

Conference Paper

Association Between Levels of Particulate Matter 2.5 (PM_{2.5}) and Tumor Necrosis Factor-Alpha (TNF- α) in Blood of Employees at Motor Vehicle Test Center

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Abstract

Exposure to fine particles can cause chronic and acute inflammation that may change cells to be abnormal. Inflammation occurs when the human body responds to exposure to particulate matter (PM) by releasing Tumor Necrosis Factor-Alpha (TNF- α), a protein that signals inflammation. The object of the present study was to analyze the correlation between human exposure to PM and blood concentrations of TNF- α in Pusat Pengujian Kendaraan Bermotor (PKB), in Ujung Menteng and Pulogadung, in East Jakarta, Indonesia. The study included 42 mechanics from PKB and 27 control participants. The independent T-test was used to assess the smoking status, obesity incidence, and age of the participants. In addition, TNF- α concentrations were analyzed using a quantitative sandwich enzyme immune assay (ELISA) technique and a Human TNF- α /TNFSFIA HS. The study used quantitative analysis to compare TNF- α concentration with the variables in both sample groups. Results showed a higher average concentration of TNF- α in the blood samples of the mechanics group than in those of the control group (p -value < 0.05), meaning that PM_{2.5} exposure may increase TNF- α concentration in human blood.

Keywords: Particulate Matter 2.5 (PM_{2.5}), Tumor Necrosis Factor-Alpha (TNF- α), Inflammation

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1. INTRODUCTION

Air pollution is harmful to the health of workers because it mixes hazardous chemicals with the air in the environment. This pollution in the air can take the form of dust/particulate matter (PM). PM is defined as the condensation phase of particulates in the air. Particulate matter 2.5 is one of a group of small particulates that consist

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of a complex mix of chemicals and contain of a mixture of solids and liquids that can include acids, organic compounds, dust, and more [9].

The potential occurrence of respiratory disorders depends on the size of the PM entering the body. The smallest PM entering the body may stick to areas in the lungs, creating an inflammatory response to the hazardous chemicals in the PM, which are treated like pathogens by the body [2, 11]. These research has correlated high levels of PM in the body with inflammation in the organs.

The human body has a comprehensive system for responding to foreign substances entering it, and part of that system is the release of cytokines, which are small proteins released by cells to interact with foreign substances entering the body. When the primary cytokine protein interacts with foreign substances, it releases Interleukin-1 (IL-1), which is produced by macrophages and lymphocytes in the body and TNF- α . TNF- α is a component of the immune system and triggers inflammation in the cells. In addition, trans membrane TNF- α has bipolar molecules that can pass signals between two ligands and receive signals between cells [6].

Transportation by motorized vehicles continues to increase, making it important to test vehicles for mechanical operation. The vehicle testing center, or Pusat Pengujian Kendaraan Bermotor (PKB), in Ujung Menteng and Pulogadung tests various types of motorized vehicles, including buses, cars, and trucks. PKB employees are exposed to PM while conducting these tests. In a previous study, the average amount of PM exposure of these employees was 272.437 $\mu\text{g}/\text{m}^3$ (\pm 100.770). The present study compared the level of TNF- α in the employee group to that in a control group, which comprised employees in the Faculty of Public Health of the University of Indonesia.

The objective of the study was to analyze the relationship between exposure to PM_{2.5} and the level of TNF- α in the blood of employees of the PKB Ujung Menteng and Pulogadung, in East Jakarta, Indonesia. Blood samples were taken and analyzed in Health Laboratory X, which is experienced in analyzing blood samples. Blood samples were analyzed using a quantitative sandwich enzyme immunoassay (ELISA) technique and HS kit.

2. METHODS

This study used quantitative analysis to identify the relationship between PM_{2.5} exposure and the level of TNF- α in the blood of employees at the PKB in Ujung Menteng and Pulogadung, in East Jakarta, Indonesia. The concentration of TNF- α is expressed as a ratio. The study was conducted between October 2015 and June 2016 and used

two groups, the group exposed to PM_{2.5} and an unexposed control group. Members of the exposed group were employees at the PKB and were mechanical test officers and field employees. The unexposed group consisted of employees in the Faculty of Public Health at the University of Indonesia, who were not exposed to vehicle emissions. The university employees were chosen for use as the control group because it was difficult to find unexposed employees at the PKB.

The study's primary data were the measurements of the concentrations of TNF- α in the blood. The technique used to analyze the blood samples was an ELISA Human/TNFSF1A HS (R & D Systems). In addition to blood samples, the researchers collected data through a questionnaire to obtain information about participants' characteristics. The study's secondary data were the concentrations of PM_{2.5} obtained in a previous study [7]. The values of these concentrations were used to confirm the existence of exposure.

The researchers used an independent t-test to identify any association between TNF- α concentration in a group and the smoking and obesity statuses of that group. In addition, researchers used an Analysis of Variance (ANOVA) test to identify any association between PM_{2.5} exposure and TNF- α concentration. Finally, a correlation test was used to evaluate the strength of the association between employees' ages and the concentration of TNF- α in their blood.

3. RESULTS

TABLE 1: TNF- α Concentration in Exposed and Unexposed Groups, Smoking and Nonsmoking Groups, and Obese and Non-obese Groups and Correlation of Age with TNF- α Concentration.

Variable	Mean TNF- α (pg/ml)	Total	Standard deviation	p value	Correlation (r)
Unexposed	1.349	27	0.656	0.001	-
Exposed	2.793	42	1.860		
Nonsmoking	2.073	31	1.547	0.487	-
Smoking	2.355	38	1.756		
Non-obese (≤ 25)	2.011	42	1.313	0.222	-
Obese (> 25)	2.565	27	2.071		
Control	1.349	27	0.656	-	-
Low exposure	2.427	11	1.429		
High exposure	2.923	31	1.995		
Age	41.72	-	8.734	-	0.114
TNF-α	2.228		1.659		

3.1. Average TNF- α Concentration in Exposed and Unexposed, Smoking and Non-smoking Groups and Obese and Non-obese Groups

Table 1 shows that the TNF- α concentrations in the exposed and unexposed groups had a p value < 0.05 , meaning that exposure to PM_{2.5} was correlated with TNF- α concentrations in employees' blood. However, in the smoking and the obese groups, the p values were > 0.05 , meaning that employees' smoking and obesity status were unrelated to the level of TNF- α concentration in their blood.

3.2. Correlation between Blood TNF- α Concentration and Level of PM_{2.5} Exposure

As Table 1 shows, comparison of TNF- α levels and employees' exposure to PM_{2.5} had a p value < 0.05 , meaning that exposure levels were correlated with TNF- α concentration in the blood.

3.3. Correlation between Age and TNF- α Concentration

Table 1 also shows that the correlation between age and TNF- α concentration in the blood was $r = 11.4\%$, although the value of belonging to the group was very weak.

4. DISCUSSION

PM_{2.5} exposure has been shown to affect the human cardiovascular system. However, to date, certain mechanisms of these effects remain unknown. Short-term PM_{2.5} exposure increases the circulation pressure in the body. In addition, long-term PM_{2.5} exposure contributes to the inflammation process in the blood, called oxidative stress, by releasing TNF- α [1]. The present study found a relationship between TNF- α concentration in the blood and PM_{2.5} exposure from diesel-engine vehicles. This result supports those in the literature showing that the body's response against chemical particles, bacteria, and foreign substances stimulates TNF- α and IL-1 activation and that TNF- α then activates neutrophils in the body, triggering eicosanoid biosynthesis.

Previous studies have also found a relationship between TNF- α concentrations in the blood and exposure to diesel-fuel combustion, which produces PM_{2.5}. They have shown that exposure to 250 $\mu\text{g}/\text{m}^3$ of PM_{2.5}, a high exposure, for four hours can increase the

TNF- α concentration in the blood. However, the present study found no relationship between exposure to 25 $\mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$, a low exposure, for four hours and the TNF- α concentration in the blood. A previous study compared the TNF- α concentration in a high-exposure group, a low-exposure group and a control group. We found the differences and relationships between the level of exposure to $\text{PM}_{2.5}$ and the TNF- α concentration in blood [8].

This study found no association between smoking and TNF- α concentration in the blood. Previous studies have found that the composition of chemical PM could be cytotoxic, causing cell division in the genes to become cancer-like. Such cell division has been correlated with the chronic inflammation triggered by the presence of TNF- α in the blood [4]. The inflammatory process caused by tobacco consumption can activate the immune system of the epithelial cells, causing secretion of pro-inflammatory TNF- α . If this behavior continues for a long time, it activates neutrophils, macrophages, T-cells, and other parts of the cell dendrites, which stimulates chronic inflammation or other disruptions of the respiratory system [10].

The present study found no correlation between obesity and TNF- α concentration in the blood.

A previous study found a positive correlation between increased TNF- α concentration in the blood and the incidence of obesity in humans and animals, which is related to the incidence of insulin resistance. In addition, leptin in the body can be a factor for obesity. The leptin protein influences the hypothalamus and is associated with body weight [5].

The present study found a weak correlation ($r = 11.4\%$) between employees' age and TNF- α concentration in their blood. Previous human studies have found that increased age causes aging in every bodily organ, including the blood vessels. Aging of the blood vessels causes endothelial dysfunction that leads to oxidative stress, which, in turn, leads to cell apoptosis [3].

5. CONCLUSIONS

Motor vehicle testing is fundamental to preventing accidents in public transportation. The present study found that employees of the motor vehicle testing facility in PKB Ujung Menteng and Pulogadung, in East Jakarta, Indonesia, were exposed to $\text{PM}_{2.5}$. In fact, both the exposed employees and an unexposed control group had average concentrations of TNF- α as high as 2.228 $\mu\text{g}/\text{m}^3$. The study found that the level of $\text{PM}_{2.5}$ exposure from vehicle emissions had a positive relationship with the concentration of

TNF- α in the blood, while employees' obesity and smoking statuses had no relationship with the concentration of TNF- α in their blood. Age had a very weak relationship with the concentration of TNF- α in employees' blood.

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