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Original Article

Increase of Cardiometabolic Biomarkers Among Vehicle Inspectors Exposed to PM0.25 and Compositions

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ABSTRACT

Background: Exposure to particulate matter (PM) emitted from vehicle exhaust might disrupt systemic function and elevate the risk of cardiovascular disease. In this study, we examined the changes of cardiometabolic biomarkers among vehicle inspectors exposed daily to PM0.25 and components.
Methods: This cross-sectional study was conducted at two vehicle inspection centers, Pulogadung and Ujung Menteng, located in East Jakarta, Indonesia. The exposed respondents were 43 workers from vehicle inspection centers, and the unexposed group consisted of 22 staff officers working in the same locations. Vehicle exhaust particulate matter was measured for eight hours using a Leland Legacy personal pump attached to a Sioutas Cascade Impactor. The used filters were 25 and 37-mm quartz filters. The particulate matter concentration was analyzed using a gravimetric method, whereas trace elements were analyzed using energy dispersive X-ray fluorescence. An EEL Smoke Stain Reflectometer analyzed black carbon.
Results: The personal exposure concentrations of PM0.25 were 10.4-fold higher than those in unexposed groups. Calcium and sulfur were the major components in the obtained dust, and their levels were 3.3- and 7.2-fold higher, respectively, in the exposed group. Based on an independent-samples *t*-test, high-density lipoprotein, triglyceride, HbA1c, total immunoglobulin E, high-sensitivity C-reactive protein, tumor necrosis factor–alpha, and nitric oxide levels were significantly different between the groups.
Conclusions: In summary, it was suggested that PM0.25 exposure from vehicle exhaust might affect cardiometabolic biomarkers change.

1. Background

Particulate and gas emissions from vehicle exhaust now become the primary source of air pollution in urban areas. Exposure to vehicle exhaust particulate, especially fine particulate matter (PM) ≤2.5 μm in aerodynamic diameter (PM2.5), has been associated linked with adverse health outcomes. These human health outcomes, including the increase in type 2 diabetes, cardiovascular disease, pulmonary cancer, and disturb reproductive function [1–4]. A cohort study conducted by the American Cancer Society stated that each 10 μg/m^3 increase in PM2.5 (fine particulate matter) exposure increases the risk of mortality because of cardiovascular disease by 12% [5].

PM2.5 in vehicle exhaust can reach the bronchioles and alveolar space and cross the pulmonary epithelium and the lung–blood barrier. The translocation of PM2.5 into the bloodstream and specific remote organs can induce the local oxidative stress and inflammation in the vascular endothelium. Then causing atherosclerotic plaque destabilization and, lastly, initiate thrombus formation [6]. In particular, exposure to PM can induce pulmonary inflammation, an initial step in systemic inflammation. Subsequently, it leads to elevated inflammation biomarker levels such as tumor necrosis factor–alpha (TNFα), IL-6, and IL-8 in the blood as

Abbreviations: PM, particulate matter; EDXRF, energy dispersive X-ray fluorescence; ELISA, enzyme-linked immunosorbent assay; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride; HbA1c, hemoglobin A1c; IgE, immunoglobulin E; hs-CRP, high-sensitivity C-reactive protein; TNFα, tumor necrosis factor–alpha; NO, nitric oxide; S, sulfur; K, potassium; Fe, iron; Ni, nickel; Cu, copper; Pb, lead; Ca, calcium; Ti, titanium; Mn, manganese; Zn, zinc.

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well as blood coagulation and plaque destabilization, which cause ischemic heart disease and myocardial infarction [7,8].

PM2.5 exposure is also correlated with glucose and lipid metabolism disorders mediated by insulin resistance, which may contribute to cardiovascular disease risk. Furthermore, the cardiometabolic effects of long-term exposure to air pollution may be majorly driven by the impairment of glucose homeostasis and, to a lesser extent, by visceral adiposity [9]. Jiang et al. [10] (2016) reported that exposure to traffic-related PM2.5 might contribute to the development or exacerbation of cardiometabolic disorders, as indicated by increases in HOMA-IR, low-density lipoprotein (LDL), and IL-6 levels; the augmentation index; and systolic and diastolic blood pressure.

Numerous studies reported the association of PM2.5 exposure and cardiometabolic disorders [11]. However, limited studies have investigated the relationship between PM0.25 and components and cardiometabolic biomarkers. PM0.25, known as quasi-ultrafine particles, has greater surface area than larger PM size fractions, be able to penetrate the respiratory tract, also translocate from the alveoli to the bloodstream, and cause more serious health effects [12,13]. The Kleeman et al. [14] (2000) study shows that the mass particle distribution from vehicle combustion has a peak in the diameter range of 0.1–0.3 μm. Thus, this study aimed to assess the personal exposure of PM0.25 and components and the resulting changes in cardiometabolic biomarkers, including markers of inflammation, glucose, and lipid metabolism disorders among vehicle inspectors.

2. Methods

2.1. Study design

This cross-sectional study was conducted on March–April 2016 at two vehicle inspection centers, Pulogadung and Ujung Menteng, located in East Jakarta, Indonesia. The exposed group included forty-three male inspectors who had been conducted daily safety and emissions testing of passenger vehicles, goods transport vehicles, and buses for at least one year. The control group included twenty-two male administration officers who unexposed to vehicle exhaust. The Ethics Committee of Universitas Indonesia approved the study, and all the participants gave informed consent before the study.

2.2. Personal PM0.25 exposure measurements

The PM was collected, referring to the US EPA IP-10 method adapted from SKC Inc. Samples were measured using a Leland Legacy pump (SKC Inc., Eighty Four, PA) and separated by size using a Sioutas Cascade impactor (SKC Inc., Eighty Four, PA), which consists of a four-tier impactor and one after the filter. This process divides particles by size into five groups. Specifically, 25-mm Polytetrafluoroethylene (PTFE) filters (Zefluor™, 0.5 μm pore size, Pall Life Sciences, Ann Arbor, MI) were installed at different stages (stages A to D). Stages A, B, C, and D collect particles >2.5, 1–2.5, 0.5–1, and 0.25–0.5 μm in size, respectively. A 37-mm quartz filter (SKC Inc., Ann Arbor, MI, USA) was installed at stage E to collects particles <0.25 μm in size. The personal pump was operated at a flow rate of 9 L/min and calibrated before and after measurement at the sampling location. The Sioutas Cascade impactor was attached to each worker's breathing zone during working hours (eight hours). Field blanks filter is loaded on a sampler for about 10 min without airflow and handled in the same manner as the samples. The PTFE and quartz filters were gravimetrically analyzed using an MT5 Microbalance (METTLER TOLEDO Inc., Columbus, OH). Before weighing, filters were conditioned in the balance room for at least 24 h. The calculation of PM concentrations began by calculating the total sample volume (V_a) as follows:

$$V_a = Q_{ave} \text{ x } T \text{ x } 10^{-3} \quad \text{Equation (1)}$$

where

V_a = total sample volume (m^3)

Q_{ave} = average sample flow rate (L/min)

T = total sample time (min)

10^3 = unit conversion factor for liters (L) into cubic meters (m^3)

The second step is a determination of the total mass of PM on filter using the postsample and presample filter weights as:

$$M_{PM} = (M_{post} - M_{pre}) \text{ x } 10^3 \quad \text{Equation (2)}$$

where

M_{PM} = total mass of PM on the filter (μg)

M_{post} = postsample filter weight (mg)

M_{pre} = presample filter weight (mg)

10^3 = unit conversion factor for milligrams (mg) to micrograms (μg)

And then calculate the PM concentration as:

$$C_{PM} = M_{PM}/V_a \quad \text{Equation (3)}$$

where:

C_{PM} = mass concentration of PM (μg/m^3)

M_{PM} = total mass of PM collected during the sampling period (μg)

V_a = total sample volume (m^3)

2.3. Black carbon analysis

Black carbon concentrations were analyzed using an EEL M43D Smoke Stain Reflectometer (Diffusion Systems Ltd, London, UK) at the National Nuclear Agency of Bandung, West Java, Indonesia. Samples were conditioned for at least 12 h at 18–25 °C and 55% relative humidity. The reflectometer lead was placed on gray and white standards to ensure the standard reflectance values.

2.4. Chemical compositions analysis

Inorganic elements contained in emissions were analyzed using an X-ray fluorescence spectrometry (Epsilon 5, PANalytical, Almelo, The Netherlands). This instrument identifies X-ray characteristics associated with photoelectric effects. Specifically, X-ray fluorescence emitted primarily from the samples is dispersed in the X-ray tube. Radiation from the X-ray tube can be used as a semi-monochromatic X-ray source to examine different characteristics of the sample. The electrons are excited by X-rays and captured by the detector for conversion into a voltage signal. Then, the characteristics of the X-rays are fed to the analyzer, and the concentrations were measured in μg/m^3.

2.5. Cardiometabolic biomarker analysis

After fasting for at least 12 hours, one tube of the ethylenediaminetetraacetic acid blood sample from each participant was collected for routine hematologic tests by Prodia Laboratory on Friday morning. Another two tubes of anticoagulant-free fasting blood were then collected and immediately centrifuged for 15 min at 3000 rpm before laboratory analyses. Total cholesterol, high-density lipoprotein cholesterol (HDL-C), LDL cholesterol, triglyceride (TG) levels were analyzed using an Advia 1800 Analyzer (Siemens, Germany), hemoglobin A1c (HbA1c) was measured by a

Table 1
Characteristics of the study participants

Variables	Exposed group (n = 43)	Control group (n = 22)	*p*
Age (years), mean ± SD	41.70 ± 9.40	43.41 ± 7.07	0.460
BMI (kg/m^2), mean ± SD	24.59 ± 3.44	23.01 ± 5.39	0.164
Smoking status			
Yes	23 (53.5%)	13 (59.1%)	0.868
No	20 (46.5 %)	9 (40.9%)	

BMI, body mass index.

Bio-Rad D10 Analyzer (Bio-Rad, Hercules, CA, USA), and immunoglobulin E (IgE) was measured using an Immulite 2000 (Siemens, Germany). Furthermore, high-sensitivity C-reactive protein (hs-CRP) was measured using a COBAS INTEGRA Analyzer (Roche Diagnostics), TNFα was detected by enzyme-linked immunosorbent assay using commercial kits (R&D System), and nitric oxide (NO) was analyzed using commercial kits (R&D System).

2.6. Statistical analysis

Differences in the mean concentrations were analyzed using an independent-samples *t*-test. Data are presented as the mean ± standard deviation unless otherwise indicated. Significance was indicated by $p < 0.05$.

3. Results

3.1. Characteristics of the subject

Table 1 shows the comparison of personal characteristics between the exposed and control groups. The age and body mass index were not different between the exposed and control groups. Meanwhile, the proportions of smokers in the exposed and control groups were 53.5% and 59.1%, respectively.

3.2. Particulate matter concentrations

The concentration of PM in each size in the current study was shown in Table 2 as mean and standard deviation. In the exposed group, the PM concentration in every size was higher than that in the control group. The concentration of PM0.25 from personal exposure measurement in the exposed group was 121.79 ± 54.42 μg/m^3. The concentrations ratio of PM2.5, PM1, and PM0.5 were 3.67 to 4.38 times higher in the exposed group than those in the unexposed group. However, the PM0.25 concentration ratio in the exposed group was 10.39 times higher than that in the control group. Furthermore, the black carbon concentration ratio was 7.12 times higher in the exposed group than that in the control group.

Table 2
Particulate matter and black carbon concentrations of the vehicle inspectors

Particulate sizes	Exposed group mean ± SD, (μg/m^3)	Control group mean ± SD, (μg/m^3)	*p*
PM2.5	272.44 ± 100.77	68.06 ± 30.51	<0.001*
PM1	223.07 ± 73.88	60.72 ± 33.72	<0.001*
PM0.5	181.02 ± 60.98	41.25 ± 21.86	<0.001*
PM0.25	121.79 ± 54.42	36.56 ± 10.17	<0.001*
Black carbon	8.31 ± 3.80	1.17 ± 0.55	<0.001*

* significant at <0.05 for an independent t-test.
PM, particulate matter; SD, standard deviation.

3.3. Chemical compositions

Fig. 1 shows the chemical composition concentration of PM0.25 and presented as mean and standard deviation. Sulfur and calcium were the major elements of the obtained particulate in the exposed group, reaching concentrations of 2.40 ± 1.29 and 2.68 ± 1.73 μg/m^3, respectively. These values were 5.52 and 1.93 times higher, respectively, than those in the control group. Other major elements obtained in the exposed group that their concentrations were higher than those in the control group were iron, potassium, and zinc.

3.4. Cardiometabolic biomarkers

The biomarkers of inflammation, glucose, and lipid metabolism disorders were measured to assess the association of personal exposure of PM0.25 and changes in cardiometabolic biomarkers. Table 3 showed differences in HDL-C, TG, HbA1c, total IgE, hs-CRP, TNFα, and NO levels between the exposed and control groups. In the vehicle inspector group who exposed to PM0.25, we observed that the level of inflammation biomarkers was 2.82 higher times than that of total IgE, 2.45 higher times than that of hs-CRP, 2.32 higher times than that of TNFα, and 1.29 higher times than that of NO than in control group. The levels of HbA1c and TG in the exposed group were 1.30 and 1.62 times higher than those in control, respectively. Contrary to the increase in the biomarkers of lipid metabolism, the level of HDL in the exposed group was 1.10 lower times than that in the control group. However, the level of total cholesterol and LDL cholesterol did not show significant differences between the exposed and control group.

4. Discussion

Our present study suggested that PM0.25 exposure from vehicle exhaust might affect cardiometabolic biomarkers change, as shown by the increases of biomarkers of inflammation, glucose, and lipid metabolism disorders levels in vehicle inspectors. Because the ratio of PM0.25 concentration was the highest among PM2.5, PM1, and PM0.5 concentration ratios, the inspectors are exposed to more PM0.25. Inhalation of PM is closely related to inflammatory and oxidative stress. The particulate of this size has greater surface area than larger PM size fractions and be able to penetrate the respiratory tract, and initiate oxidative stress and systemic inflammation, which then lead to cardiometabolic disorders [15].

During our research study, heavy-duty vehicles represented the major vehicle type in both vehicle inspection centers. Heavy-duty vehicles are known to emit PM at significantly higher levels than light-duty vehicles. A previous study stated that heavy-duty vehicles contributed more fine and ultrafine particles than light-duty vehicles [16]. The concentration ratio of PM in each size in the exposed group in the current study was 3.67 to 10.39 times higher in the exposed group than that in the unexposed group, and the $PM_{0.25}$ concentration ratio was the highest. The PM0.25 concentration among these vehicle inspectors was 2.5-fold higher than that in Jakarta traffic policemen [17]. Furthermore, the black carbon concentration ratio was 7.12 times higher in the exposed group than that in the control group. Ultrafine particles with diameters of <0.05 - 0.10 μm are known to penetrate the epithelial and vascular wall and enter the bloodstream, inducing oxidative stress and activating the inflammatory pathway in blood vessels [5]. In regards to PM2.5, the concentration among vehicle inspectors was 272.44 ± 100.77 μg/m^3, 4.2 times higher than 65 μg/m^3 of Indonesian Air Quality Standard [18].

Abundant sulfur levels in emissions have been reported as impurities of diesel fuel and lubricant additives [19]. Sulfur is also

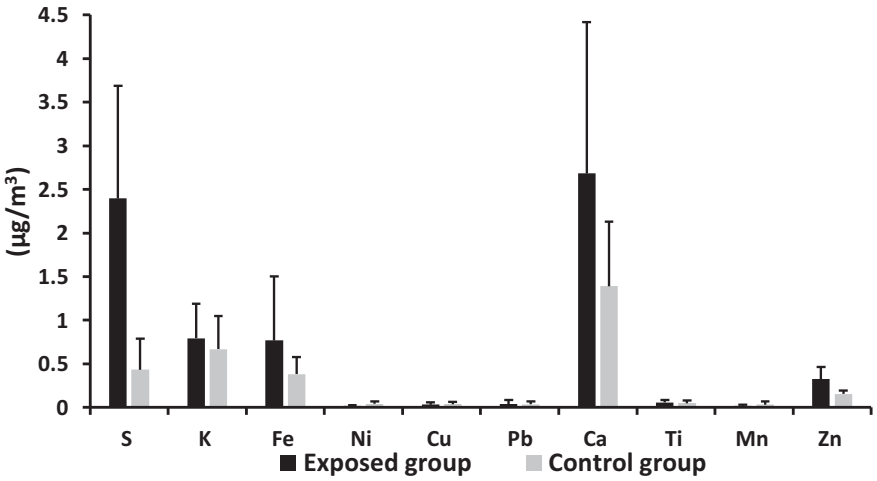

Fig. 1. Mean concentrations and standard deviation of chemical composition (µg/m^3) from PM0.25 among vehicle inspectors and control groups. Sulfur, calcium, iron, potassium, and zinc were the major elements of the obtained particulate in the exposed group.

present as a trace species in diesel fuel and lubricant oil additives. Calcium is usually absorbed in soot particles from exhaust fumes or self-ignition engines, and it was the main component of ultrafine particles in the exhaust fumes of self-ignition engines [20]. Calcium is also present in the lubricating oil at various levels depending on its consumption [21]. The other major elements, namely, iron, potassium, and zinc, might have originated from wear debris, the fuel type, engine wear residuals, and the lubricating oil and its additives [22].

Fine particulate as a risk factor for cardiovascular disease can induce the local oxidative stress and inflammation in the vascular endothelium and pulmonary region [23]. Our finding of significant differences in TNFα levels between the groups coincided with the result of a previous study conducted in Shanghai, China, every 10 µg/m^3 elevation of PM2.5 increased the TNFα concentration by 4.4% [24]. An animal study also found that the TNFα concentration was significantly higher among mice exposed daily to PM2.5; however, the elevation of TNFα was significantly higher in the fourth week of exposure than that in the first week [25]. CRP elevation is often used as a sign of inflammation caused by air pollution exposure and biomass combustion, as well as a predictor of cardiovascular disease risk [26]. In our study, vehicle inspectors have a significant elevation of hs-CRP concentrations. Previous studies concluded that total IgE levels increase as an immune response to acute coronary heart disease, and IgE may be a risk factor of ischemic heart disease [27]. A study in Shanghai found that PM2.5 exposure was associated with 3.3% increases in IgE among traffic police officers [28].

Furthermore, NO is an essential molecule of the cardiovascular system. A study reported an increase in NO levels in the early stages of hypoxia and ischemia; therefore, NO can be used as a marker of the risk of cardiovascular disease, especially that caused by fine particle exposure [29]. Similar to our findings, a cohort study reported that the 3-month average concentration of PM10 was associated with increases of serum glucose, HbA1c, LDL, and TG levels and decreased HDL levels [30]. The occurrence of systemic inflammation may lead to the disruption of fat metabolism, decreased anti-inflammatory capacity, and cholesterol transfer by HDL, and fat oxidation, all of which are associated with atherosclerotic risk [31].

This study has potential limitations that can prevent firm conclusions. The number of samples is small, where the number of participants who are vehicle inspector workers is minimal. Nevertheless, the findings of this study are consistent with the current literature on cardiometabolic disorders in subjects exposed to PM. Besides, although this study was cross-sectional, a comparison was made between the exposed group and control group. In the future, similar studies should consider using a larger sample to answer this critical question.

Table 3
Cardiometabolic biomarker concentrations in the exposed and control groups

Parameter (unit value)	Exposed group mean ± SD, (n = 43)	Control group mean ± SD, (n = 22)	*p*
Total cholesterol (mg/dL)	196.30 ± 33.14	196.60 ± 34.40	0.999
LDL cholesterol (mg/dL)	128.25 ± 29.39	121.09 ± 30.87	0.364
HDL cholesterol (mg/dL)	42.79 ± 7.34	47.13± 9.37	0.044*
Triglyceride (mg/dL)	200.25 ± 134.21	123.72 ± 67.38	0.003**
HbA1c (mmol/mol)	44.58 ± 23.19	35.00 ± 4.29	0.012*
Total IgE (µg/L)	473.47 ± 713.28	168.00 ± 211.14	0.031*
hs-CRP (mg/dL)	3.84 ± 4.19	1.57 ± 1.47	0.003**
TNFα (pg/dL)	3.05 ± 2.51	1.31 ± 0.71	0.000***
NO (pg/dL)	4.19 ± 2.32	3.26 ± 1.09	0.030*

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ for an independent t-test.
HDL, high-density lipoprotein; LDL, low-density lipoprotein; hs-CRP, high-sensitivity C-reactive protein; IgE, immunoglobulin E; NO, nitric oxide; SD, standard deviation; TNFα, tumor necrosis factor−alpha.

5. Conclusion

The results illustrated that PM concentrations were significantly higher among mechanic officers. PM0.25 exposure might affect cardiometabolic biomarker change, as shown by the increases of biomarkers of inflammation, glucose, and lipid metabolism disorders levels in vehicle inspectors.

Conflicts of interest

All authors have no conflicts of interest to declare.

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