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Association between Air Pollutants and Levels of Macrophage Inflammatory Protein-2 in Purwokerto Informal Workers

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ABSTRAK

Judul : Hubungan Pencemaran Udara dengan Kadar Protein Inflamasi Makrofag-2 pada Pekerja Informal Purwokerto

Latar belakang: Tingkat polusi udara di Indonesia telah meningkat secara signifikan dalam dekade terakhir, yang sejalan dengan peningkatan insidensi gangguan pada sistem pernapasan, termasuk Penyakit Paru Obstruktif Kronik (PPOK). Deteksi dini gangguan sistem pernapasan akibat polusi udara menggunakan penanda biologis berpotensi mencegah keparahan penyakit meskipun masih diperlukan penelitian lebih lanjut. Penelitian ini bertujuan untuk Penelitian ini bertujuan menganalisis hubungan antara pajanan polutan udara dengan kadar Macrophage Inflammatory Protein-2 (MIP-2) pada pekerja sektor informal di Purwokerto, Kabupaten Banyumas, Provinsi Jawa Tengah.

Metode: Studi belah lintang dilakukan pada 35 pekerja parkir luar ruang dan 35 pekerja informal dalam ruang di Purwokerto pada Maret 2021. Kadar *particulate matter* (PM) diukur menggunakan *particle counter* sebagai parameter tingkat polusi udara. Kadar. MIP-2 diukur dari sampel darah partisipan dengan menggunakan metode ELISA. Data dianalisis menggunakan Uji Mann-Whitney, Korelasi Spearman, dan analisis multivariat dengan *Generalized Linear Model* untuk mengevaluasi hubungan antara paparan polutan udara dan kadar MIP-2.

Hasil: Kadar polutan udara di luar ruangan lebih tinggi dibandingkan di dalam ruangan (p=0,00), dan kadar MIP-2 lebih tinggi pada pekerja di luar ruangan dibandingkan pekerja di dalam ruangan (p=0,00). Kadar debu tidak berkorelasi dengan kadar MIP-2, baik pada pekerja di dalam ruangan (r=0,03; p=0,85), pekerja di luar ruangan (r=-0,31; p=0,07), maupun secara keseluruhan (r=0,20; p=0,09). Lama kerja total dan per hari juga tidak memiliki korelasi dengan kadar MIP-2 pada pekerja. Analisis multivariat menunjukkan tidak adanya hubungan antara durasi paparan dan kadar MIP-2 setelah dikendalikan oleh variabel usia dan kadar polusi udara.

Simpulan: Terdapat perbedaan signifikan antara kadar debu dan kadar MIP-2 di lokasi luar ruangan dibandingkan dengan dalam ruangan. Pajanan polutan udara, baik dari segi tingkat maupun durasi, secara konsisten tidak berkorelasi dengan kadar MIP-2 pada pekerja. Penelitian lebih lanjut diperlukan untuk memahami interaksi antara paparan polutan udara, kadar MIP-2, dan kondisi klinis gangguan pernapasan yang disebabkan oleh polusi udara.

Kata kunci: *Microphage Inflammatory Protein-2* (MIP-2); Pekerja Informal; Penyakit Paru Obstruktif Kronik; Polusi udara

ABSTRACT

Background: *Air pollution level has significantly increased in Indonesia followed by the increase in respirational disorders such as Chronic Obstructive Pulmonary Disease (COPD) in the last decade. Early detection of air pollution-related respiratory disorders using biological markers potentially reduces the severity of these diseases, but further studies are still required. This research seeks to evaluate the relationship between exposure to air pollutants and Macrophage Inflammatory Protein-2 (MIP-2) levels among informal workers in Purwokerto, Banyumas District, Central Java Province.*

Method: *A cross-sectional study was carried out in March 2021 involving 35 informal outdoor workers and 35 indoor workers in Purwokerto. Particulate matter (PM) concentration was assessed using a particle counter, serving as an indicator of air pollution level. MIP-2 serum level was measured from participants' blood samples using the ELISA method. The Mann-Whitney test, Spearman correlation test, and multivariate analysis using the Generalized Linear Model were employed to assess the relationship between air pollutant exposure and MIP-2 serum levels.*

Result: *The levels of air pollution (p=0.00) and MIP-2 serum (p=0.00) were significantly elevated in outdoor environments compared to indoor environment. Exposure to air pollutants did not show a significant correlation with MIP-2 serum levels in outdoor workers (r=-0.31; p=0.07), indoor workers (r=0.03; p=0.85), or overall (r=0.20; p=0.09). The overall and daily working duration did not show a correlation with the MIP-2 serum levels in the workers. Multivariate analysis indicated that there was no association between the duration of exposure and MIP-2 levels when adjusted for age and air pollution level.*

Conclusions: *There were notable differences in air pollutant levels and MIP-2 serum levels between indoor and outdoor environments. Air pollutant exposure, both in duration and level, consistently did not correlate with the MIP-2 serum level of workers. Further studies are required to understand the interactions among air pollutant exposure, MIP-2 serum level, and clinical conditions of air pollution-related respiratory disorders.*

Keywords: Microphage Inflammatory Protein-2 (MIP-2); Informal workers; Chronic Obstructive Pulmonary Diseases; Air pollution

INTRODUCTION

The air quality in Indonesia has been worsening rapidly during the last decade. Particulate air pollution levels in Indonesia rose by 171% between 1998 and 2016, shifting the country from being among the world's cleanest to ranking within the twenty most polluted nations.[1] The Indonesian population will lose an average of 1.2 years of life expectancy if the current air pollution level is maintained. In highly polluted areas such as Jakarta, the average life expectancy loss can be as high as four years.[2] The majority of the reduction in life expectancy from air pollution is linked to respiratory illnesses, especially chronic obstructive pulmonary disease (COPD). Over this period, as air pollution grew, low- and middle-income countries like Indonesia experienced a 44% rise in COPD cases.[3] The World Health Organization (WHO) has projected that by 2030, COPD will rank as the third leading cause of death worldwide, with approximately 30-40% of these deaths linked to air pollution. Each year, air pollution is responsible for around 3.2 million COPD-related deaths globally.[4]

Chronic respiratory diseases associated with air pollution, such as chronic obstructive pulmonary disease (COPD) and interstitial lung disease (ILD), are generally asymptomatic and progress gradually over time.[5] As a result, these irreversible conditions tend to be diagnosed at later stages. Delayed diagnosis of pollution-attributed respiratory pathology can negatively impact disease management and prognosis. In advanced diseases, complex therapeutic interventions are often required, demanding increased time, modalities, and cost.[6] More critically, the irreversible damage sustained by late-stage patients leads to poorer outcomes and reduced quality of life. Early identification of air pollution-related respiratory injury is therefore imperative.

Early detection of air pollution-induced respiratory diseases necessitates diagnostic modalities with high accuracy that can be widely implemented. High-resolution computed tomography (HRCT), though considered the gold standard, is often costly and not readily accessible in many regions.[7,8] As an alternative, inflammatory mediators produced during the initial phases of pulmonary inflammation show promise as biomarkers for the early identification of pollution-linked lung disorders.[9] Such mediators are generated with the onset of inflammation, allowing detection prior to functional or structural pathologies.[10] Furthermore, biomarkers can be measured from routine blood draws and analyzed via simple, accessible immunoassay methods like ELISA. The practicality and availability of biomarker screening enhance the feasibility of large-scale implementation for early diagnosis and monitoring. Further research should explore and validate candidate inflammatory biomarkers, in comparison to HRCT and pulmonary function, to evaluate their accuracy in diagnosing subclinical disease.[11] Overall, biomarker-based diagnostic approaches could enable prompt identification of pollution-related respiratory injury to improve outcomes.

Ambient particulate matter (PM) air pollution originating from vehicular emissions can elicit

pulmonary inflammation and mediator release, signaling progression of respiratory pathology.[12] Fine particles between 0.5-1 μm readily permeate epithelial barriers and interact with alveolar cells. Following translocation into the circulatory system, PM induces reactive oxygen species (ROS) formation, resulting in tissue injury and localized inflammatory responses in the lung parenchyma.[13] The particular chemical components within PM can intensify oxidative stress, while the activation of epithelial cells and alveolar macrophages can heighten inflammation, demonstrated by the increased release of inflammatory cytokines like macrophage inflammatory protein-2 (MIP-2).[13,14]

MIP-2 is a key chemotactic cytokine that is upregulated during inflammatory responses. It can be produced by several cell types, such as monocytes, alveolar epithelial cells, macrophages, and hepatocytes, in response to injury or infection.[15] During pulmonary inflammation, MIP-2 is produced simultaneously with several other crucial mediators. These include IL-6, IL-8, TNF-α, IL-2, IL-17, IL-31, IL-33, and KL-6.[16] IL-6 plays a role in various processes such as immune regulation, cancer development, inflammatory responses, and blood cell formation.[17] IL-8 specifically recruits neutrophils to sites of inflammation. TNF-α plays a vital role in inflammatory responses triggered by both infectious and non-infectious causes.[18] IL-2 and IL-17 contribute to tuberculosis-associated lung inflammation, while IL-31 and IL-33 are implicated in asthma pathogenesis.[19] Elevated KL-6 has been linked to alveolar epithelial damage and fibrotic changes in the pulmonary interstitium, indicative of inflammation and fibrosis.[20]

While not exclusive to the respiratory system, MIP-2 production is markedly upregulated in the lungs during inflammatory responses, regardless of inciting etiology (infectious or noninfectious). MIP-2 has been evaluated as a potential biomarker for detecting pulmonary inflammation resulting from infection.[21] Additionally, investigations have associated smoking with elevated MIP-2 serum levels. Cigarette smoke exposure appears to increase systemic MIP-2.[22] Beyond smoking, MIP-2 serum concentrations have been linked to other respiratory diseases like lung cancer and hypertension, as well as individual characteristics including age, sex, and nutritional status. Currently, there is a lack of research directly investigating the connection between exposure to ambient air pollution and levels of circulating MIP-2.. Preliminary findings suggest air pollutant exposure may increase serum MIP-2. Given the potential for MIP-2 as an early biomarker of pollution-induced lung injury, further investigation is warranted. The objective of this research is to examine the relationship between exposure to air pollutants and serum MIP-2 levels among workers in the informal sector of Purwokerto, Indonesia. Evaluating this relationship can elucidate the utility of MIP-2 as a biomarker of effect for air pollution exposure which may demonstrate the practical use of serum MIP-2 measurement for early detection of respiration inflammation in at-risk populations.

MATERIAL AND METHOD

This cross-sectional observational study was conducted in March 2021 on Jenderal Soedirman Street, Purwokerto, Indonesia, which represented the city's largest and most high-traffic thoroughfare. A total sample of 70 male informal workers was recruited by consecutive enrollment. Participants were allocated into two groups: 35 outdoor parking attendants and 35 indoor informal workers. The sample size met the minimum required calculated via the Lemeshow formula.[23] Inclusion criteria comprised age ≤60 years, normal body mass index (18.50-24.99 kg/m2), smoking ≤10 cigarettes daily, lack of mask usage at work pre-pandemically (prior to March 2020), absence of active infections (cough, fever) or chronic respiratory conditions (e.g. asthma, emphysema), and negative history for pneumonia, tuberculosis, diabetes, and hypertension. Before participating, all individuals gave their written informed consent. The Health Research Ethics Committee of the Faculty of Medicine at Universitas Jenderal Soedirman approved the study protocol (Ref: 016/KEP/II/2021).

The primary dependent variable under investigation was serum macrophage inflammatory protein-2 (MIP-2) concentration. Venous whole blood specimens (approximately 3 mL) were collected from the median cubital vein of all study participants and transferred into tubes for processing and storage. The specimens were promptly chilled to a temperature between 1 and 5°C, then subjected to centrifugation at 3000 rpm for a duration of 5 minutes to extract the serum.. Serum aliquots were frozen at -80°C until the time of analysis. The measurement of MIP-2 levels was conducted using a commercially available sandwich ELISA kit designed specifically for detecting human MIP-2/CXCL2 through enzyme-linked immunosorbent assay (ELISA) techniques. Following the manufacturer's instructions, duplicate analyses of serum specimens were conducted. A spectrophotometer was used to measure optical densities at a wavelength of 450 nm, and concentrations were determined by interpolating from a standard curve using ELISA analysis software (arigobio.com).[24] To mitigate potential confounding effects, the researchers collected additional data on several participant characteristics. These variables included participants' age, tobacco use, adherence to personal protective equipment protocols, prior diagnoses of pulmonary conditions (including asthma and pneumonia), and nutritional status as assessed by body mass index (BMI). All these factors were subsequently integrated into multivariate regression models to account for their possible influence on the study outcomes..

The primary independent variable was ambient particulate matter air pollution, represented by fine (PM2.5) and coarse (PM10) particulate concentrations. These were quantified on-site using a particle counter at three peak traffic periods (06:00-07:00, 12:00-13:00, 16:00-17:00) across five roadside locations along the Jenderal Sudirman Street study area. The mean of the three measurements at each site provided composite PM2.5 and PM10 levels, serving as proxies of traffic-related pollution exposure. Secondary predictors included occupational exposure duration, both cumulative years employed and daily hours worked, collected via direct interviews. These temporality variables account for the dependency of pollution health effects on both exposure magnitude and duration. Incorporating both particulate levels and length of exposure enabled comprehensive characterization of study participants' air pollution exposure.

Descriptive statistics were employed to summarize the demographic and clinical characteristics of the study participants. For categorical variables, the results were expressed as counts and percentages. Continuous variables were reported using means and standard deviations. Data distributions were assessed for normality using the Shapiro-Wilk test prior to bivariate analyses. As the data demonstrated non-normal distributions that could not be corrected by transformation, nonparametric tests were employed.[25] Two methods were employed to assess the association between air pollution exposure and serum MIP-2 levels. Initially, the Mann-Whitney U test was used to compare particulate matter concentrations and serum MIP-2 levels in indoor versus outdoor environments. Subsequently, Spearman's rank correlation analysis was conducted to examine the relationship among particulate exposures, duration of occupational exposure, and serum MIP-2 concentrations within each group. A generalized linear model was utilized to explore the complex relationship between exposure variables and MIP-2, while controlling for age and additional covariates to mitigate potential confounding effects. This statistical approach facilitated the analysis of non-normal data distributions without sacrificing ease of interpretation. All statistical computations were executed using SPSS 22.0 software package.

RESULTS AND DISCUSSION

Table 1 displays the descriptive characteristics of participants from the outdoor, indoor, and combined groups in the study. An analysis of workforce demographics revealed a significant age disparity between those employed in outdoor versus indoor settings. The data indicated that a considerable majority (77.1%) of outdoor workers were in the 40-60 year age range. In contrast, only a small fraction (8.6%) of indoor workers fell within this same age category. Overall, the majority of study subjects were younger, aged 20-40 years (57.1%). All outdoor workers were smokers (100%), while the majority of indoor subjects also had a smoking history (80%). Both employees working outdoors and those working indoors reported using universal personal protective equipment (PPE). Participants in both the indoor and outdoor groups exhibited healthy body mass index (BMI) measurements and lacked any previous pulmonary disease diagnoses. Employees who work outdoors typically had longer careers in their field, with 51.4% having more than a decade of experience. In contrast, only 20% of those working indoors had surpassed the ten-year mark in their occupations.. The majority of workers labored less than eight hours per day in both the indoor (51.4%) and outdoor (71.5%) groups.

Table 1. Basic characteristics of the study subject

Characteristics	Frequency (%)		
	Outdoor	Indoor	Total
Age group			
20-40	8 (22.9)	32 (91.4)	40 (57.1)
41-60	27 (77.1)	3 (8.6)	30 (42.9)
Smoking status			
Yes	35 (100.0)	28 (80.0)	63 (90.0)
No	-	7 (20.0)	7 (10.0)
Working duration (overall)			
< 10 years	17 (48.6)	28 (80.0)	45 (64.3)
≥ 10 years	18 (51.4)	7 (20.0)	25 (35.7)
Working duration per day			
< 8 hours	25 (71.5)	18 (51.4)	43 (61.4)
≥ 8 hours	10 (28.5)	17 (48.6)	27 (38.6)

Table 2 demonstrates associations between air pollutant levels, MIP-2 serum levels, and study settings (outdoor versus indoor). Measurements of air pollution, assessed through PM2.5 and PM10 dust particle concentrations, revealed substantially higher levels in exterior environments (26.67 ± 3.31 μg/m3) than in interior spaces (16.21 ± 1.96 μg/m3). This difference was statistically significant, as indicated by a p-value of 0.000.. Similarly, the mean MIP-2 serum levels were considerably higher among individuals working in outdoor settings versus indoor settings. Outdoor workers exhibited significantly elevated serum MIP-2 levels, with a mean of 23.43 ± 15.38 ng/ml, compared to indoor workers, who had an average MIP-2 concentration of 16.41 ± 13.04 ng/ml in their serum (p-value < 0,001).

Table 2. Comparison of air pollutant and MIP-2 serum level between outdoor and indoor settings.

Variables	Setting		p-value[1]
	Outdoor (mean± SD)	Indoor (mean± SD)	
Air pollutant level (μg/m³)	26.67 ± 3.31	16.21 ± 1.96	0.000
MIP-2 serum level (ng/ml)	23.43 ± 15.38	16.41 ± 13.04	0.000

[1]Mann-Whitney test

The correlations between air pollutant exposure and MIP-2 serum levels for both indoor and outdoor workers are displayed in Table 3. No significant correlations were found between MIP-2 and air pollutant exposure in either the outdoor (r = 0.03, p = 0.85) or indoor (r = -0.31, p = 0.07) work environments. Additionally, the relationship between overall working duration and MIP-2 serum levels was

not statistically significant for outdoor (r = 0.08; p = 0.64) or indoor (r = 0.20; p = 0.26) workers. Similar findings were observed when analyzing the correlation between daily working hours and MIP-2 levels among outdoor (r = 0.07; p =0.64) and indoor (r = 0.03; p = 0.85) workers.

Table 3. Correlation of air pollutant exposure (level and duration) and MIP-2 serum level in indoor and outdoor settings.

Variables	Setting			
	Outdoor		Indoor	
	r	p-value	r	p-value
Air pollutant level	0.03	0.85	-.0.31	0.07
Overall working duration	0.08	0.64	0.20	0.26
Working duration per day	0.07	0.64	0.03	0.85

Table 4 utilized a Generalized Linear Model (GLM) to investigate the relationships between air pollution exposure levels, duration of exposure, and serum concentrations of MIP-2, while considering age as a potential confounding factor. The results, after adjusting for age, indicated no statistically significant linear associations between MIP-2 serum concentrations and air pollutant levels, overall work duration, or daily hours worked.

Table 4. Multivariate analysis of association between air pollutant exposure and MIP-2 serum level using generalized linear model (GLM).

Variables	B (coefficient)	Standard error (SE)	p-value
Age	0.02	0.01	0.17
Overall working duration	0.01	0.01	0.67
Working duration per day	0.01	0.04	0.87
Air pollutant level	0.02	0.02	0.29

This research investigated the relationship between exposure to air pollutants and MIP-2 serum concentrations in workers from both indoor and outdoor environments in Purwokerto, Banyumas District, Central Java Province. Results indicated that outdoor environments exhibited considerably higher air pollutant levels than indoor spaces. Additionally, individuals employed in outdoor settings displayed notably elevated MIP-2 serum levels compared to their indoor counterparts. Correlation analyses failed to reveal significant associations between air pollutant concentrations and MIP-2 serum levels for workers in either outdoor or indoor settings. Furthermore, no substantial correlations were observed between work duration (both overall and daily) and MIP-2 serum levels in either group of workers. When controlling for age, work duration, and air pollutant levels, linear associations with MIP-2 serum concentrations were found to be non-significant across the entire study population.

The findings of significantly higher PM2.5 and PM10 levels in outdoor versus indoor settings are expected given the differing exposure conditions. Outdoor areas are directly exposed to various air pollution sources.[26] In this study, the outdoor worksite's location on a major roadway in Purwokerto City with high motor vehicle traffic likely contributed to the considerably elevated dust levels. On the other hand, the indoor environment was situated in the same area but within a concrete structure, which served as a physical shield, protecting employees from dust exposure. Furthermore, the majority of indoor spaces were equipped with air conditioning units that filtered airborne particles, thus lowering indoor dust concentrations.. Proper ventilation circulates and replaces indoor air, further decreasing dust accumulation and pollutant exposure.[27] The indoor building infrastructure and ventilation likely explain the lower indoor PM2.5 and PM10 levels compared to the outdoor space directly exposed to traffic emissions.

The finding of significantly higher MIP-2 serum levels in outdoor versus indoor workers can be explained by several potential mechanisms. First, outdoor dust levels were higher than indoor levels, likely inducing more airway inflammation and increasing MIP-2 production. Long-term exposure to airborne particles from outdoor pollution can lead to oxidative damage in respiratory cells. This process increases the production of reactive oxygen species and oxidative stress, while also triggering both innate and adaptive immune responses.[28,29] This respiratory tract irritation from prolonged outdoor PM exposure may explain the higher outdoor worker MIP-2 levels. Additionally, the outdoor workforce was significantly older compared to those working indoors. Higher age has been linked to increased individual MIP-2 levels. It is probable that older people have encountered more instances of illness throughout their lives, resulting in accumulated damage to cells in their lungs and respiratory system.[30] Several non-respiratory illnesses like liver injury have been linked to increased serum MIP-2.[31] The deterioration of lung tissue may be hastened by these conditions associated with aging, potentially explaining the elevated MIP-2 levels observed in older individuals working outdoors compared to their younger counterparts employed indoors.

The lack of significant correlation between MIP-2 serum levels and particulate matter exposure found in this study may be attributable to several factors based on recent literature. MIP-2 responses tend to be acute and transient, so precisely-timed sampling could be needed to capture peak levels.[32] High inter-individual variability in MIP-2 reactions to the same particulate matter exposure may obscure population-level associations.[33] Additionally, particulate matter chemical composition and soluble metal content, rather than just particle mass, can influence MIP-2 induction. Finally, co-exposure to gaseous pollutants like ozone or cigarette smoke can modify airway cytokine responsiveness.[34] Therefore, the transient, variable nature of MIP-2 responses, the chemical heterogeneity of particulate matter, and co-exposures likely

contributed to the lack of correlation between MIP-2 and particulate levels seen in this cross-sectional study.

This study found no correlation between overall working duration or daily work duration and MIP-2 serum levels in outdoor and indoor workers. The concentration of MIP-2 serves as an indicator of inflammatory processes and pulmonary damage resulting from exposure to airborne pollutants.[35] The severity of respiratory effects caused by air pollutants is determined by both the pollutant concentration and the length of time an individual is exposed to them.[36] This research, despite involving prolonged (chronic) exposure, featured relatively low air pollutant concentrations that were significantly below maximum permissible limits. Extended exposure to such low-level air pollution might not be sufficient to trigger significant pathological alterations in the respiratory system, which could explain the comparatively low MIP-2 levels detected.[37] The absence of a correlation between work duration and MIP-2 concentrations supports this observation. Additionally, after accounting for confounding factors, multivariate analysis revealed no significant linear relationship among air pollutant concentrations, overall work duration, and MIP-2 levels.

One notable advantage of this research was the use of ELISA, an internationally recognized technique, to quantify MIP-2 serum concentrations. This method is not commonly employed for identifying respiratory conditions in Indonesia. However, there were some limitations. Although the sample size was sufficient for conducting bivariate analysis, it may have been inadequate to detect relationships between variables in multivariate analysis. Additionally, measurement of confounding variables relied on participants' recall during direct interviews, which is prone to recall bias and may not accurately capture participants' true conditions.

CONCLUSIONS

This research examined the relationship between air pollution exposure and the levels of the chemokine MIP-2 in blood serum among individuals employed in diverse occupational environments. The findings demonstrated that participants in different occupational environments experienced differential air pollution exposure and exhibited differing MIP-2 serum concentrationsDespite bivariate analysis revealing unexpected, non-significant correlations between exposure to air pollutants and MIP-2 levels, the current data did not allow for conclusive determinations regarding how air pollution affects MIP-2 as an indicator of respiratory disease. Further inquiry with expanded sample size, improved quantification of air pollution exposure, more accurate measurement of confounding variables, for instance, comparison between groups with similar age characteristics of participants, investigation of the interrelationships between exposure, exposure duration, MIP-2 levels, and other determinants is still required. Such research will facilitate characterization of the potential utility of MIP-2 as an indicator of pulmonary disorders resulting from air pollution. Additional work is needed to firmly establish the association between air pollution and MIP-2.

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