffic AE with MetS differed from the other sources of AE. Although these relationships were positive, the upward trends of the curve were different, with industrial AE and traffic AE possible contributing more to the total association of AE with MetS. Further studies with an improved study design are needed to verify our findings. Elucidating the biological mechanisms involved with the associated between AE and MetS are challenging and unclear. Several hypotheses have been proposed. First, Méndez-Lázaro et al. (2018) proposed that high temperature stimulates the body's heat stress and changes the physiological processes related to cell inflammation and damage. Furthermore, a previous study explored the mechanisms for the association between temperature and heart failure and reported that ambient temperature was associated with higher levels of B-type natriuretic peptide and C-reactive protein, which are systemic inflammatory factors. More relevant mechanistic studies capable of achieving greater precision regarding how anthropogenic heat is related to human health are obviously necessary.

In our study, we found the association between total AE and MetS was no significant statistical significance when modified by sex. Several previous studies found the relationships were more pronounced among females (Chen et al., 2018; Yu et al., 2010). Chen et al. (2018) found that hot temperature was more associated with increased cardiopulmonary disease mortality risks in females, and their potential vulnerabilities to the adverse effect of temperature might explained the modified phenomenon. Perhaps because in our study, males and females played different roles in AE contribution. Furthermore, we also observed that the young and middle-aged group exhibited a stronger association between AE and MetS. Alahmad et al. (2020) also found that the young and middle-aged group is at a very high risk of death because of hot temperature. However, Zanobetti et al. (2013) reported the elderly are most likely to suffer from heat death when stratified by age. A possible reason for this is that the working age group, particularly those who were less than 60 years old, are more commonly involved in industrial, traffic, and construction work. Therefore, additional underlying mechanism studies need to explore the modification of sex/age on the relationship between AE and MetS.

Our study has several strengths. First, we employed a large population-based sample, which is well-powered to assess associations between AE and MetS. Second, we carried out an accurate assessment of the anthropogenic heat emissions with advanced technology and instrumentation. Third, we conducted several sensitivity analyses which showed the estimated associations were stable. Finally, our study is the first to evaluate the association of anthropogenic heat emissions with metabolic syndrome and present the positive dose-response relationship and apparent threshold effect.

Despite these strengths, our study also has limitations. First, we cannot identify any causal relationship between AE and MetS due to the cross-sectional nature of our design. Second, some covariates in the self-report questionnaire may lead to recall bias. Third, other confounding factors like traffic noise and total energy intake were not measured in this research. However, we have adjusted for many demographic and socioeconomic characteristics that could influence the observed associations. Hence, more comprehensive information should be collected and taken into consideration in future research.

5. Conclusions

We are among the first to report that the flux of anthropogenic heat emissions is positively related to metabolic syndrome and that these associations are significantly stronger in the subgroups who are young and middle-aged. Our study provides a novel and valuable contribution to the evidence given the limited data on health association of anthropogenic heat emissions. Further longitudinal studies are needed to confirm our results. These findings provide more evidence that reducing AE is essential to optimize air quality and mitigate AE health effects, and these findings may be especially useful for policy formulation and city planning.

Declaration of competing interest

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

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