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Evaluation of the effects of short-term $PM_{2.5}$ exposure on triglyceride-glucose metrics in a population in eastern China

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Abstract

Context The triglyceride-glucose (TyG) index, a novel health indicator, has been widely employed to assess insulin resistance (IR). However, its relationship with fine particulate matter (PM) exposure remains inadequately investigated.

Objective This study endeavors to probe the association between $PM_{2.5}$ and TyG within the population of eastern China and to determine whether there are disparities in this association among diverse subgroups.

Methods We conducted an ecological study on a cohort comprising 39,011 individuals who had undergone at least two physical examinations between 2017 and 2019 at the First Affiliated Hospital of Nanjing Medical University, China. TyG levels concerning short-term $PM_{2.5}$ exposure were examined using a generalized additive model.

Results In the overall population, at lags of 0–7 and 0–14 days in the single-pollutant model, it was observed that a 10 μg/m^3 rise in $PM_{2.5}$ corresponded to a 0.0021 elevation in TyG levels. In the multi-pollutant models, at 0–7 and 0–14 days lags, a comparable increase in $PM_{2.5}$ resulted in an increase in TyG of 0.0073 and 0.0044, respectively. The association remained significant in the subgroup analyses.

Conclusion $PM_{2.5}$ exposure is related to the TyG index. Controlling air pollution might contribute to maintainin normal lipid metabolism function.

Keywords Triglyceride-glucose, Insulin resistance, $PM_{2.5}$, Environmental exposure

Introduction

Despite the decline in fine particulate matter (PM) levels in recent years, $PM_{2.5}$ still leads to 1 million premature deaths annually [1]. Previous studies have demonstrated that $PM_{2.5}$ can trigger oxidative stress, inflammatory injury, immune response, and insulin resistance (IR) [2]. A positive correlation has been observed between exposure to $PM_{2.5}$ and adverse outcomes such as lung cancer, stroke, preterm birth, and cardiovascular diseases (CVD) [3–6].

The triglyceride-glucose (TyG) index has been employed to assess the body's insulin sensitivity [7, 8]. It is not only associated with the incidence and prognosis of different types of diabetes, obesity, and CVD but also

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increases the risk of all-cause death and CVD mortality [9–13]. In addition, in cohorts of people with primary and secondary prevention for CVD, the TyG index can also be utilized as a biomarker to forecast the occurrence and prognosis of the disease [9].

Previous research has demonstrated that exposure to $PM_{2.5}$ can increase the level of fasting blood glucose (FBG) and decrease the level of triglyceride (TG) [14, 15]. We hypothesized that $PM_{2.5}$ and TyG may have a particular association, but it has not been thoroughly examined. Thus, we conducted an ecological study on a cohort of individuals in eastern China to explore the association between $PM_{2.5}$ and the TyG index, and to determine whether this relationship varies among different subgroups.

Subjects and methods

Study population

We recruited a cohort of 39,011 subjects who participated in regular physical examinations at the First Affiliated Hospital of Nanjing Medical University between 2017 and 2019. They should (a) possess at least two repeated physical examination records, and (b) have results of lipid-related indicators and blood glucose tests. The exclusion criteria were as follows: subjects who (a) suffered from mental illness, (b) had severe occupational exposure, or (c) were unable to provide complete data required for the study. We collected their demographic characteristics, medical history, smoking status, menopausal status, and occupational status.

Environmental exposure

We collected the daily concentrations of six ambient air pollutants in Nanjing from the China Air Quality Online Monitoring and Analysis Platform (https://www.aqistudy.cn/) during the period from 2016 to 2019. There were thirteen fixed-site air monitoring stations in Nanjing. We mainly focused on six types of air pollutants, namely $PM_{2.5}$(μg/m^3), PM_{10}(μg/m^3), NO_2(μg/m^3), SO(μg/m^3), CO(mg/m^3), and O_3(μg/m^3). Additionally, we collected information on local meteorological factors, including daily average temperature (°C) and daily average relative humidity (%) through the National Meteorological Data Sharing Center (http://data.cma.cn/).

Definitions

The TyG index was calculated using the following formula: $\mathrm{TyG} = \ln\left[TG\,(mg/dl) * FBG\,(mg/dl)\,/2\right]$ [16, 17]. Individuals with a body mass index (BMI) less than 24 kg/m^2, were classified as underweight or normal weight, whereas those with a BMI was greater than 24 kg/m^2 were defined as overweight. Smoking history was categorized into three groups: never, current, and former [18]. Based on the nature of the company and workplace, occupational status was classified into four groups: laborer, professional/executive, sales, and others. We regarded the season as constant if an individual underwent two physical examinations during the same season. However, if the physical examinations took place in different seasons, it was assumed that a seasonal change had occurred [14]. The remaining definitions are provided in Supplementary Methods 1.

Statistical analysis

Considering that exposure to $PM_{2.5}$ often results in lag effects on health outcomes [14, 19], we applied the generalized additive model (GAM) to analyze the short-term lag effects of $PM_{2.5}$ on health outcomes [20]. Differences in TyG levels exhibited an approximately normal distribution. Thus, to examine the impact of short-term $PM_{2.5}$ exposure on TyG, GAM of the Gaussian distribution family was adopted. Supplementary Methods 2 describes how the GAM was constructed [21].

We incorporated the findings from previous research conducted by our group, such as the studies by studies by Zhang M et al. [14] and Liu Q et al. [15], which demonstrated that the health effects of $PM_{2.5}$ were only observed at lags of 0–7, 0–14, 0–21, and 0–28 days, along with the characteristics of the lagged effects of $PM_{2.5}$ exposure on health [22, 23]. Consequently, specific times were selected for analysis. By determining the concentration of $PM_{2.5}$ at lags of 0–7 days, 0–14 days, 0–21 days, and 0–28 days, the possible lag effects of $PM_{2.5}$ were evaluated.

Both the single-pollutant and multi-pollutant models were established. The covariates adjusted in the single-pollutant models encompassed age, sex, BMI, smoking status, seasonal change, intervals between physical examinations, meteorological parameters, and occupational status. Building upon this, other air pollutants that excluded the problem of multicollinearity were incorporated as covariates to formulate the multifactorial pollutant models [14]. Age and BMI were determined by averaging two measurements. The differences between the two measurements for TG and FBG levels and environmental factors indicated changes over the study period.

Spearman rank correlation analysis was employed to identify, the multicollinearity of environmental factors. If the absolute value of the correlation coefficient $|r| \geq 0.7$, the corresponding variable was removed; if $|r| < 0.7$, it was retained in the model [24, 25]. Sensitivity studies were conducted to evaluate the stability of the relationships between $PM_{2.5}$ and TyG. Firstly, the relationship of $PM_{2.5}$ with TyG was reanalyzed within the subgroup of the healthy population. Secondly, we separately constructed the $PM_{2.5}$ single-pollutant and multi-pollutant models. Thirdly, in order to control for confounders

Table 1 Demographic characteristics of the study population

Characteristics	Statistics
Sex	
Male, n(%)	22,598(57.93%)
Female, n(%)	16,413(42.07%)
Age (years)	
Age[a], mean ± SD	44.72 ± 14.53
Age[b], mean ± SD	45.70 ± 14.53
Menopausal [c]	
Premenopausal, n(%)	11,507(70.11%)
Postmenopausal, n(%)	4906(29.89%)
BMI (Kg/m^2)	
BMI[a], mean ± SD	23.84 ± 3.44
BMI[b], mean ± SD	23.98 ± 3.23
Smoking	
Never, n(%)	33,126(84.91%)
Current, n(%)	5291(13.56%)
Former, n(%)	594(1.52%)
Hypertension	
Yes, n(%)	5173(13.26%)
No, n(%)	33,838(86.74%)
Diabetes	
Yes, n(%)	1272(3.26%)
No, n(%)	37,739(96.74%)
TyG	
TyG[a], mean±(SD	8.62 ± 0.61
TyG[b], mean ± SD	8.64 ± 0.59
Interval time (days), mean ± SD	366.97 ± 50.34
CHD	
Yes, n(%)	522(1.34%)
No, n(%)	38,489(98.66%)
Seasonal change	
Yes, n(%)	12,239(31.37%)
No, n(%)	26,772(68.63%)
Occupation status	
Laborer, n(%)	2210(5.67%)
Professional/executive/, n(%)	22,672(58.12%)
Sales, n(%)	13,595(34.85%)
Other types, n(%)	534(1.37%)
$PM_{2.5}$ (μg/m^3), mean ± SD	41.47 ± 29.11
PM_{10}(μg/m^3), mean ± SD	76.36 ± 44.36
SO_2(μg/m^3), mean ± SD	11.64 ± 5.78
NO_2(μg/m^3), mean ± SD	43.55 ± 18.32
O_3(μg/m^3), mean±(SD	78.75 ± 42.51
CO(mg/m^3), mean ± SD	0.86 ± 0.31
Temperature (℃), mean ± SD	16.83 ± 9.26
Relative humidity (%), mean ± SD	72.85 ± 14.39

[a] The first time that people participated in routine physical examinations

[b] The second time that people participated in routine physical examinations

[c] Only female subjects

Note TyG refers to the triglyceride-glucose index, and CHD stands for coronary heart disease

related to occupational exposure, we classified occupational status into four categories.

Subgroup analyses were carried out to assess the potential influence of age, sex, smoking history, BMI, and menopausal status on the association between $PM_{2.5}$ and TyG. Additionally, heterogeneity testing among subgroups was performed using the formula $|\beta 1 - \beta 2|/\sqrt{SE1^2 + SE2^2}$, where $\beta 1$ and $\beta 2$ represent estimated effects, and $SE1$ and $SE2$ denote standard errors. A statistically significant difference was indicated by a value exceeding 1.96 [26]. Data analyses were conducted with R version 4.3.1 with a significance level set at 0.05.

Results

General characteristics of the study population

As shown in Table 1, a total of 39,011 individuals were enrolled in our study, among whom 22,598 (57.93%) were males and 16,413 (42.07%) were females. Specifically, there were 33,126 nonsmokers (84.91%), 5,291 current smokers (13.56%), and 597 former smokers (1.52%). At baseline, the mean age was 44.72 years, and the mean BMI was 23.84 kg/m^2. The average TyG levels at baseline and during the second physical examination were 8.62 and 8.64, respectively. After excluding individuals with diabetes (n = 1,272, 3.26%), hypertension (n = 5173, 13.26%), and coronary heart disease (CHD) (n = 522, 1.34%), a healthy group composed 33,130 individuals (84.92%) was identified for subgroup analysis.

Environmental factors

Table 1 presents the median daily average concentrations of air pollutants. The values are 34.00 μg/m^3 for $PM_{2.5}$, 65.00 μg/m^3 for PM_{10}, 39.00 μg/m^3 for NO_2, 10.00 μg/m^3 for SO_2, 0.80 mg/m^3 for CO, and 69.00 μg/m^3 for O_3. The average temperature recorded was 17.20 °C, and the average relative humidity was 73.00%.

The results of Spearman rank correlation analysis are presented in Table 2. In this table, the r-value between PM_{10} and $PM_{2.5}$ was 0.91, and the r-value between NO_2 and $PM_{2.5}$ was 0.70. As a consequence, PM_{10} and NO_2 were excluded from the multi-pollutant models.

Exposure-response curves for PM2.5 and TyG

The exposure-response curves of both the single-pollutant and multi-pollutant models exhibited a linear trend at lags of 0–7 days and 0–14 days in both the entire population and the subgroup of the healthy population (Figs. 1 and 2). However, the curves showed inverted parabolic shapes at lags of 0–21 days and 0–28 days. Moreover, a flat trend was noticed when the difference in $PM_{2.5}$ concentration was 0 μg/m^3.

Table 2 Spearman correlation coefficient between the meteorological factors and air pollutant concentrations

	$PM_{2.5}$	PM_{10}	SO_2	NO_2	O_3	CO	Temperature	Relative humidity
$PM_{2.5}$	1	0.9067*	0.5103*	0.7024*	-0.2156*	0.6556*	-0.4885*	-0.1808*
PM_{10}		1	0.6607*	0.7712*	-0.1285*	0.6307*	-0.4123*	-0.4392*
SO_2			1	0.6273*	-0.0085*	0.6425*	-0.2131*	-0.5633*
NO_2				1	-0.3283*	0.6311*	-0.4594*	-0.3152*
O_3					1	-0.1820*	0.5776*	-0.2990*
CO						1	-0.2413*	-0.0999*
Temperature							1	0.0931*
Relative humidity								1

*: $P < 0.05$

Association of PM2.5 with TyG

In both single-pollutant and multi-pollutant models, an increase of 10 μg/m³in $PM_{2.5}$ was associated with an increase in TyG. In the entire population, within the single-pollutant models, TyG increased by 0.0021 at 0–7 days and 0.0021 at 0–14 days. In the multi-pollutant models, it increased by 0.0073 and 0.0044, respectively (Table 3). In the subgroup population, for the single-pollutant models, the TyG increased by 0.0025 at 0–7 days and by 0.0026 at 0–14 days. In the multi-pollutant models, it increased by 0.0075 and 0.0046, respectively (Table 3). These associations remained significant after adjusting for occupational status in the sensitivity analyses (Supplementary Table 1).

Subgroup analysis

Subgroup analyses were conducted to investigate the relationship between $PM_{2.5}$ and TyG in different groups categorized by sex, age, smoking history, BMI, and menopausal status. In the entire population, a significant association between $PM_{2.5}$ and TyG was observed in various sex subgroups at lags of 0–7 days and 0–14 days. Specifically, at a lag of 0–7 days, $PM_{2.5}$ showed a significant association with TyG across different age and BMI subgroups. Furthermore, within the healthy population subgroup, $PM_{2.5}$ displayed a significant association with TyG across sex, age, and BMI subgroups at a lag of 0–7 days (Tables 4 and 5; Fig. 3). Since only 1.52% of the participants were former smokers, we did not take former smokers into account in the subgroup heterogeneity effect analysis. Only heterogeneity between subgroups of menopausal status was observed at lags of 0–7 days and 0–14 days in the entire population (Supplementary Table 2). This might be attributed to the fact of premenopausal females have higher estrogen levels than postmenopausal females and that $PM_{2.5}$ possesses estrogenic activity [27–29].

Discussion

Our study revealed that TyG levels were positively correlated with $PM_{2.5}$ concentrations in a cohort of the Chinese population that repeatedly underwent physical examinations. To the best of our knowledge, this is one of the most extensive population-based cohort studies exploring the relationship between $PM_{2.5}$ and TyG levels.

TyG not only reflects IR, but also predicts the risk of CVD and is related to sudden cardiac arrest [30, 31]. Exposure to $PM_{2.5}$ can induce lung oxidative stress, prompt unique changes in lipid composition, increase fatty acids like palmitic acid esters, myristate esters, and palmitoyl esters, and result in blood vessel damage [32–34]. In a cohort study of 76 healthy elderly individuals in China [35], it was demonstrated that $PM_{2.5}$ was positively associated with elevated IR index assessed by the homeostasis model and the insulin action index. Li et al. also reported that increased exposure to $PM_{2.5}$ was associated with an increased risk of CVD mortality [36].

Although only a limited number of studies have directly demonstrated the association between $PM_{2.5}$ exposure and the TyG index, a large quantity of research, like the ones described above, has indicated that an increased $PM_{2.5}$ concentration is a risk factor for elevated IR and a high incidence of CVD. For this reason, our findings that short-term exposure to $PM_{2.5}$ is positively correlated with TyG levels were somewhat consistent with previous studies.

After inhalation, $PM_{2.5}$ mainly deposits in the lungs. The body then releases inflammatory factors such as nitric oxide and interleukins, which trigger an inflammatory response [37]. Systemic inflammatory responses and oxidative stress are signaling abnormalities that characterize IR [38]. In a mouse model, $PM_{2.5}$ exacerbates IR by inhibiting the insulin-PI3K-Akt signaling pathway in the liver, resulting in increased FBG levels and consequently a higher TyG index. In addition, $PM_{2.5}$ can also enter the gastrointestinal tract, carrying microorganisms that induce a pro-inflammatory response in the immune system, increase intestinal permeability, and disrupt the balance of gut microbiota. These effects may contribute to the development of IR and elevate FBG levels, subsequently increasing the TyG index [39, 40]. Additionally, IR impairs lipid metabolism, resulting in increased fatty acid release and enhanced triglyceride synthesis in the liver and adipose tissue [41].Moreover, exposure to $PM_{2.5}$ significantly affects sphingolipid metabolism. An animal

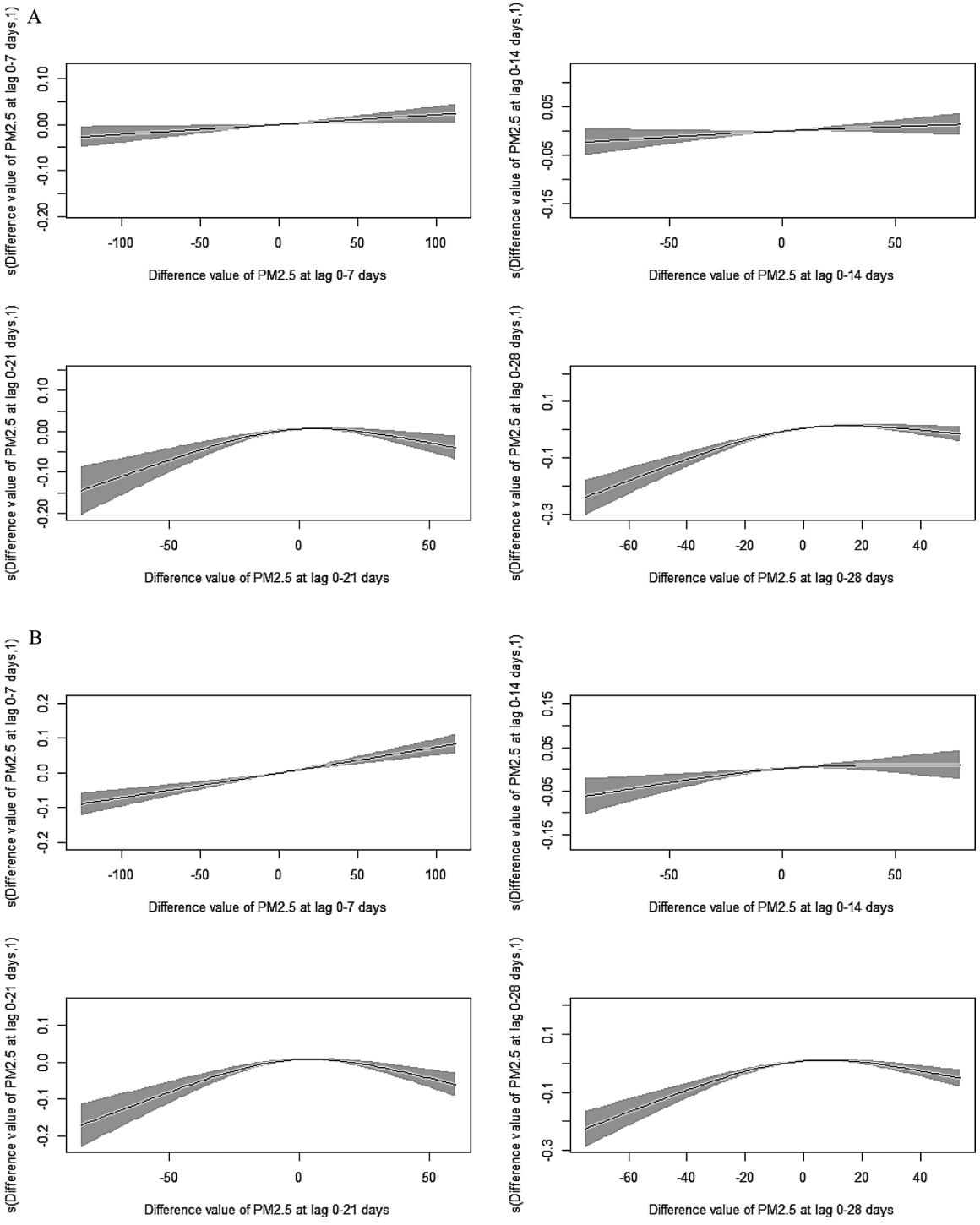

Fig. 1 The exposure-response curves for the effects of $PM_{2.5}$ on the triglyceride-glucose index in the entire population. The x-axis represents the difference value of $PM_{2.5}$ at different lag times, while the y-axis represents the contribution of the smooth term to the fitted values. **A**: Single-pollutant model; adjusted for the interval time, sex, age, BMI, smoking status, occupational status, season, temperature, and relative humidity. **B**: Multipollutant model; adjusted for SO_2, O_3, and CO based on single-pollutant models. *Note* With lags of 0–21 days and 0–28 days, as the difference in $PM_{2.5}$ concentration increases, the curve shows a trend of first rising and then falling, peaking around '0'. This indicates a non-linear relationship between TyG and $PM_{2.5}$ exposure response as the lag time increases

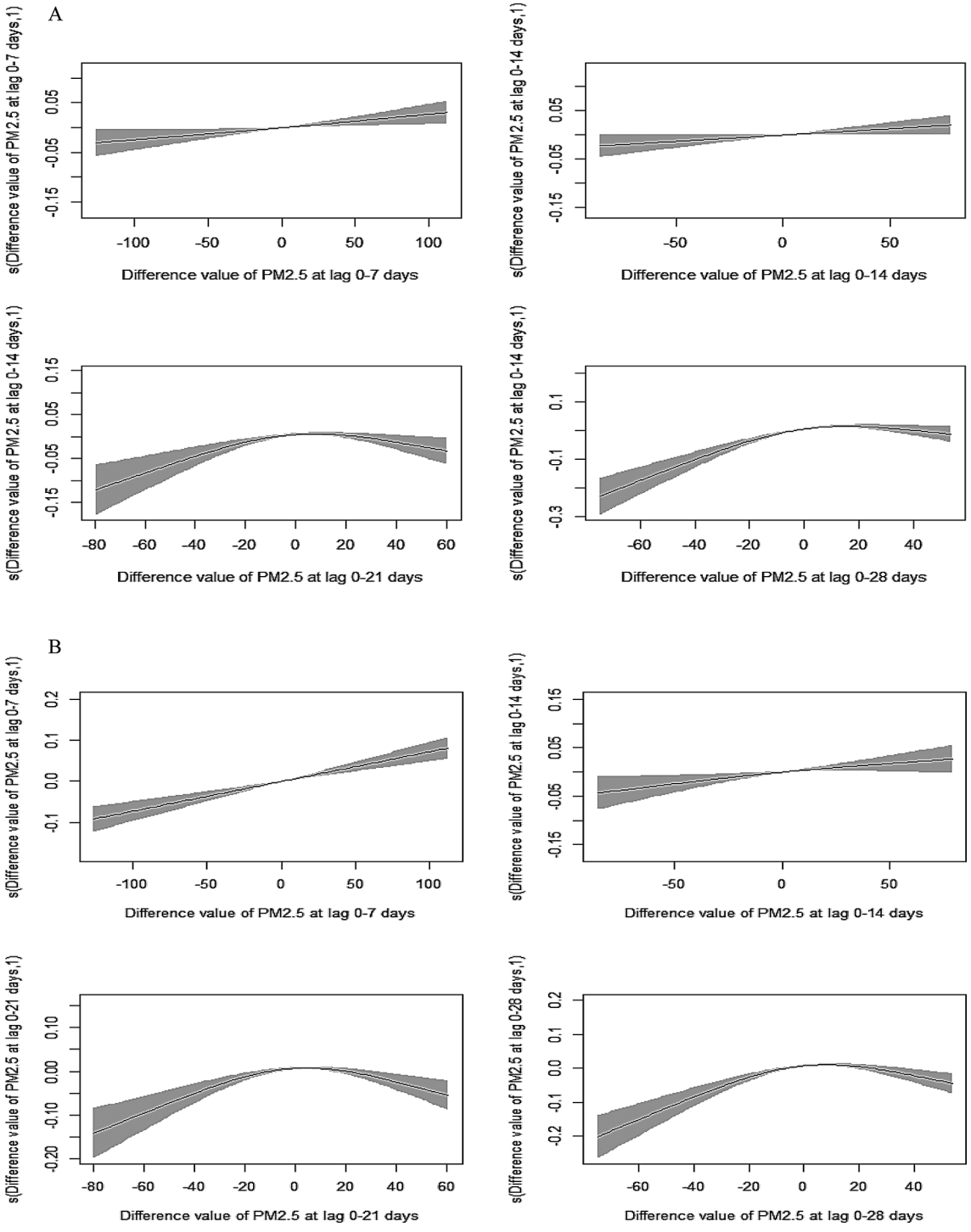

Fig. 2 The exposure-response curves for the effects of $PM_{2.5}$ on the triglyceride-glucose index in the healthy population. The x-axis represents the difference value of $PM_{2.5}$ at different lag times, while the y-axis represents the contribution of the smooth term to the fitted values. **A**: Single-pollutant model; adjusted for interval time, sex, age, BMI, smoking status, occupational status, season, temperature, and relative humidity. **B**: Multipollutant model; adjusted for SO_2, O_3, and CO based on single-pollutant models. *N*ote: With lags of 0–21 days and 0–28 days, as the difference in $PM_{2.5}$ concentration increases, the curve shows a trend of first rising and then falling, peaking around '0'. This indicates a non-linear relationship between TyG and $PM_{2.5}$ exposure response as the lag time increases

Table 3 Estimated changes (95% confidence intervals) in triglyceride-glucose index levels for a 10 μg/m^3 increase in $PM_{2.5}$

Lag time	Entire population		Normal population	
	Single model[a]	Multi-model[b]	Single model[a]	Multi-model[b]
Lag0-7 days	0.0021(0.0004,0.0039)	0.0073(0.0051,0.0095)[c]	0.0025(0.0008,0.0044)	0.0075(0.0050,0.0098) [c]
Lag0-14 days	0.0021(-0.0004,0.0046)	0.0044(0.0016,0.0073)[c]	0.0026(0.0001,0.0050)	0.0046(0.0019,0.0076) [c]
Lag0-21 days	0.5106(0.0604,0.9608)	0.3930(-0.0927,0.8787) [c]	0.4675(0.0117,0.9233)	0.3442(-0.1496,0.8380) [c]
Lag0-28 days	1.7470(1.288,2.2047)	0.9589(0.4652,1.4526) [c]	1.7080(1.2251,2.1908)	0.9518(0.4311,1.4726) [c]

[a] Adjusted for the interval time, sex, age, BMI, smoking history, season, temperature, and relative humidity

[b] Adjusted for SO_2, O_3, and CO based on the single-pollutant models

[c] The results between the single-pollutant and multi-pollutant models were significantly different ($P < 0.05$)

Table 4 Estimated changes (95% confidence intervals) in triglyceride-glucose index levels for a 10 μg/m^3 increase in $PM_{2.5}$ among the entire population[a]

Characteristic	Categorization	Lag 0–7 days	Lag 0–14 days	Lag 0–21 days	Lag 0–28 days
Sex	Male	0.0071(0.0041,0.0101)	0.0050(0.0010,0.0089)	0.5589 (-0.1578,1.2755)	0.9927(0.2933,1.6921)
	Female	0.0084(0.0052,0.0015)	0.0041(0.0001,0.0082)	0.2020(-0.4456,0.8496)	1.0640(0.3584,1.7697)
Smoking	Never	0.0067(0.0044,0.0091)	0.0038(0.0007,0.0068)	0.3391(-0.1809,0.8591)	0.9498(0.4206,1.4790)
	Current	0.0115(0.0053,0.0177)	0.0099(0.0016,0.0181)	0.9072(0.1033,1.7111)	1.1862(-0.1722,2.5447)
	Former	-0.01576(-0.0385,0.0070)	-0.0239 (-0.0524,0.0046)	-0.2805(-2.8176,2.2566)	-1.3732 (-4.8567,0.2110)
Age(years)	< 65	0.0070(0.0047,0.0092)	0.0045 (0.0016,0.0075)	0.4116(-0.0935,0.9162)	0.9714 (0.4561,1.4866)
	≥ 65	0.0099 (0.0011,0.0187)	0.0037(-0.0083,0.0156)	-0.5678(-2.4966,1.3600)	0.2252 (-1.8467,2.2971)
BMI(kg/m^2)	< 24	0.0077(0.0049,0.0105)	0.0033(-0.0003,0.0069)	0.1690 (-0.4290,0.7670)	0.9295 (0.2931,1.5660)
	≥ 24	0.0068(0.0033,0.0102)	0.0057(0.0012,0.0102)	0.5460(-0.2386,1.3305)	1.0630 (0.2854,1.8407)
Menopausal	Premenopausal	0.0104(0.0067,0.0141)	0.0067(0.0021,0.0114)	0.3216(-0.4240,1.0672)	1.2410(0.4230,2.0582)
	Postmenopausal	0.0014(-0.0053,0.0081)	-0.0053(-0.0138,0.0032)	-0.5550(-1.7592,0.64593)	0.3364(-1.0267,1.6995)

[a] The analyses were based on multipollutant models

Table 5 Estimated changes (95% confidence intervals) in triglyceride-glucose index levels for a 10 μg/m^3 increase in $PM_{2.5}$ among the normal population [a]

Characteristic	Categorization	Lag 0–7 days	Lag 0–14 days	Lag 0–21 days	Lag 0–28 days
Sex	Male	0.0066(0.0033,0.0099)	0.0040 (-0.0003,0.0083)	0.3800 (-0.3664,1.1264)	0.8401(0.0867,1.5935)
	Female	0.0093(0.0060,0.0126)	0.0054 (0.0012,0.0096)	0.3378(-0.3312,1.0067)	1.1410(0.4109,1.8719)
Smoking	Never	0.0072(0.0047,0.0096)	0.0044 (0.0012,0.0076)	0.3255 (-0.2025,0.8535)	0.9815(0.4233,1.5397)
	Current	0.0010(0.0036,0.0167)	0.0065(-0.0024,0.0153)	0.1590(-1.2249,1.5428)	0.6858(-0.7706,2.1421)
	Former	-0.0067(-0.0331,0.0197)	-0.0196 (-0.0527,0.0134)	-1.9815 (-5.8660,1.9093)	-1.958(-5.8162,1.9007)
Age(years)	< 65	0.0074(0.0050,0.0097)	0.0048(0.0017,0.0078)	0.3781(-0.1271,0.8832)	0.9779(-0.4438,1.5120)
	≥ 65	0.0116(-0.0010,0.0243)	0.0084 (-0.0084,0.0253)	0.0054(-2.1739,2.1847)	0.5608(-1.8497,2.9713)
BMI(kg/m^2)	< 24	0.0078(0.0049,0.01066)	0.0036 (-0.0001,0.0073)	0.2271(-0.3880,0.8422)	0.9547(0.0300,1.6096)
	≥ 24	0.0071(0.0033,0.0109)	0.0059(0.0009,0.0108)	0.5014(-0.3258,1.9174)	1.0500(0.1927,1.9066)
Menopausal	Premenopausal	0.0107(0.0070,0.0144)	0.0072(0.0026,0.0118)	0.3735(-0.3778,1.1248)	1.2940(0.4710,2.1177)
	Postmenopausal	0.0035(-0.0041,0.0110)	-0.0021(-0.0117,0.0074)	-0.0330(-1.3945,1.3285)	0.6145(-0.9125,2.1415)

[a] The analyses were based on multipollutant models

study demonstrated that sphingolipids play an essential role in the development of IR [42]. Studies also indicated that $PM_{2.5}$ may impact the voltage-gated chloride channel Clcn1, influence muscle contraction pathways and contribute to dyslipidemia, consequently increasing the TyG index [43, 44].

$PM_{2.5}$ exhibits a lag effect on TyG-related indicators, such as FBG and TG [14, 45]. Therefore, our study investigated the influence of $PM_{2.5}$ exposure on TyG across four specific lag periods. We noticed a significant impact of $PM_{2.5}$ exposure on TyG in both the overall population and the subgroup of healthy individuals, particularly at lag periods of 0–7 days and 0–14 days. However, some studies in the general population have shown a weak correlation between the TyG index and coronary artery calcification burden, with no association with mortality, suggesting the need for more comprehensive research [46, 47]. Considering that environmental pollutants are usually mixed in nature, we adopted the multi-pollutant models by taking CO, O_3, and SO_2 into account. Incorporating these pollutants significantly strengthened the relationship between $PM_{2.5}$ and TyG levels. This might be explained by the fact that exposure to O_3 and SO_2 can increase TyG levels [48, 49], which enhances the effects

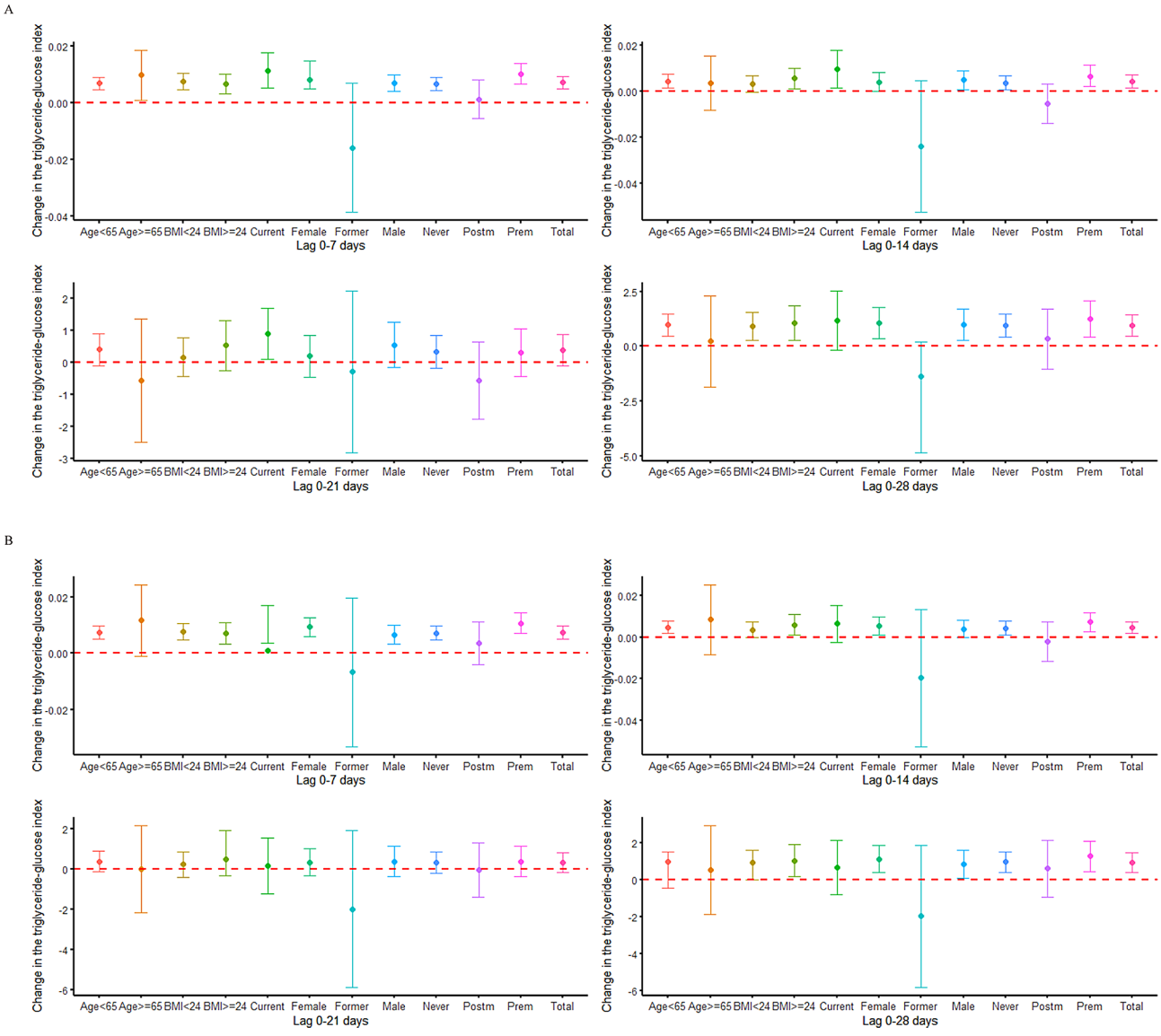

Fig. 3 Subgroup analysis of the estimated changes (95% confidence intervals) in triglyceride-glucose index levels. **A**: Entire population; adjusted for the interval time, sex, age, BMI, smoking status, occupational status, season, temperature, relative humidity, SO_2, O_3, and CO. **B**: Healthy population; adjusted for the interval time, sex, age, BMI, smoking status, occupational status, season, temperature, relative humidity, SO_2, O_3, and CO. Note. "Prem" stands for premenopausal, and "Postm" stands for postmenopausal

of $PM_{2.5}$. Although the relationship between CO and TyG has not well-established, Gao's findings in Beijing demonstrated a positive correlation between the risk of CHD and the concentrations of $PM_{2.5}$, SO_2, and CO [50], which may partly explain the association between CO and TyG.

Our research can assist clinicians in identifying IR among individuals affected by air pollution, particularly when there are no obvious symptoms of diabetes [51]. Regular monitoring of the TyG index enables early preventive interventions to slow or reverse the development of IR. Additionally, our research may promote the development of personalized treatment strategies for metabolic abnormalities induced by air pollution. For individuals with elevated TyG indices, improving air quality and adopting lifestyle modifications may help reduce IR and alleviate CVD risk [52, 53].

The effect of $PM_{2.5}$ on TyG was observed at lags of 0–7 and 0–14 days, when stratified by sex and at a lag of 0–7 days when stratified by BMI. With the prolonged lag time, the correlation became nonsignificant in each subgroup. This suggests that TyG is more likely to be affected by short-term exposure to $PM_{2.5}$. No heterogeneity was observed between subgroups except for those related to menopausal status. In the entire population, the effect of $PM_{2.5}$ was more pronounced in premenopausal females at lags of 0–7 days and 0–14 days. We speculate that this

might be due to the higher estrogen levels in premenopausal women, which alters the effect of $PM_{2.5}$ exposure on TyG [27–29]. However, this hypothesis requires further investigation. In the sensitivity analysis shown in Supplementary Table 1, we found that the lag effect still weakens as the lag time prolongs. This reminds us to pay more attention to the short-term effects of air pollutants. It has been suggested that overweight individuals are more susceptible to $PM_{2.5}$ [54–56]. Overweight individuals may exhibit higher resting tidal volume and minute ventilation, potentially leading to elevated $PM_{2.5}$ inhalation rates [57]. However, in our subgroup analysis by BMI, there was no heterogeneity among the groups at a lag of 0–7 days. This might be related to differences in study populations and regions as well as some interaction between BMI and TyG.

Our study acknowledges several inherent limitations. First, the environmental data obtained from fixed monitoring stations may not accurately capture individual exposure levels, thereby potentially obscuring the interpersonal variations in exposure. Second, although our dataset is relatively comprehensive, it does not encompass crucial variables such as drinking history, physical activity, socioeconomic status, dietary habits, or blood concentrations of lead and cadmium—factors that Tai J et al. [58] have demonstrated to be correlated with the TyG index. Finally, owing to the inherent limitations of ecological studies, the conclusions drawn may not always be completely reliable or accurate. Further rigorous research is required to validate any potential causal relationships.

Conclusion

The TyG index was positively correlated with short-term $PM_{2.5}$ exposure, and this relationship did not vary across subgroups except for that of the menopausal status subgroups. Our findings suggest that controlling air pollution is essential for cardiovascular disease prevention. To alleviate this risk, we recommend the implementation of stricter air quality regulations, the promotion of clean energy, and the development of urban green spaces. Public health campaigns that raise awareness of the impact of air pollution on lipid metabolism could further assist in reducing exposure and improving overall health outcomes.

Abbreviations

TyG	The triglyceride-glucose
IR	Insulin resistance
PM	Fine particulate matter
CVD	Cardiovascular diseases
FBG	Fasting blood glucose
TG	Triglyceride
BMI	Body mass index
GAM	Generalized additive model
CHD	Coronary heart disease
PVAT	Perivascular adipose tissue

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04489-y.

Supplementary Material 1

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Not applicable.

Author contributions

Z.G., Y.C. and Q.Z. conceived the study, analyzed the data and drafted the manuscript; X.S. and J.W. participated in the study design; X.L. implemented the field investigation; X.L., Q.Z. and J.W. participated in the study design and helped draft the manuscript. All authors contributed to the study and have read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board (ethics committee) of Nanjing Medical University. Written informed consent was obtained from study participants. The study was conducted in accordance with the Declaration of Helsinki. Participants are aware of the potential health impacts of PM exposure.

Consent for publication

Not applicable.

Clinical trial number

Not applicable.

Competing interests

The authors declare no competing interests.

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