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Long-term effects of ambient air pollutants to blood lipids and dyslipidemias in a Chinese rural population☆

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ABSTRACT

Both air pollution and dyslipidemias contributed to large number of deaths and disability-adjusted life lost years. Long-term air pollution exposure was related to changed blood lipids and risk of dyslipidemias. This study was designed to evaluate relationships between air pollutants, blood lipids and prevalence of dyslipidemias in a Chinese rural population exposed to high-level air pollution based on baseline data of The Henan Rural Cohort study. An amount of 39,057 participants from rural areas in China were included. The 3-year average exposure of air pollutants ($PM_{2.5}$, PM_{10}, NO_2) was estimated by a spatiotemporal model. Logistic and linear regression models were employed to explore relationships between air pollutants, blood lipids (TC, TG, HDL-C and LDL-C) and prevalence of dyslipidemias. The three-year concentration of $PM_{2.5}$, PM_{10} and NO_2 was 72.8 ± 2.3 μg/m^3, 131.5 ± 5.7 μg/m^{3}and 39.1 ± 3.1 μg/m^3, respectively. Overall, increased air pollution exposure was related to increased TC and LDL-C, while decreased TG and HDL-C. Each 1-μg/m^3 increment of $PM_{2.5}$ was related to 0.10% (0.07%−0.19%) increase in TC, 0.63% (0.50%−0.77%) increase in LDL-C, 2.93% (2.70%−3.16%) decrease in TG, 0.49% (0.38%−0.60%) decrease in HDL-C; and 5.7% (95%CI: 3.7%−7.6%), 4.0% (95%CI: 2.1%−6.0%) and 3.8% (95%CI: 2.5%−5.1%) increase in odds for hypercholesterolemia, hyperbetalipoproteinemia and hypoalphalipoproteinemia, respectively. Stronger associations were found in male and older participants. Findings suggest that air pollutants were associated with changed blood lipid levels and higher risk of dyslipidemias among rural population. Male and elder people should pay more attention to personal safety protection.

1. Introduction

Dyslipidemias referring to the elevated levels of total cholesterol (TC), triglyceride (TG), and low-density lipoprotein cholesterol (LDL-C) and decreased level of high-density lipoprotein cholesterol (HDL-C), is a modifiable and crucial risk factor for cardiovascular disease, myocardial infarction and ischemic stroke (Franssen et al., 2011; Lee et al., 2012; Toth, 2008). Current evidence suggested that blood lipid levels was associated with total mortality, cardiovascular disease mortality, and risk of ischemic stroke (Anderson et al., 1987; Tirschwell et al., 2004). According to Global Burden of Disease Study 2017 (GBD, 2018), high LDL-C accounted for 4.32 million deaths and 94.9 million disability-adjusted life lost years (DALYs). Dyslipidemias has been a severe threat for all general population. According to China Nutrition and Health Survey (CNHS) between 2010 and 2012, the prevalence of dyslipidemia reached 40.8% in rural population compared that in urban population was 39.9%, and the prevalence of hypercholesterolemia, hypertriglyceridemia, hypoalphalipoproteinemia and hyperbetalipoproteinemia in rural

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population has reached to 4.3%, 12.2%, 19.7%, and 11.0%, respectively (Jile and Wang., 2016).

Hazards of air pollution was an essential public problem worldwide. A report published on Nature indicated that about 3.45 million die on account of air pollution (Zhang et al., 2017); and Global Burden of Disease Study (GBD, 2018) demonstrated particulate matter (PM) pollution led to 4.58 million (95% uncertainty interval [UI]: 4.13–5.03) deaths and 143 million (95%UI: 129–159) DALYs in 2017. Lots of studies investigated relationships between air pollutants and blood lipids, but the results were different (McGuinn et al., 2019; Poursafa et al., 2014; Shanley et al., 2016; Vecchi et al., 2004; Wallwork et al., 2017; Yitshak Sade et al., 2016). A recent study in U.S. showed that each 1-μg/m^3 increment of particles with diameters ≤2.5 μm ($PM_{2.5}$) corresponded to 1.62 (95% confidence interval [CI]:1.13–2.11) mg/dL, 3.29 (95%CI: 1.67, 4.92) mg/dL, 1.70 (95%CI: 1.02, 2.37) mg/dL and 0.61 (95%CI: 0.07, 1.13) mg/dL increase in TC, TG, LDL-C and HDL-C, respectively (McGuinn et al., 2019). Besides, another study based on two cohorts in Europe showed that an interquartile range (7.4 μg/m^3) increase of nitrogen dioxide (NO_2) was related to 2.2% (95%CI: 1.6%–2.7%) increase in TG and 0.5% (95%CI: 0.3%–0.8%) increase in HDL-C (Cai et al., 2017). However, Wallwork et al. (2017) demonstrated that each 1-μg/m^3 increment of $PM_{2.5}$ corresponded to higher risk of hypertriglyceridemia (hazard ratio (HR) = 1.14, 95%CI: 1.00, 1.30), whereas not with hypoalphalipoproteinemia (HR = 0.98, 95%CI: 0.85, 1.13).

Air pollution remained a serious threaten for China. As Global Burden of Disease Study reported, $PM_{2.5}$ accounted for 1.1 million (95%UI:1.0–1.8) deaths in China, compared with 88,400 (95%UI: 66,800-115,000) deaths in U.S. (Cohen et al., 2017). However, few researches studied the associations in China population, let alone the application to rural areas. In Guo et al. (2016) study, the mean concentrations of $PM_{2.5}$ in urban and rural areas was 71.7 μg/m^3 and 68.9 μg/m^3, respectively, both were much higher than World Health Organization (WHO) recommended. Residential energy use, dust storms, agriculture and traffic are the main sources of $PM_{2.5}$ in rural Asian environments (Faizan and Thakur, 2019; Gautam et al., 2016). Lelieveld et al. (2015) reported that agriculture was an important anthropogenic source category contributing one-fifth premature mortality linked to outdoor air pollution. In rural areas, lower household income, lower education levels, limited availability of food and limited access to health care resources make rural population more susceptible to adverse effect of air pollution compared to urban population. Therefore, it was of great significance to explore associations between air pollution, blood lipids and dyslipidemias in Chinese rural population which accounted for over 50% population.

To address this issue, current study aimed to explore associations between air pollutants ($PM_{2.5}$, particles with diameters ≤ 10 μm (PM_{10}), NO_2), blood lipid levels and dyslipidemias in a rural region using baseline data of The Henan Rural Cohort study. Then, we examine potential modification effects of age, sex, BMI and lifestyle characteristics on these associations.

2. Materials and methods

2.1. Study population

Population from The Henan Rural Cohort study (Registration number: ChiCTR-OOC-15006699) were included. Briefly, participants were selected from five rural areas (Suiping, Yuzhou, Xinxiang, Tongxu and Yima) of Henan province in China, by using multistage stratified random cluster sampling. Detailed information has been reported (Liu et al., 2019; Tian et al., 2018; Zhang et al., 2018a). During 2015–2017, 39,259 participants aged from 18 to 79 years were included, with a response rate of 93.7%. Among all participants, 138 participants without blood lipids data and 64 participants without key demographic characteristics were excluded, leaving 39,057 participants available for the present analyses ultimately. Informed consents were acquired from each individual before investigation. Current study was approved by the Zhengzhou University Life Science Ethics Committee (Code: [2015] MEC (S128)).

2.2. Data collection

Baseline information including demographic characteristics (age, sex, incomes, education, etc.), lifestyle (smoking, alcohol drinking, vegetable and fruit intake, meat and poultry intake, physical activity, etc.) and family history (diabetes, hypertension, dyslipidemia, stroke, coronary heart disease, etc.) were gathered using standardized questionnaires. Vegetable and fruit intake was classified by a person who consumed an average of 500 g vegetable and/or fruit per day; meat and poultry intake was classified by a person who took an average of 75 g meat of livestock and poultry per day according to dietary guidelines for Chinese residents. Physical activity was evaluated by International Physical Activity Questionnaire (IPAQ2001). Details of the measurements and definitions have been reported (Liu et al., 2019; Liu et al., 2018; Tian et al., 2018). Meanwhile, data of height and weight were measured twice by two investigators independently on the day of the investigation, then body mass index (BMI, kg/m^2) was obtained.

Blood samples were obtained from individuals after at least 8 h overnight fasting. HDL-C and LDL-C was analyzed by the direct assay method. TC by cholesterol oxidase method and TG by enzymatic method, using CobasC501, (Roche Diagnostics GmbH, Switzerland).

2.3. Exposure data

Concentrations of $PM_{2.5}$, PM_{10} and NO_2 were estimated using a spatiotemporal model with satellite remote sensing, meteorological data and land use information at a 0.1-degree spatial resolution. Detailed measurement methods have been reported (Chen et al., 2018a; Chen et al., 2018b). We employed a random forests model to predict air pollutants concentrations using ground-level measurements, aerosol optical depth data and some spatiotemporal predictors (afforested cover, temperature, rainfall, etc.). The adjusted coefficient of determination (R^2) and Root Mean Squared Error (RMSE) for annual prediction was 0.82 and 9 μg/m^3. Individual concentrations of $PM_{2.5}$, PM_{10} and NO_2 were estimated according to residential addresses. Three-year average air pollution exposure before participants participated in this survey was defined as long-term exposure, while five-year average air pollution exposures were used in sensitivity analyses.

2.4. Definition of dyslipidemia

According to Guidelines on Prevention and Treatment of Dyslipidemia for Chinese Adults (Chinese guidelines, 2007), participants were defined as (a) hypercholesterolemia when TC ≥ 6.22 mmol/L; (b) hypertriglyceridemia when TG ≥ 2.26 mmol/L; (c) hypoalphalipoproteinemia when HDL-C <1.04 mmol/L; (d) hyperbetalipoproteinemia when LDL-C ≥4.14 mmol/L.

2.5. Statistical analysis

Effects(β) and 95%CIs were obtained by multiple linear regression analyses for each 1-μg/m^3 increment of $PM_{2.5}$, PM_{10}, NO_2 and

naturally log-transformed blood lipids. Then percent differences with corresponding 95%CIs were calculated by using $100 \times [\exp(\beta)-1]$. Meanwhile, odds ratios (ORs) with 95%CIs were quantified from binary logistic regression model to evaluate relationships between air pollutants and dyslipidemias. In crude model, no covariates were included. In adjusted model, age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, physical exercise, fruit and vegetable intake, meat and poultry intake and dyslipidemia family history were included as covariates.

In addition, a series of stratified analyses were performed to estimate the underlying modification effects of sex (male *v.s.* female), age (<65 years *v.s.* ≥65 years), BMI (<25 kg/m^2 *v.s.* ≥25 kg/m^2) and lifestyle characteristics. The statistical difference was tested by including an interaction term in the multivariate regression based on adjusted model.

We undertook a string of sensitivity analyses to test the steadiness of findings. First, we estimated associations of air pollutants, blood lipids and dyslipidemias by excluding individuals who took lipid-lowering drugs. Second, we evaluated the associations after excluded participants with hypertension and/or diabetes. Third, we explored the associations of different exposure term (one year and five years). Then, we estimated associations of air pollutants, blood lipids and dyslipidemias by excluding smokers and/or drinkers.

Binary logistic regression and linear regression analyses were performed using SAS, stratified analyses were performed using R, and statistically significance was defined as $P < 0.05$.

3. Results

3.1. General characteristics

Table 1 displays all general characteristics of all objects. In our study, 15,365 males and 23,692 females were included. The mean age was 56 years, while the average BMI was 24.8 kg/m^2. Among all, 89.76% participants were married or cohabited. Only 15.35% of participants had an education of high school or higher. About 19% participants were current smokers, while 17.93% participants were current alcohol drinkers. Meanwhile, only 3.53% participants had a family history of dyslipidemia. The mean concentrations of TC, TG, HDL-C and LDL-C were 4.8 mmol/L, 1.7 mmol/L, 1.3 mmol/L, 2.9 mmol/L, respectively. The prevalence of hypercholesterolemia, hypertriglyceridemia, hypoalphalipoproteinemia, and hyperbetalipoproteinemia were 7.2%, 18.8%, 19.1%, and 6.7%, respectively.

Table 1
General characteristics of participants in The Henan Rural Study.

Variable	Total n = 39,057	Men n = 15,365	Women n = 23,692
Age (years) , mean ± SD[#]	56 ± 12	57 ± 12	55 ± 12
BMI (kg/m^2) , mean ± SD[a#]	24.8 ± 3.6	24.5 ± 3.5	25.0 ± 3.6
Marital status, n (%)			
Married/cohabitation	35059 (89.8)	13819 (89.9)	21240 (89.6)
Unmarried/divorced/widowed	3998 (10.2)	1546 (10.1)	2452 (10.4)
Per capita monthly income, n (%)[#]			
< 500RMB	13950 (35.7)	5571 (36.3)	8379 (35.4)
500RMB~	12849 (32.9)	4891 (31.8)	7958 (33.6)
≥1000RMB	12258 (31.4)	4903 (31.9)	7355 (31.0)
Education level, n (%)[#]			
Elementary school or below	17504 (44.8)	5202 (33.9)	12302 (51.9)
Junior high school	15556 (39.8)	7100 (46.2)	8456 (35.7)
High school or above	5997 (15.4)	3063 (19.9)	2934 (12.4)
Smoking, n (%)[#]			
Nonsmoker	28470 (72.9)	4866 (31.7)	23604 (99.6)
Former smoker	3165 (8.1)	3142 (20.4)	23 (0.1)
Current smoker	7422 (19.0)	7357 (47.9)	65 (0.3)
Drinking, n (%)[#]			
Nondrinking	30234 (77.4)	7219 (47.0)	23015 (97.1)
Former drinking	1822 (4.7)	1758 (11.4)	64 (0.3)
Current drinking	7001 (17.9)	6388 (41.6)	613 (2.6)
Meat and poultry intake, n (%)[#]			
<75 g/d	31638 (81.0)	11540 (75.1)	20098 (84.8)
≥75 g/d	7419 (19.0)	3825 (24.9)	3594 (15.2)
Vegetable and fruit intake, n (%)[a#]			
<500 g/d	22765 (58.3)	8805 (57.3)	13960 (58.9)
≥500 g/d	16292 (41.7)	6560 (42.7)	9732 (41.1)
Physical activity, n (%)[#]			
Low	12627 (32.3)	5468 (35.6)	7159 (30.2)
Moderate	14745 (37.8)	4275 (27.8)	10470 (44.2)
High	11685 (29.9)	5622 (36.6)	6063 (25.6)
Family history of hyperlipidemia, n (%)[#]	1379 (3.5)	447 (2.9)	932 (3.9)
TC (mmol/L), mean ± SD[#]	4.8 ± 1.0	4.6 ± 1.0	4.8 ± 1.0
TG (mmol/L), mean ± SD[#]	1.7 ± 1.1	1.7 ± 1.2	1.7 ± 1.1
HDL-C (mmol/L), mean ± SD[#]	1.3 ± 0.3	1.3 ± 0.3	1.4 ± 0.3
LDL-C (mmol/L), mean ± SD[#]	2.9 ± 0.8	2.8 ± 0.8	2.9 ± 0.8
Hypercholesterolemia, n (%)[#]	2825 (7.2)	831 (5.4)	1994 (8.4)
Hypertriglyceridemia, n (%)	7330 (18.8)	2815 (18.3)	4515 (19.1)
Hypoalphalipoproteinemia, n(%)[#]	7477 (19.1)	3910 (25.5)	3567 (15.1)
Hyperbetalipoproteinemia, n (%)[#]	2624 (6.7)	890 (5.8)	1734 (7.3)

Abbreviations: SD, standard deviation; BMI, body mass index; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.
[#]Statistically significant difference between men and women (p < 0.01).
[a] Missing data.

Table 2
Three-year average concentrations of air pollutants.

Exposure	Mean	SD	Interquartile rang	Min	*P25*	*P50*	*P75*	Max
NO_2 ($\mu g/m^3$)	39.1	3.1	5.0	31.0	35.3	35.9	40.3	43.3
$PM_{2.5}$ ($\mu g/m^3$)	72.8	2.3	3.5	68.0	69.8	70.5	73.3	77.0
PM_{10} ($\mu g/m^3$)	131.5	5.7	9.2	122.4	123.9	125.4	133.1	139.8

Abbreviations: SD, standard deviation.

Table 2 summarizes the three year average air pollution concentration of study participants. The three year average exposure of NO_2 was 39.1 ± 3.1 $\mu g/m^3$, with a range of 31.0–43.3 $\mu g/m^3$, $PM_{2.5}$ was 72.8 ± 2.3 $\mu g/m^3$, ranging from 68.0 to 77.0 $\mu g/m^3$, and PM_{10} was 131.5 ± 5.7 $\mu g/m^3$, ranging from 122.4 to 139.8 $\mu g/m^3$.

3.2. Associations between air pollutants and blood lipids

Fig. 1, Table S2 displays the associations between air pollutants and blood lipids. Higher $PM_{2.5}$, PM_{10}, and NO_2 exposure were related to elevated TC and LDL-C, while related to decrease TG and HDL-C. Each 1 $\mu g/m^3$ increment of $PM_{2.5}$ was related to 0.10% (95% CI: 0.07%–0.19%) increment in TC and 0.63% (95%CI: 0.50%–0.77%) increment in LDL-C, while a 2.93% (95%CI: 2.70%–3.16%) decrease in TG and 0.49% (95%CI: 0.38%–0.60%) decrease in HDL-C in adjusted model.

Fig. 2 and Tables S3–S5 presents detailed stratified results by sex, age, BMI and lifestyle characteristics. Significant interactions of sex and age were evident, while few significant interactions of BMI existed. The estimated effects of air pollution among males were stronger than females. Besides, we observed stronger associations among participants over 65 years. Furthermore, statistically significant interaction of BMI on associations between air pollution and LDL-C was presented only in overweight individuals. As for lifestyle characteristics, few significant interactions of meat and poultry intake existed between air pollutants and TG. Individuals of excessive meat and poultry intake were susceptible to the effects of air pollutants. We also observed significant interactions with vegetable and fruit intake and physical activity.

3.3. Associations between air pollutants and dyslipidemias

Table 3 displays the associations between air pollutions and dyslipidemias. In crude and adjusted model, higher $PM_{2.5}$, PM_{10}, and NO_2 exposure was associated with higher risk of hypercholesterolemia, hyperbetalipoproteinemia and hypoalphalipoproteinemia. Each 1 $\mu g/m^3$ increase of $PM_{2.5}$ was related to increased risks of hypercholesterolemia (1.057, 95%CI: 1.037–1.076), hyperbetalipoproteinemia (1.040, 95%CI: 1.021–1.060) and hypoalphalipoproteinemia (1.038, 95%CI: 1.025–1.051) in adjusted model.

In stratified analyses (Tables 4–6), we detected statically significant interactions of sex and age. Apart from hypoalphalipoproteinemia, air pollutants brought about more serious adverse effects among males. Besides, participants over 65 years had statistically stronger associations than the others. However, there were none statistically significant interactions between air pollutions and BMI. We also observed similar significant interactions of lifestyle characteristics between air pollutants and dyslipidemias. Consist with associations between $PM_{2.5}$ and TC levels, a relatively higher OR of $PM_{2.5}$ and hypercholesterolemia were observed among adequate vegetable and fruit intake

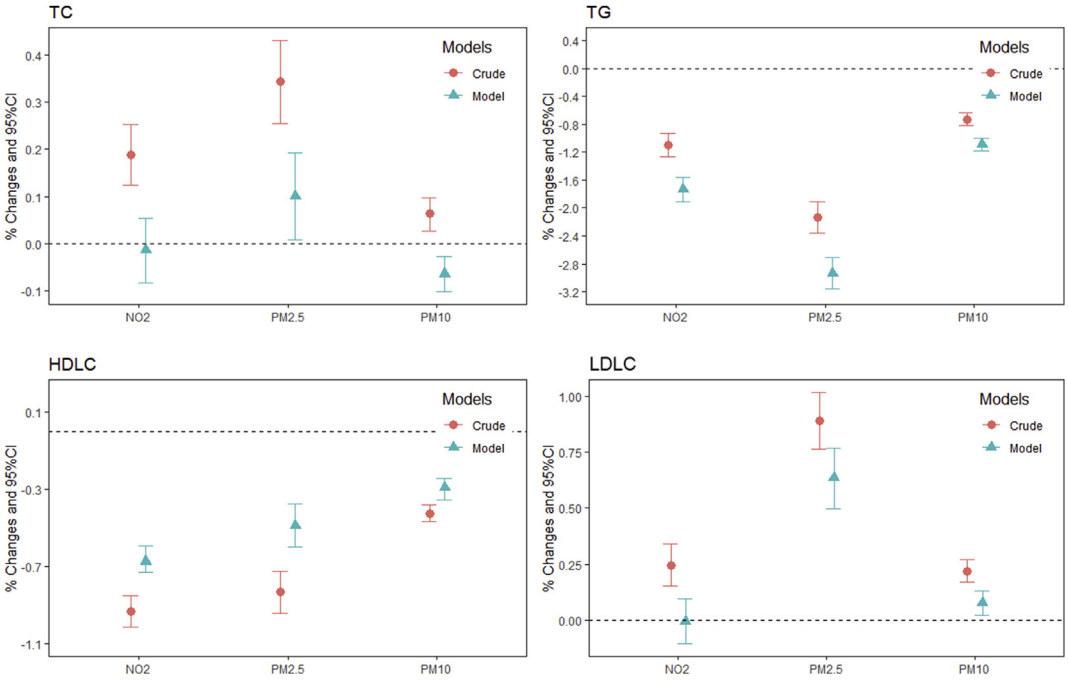

Fig. 1. Associations between per 1-$\mu g/m^3$ increment of air pollutions and blood lipid levels. Abbreviations: CI, confidence interval; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. Model: Covariates included age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, meat and poultry intake, vegetable and fruit intake, physical activities and family history of dyslipidemia.

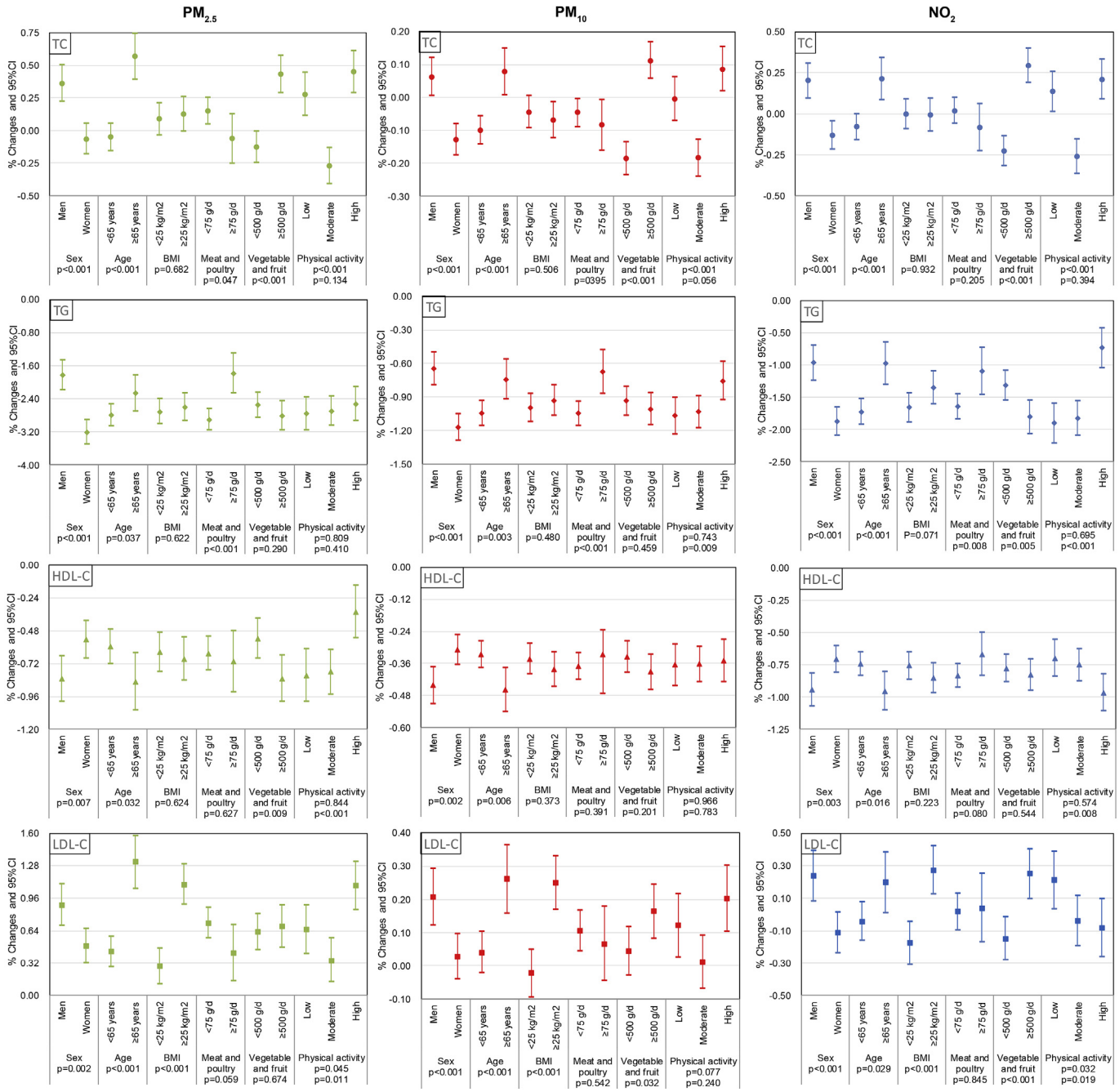

Fig. 2. Interactions of sex, age, BMI and lifestyle characteristic on associations between per 1-μg/m^3 increment of air pollutions and blood lipid levels. Abbreviations: CI, confidence interval; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. Covariates included age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, meat and poultry intake, vegetable and fruit intake, physical activities and family history of dyslipidemia.

individuals.

3.4. Sensitivity analysis

Similar with the whole results of full data analyses, sensitivity analyses showed that concentration of air pollutants was related to changed blood lipids and increased risk of dyslipidemias, where participants of taking lipid lowering drugs, participants with hypertension and/or diabetes and smokers and/or drinkers were excluded, and data of different term air pollution exposure were used. The detailed results are displayed in Table S6 and Table S7.

4. Discussion

This study provided new evidence on the deleterious effects of $PM_{2.5}$, PM_{10} and NO_2 on blood lipids and dyslipidemias, which had implications for prevention of cardiovascular disease (CVD) events. Among 39,057 participants of our study, high $PM_{2.5}$, PM_{10} and NO_2 were related to changed blood lipids and higher risk of hypercholesterolemia, hyperbetalipoproteinemia and hypoalphalipoproteinemia. Besides, male and older individuals were more susceptive to the adverse effects of air pollutants.

In accordance with previous studies (Bind et al., 2016; McGuinn et al., 2019; Shanley et al., 2016; Sorensen et al., 2015; Yang et al.,

Table 3
Associations between per 1-μg/m^3 increment of air pollutions and dyslipidemias.

	Hypercholesterolemia	Hypertriglyceridemia	Hypoalphalipo-proteinemia	Hyperbetalipo-proteinemia
	OR (95%CI)	OR (95%CI)	OR (95%CI)	OR (95%CI)
$PM_{2.5}$				
Crude Model	1.081 (1.063, 1.099)	0.944 (0.934, 0.955)	1.056 (1.044, 1.067)	1.060 (1.043, 1.079)
Adjusted Model	1.057 (1.037, 1.076)	0.912 (0.901, 0.924)	1.038 (1.025, 1.051)	1.040 (1.021, 1.060)
PM_{10}				
Crude Model	1.024 (1.017, 1.031)	0.981 (0.975, 0.985)	1.028 (1.023, 1.032)	1.009 (1.002, 1.016)
Adjusted Model	1.011 (1.003, 1.019)	0.967 (0.962, 0.972)	1.022 (1.016, 1.027)	0.997 (0.989, 1.005)
NO_2				
Crude Model	1.056 (1.042, 1.069)	0.973 (0.965, 0.981)	1.063 (1.054, 1.071)	1.000 (0.988, 1.013)
Adjusted Model	1.033 (1.019, 1.048)	0.948 (0.939, 0.957)	1.051 (1.041, 1.061)	0.976 (0.963, 0.990)

AbbreviationsOR, odds ratio; CI, confidence interval.
Adjusted Model: Covariates included age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, meat and poultry intake, vegetable and fruit intake, physical activities and family history of dyslipidemia.

Table 4
Interactions of sex, age, BMI and lifestyle characteristic on associations between per 1-μg/m^3 increment of $PM_{2.5}$ and dyslipidemias.

	Hypercholesterolemia	$P_{interaction}$	Hypertriglyceridemia	$P_{interaction}$	Hypoalphalipo-proteinemia	$P_{interaction}$	Hyperbetalipo-proteinemia	$P_{interaction}$
	OR (95%CI)		OR (95%CI)		OR (95%CI)		OR (95% CI)	
Sex								
Men	1.106 (1.071, 1.141)	–	0.956 (0.938, 0.975)	–	1.062 (1.045, 1.080)	–	1.082 (1.050, 1.115)	–
Women	1.034 (1.011, 1.056)	<0.001	0.903 (0.889, 0.917)	<0.001	1.035 (1.018, 1.053)	0.027	1.019 (0.997, 1.043)	<0.001
Age								
<65 years	1.033 (1.011, 1.056)	–	0.918 (0.905, 0.931)	–	1.046 (1.031, 1.060)	–	1.016 (0.993, 1.039)	–
≥65 years	1.102 (1.069, 1.136)	<0.001	0.943 (0.919, 0.966)	0.063	1.058 (1.033, 1.084)	0.394	1.097 (1.062, 1.132)	<0.001
BMI								
<25 kg/m^2	1.070 (1.043, 1.098)	–	0.912 (0.894, 0.930)	–	1.037 (1.018, 1.056)	–	1.031 (1.003, 1.058)	–
≥25 kg/m^2	1.042 (1.017, 1.068)	0.125	0.931 (0.916, 0.945)	0.098	1.056 (1.040, 1.073)	0.120	1.051 (1.025, 1.077)	0.282
Meat and poultry								
<75 g/d	1.051 (1.030, 1.072)	–	0.916 (0.904, 0.929)	–	1.049 (1.035, 1.064)	–	1.041 (1.019, 1.063)	–
≥75 g/d	1.077 (1.033, 1.123)	0.283	0.947 (0.924, 0.971)	0.018	1.045 (1.020, 1.071)	0.770	1.043 (1.002, 1.087)	0.908
Vegetable and fruit								
<500 g/d	1.026 (1.002, 1.050)	–	0.929 (0.914, 0.945)	–	1.046 (1.029, 1.063)	–	1.043 (1.018, 1.068)	–
≥500 g/d	1.101 (1.070, 1.132)	<0.001	0.916 (0.899, 0.933)	0.258	1.051 (1.032, 1.071)	0.674	1.039 (1.009, 1.071)	0.873
Physical activity								
Low	1.081 (1.050, 1.113)	–	0.917 (0.898, 0.937)	–	1.061 (1.039, 1.083)	–	1.036 (1.004, 1.070)	–
Moderate	1.000 (0.971, 1.030)	<0.001	0.924 (0.907, 0.941)	0.518	1.051 (1.032, 1.071)	0.483	0.998 (0.970, 1.028)	0.081
High	1.099 (1.063, 1.137)	0.453	0.930 (0.909, 0.952)	0.355	1.030 (1.007, 1.053)	0.049	1.113 (1.075, 1.152)	0.002

AbbreviationsOR, odds ratio; CI, confidence interval. Covariates included age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, meat and poultry intake, vegetable and fruit intake, physical activities and family history of dyslipidemia.

Table 5
Interactions of sex, age, BMI and lifestyle characteristic on associations between per 1-μg/m^3 increment of PM_{10} and dyslipidemias.

	Hypercholesterolemia	$P_{interaction}$	Hypertriglyceridemia	$P_{interaction}$	Hypoalphalipo-proteinemia	$P_{interaction}$	Hyperbetalipo-proteinemia	$P_{interaction}$
	OR (95%CI)		OR (95%CI)		OR (95%CI)		OR (95% CI)	
Sex								
Men	1.033 (1.019, 1.047)	–	0.985 (0.977, 0.993)	–	1.032 (1.025, 1.039)	–	1.012 (1.000, 1.026)	–
Women	1.002 (0.993, 1.011)	<0.001	0.963 (0.957, 0.969)	<0.001	1.020 (1.013, 1.027)	0.012	0.990 (0.981, 0.999)	0.004
Age								
<65 years	1.004 (0.995, 1.013)	–	0.968 (0.963, 0.974)	–	1.024 (1.018, 1.030)	–	0.992 (0.983, 1.001)	–
≥65 years	1.026 (1.013, 1.039)	0.004	0.983 (0.974, 0.993)	0.005	1.031 (1.021, 1.042)	0.215	1.011 (0.998, 1.024)	0.014
BMI								
<25 kg/m^2	1.017 (1.007, 1.028)	–	0.968 (0.960, 0.975)	–	1.021 (1.014, 1.029)	–	0.998 (0.987, 1.009)	–
≥25 kg/m^2	1.005 (0.995, 1.016)	0.096	0.974 (0.968, 0.980)	0.182	1.029 (1.022, 1.036)	0.121	0.998 (0.988, 1.008)	0.954
Meat and poultry								
<75 g/d	1.008 (1.000, 1.017)	–	0.969 (0.963, 0.974)		1.027 (1.021, 1.033)	–	0.996 (0.987, 1.004)	–
≥75 g/d	1.023 (1.006, 1.041)	0.120	0.980 (0.971, 0.990)	0.037	1.021 (1.011, 1.031)	0.287	1.007 (0.991, 1.024)	0.218
Vegetable and fruit								
<500 g/d	0.995 (0.986, 1.005)	–	0.972 (0.966, 0.979)	–	1.027 (1.020, 1.034)	–	0.992 (0.982, 1.001)	–
≥500 g/d	1.034 (1.022, 1.046)	<0.001	0.971 (0.963, 0.978)	0.729	1.024 (1.017, 1.032)	0.582	1.007 (0.996, 1.020)	0.042
Physical activity								
Low	1.020 (1.008, 1.033)	–	0.966 (0.958, 0.974)	–	1.026 (1.018, 1.035)	–	0.999 (0.986, 1.012)	–
Moderate	0.991 (0.980, 1.003)	<0.001	0.970 (0.963, 0.977)	0.464	1.023 (1.016, 1.031)	0.622	0.986 (0.975, 0.997)	0.126
High	1.028 (1.013, 1.042)	0.461	0.980 (0.971, 0.991)	0.021	1.029 (1.019, 1.039)	0.688	1.016 (1.001, 1.030)	0.085

Abbreviations: OR, odds ratio; CI, confidence interval. Covariates included age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, meat and poultry intake, vegetable and fruit intake, physical activities and family history of dyslipidemia.

Table 6
Interactions of sex, age, BMI and lifestyle characteristic on associations between per 1-μg/m^3 increment of NO_2 and dyslipidemias.

	Hypercholesterolemia	$P_{interaction}$	Hypertriglyceridemia	$P_{interaction}$	Hypoalphalipo-proteinemia	$P_{interaction}$	Hyperbetalipo-proteinemia	$P_{interaction}$
	OR (95%CI)		OR (95%CI)		OR (95%CI)		OR (95% CI)	
Sex								
Men	1.075 (1.049, 1.102)	–	0.980 (0.966, 0.994)	–	1.068 (1.054, 1.082)	–	1.010 (0.987, 1.033)	–
Women	1.016 (0.999, 1.032)	<0.001	0.942 (0.931, 0.953)	<0.001	1.049 (1.035, 1.062)	0.033	0.962 (0.946, 0.979)	<0.001
Age								
<65 years	1.022 (1.003, 1.038)	–	0.948 (0.938, 0.958)	–	1.055 (1.044, 1.066)	–	0.973 (0.957, 0.990)	–
≥65 years	1.057 (1.033, 1.081)	0.011	0.984 (0.966, 1.001)	<0.001	1.069 (1.050, 1.089)	0.192	0.988 (0.965, 1.011)	0.285
BMI								
<25 kg/m^2	1.041 (1.021, 1.062)	–	0.948 (0.935, 0.962)	–	1.048 (1.034, 1.063)	–	0.978 (0.958, 0.998)	–
≥25 kg/m^2	1.025 (1.007, 1.045)	0.246	0.961 (0.950, 0.972)	0.145	1.065 (1.053, 1.078)	0.077	0.978 (0.960, 0.997)	0.983
Meat and poultry								
<75 g/d	1.028 (1.013, 1.044)	–	0.952 (0.942, 0.962)	–	1.061 (1.050, 1.072)	–	0.972 (0.978, 0.988)	–
≥75 g/d	1.057 (1.025, 1.092)	0.105	0.970 (0.952, 0.988)	0.071	1.048 (1.029, 1.067)	0.234	1.003 (0.973, 1.035)	0.071
Vegetable and fruit								
<500 g/d	1.005 (0.988, 1.023)	–	0.962 (0.951, 0.974)	–	1.061 (1.047, 1.074)	–	0.962 (0.946, 0.979)	–
≥500 g/d	1.075 (1.053, 1.098)	<0.001	0.949 (0.936, 0.962)	0.120	1.055 (1.041, 1.070)	0.594	1.005 (0.983, 1.028)	0.002
Physical activity								
Low	1.054 (1.031, 1.078)	–	0.942 (0.927, 0.956)	–	1.051 (1.035, 1.068)	–	0.994 (0.971, 1.018)	–
Moderate	0.994 (0.972, 1.016)	<0.001	0.948 (0.935, 0.961)	0.497	1.050 (1.035, 1.065)	0.919	0.964 (0.943, 0.985)	0.055
High	1.058 (1.031, 1.085)	0.859	0.988 (0.971, 1.005)	<0.001	1.078, (1.060, 1.097)	0.028	0.979 (0.954, 1.004)	0.375

Abbreviations: OR, odds ratio; CI, confidence interval. Covariates included age, sex, BMI, education, marital status, family income, smoking, alcohol drinking, meat and poultry intake, vegetable and fruit intake, physical activities and family history of dyslipidemia.

2018; Yitshak Sade et al., 2016), we found higher air pollution was related to increased TC and LDL-C, while decreased HDL-C. For instance, a study investigated in Chinese urban areas demonstrated higher $PM_{2.5}$ corresponded to elevated TC and LDL-C, while decreased HDL-C, and the effects of PM_{10} and NO_2 were similar (Yang et al., 2018). A recent study performed in U.S. showed that each 1 μg/m^3 increment of $PM_{2.5}$ corresponded to 1.62% (95%CI: 1.13, 2.11) increment in TC and 1.70% (95%CI: 1.02, 2.37) increment in LDL-C (McGuinn et al., 2019). Similar results were reported in Denmark that a 6.7 μg/m^3 increment of NO_2 and a 1 μg/m^3 increment of $PM_{2.5}$ was related to 0.68 mg/dL (95%CI: 0.22; 1.16) and 0.78 mg/dL (95%CI: 0.22; 1.34) higher level of TC, respectively (Sorensen et al., 2015). A large and longitudinal survey in U.S. showed that an increment of 11.1 μg/m^3 PM_{10} corresponded to 1.43% (95%CI: 1.21, 1.66) and 1.18% (95%CI: 0.81, 1.56) higher TC and LDL-C (Shanley et al., 2016).

However, associations between air pollutions and TG remained inconsistent with previous studies. This study suggested that increased air pollution was associated with decreased TG, however, previous studies in China (Yang et al., 2018), U.S. (Bind et al., 2016; Shanley et al., 2016) and Israel (Yitshak Sade et al., 2016) showed that air pollution was related to increased TG. Another longitudinal investigation performed in U.S. revealed each 11.1 μg/m^3 increment of PM_{10} was related to 2.43% (95%CI: 1.09, 3.76) increased of TG (Bind et al., 2016). Yang et al. also reported that each 10 μg/m^3 increment of $PM_{2.5}$, PM_{10}, and NO_2 was related to 1.1%, 4.7% and 6.0% increase of TG in Chinese urban population, respectively, whereas only PM_{10} increment was related with increased risk of hypertriglyceridemia (Yang et al., 2018). In addition, another study showed both short- and intermediate-term expose to $PM_{2.5}$ and PM_{10} were related to increased TG (Yitshak Sade et al., 2016). Confounders like indoor exposure, noise, food intake and life style, contributed to lipid metabolism of the rural population, along with air pollution. Firstly, the type of food intake in rural population was quite unitary, with higher intake of cereals, eggs and salted products, while insufficient intake of aquatic products/seafood. According to CNHS 2010–2012, intake of meat, dairy, eggs, fishes and shrimps among rural population was significantly insufficient compared to urban populations (Guo et al., 2017). Secondly, people in rural areas accustomed to cook food by burning wood, coal and straw, instead of clean energy, resulting in elevated residential air pollution levels. Besides, rural residents had a higher physical activity level both in working and leisure time which could reduce TG level but increase the air inhaled and exposure dose. Different exposure source and different lifestyle complicated the associations between TG levels and ambient air pollution.

All these above findings supported that air pollution was associated to adverse changes on blood lipids. Several biological mechanisms linked air pollution to changed blood lipids and prevalence of dyslipidemia. Some evidence suggested that inhaled air pollution could evoke inflammation and oxidative stress, interfering with lipids metabolism and oxidation, and contributing to altered blood lipid levels and dyslipidemias (Araujo and Nel, 2009; Poursafa et al., 2014; Xu et al., 2011). Additionally, intervention and experimental studies provided evidence that inhaled air pollution elicited decreases in DNA methylation, especially on genes related to lipid metabolism and inflammation pathways (Bind et al., 2014; Chen et al., 2016; Mendez et al., 2013).

Sex and age can modify these detrimental effects of air pollutants on blood lipids. Consist with Shanley et al. (2016) study, stronger associations of PM_{10} with triglyceride and total cholesterol were presented among males. However, females were found to be more susceptive in Yang et al. (2018) study among urban population. In addition, Sorensen et al. (2015) reported sex had no modification of associations between air pollutants and TC. Different biological and lifestyle characteristics may attribute to the sexual differences. For example, smoking and drinking can affect the adverse effects of air pollutants, while the rates of smoking and drinking in females were much lower than males in China. Besides, males in rural areas spent more time outdoors for farming or leisure and exposed to more ambient air pollution. Some research also suggested inhaled air pollution can act as a potential exogenous hormone, activing estrogen-disrupting effects, playing a role in reactive oxygen species generating and oxidative stress inducting (Bell et al., 2017; Chen et al., 2013; Miller, 1994). Besides, consist with existing evidence, higher ORs and lager percent changes between $PM_{2.5}$, PM_{10}, NO_2, blood lipids and dyslipidemias were detected among older individuals. Both Wang et al. (2018) and Yang et al. (2018) reported higher susceptibility to air pollution among elderly. However, studies performed in U.S. and Denmark showed

that age didn't influence the detrimental effects of air pollutants on blood lipids (Shanley et al., 2016; Sorensen et al., 2015). Undesirable changes in body composition related to aging can lead to plenty of metabolic complications, such as elevated secretion of large numbers of cytokines following the endocrine function of adipose tissue, result in proinflammatory condition and interfere with lipid metabolism (Cartier et al., 2009; Liu and Li, 2015). Besides, some sociological characteristics of elderly could indirectly affect associations between air pollutants and blood lipids. For instance, Zhang et al. (2018b) reported that the elderly had a low level of local health services utilization, which may exacerbate the adverse effects of air pollutants on blood lipids.

Additionally, further analyses of current study (see Fig. 2, Tables S3–S5) showed that low physical exercise and excessive meat and poultry intake participants had stronger associations between air pollution, blood lipids, and dyslipidemias. Literature showed that unhealthy lifestyles like lack of exercise, high-sugar and high-fat foods intake could trigger systemic inflammation and oxidative stress (Delfino et al., 2011; Lin et al., 2017). However, a relatively higher OR of $PM_{2.5}$ and hypercholesterolemia were observed among adequate vegetable and fruit intake individuals (1.101, 95% CI: 1.070, 1.132) than others (1.026, 95% CI: 1.002, 1.050). Similar results was reported in Li et al.'s study where they found participants who intake of adequate fruit and vegetable had a higher risk of hypertension (Li et al., 2019). The possible explanation might be Chinese people's cooking habits, which can destruct and reduce the antioxidants in vegetables and fruits during cooking (Lin et al., 2017). Besides, differences in food supply, food choice, and consumption custom in different populations influenced vegetables and fruits intake, resulting in insufficient supplementation of antioxidants in rural population (Jia et al., 2018). As prior literature reported, the rate of intake of fruits among Chinese residents was low, especially in rural population (He et al., 2016).

Limitations in our study couldn't be neglected. Firstly, as the research data was the baseline data of cohort studies and the cross-sectional study design, it was difficult to verify conclusions on causality between air pollutants, blood lipids and dyslipidemias. A longitudinal design with repeated measures was expected to confirm our results. Secondly, some unavailable potential confounders, such as temperature, humidity and noise were not adjusted in the study. Also, the information of health status was not available in our study, like acute infection and inflammation, which may influence the effects of air pollutants on blood lipids and dyslipidemias as it can affect blood lipid metabolism (Nigam, 2011; Yang et al., 2018). Thirdly, the information of dyslipidemia family history was collected by participants' self-report, and the reply to this question may be inaccurate since the lack of medical diagnosis and people's poor knowledge about dyslipidemia. Fourthly, generalizability of current results may be limited as the concentrations of air pollution in current study was high, where the three years average exposure of NO_2, $PM_{2.5}$ and PM_{10} were 39.1 ± 3.1 μg/m^3, 72.8 ± 2.3 μg/m^3, and 131.5 ± 5.7 μg/m^3, respectively. Evidence indicated that the sensitivity of residents in high-pollution areas often tended to be reduced as these adverse effects of air pollutants among vulnerable subjects may have reached maximum level (Cao et al., 2011).

5. Conclusions

In conclusion, high $PM_{2.5}$, PM_{10}, and NO_2 exposure were related to changes of blood lipid levels and higher risk of hypercholesterolemia, hyperbetalipoproteinemia and hypoalphalipoproteinemia in Chinese rural populations. In particular, male and older individuals were more susceptive to the adverse effects of air pollutants. Our findings added new evidence on relationships between $PM_{2.5}$, PM_{10}, and NO_2 exposure, blood lipids and dyslipidemias in high-exposure rural areas. Further longitudinal studies based on individual exposures are urgently warranted to verify our findings.

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Declaration of competing interest

None declared.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envpol.2019.113403.

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