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Effects of short-term $PM_{2.5}$ exposure on blood lipids among 197,957 people in eastern China

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Globally, air pollution is amongst the most significant causes of premature death. Nevertheless, studies on the relationship between fine particulate matter ($PM_{2.5}$) exposure and blood lipids have typically not been population-based. In a large, community-based sample of residents in Yixing city, we assessed the relationship between short-term outdoor $PM_{2.5}$ exposure and blood lipid concentrations. Participants who attended the physical examination were enrolled from Yixing People's hospital from 2015 to 2020. We collected general characteristics of participants, including gender and age, as well as test results of indicators of blood lipids. Data on daily meteorological factors were collected from the National Meteorological Data Sharing Center (http://data.cma.cn/) and air pollutant concentrations were collected from the China Air Quality Online Monitoring and Analysis Platform (https://www.aqistudy.cn/) during this period. We applied generalized additive models to estimate short-term effects of ambient $PM_{2.5}$ exposure on each measured blood lipid-related indicators and converted these indicators into dichotomous variables (non- hyperlipidemia and hyperlipidemia) to calculate risks of hyperlipidemia associated with $PM_{2.5}$ exposure. A total of 197,957 participants were included in the analysis with mean age 47.90 years (± SD, 14.28). The increase in $PM_{2.5}$ was significantly associated with hyperlipidemia (odds ratio (OR) 1.003, 95% CI 1.001–1.004), and it was still significant in subgroups of males and age < 60 years. For every 10 μg/m^3 increase in $PM_{2.5}$, triglyceride levels decreased by 0.5447% (95% CI − 0.7873, − 0.3015), the low-density lipoprotein cholesterol concentration increased by 0.0127 mmol/L (95% CI 0.0099, 0.0156), the total cholesterol concentration increased by 0.0095 mmol/L (95% CI 0.0053, 0.0136), and no significant association was observed between $PM_{2.5}$ and the high-density lipoprotein cholesterol concentration. After excluding people with abnormal blood lipid concentrations, the associations remained significant except for the high-density lipoprotein cholesterol concentration. $PM_{2.5}$ was positively correlated with low-density lipoprotein cholesterol and total cholesterol, and negatively correlated with triglyceride, indicating $PM_{2.5}$ can potentially affect health through blood lipid levels.

Abbreviations

SD	Standard deviation
IQR	Interquartile range
95%CI	95% Confidence interval
OR	Odds ratio

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$PM_{2.5}$	Particulate matter with an aerodynamic matter smaller than 2.5 μm
PM_{10}	Particulate matter with an aerodynamic matter smaller than 10 μm
NO_2	Nitrogen dioxide
SO_2	Sulphur dioxide
O_3	Ozone
CO	Carbon monoxide
GAM	Generalized additive model
AIC	Akaike information criterion

Exposure to fine particulate matter ($PM_{2.5}$) has been linked to a substantial disease burden globally[1]. Although some research has shown that short-term exposure to $PM_{2.5}$ is positively correlated with mortality from respiratory diseases as well as an increased risk of cardiovascular disease[2,3], the mechanisms and other impacts of $PM_{2.5}$ exposure on health is still unclear.

Previous studies have reported that $PM_{2.5}$ exposure may increase the incidence of non-alcoholic fatty liver disease[4]. A previous study among senior citizens found that individuals exposed to long-term $PM_{2.5}$ exposure had an increased incidence of dementia[5]. Other studies found long-term $PM_{2.5}$ exposure was associated with increased serum triglyceride and decreased high-density lipoprotein cholesterol concentration in elderly males[6]. Other research in children and adolescents suggests that long-term $PM_{2.5}$ exposure was positively associated with the total cholesterol concentration and risk of hypercholesterolemia[7]. Whether $PM_{2.5}$ exposure and blood lipids are epidemiologically related is debated and few large studies have investigated this relationship at the population-level.

Although there have been several studies on $PM_{2.5}$ exposure and blood lipids, most of these studies are based on long-term $PM_{2.5}$ exposure, and few studies have explored the association between short-term $PM_{2.5}$ exposure and blood lipids. To further the understanding of the relationship between short-term $PM_{2.5}$ exposure and blood lipids, we collected test results of blood lipid-related indicators through routine physical examinations from a community-based sample of 197,957 residents in Yixing city. We also assessed for a range of environmental factors during the same period.

Methods

Study population. This cross-sectional study was performed in Yixing city, located in eastern China, with a population of approximately 1.3 million. The study population was not selected based on disease status; participants who attended a routine physical examination at Yixing People's Hospital from 2015 to 2020 were eligible and enrolled in the study. No subjects repeatedly took part in the study. Inclusion criteria: (1) participants who were tested for lipid-related indicators (2) participants were local residents Exclusion criteria: (1) participants who took lipid-lowering drugs (2) participants who were workers exposed to dust. We collected participant characteristics, blood lipid-related indicators, including total cholesterol, triglyceride, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol. Blood samples were obtained from individuals after at least 8 hours overnight fasting. High-density lipoprotein cholesterol and low-density lipoprotein cholesterol was analyzed by the direct assay method. Total cholesterol by cholesterol oxidase method and triglyceride by enzymatic method, using CobasC501, (Roche Diagnostics GmbH, Switzerland).

Binary and continuous outcomes. According to Chinese guidelines for the prevention and treatment of dyslipidemia in adults[8], the normal range of these indicators are: (1) total cholesterol < 6.2 mmol/L; (2) triglyceride < 2.3 mmol/L; (3) low-density lipoprotein cholesterol < 4.1 mmol/L; and (4) high-density lipoprotein cholesterol > 1.0 mmol/L. We calculated the number of participants with normal blood lipid-related indicators separately. Participants with abnormalities in either indicator were defined as having hyperlipidemia.

Data on meteorological factors and air pollutants. The exposure data were obtained from a fixed monitoring station ((120.35′E, 31.62′N)) for the city. The quality control methods of the monitoring stations include climate limit value check, station extreme value check, time consistency check, space consistency check and manual check. We collected daily average meteorological factors, including atmospheric pressure (hPa), temperature (°C), wind speed (m/s), and relative humidity (%) during January 8, 2015 and December 31, 2020 from the National Meteorological Data Sharing Center (http://data.cma.cn/). Data on daily average air pollutant concentrations, including $PM_{2.5}$, PM_{10} (particles of less than 10 μm diameter), sulphur dioxide (SO_2), nitrogen dioxide (NO_2), ozone (O_3), and carbon monoxide (CO) were collected from the China Air Quality Online Monitoring and Analysis Platform (https://www.aqistudy.cn/). Except that the unit of CO concentration was mg/m^3, the unit of other pollutants was $\mu g/m^3$.

Statistical analysis. A generalized additive model (GAM) was applied to explore the relationship between short-term ambient $PM_{2.5}$ exposure and blood lipid-related indicators similar to prior studies[9,10]. GAMs are useful for evaluating the impact of air pollution on human health[11]. Among the four indicators, triglyceride were not normally distributed. Total cholesterol, low-density lipoprotein cholesterol and high-density lipoprotein cholesterol were all normally distributed. We performed natural log conversions of triglyceride to achieve a approximate normal distribution. To account for potential confounders, adjusted covariates in the GAM model included day of the week, time, sex, age, and meteorological factors. To address multiple collinearities, Spearman rank correlation coefficients between environmental factors were calculated; the model only included variables with $|r|<0.7$[12]. We applied a thin plate spline function in order to control for nonlinear effects of meteorological factors[13]. Minimum Akaike information criterion (AIC) values corresponded to the optimal degree of

freedom[10]. Considering lag effects of $PM_{2.5}$ on blood lipids, we calculated 2- to 8-day moving averages (lag 0–1 day to lag 0–7 days) of the daily average concentration of $PM_{2.5}$ to capture cumulative lag effects. For example, if a person attended the physical examination on January 9, we collected the daily average concentration of $PM_{2.5}$ from January 2 to January 9, and then calculated the average concentration from January 8 to January 9 as the 2-day moving average. The n-day moving average concentration was applied to estimate personal short-term $PM_{2.5}$ exposure level. Minimizing the AIC value was applied to identify the optimal lag time[14–16]. We expressed the effects as the estimated changes in blood lipid-related indicators and their 95% confidence intervals (CIs) for a 10 μg/m^3 increase in ambient $PM_{2.5}$ concentration[15]. We also converted lipid-related indicators into dichotomous variables (normal and abnormal) to calculate risks of hyperlipidemia associated with $PM_{2.5}$ exposure, and expressed them as the odds ratio (OR) as well as their 95% CIs for 10 μg/m^3 rise in outdoor $PM_{2.5}$ concentration. In addition, we analyzed the relationship between other air pollutions (including PM_{10}, SO_2, NO_2, O_3, and CO) and blood lipids using similar approaches.

We performed two sensitivity analyses to examine the robustness of the associations between $PM_{2.5}$ and blood lipid-related indicators. First, we constructed single- and multi-pollutant models for $PM_{2.5}$, respectively. Second, individuals with abnormal indicators were excluded to estimate the effects of $PM_{2.5}$ among the population with normal indicators. A subgroup analysis was also performed to explore if the effect was modified by sex or age. The heterogeneity effects between subgroups were evaluated using the formula: $|\beta_1 - \beta_2|/\sqrt{SE_1{}^2 + SE_2{}^2}$, where β_1 and β_2 are the estimated effects, and SE_1 and SE_2 are their standard errors, respectively. When the value was larger than 1.96, the difference was considered statistically significant[15].

All analyses were performed with the "mgcv" and "ggplot2" packages in R software version 4.1.2 (https://www.r-project.org/). The significance level was set at 0.05.

Ethics statement. This study was approved by ethics committee of Yixing people's hospital.

Results

Characteristics of study participants. Of 206,452 participants eligible for the study, 205,945 attended a physical examination. In total, 7988 participants were excluded for various reasons; 1944 (0.90%) participants were excluded because they were not local residents while 4562 participants were not tested for lipid-related indicators. Lastly, 1482 participants were taking lipid-lowering drugs during the study period and were also excluded (Fig. 1). After exclusions, a total of 197,957 people were included in the analysis. Mean age was 47.90 years (± SD, 14.28) and 55.61% of participants were male. The mean values of total cholesterol, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol was 4.94 mmol/L (± 0.94), 2.73 mmol/L (± 0.67), and 1.31 mmol/L (± 0.31), respectively. The median triglyceride values were 1.35 mmol/L ((interquartile range [IQR], 0.92, 2.04) (Table 1). The number of participants with normal levels was 133,080 for triglyceride, 132,643 for low-density lipoprotein cholesterol, 132,308 for high-density lipoprotein cholesterol and 133,172 for total cholesterol.

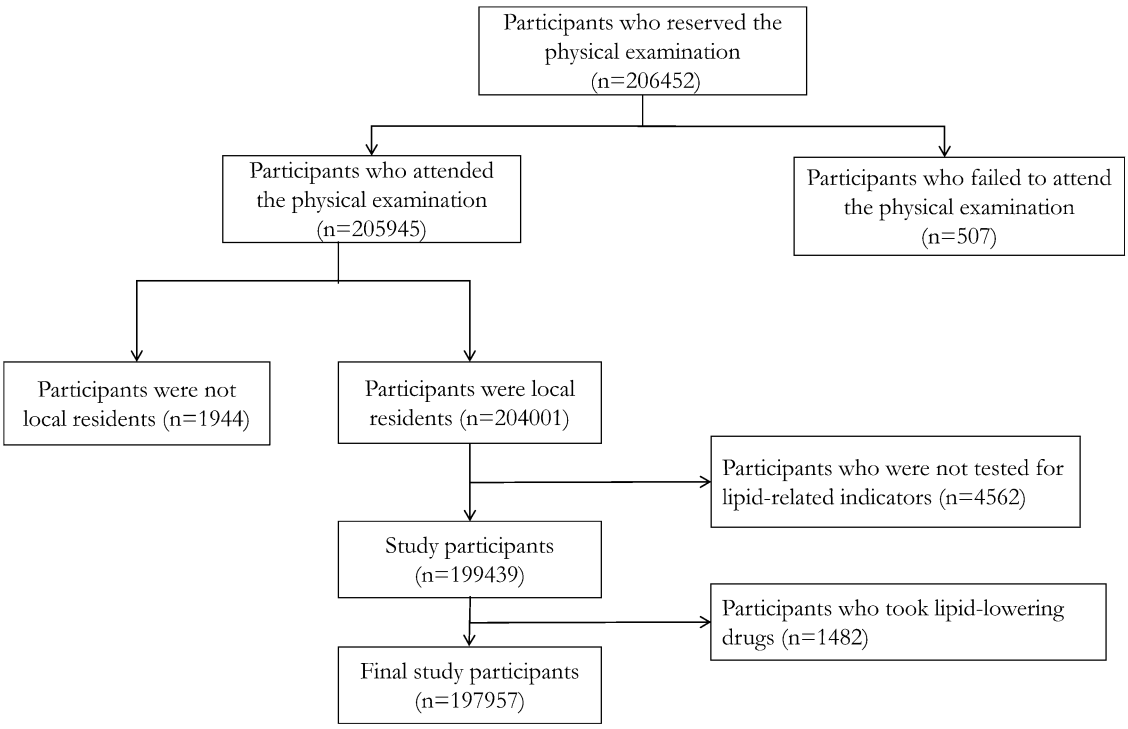

Figure 1. Flowchart of participants enrolled in the study from eastern China.

Variables	N (%)	Median (IQR)	Mean (±SD)
Sex			
Male	110,090 (55.61)		
Female	87,867 (44.39)		
Age		48.00 (37.00–57.00)	47.90 ± 14.28
< 60 years	156,195 (78.90)	44.00 (34.00–51.00)	42.46 ± 10.20
≥ 60 years	41,762 (21.10)	66.00 (63.00–72.00)	68.27 ± 7.19
Hyperlipidemia	64,448 (32.56)		
Low-density lipoprotein cholesterol (mmol/L)		2.66 (2.22–3.13)	2.73 ± 0.67
High-density lipoprotein cholesterol (mmol/L)		1.27 (1.08–1.49)	1.31 ± 0.31
Total cholesterol (mmol/L)		4.87 (4.30–5.50)	4.94 ± 0.94
Triglyceride (mmol/L)		1.35 (0.92–2.04)	1.72 ± 1.44
Meteorological factors			
Temperature (°C)		18.10 (9.60–24.70)	17.47 (± 8.97)
Atmospheric pressure (hPa)		1016.30 (1008.00–1023.40)	1016.16 (± 9.35)
Wind speed (m/s)		2.10 (1.50–2.60)	2.14 (± 0.83)
Relative humidity (%)		74.00 (64.00–83.00)	73.40 (± 13.37)
Air pollutants			
$PM_{2.5}$ (μg/m^3)		38.00 (26.00–57.00)	45.56 (± 28.45)
PM_{10} (μg/m^3)		67.00 (48.00–95.00)	76.64 (± 41.12)
SO_2 (μg/m^3)		11.00 (8.00–18.00)	13.86 (± 8.81)
NO_2 (μg/m^3)		38.00 (28.00–51.00)	41.76 (± 17.82)
CO (mg/m^3)		0.90 (0.70–1.10)	0.96 (± 0.33)
O_3 (μg/m^3)		95.00 (63.00–140.00)	103.50 (± 51.59)

Table 1. Characteristics of the study population.

Characteristics of meteorological factors and air pollutants. The median daily average meteorological factors and air pollutant concentrations was 18.10 °C for temperature, 1016.30 hPa for atmospheric pressure, 2.10 m/s for wind speed, 74% for relative humidity, 38.00 μg/m^3 for $PM_{2.5}$, 67.00 μg/m^3 for PM_{10}, 11.00 μg/m^3 for SO_2, 38.00 μg/m^3 for NO_2, 0.90 mg/m^3 for CO, and 95.00 μg/m^3 for O_3 (Table 1). $PM_{2.5}$ was positively correlated with atmospheric pressure, PM_{10}, SO_2, NO_2, and CO, and negatively correlated with temperature and wind speed ($P < 0.05$). Because Spearman rank correlation coefficients between $PM_{2.5}$ and PM_{10} and CO were larger than 0.7, the above two air pollutants were excluded from the final model. The absolute value of the correlation coefficient between temperature and atmospheric pressure was larger than 0.7, atmospheric pressure was removed from the model (Supplementary Table 1). The df of meteorological factors in the analyze of effects of $PM_{2.5}$ on blood lipids were shown in Supplementary Table 2.

$PM_{2.5}$ and blood lipids in entire population. We applied lag 0–6 days, 0–7 days, 0–5 days and 0–7 days for triglyceride, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol and total cholesterol in the entire population respectively. For a 10 μg/m^3 increase in $PM_{2.5}$, the triglyceride decreased by 0.5447% (95% CI − 0.7873, − 0.3015), the low-density lipoprotein cholesterol concentration increased by 0.0127 mmol/L (95% CI 0.0099, 0.0156) and the total cholesterol concentration increased by 0.0095 mmol/L (95% CI 0.0053, 0.0136) and no significant association was observed between $PM_{2.5}$ and the high-density lipoprotein cholesterol concentration(Table 2). The associations remained significant of low-density lipoprotein cholesterol and total cholesterol by the subgroups of males, females, age < 60 years and age ≥ 60 years. Of triglyceride, the associations remained significant in the subgroups of females, age < 60 years and age ≥ 60 years, and the effect of short-term $PM_{2.5}$ exposure on the low-density lipoprotein cholesterol concentration and total cholesterol concentration could be modified by age, the effects was stronger for the subgroup of age ≥ 60 years (Fig. 2, Supplementary Table 3).

$PM_{2.5}$ and blood lipids in persons with normal blood lipid levels. In persons with normal test results, for a 10 μg/m^3 increase in $PM_{2.5}$, the triglyceride decreased by 0.5184% (95% CI − 0.7235, − 0.3128), the low-density lipoprotein cholesterol concentration increased by 0.0096 mmol/L (95% CI 0.0068, 0.0124) and the total cholesterol concentration increased by 0.0057 mmol/L (95% CI 0.0019, 0.0095). No significant association was observed between $PM_{2.5}$ and the high-density lipoprotein cholesterol concentration (Table 2). The associations remained significant of low-density lipoprotein cholesterol and triglyceride in the subgroups of males, females, age < 60 years and age ≥ 60 years and remained significant of the total cholesterol concentration in the subgroups of males, age < 60 years and age < 60 years (Fig. 3). After excluding participants with abnormal test results, short-term $PM_{2.5}$ exposure and its effect on triglyceride could be modified by age, the effects were stronger for the subgroup of age ≥ 60 years (Supplementary Table 3).

Indicators	Complete study population[a]		Persons with normal blood lipid levels[a]	
	Single-pollutant model	Multi-pollutant model[b]	Single-pollutant model	Multi-pollutant model[b]
Triglyceride (%)	− 0.5447 (− 0.7873, − 0.3015)	0.3081 (− 0.0495, 0.6669)	− 0.5184 (− 0.7235, − 0.3128)	0.2006 (− 0.1024, 0.5045)
Low-density lipoprotein cholesterol (mmol/L)	0.0127 (0.0099, 0.0156)	0.0194 (0.0152, 0.0237)	0.0096 (0.0068, 0.0124)	0.0134 (0.0093, 0.0175)
High-density lipoprotein cholesterol (mmol/L)	0.0002 (− 0.0010, 0.0013)	− 0.0022 (− 0.0039, − 0.0005)	− 0.0002 (− 0.0014, 0.0011)	− 0.0017 (− 0.0036, 0.0001)
Total cholesterol (mmol/L)	0.0095 (0.0053, 0.0136)	0.0284 (0.0224, 0.0345)	0.0057 (0.0019, 0.0095)	0.0209 (0.0153, 0.0265)

Table 2. Estimated changes in the blood lipids for every 10 μg/m^3 increase in $PM_{2.5}$. [a]Persons with normal blood lipid levels were people with total cholesterol < 6.2 mmol/L, triglyceride < 2.3 mmol/L, low-density lipoprotein cholesterol < 4.1 mmol/L and high-density lipoprotein cholesterol > 1.0 mmol/L. We applied lag 0–6 days for triglyceride, lag 0–7 days for low-density lipoprotein cholesterol, lag 0–5 days for high-density lipoprotein cholesterol, and lag 0–7 days for total cholesterol. [b]Adjusted for SO_2, NO_2 and O_3.

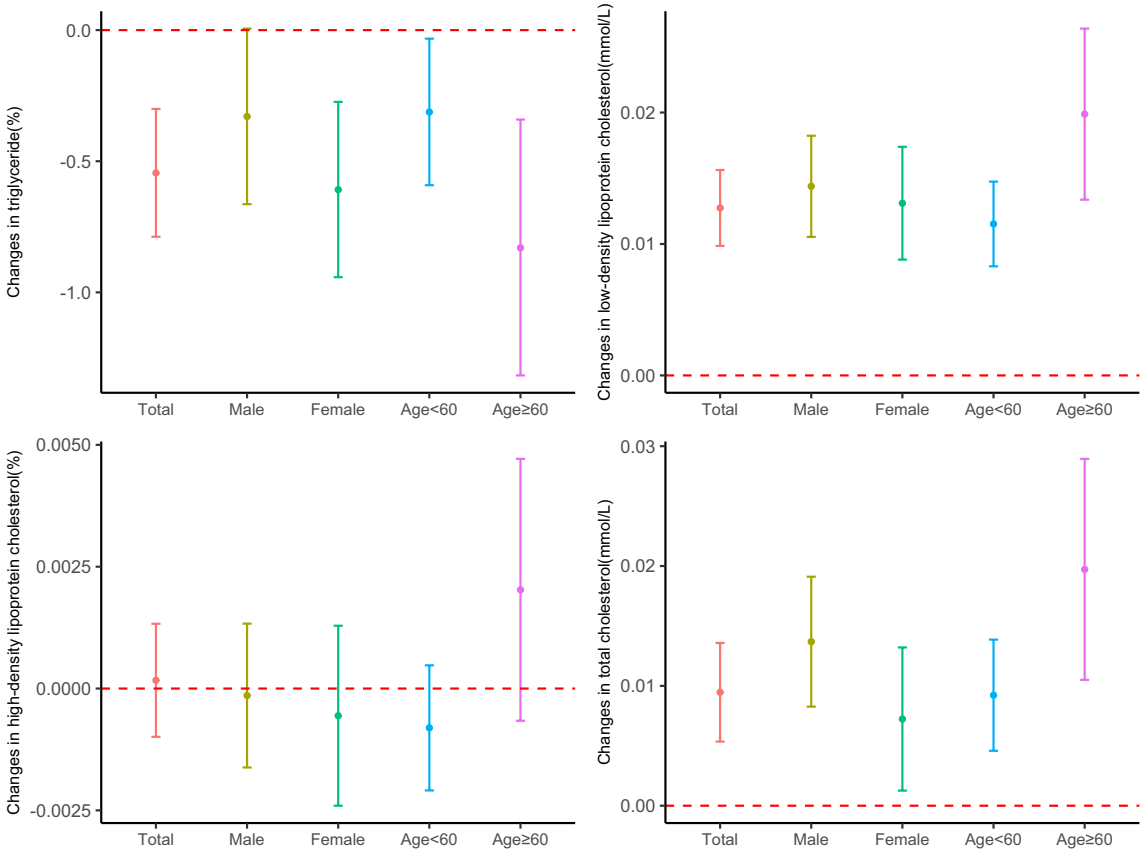

Figure 2. Estimated changes (95% confidence intervals) in the blood lipids for every 10 μg/m^3 increase in $PM_{2.5}$ among the entire population. We applied lag 0–6 days for triglyceride, lag 0–7 days for low-density lipoprotein cholesterol, lag 0–5 days for high-density lipoprotein cholesterol, and lag 0–7 days for total cholesterol.

The effects of $PM_{2.5}$ on the blood lipid at different lag days. The associations between $PM_{2.5}$ and triglyceride, low-density lipoprotein cholesterol concentration and total cholesterol concentration were robust at different lag days. And the effects of $PM_{2.5}$ exposure on triglyceride, low-density lipoprotein cholesterol concentration, and total cholesterol concentration were strongest at lag 0–4 days, lag 0–7 days, lag 0–4 days and lag 0-7 days. However, no significant association was observed between $PM_{2.5}$ and the high-density lipoprotein cholesterol concentration at different lag days. (Table 3).

The effects of $PM_{2.5}$ on the blood lipids in multi-pollutant models. For a 10 μg/m^3 increase in $PM_{2.5}$, the low-density lipoprotein cholesterol concentration increased by 0.0194 mmol/L (95% CI 0.0152, 0.0237), the high-density lipoprotein cholesterol concentration decreased by 0.0022 mmol/L (95% CI − 0.0039, − 0.0005) and the total cholesterol concentration increased by 0.0284 mmol/L (95% CI 0.0224, 0.0345). No significant association was observed between $PM_{2.5}$ and the triglyceride. In persons with normal test results, for a

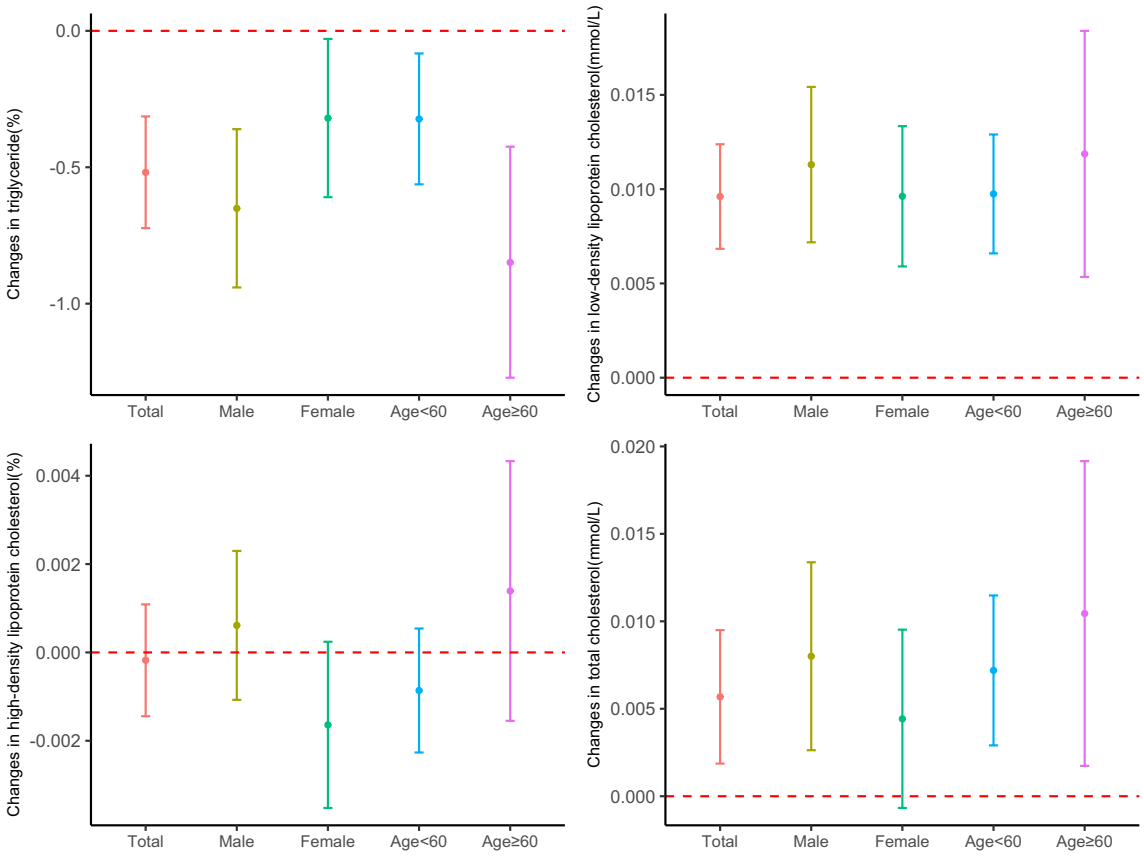

Figure 3. Estimated changes (95% confidence intervals) in the blood lipids for every 10 μg/m^3 increase in $PM_{2.5}$ among persons with normal blood lipid levels. We applied lag 0–6 days for triglyceride, lag 0–7 days for low-density lipoprotein cholesterol, lag 0–5 days for high-density lipoprotein cholesterol, and lag 0–7 days for total cholesterol.

10 μg/m^3 increase in $PM_{2.5}$, the low-density lipoprotein cholesterol concentration increased by 0.0134 mmol/L (95% CI 0.0093, 0.0175) and the total cholesterol concentration increased by 0.0209 mmol/L (95% CI 0.0153, 0.0265). No significant association was observed between $PM_{2.5}$ and the triglyceride and high-density lipoprotein cholesterol concentration (Table 2).

The effects of $PM_{2.5}$ on hyperlipidemia. We converted lipid-related indicators into binary variables (non- hyperlipidemia and hyperlipidemia) to calculate risks of hyperlipidemia associated with $PM_{2.5}$ exposure. As a result, when $PM_{2.5}$ increased 10 μg/m^3, the OR (95% CIs) was 1.003 (95% CI 1.001, 1.004), and it was still significant in the subgroups of males and age < 60 years (Supplementary Table 4). We also converted lipid-related indicators into binary variables (normal and abnormal) to calculate the OR and 95% CI of blood lipids for every 10 μg/m^3 increase in $PM_{2.5}$. We applied lag 0–6 days for triglyceride, lag 0–3 days for low-density lipoprotein cholesterol, lag 0–5 days for high-density lipoprotein cholesterol and lag 0–7 days for total cholesterol. When $PM_{2.5}$ increased 10 μg/m^3, the OR (95% CIs) for triglyceride, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol and total cholesterol was 0.998 (95% CI 0.996, 0.999), 1.001 (95% CI 1.000, 1.001), 1.002 (95% CI 1.001, 1.003) and 1.003 (95% CI 1.001, 1.004) (Supplementary Table 5).

The effects of other air pollutions (including PM_{10}, SO_2, NO_2, O_3, and CO) on the blood lipids are shown in the Supplementary Appendix (Supplementary Tables 6–15).

Discussion

In this study, we found that $PM_{2.5}$ was positively correlated with low-density lipoprotein cholesterol concentration and total cholesterol concentration, while being negatively correlated with triglyceride. Findings from our study provide evidence of the potential harmful effects of $PM_{2.5}$ exposure on blood lipids. To our knowledge, this is the largest population-based study to explore the association between short-term $PM_{2.5}$ exposure and blood lipids, and will provide new empirical for the effect of short-term air pollutant exposure on health.

Previous studies have been heterogeneous with some showing similar results[17,18] while others harmful. Study design and differential exposure may partially explain these differences. For example, long-term exposure to $PM_{2.5}$ was positively associated with triglyceride concentration in a study in Perth[6]. Distinct durations of exposure may partially explain this inconsistency and our analysis specifically evaluated short-term effect of $PM_{2.5}$ exposure while many studies concentrated on longer-term effect of $PM_{2.5}$[6]. Previous studies in rural areas have

Indicators and lag days	Entire population	Persons with normal blood lipid levels[a]
Triglyceride (%)		
0–1 days	− 0.3718 (− 0.5286, − 0.2147)	− 0.2735 (− 0.4070, − 0.1399)
0–2 days	− 0.5087 (− 0.6875, − 0.3296)	− 0.4713 (− 0.6235, − 0,.3188)
0–3 days	− 0.5394 (− 0.7371, − 0.3414)	− 0.5895 (− 0.7580, − 0.,4206)
0–4 days	− 0.5895 (− 0.8039, − 0.3745)	− 0.6790 (− 0.8621, − 0.4956)
0–5 days	− 0.5102 (− 0.7400, − 0.2798)	− 0.5734 (− 0.7701, − 0.3764)
0–6 days	− 0.5447 (− 0.7873, − 0.3015)	− 0.5184 (− 0.7235, − 0.3128)
0–7 days	− 0.5376 (− 0.7873, − 0.2874)	− 0.5297 (− 0.7341, − 0.3185)
Low-density lipoprotein cholesterol (mmol/L)		
0–1 days	0.0055 (0.0037, 0.0073)	0.0020 (0.0002, 0.0037)
0–2 days	0.0051 (0.0030, 0.0071)	0.0011 (-0.0009, 0.0031)
0–3 days	0.0064 (0.0041, 0.0086)	0.0022 (0.0000, 0.0044)
0–4 days	0.0074 (0.0049, 0.0099)	0.0038 (0.0014, 0.0062)
0–5 days	0.0082 (0.0056, 0.0108)	0.0050 (0.0024, 0.0076)
0–6 days	0.0104 (0.0076, 0.0132)	0.0071 (0.0044, 0.0099)
0–7 days	0.0127 (0.0099, 0.0156)	0.0096 (0.0068, 0.0124)
High-density lipoprotein cholesterol (mmol/L)		
0–1 days	0.0005 (− 0.0003, 0.0013)	0.0002 (− 0.0006, 0.0011)
0–2 days	0.0004 (− 0.0005, 0.0013)	0.0001 (− 0.0009, 0.0011)
0–3 days	0.0004 (− 0.0006, 0.0014)	0.0001 (− 0.0009, 0.0012)
0–4 days	0.0008 (− 0.0003, 0.0019)	0.0005 (− 0.0007, 0.0017)
0–5 days	0.0002 (− 0.0010, 0.0013)	− 0.0002 (− 0.0014, 0.0011)
0–6 days	0.0000 (− 0.0013, 0.0012)	− 0.0005 (− 0.0019, 0.0008)
0–7 days	0.0005 (− 0.0008, 0.0018)	− 0.0002 (− 0.0016, 0.0011)
Total cholesterol (mmol/L)		
0–1 days	0.0067 (0.0041, 0.0092)	0.0033 (0.0010, 0.0056)
0–2 days	0.0073 (0.0044, 0.0102)	0.0031 (0.0005, 0.0057)
0–3 days	0.0076 (0.0044, 0.0108)	0.0032 (0.0002, 0.0061)
0–4 days	0.0080 (0.0046, 0.0114)	0.0039 (0.0007, 0.0070)
0–5 days	0.0083 (0.0045, 0.0120)	0.0051 (0.0018, 0.0085)
0–6 days	0.0086 (0.0047, 0.0126)	0.0054 (0.0017, 0.0091)
0–7 days	0.0095 (0.0053, 0.0126)	0.0057 (0.0019, 0.0095)

Table 3. Estimated changes in the blood lipids for every 10 μg/m^3 increase in $PM_{2.5}$ at different lag days. Adjusted for time, day of the week, sex, age, temperature, wind speed and relative humidity. [a]Persons with normal blood lipid levels were people with total cholesterol < 6.2 mmol/L, triglyceride < 2.3 mmol/L, low-density lipoprotein cholesterol < 4.1 mmol/L and high-density lipoprotein cholesterol > 1.0 mmol/L.

demonstrated that short-term $PM_{2.5}$ exposure was positively associated with triglyceride concentration[19] and negatively associated with total cholesterol concentration[20], inconsistent with our results. Differences in lifestyle and air quality between rural and urban areas may influence outcomes[17,18,21]. For example, Omega-3 fatty acids may attenuate cardiovascular effects of short-term exposure to ambient air pollution[22]. In our study, $PM_{2.5}$ was negatively correlated with triglyceride in the single pollutant model, and positively correlated with triglyceride in the multi-pollutant model, regardless of the overall population or the population with normal blood lipids. This suggested that other air pollutants may alter the associations between $PM_{2.5}$ and triglyceride, which requires further research.

In our study, $PM_{2.5}$ was positively associated with the low-density lipoprotein cholesterol concentration. Most previous studies investigated long-term, rather than short-term, exposure effects of $PM_{2.5}$ to low-density lipoprotein cholesterol concentration[23–25]. Our study provides new evidence for the effect of short-term exposure. Long-term $PM_{2.5}$ exposure was shown to be negatively associated with high-density lipoprotein cholesterol concentration[18], inconsistent with our findings, indicating that differential exposure durations (short- versus long-term) may also have an impact on the results.

Previous studies have been heterogenous concerning the effect of $PM_{2.5}$ on total cholesterol concentration. A study in Shanghai showed no significant association between total cholesterol and $PM_{2.5}$[26]. However, another study is consistent with our results[27], showing $PM_{2.5}$ exposure was associated with an elevated total cholesterol concentration. The difference of exposure durations may explain the inconsistency because we evaluated the short-term effect of $PM_{2.5}$, while the study in shanghai explored the long-term exposure. A study among college students[20] showed that short-term $PM_{2.5}$ exposure was negatively associated with total cholesterol concentration, the inconsistency may be attributed to the difference of sample size and age. Recent research showed that

long- term $PM_{2.5}$ exposure was negatively correlated with the risk of hyperlipidemia[28], however, in our study, the OR of every 10 μg/m^3 increase in $PM_{2.5}$ for hyperlipidemia population was 1.009, which suggested that short-term $PM_{2.5}$ exposure was a risk factor for hyperlipidemia. Different life-styles and areas may explain the inconsistency. In our study, the effect of short-term $PM_{2.5}$ exposure on the low-density lipoprotein cholesterol concentration and total cholesterol concentration could be modified by age and the older were more susceptible to $PM_{2.5}$ exposure, which may be due to hypometabolism and/or hypoimmunity. Previous studies support these findings[29–31].

Our study has several limitations. First, our study was a time-series study, limiting our ability to account for reverse causation or time-specific confounding. Second, the fixed environmental monitoring station was used to estimate personal $PM_{2.5}$ exposure, which cannot be equated entirely with individual exposure. Lastly, although our dataset was large and community-based, we did not have available several other characteristics which may be associated with $PM_{2.5}$ exposure and blood lipid-related indicators, such as exercise, smoking, and medical history. Therefore, unmeasured and residual confounding is possible.

Conclusions

$PM_{2.5}$ was positively correlated with low-density lipoprotein cholesterol and total cholesterol, and negatively correlated with triglyceride, indicating $PM_{2.5}$ can potentially affect health through blood lipid levels.

Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Author contributions

Q.L., Z.W. and Z.L. conceived the study, analyzed the data and drafted the manuscript; Q.L. and X.P. participated in the study design; X.M., J.L. and C.W. implemented the field investigation; B.T., L.M., L.Z., W.L. and B.Z. 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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Competing interests

The authors declare no competing interests.

Additional information

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