

Long-term air pollution exposure and serum lipids and blood sugar: A longitudinal cohort study from the electricity generating authority of Thailand study

Kanawat Paoin¹, Kayo Ueda^{1,2, *}, Prin Vathesatogkit³, Thammasin Ingviya⁴, Suhaimee Buya⁵, Arthit Phosri⁶, Xerxes Tesoro Seposo⁷, Nisakron Thongmung⁸, Teerapat Yingchoncharoen³, Akiko Honda^{1,2}, Hirohisa Takano^{1,2}, Piyamitr Sritara³

¹ Department of Environmental Engineering, Graduate School of Engineering, Kyoto University, Kyoto, Japan

² Graduate School of Global Environmental Sciences, Kyoto University, Kyoto, Japan

³ Department of Internal Medicine, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

⁴ Department of Family and Preventive Medicine, Faculty of Medicine, Prince of Songkla University, Songkhla, Thailand

⁵ Medical Data Center for Research and Innovation, Faculty of Medicine, Prince of Songkla University, Songkhla, Thailand

⁶ Department of Environmental Health Sciences, Faculty of Public Health, Mahidol University, Bangkok, Thailand

⁷ School of Tropical Medicine and Global Health, Nagasaki University, Nagasaki, Japan

⁸ Research Center, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

*Corresponding author; C1-3-366, Kyoto Daigaku-Katsura, Nishikyo-ku, Kyoto 615-8540, Japan. Tel.: +81 7 5383 3341;

Fax: +81 7 5383 3344; E-mail: uedak@health.env.kyoto-u.ac.jp

Abstract

Only a few studies have investigated the association between long-term exposure to air pollution and alterations of serum lipids and blood sugar level in developing countries. The present longitudinal study examined associations between long-term air pollution exposure and serum lipids [total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C)] and fasting glucose (FG) in workers of the Electricity Generating

Authority of Thailand (EGAT) in the Bangkok metropolitan region (BMR) of Thailand. We performed secondary analyses using the data obtained from 1,839 participants (mean age, 58.3 years as of 2002) of the EGAT1 cohort study (2002-2012). The average concentration of each air pollutants (PM₁₀, O₃, NO₂, SO₂, and CO) at the district level in BMR from 2002 to 2012 were estimated using the ordinary kriging method. Exposure periods were averaged to 3 months prior to laboratory testing. Linear mixed effects models were used to estimate associations between air pollution and serum lipids and blood sugar. After controlling for potential confounders, an interquartile range increment of PM₁₀, SO₂, and CO was associated with elevated LDL-C [6.6% (95%CI: 4.3, 9.0), 11.1% (7.2, 15.2), and 1.9% (1.1, 2.7), respectively] and FG [2.8% (1.5, 4.2), 6.8% (4.5, 9.1), and 1.1% (0.6, 1.5), respectively]. In addition, PM₁₀, SO₂, and CO were inversely associated with HDL-C [-1.8% (-3.7, 0.1), -3.3% (-6.2, -0.3), and -1.1 (-1.7, -0.5), respectively]. O₃ was negatively associated with TC, LDL-C, TG, and FG. These findings suggest inhalation of air pollutants may increase the risk of impaired metabolism of glucose and lipids.

Key words: Long-term exposure, air pollution, longitudinal study, serum lipids, blood sugar

1. Introduction

The prevalence of impaired serum lipids and elevated blood glucose, which are the risk factors for cardiovascular disease (CVD), is rapidly increasing worldwide over the past decades (O'Neill & Driscoll, 2015). Previous studies have shown that environmental factors, especially air pollutants, are associated with CVD risk factors (i.e., obesity, diabetes mellitus, hypertension, dyslipidemia) (Brook et al., 2004, 2010; Rajagopalan & Brook, 2012). Inhaled air pollutants can trigger CVD through various mechanisms, including systemic physiological responses such as oxidative stress, inflammation (Brook et al., 2004, 2010; Pope et al., 2004), insulin resistance (Rajagopalan & Brook, 2012), and altered autonomic nervous system activity (Brook et al., 2010; Rajagopalan & Brook, 2012). These

mechanisms are related to CVD risk factors, including atherosclerosis, elevated blood pressure, abnormal serum lipid levels, and high plasma glucose (Brook et al., 2010; Rajagopalan & Brook, 2012; Yang et al., 2018). Exposure to particulate matter (PM) has been linked to impaired metabolism of glucose and lipids and mediated by insulin resistance, which can lead to the development of CVD (Brook et al., 2010; Chen et al., 2013; Rajagopalan and Brook, 2012; Sade et al., 2016).

Long-term exposure to PM less than 2.5 μm in diameter ($\text{PM}_{2.5}$) and PM less than 10 μm in diameter (PM_{10}) is reportedly associated with elevated serum lipids (Chuang et al., 2011; Gaio et al., 2019; Mao et al., 2020; Poursafa et al., 2017; Shanley et al., 2016; Yang et al., 2018; Zhang et al., 2021) and blood sugar (Cai et al., 2019; Chuang et al., 2011; Yu et al., 2019). Changes in serum lipids and fasting glucose (FG) are also related to average air concentrations of nitrogen dioxide (NO_2) and ozone (O_3) (Chuang et al., 2011; Gaio et al., 2019; Kim et al., 2019; Zhang et al., 2021). However, longitudinal studies on associations between air pollutants and serum lipids and blood sugar (A. Li et al., 2019; Sade et al., 2016; Zhang et al., 2021) are still somewhat limited, especially in Southeast Asian countries, with most such studies being cross-sectional in design (Cai et al., 2019; Chuang et al., 2011; Mao et al., 2020; Poursafa et al., 2017; Shanley et al., 2016; Yang et al., 2018; Yu et al., 2019). Therefore, the present study longitudinally examined associations between long-term exposure to air pollutants and serum lipids and blood sugar using data from an ongoing cohort study targeting workers of the Electricity Generating Authority of Thailand (EGAT) in the Bangkok metropolitan region (BMR) of Thailand.

2. Methods

2.1 Study design and participants

The population of the EGAT cohort study has been described previously (Vathesatogkit et al., 2012a). The present longitudinal study analyzed data obtained from workers of the EGAT in BMR from 2002

to 2012. Health data were obtained from the EGAT1 study, which began recruiting participants in 1985. The initial survey of the EGAT1 study mainly covered the details of established CVD risk factors, including factors related to nutrition and toxicological mechanisms (Vathesatogkit et al., 2012a). The EGAT1 cohort was surveyed with follow-up questionnaires in 1997, 2002, 2007, and 2012. The protocol of the EGAT cohort study was approved by the Ethics Committee of Ramathibodi Hospital.

We extracted data for 2,129 participants (age 52-71 years as of 2002) who lived in BMR in 2002 and met the following criteria: (1) participated in follow-up surveys at least twice (2002 and 2007) or three times (2002, 2007, and 2012) (Adar et al., 2013; Duan et al., 2019; Kaufman et al., 2016; A. Li et al., 2019; Wang et al., 2019; Wilker et al., 2013; Zhang et al., 2021), and (2) lived in Bangkok, Nonthaburi, Samut Prakan, or Pathum Thani during the study period. We excluded 290 participants who died or were lost to follow-up before 2007, or who moved from the study area in 2007.

2.2 Data collection and physical examination

Physical examinations were performed by clinicians at the time of each survey. Data on physical examinations and sociodemographic characteristics such as sex, age, blood pressure, heart rate, weight, height, waist and hip circumferences, body mass index (BMI), waist/hip ratio, smoking status, alcohol drinking, exercise habits, education level, income, prevalence of disease (e.g., hypertension, diabetes, and hypercholesterolemia), treatment status, and medication use were collected (Vathesatogkit et al., 2012a).

2.3 General chemistry

All blood samples were collected in a fasting state during the surveys. Laboratory tests were subsequently carried out and included the following: total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and FG (Vathesatogkit et al., 2012a).

2.4 Exposure assessment

Hourly PM₁₀ and other gaseous pollutant concentrations, including sulfur dioxide (SO₂), NO₂, O₃, and carbon monoxide (CO), were obtained from the Pollution Control Department, a governmental agency responsible for air quality in Thailand. Meteorological data including daily average temperature (°C) and relative humidity (%) were obtained from the Thai Meteorological Department throughout the study period. We used meteorological data from only one meteorological station located in Bangkok, where complete data on temperature and relative humidity from 2002 through 2012 were available. In addition, almost all study participants lived in Bangkok and Nonthaburi, with the latter having no meteorological station. Hence, we applied meteorological data from the methodological station in Bangkok to represent environmental conditions for all participants.

Exposure to daily average PM₁₀, SO₂, NO₂, and CO and daily maximum 8-hr average O₃ were estimated using the ordinary kriging method (Geniaux et al., 2017; Leem et al. 2006; Liu et al. 1996). Air pollutant concentrations were measured at 22 background and 7 traffic monitoring sites in BMR from 2002 to 2012, and daily average values of all discontinuous site-specific measurements were calculated. For prediction, grids of 100x100 meters were generated, and the average concentration of each air pollutant at the sub-district level was estimated based on concentrations from the grids closest to the centroid of each sub-district (Paoia et al., 2021). For each air pollutant, a separate model was developed and validated to estimate grid-specific air pollutant concentrations.

Good model performance from 2002 to 2012 was achieved, with a leave-one-out cross validation R² value of 0.99 for PM₁₀, O₃, NO₂, and SO₂, and 0.98 for CO (Paoia et al., 2021). Model predictions had little bias, with cross-validated slopes (predicted vs. observed) of 0.99 for PM₁₀, O₃, NO₂, and SO₂, and 0.98 for CO (Paoia et al., 2021). Long-term exposure to air pollution was determined as a 3-month average concentration preceding laboratory testing according to previous studies (Bell et al., 2017; Sade et al., 2016; Zhang et al., 2021).

2.5 Covariates

All potential covariates were selected a priori, including age (years), sex (male/female), BMI (kg/m²), smoking status (never smokers/former smokers/current smokers), alcohol drinking (non-users/former users/occasional users/current users), exercise habits (<3 times per week/≥3 times per week), education level (0th-8th, 9th-12th, >12th Grade), income (<10,000 Thai baht/10,000-20,000 Thai baht/20,000-50,000 Thai baht/>50,000 Thai baht), hypertension (yes/no), diabetes (yes/no), hypercholesterolemia (yes/no), hypertensive medication use (yes/no), and antidiabetic medication use (yes/no).

2.6 Statistical Analysis

We analyzed associations between long-term air pollution exposure (PM₁₀, O₃, NO₂, SO₂, and CO) and outcome variables, i.e., serum lipids (TC, TG, LDL-C, and HDL-C) and plasma FG, using linear mixed effects models. Variability over time within each participant was defined as a random effect. Values of outcome variables were log-transformed to stabilize the variances.

A wide range of covariates were selected according to previous reports (Cai et al., 2019; Chuang et al., 2011; Curto et al., 2019; Mao et al., 2020; McGuinn et al., 2019; Yang et al., 2018; Zhang et al., 2021). The missing data of all covariates was less than 10%. The following five models were generated by gradually including these covariates: a crude model (Model I); Model II, adjusted for age and sex only; Model III, adjusted for age, sex, BMI, hypertension, diabetes, hypercholesterolemia, hypertensive medication use, antidiabetic medication use, exercise habits, smoking status, and alcohol drinking; a main model (Model IV), adjusted for all covariates in Model III, education level, and income. We assumed that participants with treatment status may have more severity of diseases than participants without treatment status (Joseph & Schuna 1990). Owing to these reasons, we controlled the treatment status in the main model.

As a sensitivity analysis, the main models were tested by different exposure window using 1-month average of air pollution concentrations. We also adjusted for 3-month average temperature, relative humidity and short-term exposure to each air pollutant (averaged from the current day to 1 day before) (Model V) since some previous studies observed the associations between short-term exposure

to air pollution and serum lipids and blood sugar (Kim et al., 2019; A. Li et al., 2019; Wu et al., 2020). If more than one pollutant had a significant effect on an outcome, two pollutant models were applied for each pollutant to evaluate the robustness of the single pollutant model. We also added natural cubic spline with 3 degrees of freedom to each pollutant in our main model, and then plotted the residuals of fitted values against observed values of all pollutants and all serum lipids and blood sugar to check non-linear associations.

The main models (Model IV) were also stratified by income group, including high income (> 50,000 Thai baht per month, n = 876) and low to middle income ($\leq$ 50,000 Thai baht per month, n = 815) at the baseline in 2002 to check whether the associations remain or not. Values of outcome variables are presented as percentage change per interquartile range (IQR) increment for PM₁₀, O₃, NO₂, SO₂, and CO, with 95% confidence intervals (CIs). All statistical analyses were performed using the R Program (version 3.6.1). A P-value less than 0.05 was considered statistically significant.

3. Results

Basic characteristics of study participants at baseline are shown in Table 1. Among 1839 participants analyzed, more than 70% of EGAT1 participants were male (age range, 52 - 71 years as of 2002), roughly 95% lived in Bangkok and Nonthaburi, and more than 45% were high-income earners (>50,000 baht/month) with high education levels (>12th Grade). All participants had been retired since 2007 (age >55 years). As of 2002, more than 45% of participants had hypertension and hypercholesterolemia, and roughly 20% had diabetes. The missing data of each variable was less than 1%, except income (8%). The excluded participants (N = 290), who died or were lost to follow-up before 2007, or who moved from the study area in 2007, had higher mean age and prevalence of hypertension and diabetes than study participants (Table S1).

The median follow-up period was 5 years. 1839 participants with 4415 TC, HDL, TG, FG recorded, and 4388 LDL recorded were included.

< INSERT TABLE 1 >

Daily concentrations of environmental variables are presented as mean with standard deviation (SD), range, and IQR in Table 2. The average concentration of PM₁₀ during the study period nearly exceeded the annual PM₁₀ standard limit in Thailand (PM₁₀ <50 µg/m³), while those of NO₂ and SO₂ were lower than the respective annual standard limits. No annual standard limits have been set for O₃ and CO in Thailand. The temperature range was 18.9°C to 33.7°C, and the range of relative humidity was 45.0% to 97.0% (Table 2). Air pollutants were positively correlated with one another (P<0.05) and negatively correlated with temperature and relative humidity (P<0.05) (Table 3).

< INSERT TABLE 2 >

< INSERT TABLE 3 >

Table 4 presents estimated percent changes in outcome variables for an increment of IQR increase in 3-month average PM₁₀, O₃, NO₂, SO₂, and CO in the main model (Models IV). PM₁₀ was associated with higher levels of TC, LDL-C, TG, and FG (5.6% (95%CI: 4.0, 7.2), 6.6% (95%CI: 4.3, 9.0), 2.9% (95%CI: -0.9, 6.8) and 2.8% (95%CI: 1.5, 4.2), per 16.3 µg/m³, respectively), but lower HDL-C levels (-1.8% (95%CI: -3.7, 0.10)). SO₂ was related to increase in TC, LDL-C, TG, and FG

(10.2% (95%CI: 7.5, 13.0), 11.1% (95%CI: 7.2, 15.2), 4.3% (95%CI: -1.8, 10.7), 6.8% (95%CI: 4.5, 9.1), per 5.3 ppb, respectively), and decrease in HDL-C (3.3% (95%CI: 0.3, 6.2)). CO was also associated with TC, LDL-C, TG, and FG (1.8% (95%CI: 1.3, 2.3), 1.9% (95%CI: 1.1, 2.7), 1.1% (95%CI: -0.2, 2.3) and 1.1% (95%CI: 0.6, 1.5) per 0.2 ppm, respectively), and a decrease in HDL-C (1.1% (95%CI: 0.5, 1.7)). In contrast, TC, LDL-C, TG, and FG, but not HDL-C, were inversely associated with O₃.

< INSERT TABLE 4 >

The results of 1-month average air pollution concentrations were not substantially different from our main results (3-month average air pollution concentrations) (Table S2). After adjusting for short-term air pollution exposure and 3-month average temperature and relative humidity in Model V (Table S3), most of the observed associations between serum lipids/blood sugar and 3-month average concentration of each pollutant became weaker. Associations between all air pollutants and outcome variables were essentially unchanged in two-pollutant models (Table S4). We did not observe any departure from the linear assumption, especially for air pollutants and serum lipids (Figure S1 to Figure S5).

After stratifying by income groups, the associations remained the same, except for PM₁₀ and FG which showed a stronger association in the group of low-middle income (Table 5).

< INSERT TABLE 5 >

4. Discussion

In this study, we examined associations between long-term exposure to air pollution and serum lipids and blood sugar in EGAT workers in BMR. PM₁₀, SO₂, and CO were associated with increases in TC, LDL-C, TG, and FG, and with a decrease in HDL-C levels. In contrast, O₃ was negatively associated with TC, LDL-C, TG, and FG. We did not observe any association of NO₂ with any of these.

4.1 Comparison with other studies and interpretations

Consistent with our current findings, previous studies have reported that long-term exposure to PM_{2.5} and PM₁₀ is associated with altered lipid levels, including elevated TC, LDL-C, TG, and decreased HDL-C levels (Chuang et al., 2011; Shanley et al., 2016; Gaio et al., 2019; Poursafa et al., 2017; Yang et al., 2018; Zhang et al., 2021). One hypothesis is that inhalation of air pollutants, especially for PM may induce systemic inflammatory response and oxidative stress (Brook et al., 2004, 2010; Pope et al., 2004), leading to altered lipid metabolism (Araujo et al., 2008; Chen et al., 2013; Sun et al., 2005; Zhang et al., 2003), lipid oxidation (Brucker et al., 2013; Chen et al., 2013; Soares et al., 2009), and lower HDL-C levels (J. Li et al., 2019). Another plausible mechanism is PM may cause abnormalities of DNA methylation at specific genes which related to lipid metabolism and inflammation pathways (Bind et al., 2014; Chen et al., 2016; H. Li et al., 2018). All of these could contribute to exacerbated atherosclerosis (Shanley et al., 2016).

Only a few studies have reported the associations between long-term exposure to gaseous pollutants (i.e., NO₂, O₃, and SO₂) and serum lipids (Chuang et al., 2011; Kim et al., 2019; Zhang et al., 2021). In the present study, we found that long-term exposure to SO₂ and CO is associated with increased TC, LDL-C, TG, and decreased HDL-C. Wu et al. (2017) observed long-term exposure to SO₂ was also associated with decreases in HDL-C and apolipoprotein A1 although CO was not associated with lipid markers. Long-term exposure to gaseous pollutants (i.e., CO and SO₂) has the

potential to disrupt cholesterol balance and increase the risk of inflammation and thrombosis, contributing to decrease the HDL-C levels and increase the LDL-C and TG levels (Wu et al., 2017).

FG is primarily used to assess plasma glucose levels after overnight fasting. Chuang et al. (2011) suggested that the close relationship between high blood sugar and an elevated atherosclerotic risk may be associated with exposures to PM. Most of previous studies have consistently found similar results that long-term exposure to PM_{2.5} and PM₁₀ is associated with elevated FG (Cai et al., 2019; Chuang et al., 2011; Yu et al., 2019). Particulate air pollution may cause an imbalance in the autonomic nervous system, which directly affects insulin resistance (Andersen et al., 2012; Liu et al., 2016; Rajagopalan & Brook, 2012). Systemic inflammatory response is another potential mechanism underlying the association between long-term PM_{2.5} exposure and FG (Yu et al., 2019).

Chuang et al. 2011 reported that long-term exposure to NO₂ and O₃ were positively associated with FG, but not for SO₂ and CO. On the other hand, our results showed the positive associations between long-term exposure to SO₂ and CO with FG. Previous animal study observed that SO₂ induced alterations in glycogen metabolism of rats in liver resulting in the increase of glucose-6-phosphatase (Fatma et al., 2015), which lead to higher concentration of blood glucose level (Argaud et al., 1997). Additionally, elevated blood glucose was also observed in the CO-poisoned rat (Penney et al., 1990). Two hours after CO exposure (post-CO-exposure) induced hyperglycemia even in the presence of elevated plasma insulin activity, suggests the lack of the insulin control of blood glucose in rats (Penney et al., 1990). Nevertheless, the evidence of long-term CO exposure and blood glucose is still limited in both epidemiological and toxicological studies.

In our study, O₃ was negatively associated with TC, LDL-C, TG, and FG. There are inconsistent results of the associations between O₃ with serum lipids and blood sugar (Chuang et al., 2011; Kim et al., 2019; A. Li et al., 2019; Yang et al., 2018; Zhang et al., 2021). Some studies observed the adverse effects of O₃ on serum lipids (A. Li et al., 2019; Zhang et al., 2021) and FG

(Chuang et al., 2011). Other studies observed beneficial (Yang et al., 2018) and no associations (Kim et al., 2019) between long-term O₃ exposure and serum lipids and FG. Furthermore, our results of NO₂ are also inconsistent with some previous studies (Chuang et al., 2011; Kim et al., 2019; Zhang et al., 2021). The possible reasons of inconsistent results may be due to the difference in study designs (cross-sectional and longitudinal studies), groups of the population, and a lack of adjustment for potential confounders such as education level, income, disease prevalence, medication use, and exercise habit in previous studies (Chuang et al., 2011; Zhang et al., 2021). Further epidemiological studies are needed to examine the association between gaseous pollutants (i.e., O₃, NO₂, SO₂, and CO) with serum lipids and blood sugar in different locations and characteristics of population.

4.2 Exposure window

Results of previous studies have been inconsistent as to why long-term exposure to air pollutants is associated with serum lipids and blood sugar. Bell et al. (2017) found no association between 1-year average PM_{2.5} and HDL-C, although 3-month average PM_{2.5} was associated with HDL-C. Sade et al. (2016) also reported a strong association between 3-month average PM_{2.5} exposure and decreased HDL-C. Hemoglobin A1c (HbA1c) reflects variations in plasma glucose levels over roughly 3 months; short-term air pollution exposure was not associated with HbA1c (Sade et al., 2016). As for serum lipids and blood sugar, no or weak associations with short-term exposure to air pollution have been reported (Kim et al., 2019; A. Li et al., 2019; Wu et al., 2020). In the present longitudinal study, we focused on long-term exposure (i.e., 3 months) to assess whether exposure to air pollutants over a longer period of time might play a more prominent role in altering serum lipids and blood sugar.

4.3 Model adjustment

Vathesatogkit et al. (2012b) revealed long-term effects of income and education level on cardiovascular risk factors. Therefore, we adjusted for them in our main model (Model IV). We further adjust for short-term exposure to each air pollutant and 3-month average temperature and relative

humidity (Model V) because some previous studies reported the short-term associations between air pollution exposure and serum lipids and blood sugar (Kim et al., 2019; A. Li et al., 2019; Wu et al., 2020). Short-term air pollution exposure may trigger the daily variation while long-term exposure contributes to the chronic change in the variables examined. Hence, we controlled the short-term associations to focus only long-term associations in this study. While the values of serum lipids and blood sugar decreased after these adjustments, associations between air pollutants and serum lipids/blood sugar essentially remained the same. Hence, the alterations in serum lipids and blood sugar were not affected by long-term air pollution exposure alone, but also affected by other factors that we controlled in the models.

4.4 Strengths and limitations

In the present study, we collected a large amount of data including BMI, education level, income, exercise habits, alcohol consumption, and smoking status, and controlled for these variables in our models. Furthermore, we used the ordinary kriging method, which allowed us to evaluate the spatial representativeness of monitoring stations and improve the accuracy of air pollution exposure estimates.

Our study has several limitations. First, due to data restrictions in Thailand, we could not include traffic variables or land use data in ordinary kriging. However, data from traffic monitoring stations were used to assess exposures to traffic-related air pollutants such as NO₂ and CO. In addition, the average air pollution concentrations longer than 6-month preceding laboratory testing in 2002 would be difficult to generate according to a very few numbers of monitoring stations in Thailand before 2002. Second, participants of this study were EGAT workers aged 50 years and older (as of 2002), whose lifestyles may have differed from the general Thai population given their higher income and education levels. Therefore, our study population does not represent the general population. Moreover, the results of the present study may underestimate the effects of air pollutants on serum

lipids and blood sugar, as people with low SES are more likely to suffer greater health effects from air pollutants due to higher exposure to air pollution and compromised health status due to material deprivation and environmental equity. Third, we included only participants who had been followed-up at least 2 times during study period. The study participants might be healthier than the participants who had been excluded because they were lost followed-up or dead. Therefore, the results of this study might be underestimated. Finally, data on food consumption, a potential confounder, were not available. However, we adjusted for other covariates related to SES, lifestyle, and eating habits, such as income, education level, BMI, hypertension, diabetes, and hypercholesterolemia using linear mixed models.

5. Conclusions

Long-term exposure to air pollutants, especially for PM₁₀, SO₂, and CO was associated with impaired serum lipids and elevated blood sugar in participants of the EGAT cohort study. A further study will be necessary to elucidate mechanisms underlying these associations.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Ethics and Consent

Approval for the study was obtained from the ethics committee of Ramathibodi Hospital and the Graduate School of Engineering, Kyoto University.

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529 Table 1. Basic characteristics of study participants at baseline (in 2002) (N = 1,839)

Variables	Number (%)
Sex, n (%)	
Male	1,357 (73.8)
Female	482 (26.2)
Age, years	
Mean $\pm$ SD	58.9 $\pm$ 4.7
Range	52 – 71
Body mass index, kg/m ²	
Mean $\pm$ SD	24.6 $\pm$ 3.4
Smoking status, n (%)	
Never smokers	890 (48.4)
Former smokers	691 (37.5)
Current smokers	253 (13.8)
Missing data	5 (0.3)
Alcohol drinking, n (%)	
Non-users	584 (31.8)
Former users	310 (16.9)
Occasional users (<1 day/week)	460 (25.0)
Current users	479 (26.0)
Missing data	6 (0.3)
Exercise, n (%)	
<3 times/week	766 (41.6)
$\geq$ 3 times/week	1066 (58.0)
Missing data	7 (0.4)
Education levels, n (%)	
0 – 8th Grade	444 (24.2)
9th – 12th Grade	570 (31.0)
>12th Grade	819 (44.5)
Missing data	6 (0.3)
Income (monthly), n (%)	
<10000 Baht	135 (7.3)
10000 – 20000 Baht	151 (8.2)
20000 – 50000 Baht	529 (28.8)
>50000 Baht	876 (47.6)
Missing data	148 (8.1)
Prevalence of disease, n (%)	
Hypertension	838 (45.6)
Diabetes	382 (20.8)

530	Hypercholesterolemia	910 (49.5)
531	Treatment status, n (%)	
	Hypertension	487 (26.5)
	Diabetes	249 (13.6)
532	City of residence, n (%)	
	Bangkok	874 (47.5)
533	Nonthaburi	887 (48.2)
	Samut Prakan	40 (2.2)
534	Pathum Thani	38 (2.1)
	Serum lipids & blood sugar, Mean $\pm$ SD	
535	TC (mg/dl)	241.9 $\pm$ 43.2
	LDL-C (mg/dl)	158.2 $\pm$ 39.7
	HDL-C (mg/dl)	54.7 $\pm$ 15.0
536	TG (mg/dl)	149.2 $\pm$ 97.3
	FG (mg/dl)	106.8 $\pm$ 28.7
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Table 2. Summary statistics for daily average concentration of environmental variables during the study period

Environmental Variable	Mean $\pm$ SD	Range	IQR
PM ₁₀ ($\mu\text{g}/\text{m}^3$)	47.5 $\pm$ 18.1	20.5 – 140.8	16.3
O ₃ (ppb)	30.1 $\pm$ 5.3	7.0 – 41.5	7.0
NO ₂ (ppb)	19.7 $\pm$ 4.4	12.3 – 45.0	4.6
SO ₂ (ppb)	6.5 $\pm$ 3.7	1.2 – 18.9	5.3
CO (ppm)	0.7 $\pm$ 0.2	0.2 – 2.1	0.2
Temperature ($^{\circ}\text{C}$)	29.1 $\pm$ 1.6	18.9 – 33.7	1.9
Relative humidity (%)	72.5 $\pm$ 8.4	45.0 – 97.0	11.0

Table 3. Correlation coefficients between air pollutants and environmental variables during the study period

	PM ₁₀	O ₃	NO ₂	SO ₂	CO
O ₃	0.27				
NO ₂	0.47	0.34			
SO ₂	0.1	0.08	0.08		
CO	0.3	0.16	0.57	0.11	
Temperature	-0.11	-0.06	-0.29	-0.07	-0.25
Relative humidity	-0.33	-0.46	-0.21	-0.17	-0.08

559 Table 4. Estimated percent change (95% CI) in outcome variables for 3-month average concentrations
 560 of air pollutants in the main model (Model IV)

	PM₁₀ (µg/m³)	O₃ (ppb)	NO₂ (ppb)	SO₂ (ppb)	CO (ppm)
TC (mg/dl)	5.6 (4.0, 7.2)*	-2.4 (-3.2, -1.6)*	-0.1 (-0.9, 0.7)	10.2 (7.5, 13.0)*	1.8 (1.3, 2.3)*
LDL-C (mg/dl)	6.6 (4.3, 9.0)*	-2.3 (-3.5, -1.2)*	0.1 (-1.1, 1.2)	11.1 (7.2, 15.2)*	1.9 (1.1, 2.7)*
HDL-C (mg/dl)	-1.8 (-3.7, 0.1)	-0.3 (-1.3, 0.7)	-0.7 (-1.7, 0.4)	-3.3 (-6.2, -0.3)*	-1.1 (-1.7, -0.5)*
TG (mg/dl)	2.9 (-0.9, 6.8)	-2.2 (-4.1, -0.2)*	1.0 (-1.0, 3.1)	4.3 (-1.8, 10.7)	1.1 (-0.2, 2.3)
FG (mg/dl)	2.8 (1.5, 4.2)*	-1.9 (-2.6, -1.2)*	0.4 (-0.3, 1.1)	6.8 (4.5, 9.1)*	1.1 (0.6, 1.5)*

561 Significance indicated by *P < 0.05. Model IV (main model): adjusted for age, sex, BMI, smoking status, alcohol
 562 drinking, exercise habits, hypertension, diabetes, hypercholesterolemia, and treatment of hypertension and
 563 diabetes, education level and income.

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576 Table 5. Estimated percent change (95% CI) in outcome variables in 3-month average air pollutants

577 stratified by income group

	PM ₁₀ (µg/m ³)	O ₃ (ppb)	NO ₂ (ppb)	SO ₂ (ppb)	CO (ppm)
TC					
High	5.0 (2.9, 7.1)*	-1.7 (-2.8, -0.6)*	-0.4 (-1.5, 0.6)	8.9 (5.3, 12.7)*	1.2 (0.5, 1.9)*
Low-middle	6.1 (3.6, 8.6)*	-2.8 (-4.1, -1.6)*	0.6 (-0.7, 1.8)	10.5 (6.4, 14.7)*	2.1 (1.4, 2.9)*
LDL-C					
High	6.3 (3.3, 9.4)*	-1.7 (-3.3, -0.2)*	-0.4 (-1.8, 1.2)	10.6 (5.3, 16.2)*	1.1 (0.1, 2.2)*
Low-middle	6.5 (3.0, 10.1)*	-2.5 (-4.3, -0.7)*	0.7 (-1.1, 2.5)	9.6 (3.8, 15.7)*	2.2 (1.1, 3.2)*
HDL-C					
High	-0.9 (-3.3, 1.7)	-0.0 (-1.3, 1.3)	-0.84 (-2.1, 0.5)	-2.8 (-6.8, 1.3)	-0.9 (-1.7, -0.1)*
Low-middle	-1.9 (-4.7, 1.0)	-0.8 (-2.3, 0.8)	-0.4 (-2.0, 1.2)	-2.3 (-6.5, 2.1)	-0.8 (-1.6, 0.1)
TG					
High	4.4 (-0.7, 9.7)	-2.1 (-4.5, 0.5)	0.6 (-1.9, 3.3)	1.9 (-6.3, 10.8)	1.3 (-0.4, 3.0)
Low-middle	0.4 (-5.1, 6.2)	-1.7 (-4.7, 1.3)	2.2 (-0.9, 5.3)	4.6 (-4.1, 14.1)	0.5 (-1.2, 2.2)
FG					
High	0.66 (-1.0, 2.3)	-1.6 (-2.4, -0.7)*	0.2 (-0.7, 1.0)	5.6 (2.7, 8.6)*	0.5 (-0.1, 1.1)
Low-middle	4.8 (2.6, 7.1)*	-2.4 (-3.5, -1.2)*	0.8 (-0.3, 2.0)	7.8 (4.2, 11.5)*	1.3 (0.6, 1.9)*

578 Significance indicated by *P < 0.05. Potential covariates in the adjusted models include: age, sex, BMI, smoking

579 status, alcohol drinking, exercise habits, hypertension, diabetes, hypercholesterolemia, treatment of hypertension

580 and diabetes, education level.

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