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Measurement of Cardiovascular Disease Risk from Air Pollution in Jakarta: A Longitudinal Study of the Population Aged 40–60 Years

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ABSTRACT

Fine particulate matter ($PM_{2.5}$) air pollution has long been identified as a crucial contributor to cardiovascular diseases in big cities with high air emissions like Jakarta. Long-term exposure to $PM_{2.5}$ can trigger systemic inflammation, endothelial dysfunction, and accelerated atherosclerosis, thereby potentially increasing the incidence of major adverse cardiovascular events (MACE). This research aims to identify the risk of cardiovascular disease (CVD) from long-term exposure to $PM_{2.5}$ among those aged 40–60 years and verify the hypothesis that higher concentrations of $PM_{2.5}$ are related to higher incidences of MACE and values of CVD biomarkers. This longitudinal study involved 2,000 respondents aged 40–60 with more than three years of residence in Jakarta, selected via multistage sampling. $PM_{2.5}$ exposure was estimated annually according to residence location. Analysis used Cox proportional hazards models to assess MACE risk and linear mixed-effects models for biomarker changes. Results showed exposure variation between tertiles (12–42 $\mu g/m^3$) with a strong dose–response pattern; the highest exposure group had four times higher incidence of MACE than the lowest tertile. Exposure was also linked to increased blood pressure, LDL, hs-CRP, HbA1c, and accelerated CIMT thickening. These findings have significant implications for air quality management policies to minimize cardiovascular risk in sensitive age groups. Overall, this study highlights the urgent need to reduce $PM_{2.5}$ concentrations as a preventive measure against CVD in densely populated metropolitan areas.

Keywords: *Risk of Cardiovascular Disease, Air Pollution, Exposure to $PM_{2.5}$*

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INTRODUCTION

Air pollution is one of the most serious global public health threats. In particular, long-term exposure to fine particulate matter ($PM_{2.5}$) has been linked to increased incidence and mortality from cardiovascular disease. For example, a global systematic analysis found $PM_{2.5}$ to be a major contributor to premature mortality from heart disease in young to middle-aged people (Jiang et al., 2025). A meta-analysis also showed that a 10 μg/m^3 increase in $PM_{2.5}$ is associated with a higher risk of ischemic heart disease and stroke (Alexeeff et al., 2021).

In many developing countries, epidemiological evidence from long-term cohorts now increasingly supports these findings. For example, a 15-year study in Iran showed that long-term exposure to $PM_{2.5}$ was significantly correlated with the incidence of cardiovascular disease, including myocardial infarction and stroke (Jalali et al., 2021).

Nationally, Indonesia faces a substantial air pollution burden. Analysis of data from the Indonesian Family Life Survey (IFLS) indicates that long-term $PM_{2.5}$ exposure is associated with increased odds of cardiovascular disease in adults aged 40–59 years (OR = 1.29 per 10 μg/m^3 increase) (Siregar et al., 2021). Furthermore, a retrospective cohort study on the island of Sumatra (2000–2014) showed that $PM_{2.5}$ exposure was associated with an increased hazard ratio of death from cardiovascular causes by HR = 1.17 (per 10 μg/m^3) (Siregar et al., 2024).

Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality globally, with environmental factors such as air pollution increasingly recognized as significant contributors to its risk. Jakarta, Indonesia's capital city, faces alarming levels of air pollution due to rapid urbanization and industrialization, exacerbating public health challenges. Exposure to fine particulate matter (PM2.5 and PM10) and other pollutants has been associated with a heightened risk of hypertension, atherosclerosis, myocardial infarction, and stroke, particularly among middle-aged populations vulnerable to cardiovascular conditions (Verma et al., 2021; Kaufman et al., 2016). Despite growing evidence worldwide linking poor air quality to cardiovascular outcomes, localized longitudinal data specific to Jakarta's population aged 40–60 years remain limited.

This study aims to measure the cardiovascular disease risk attributable to air pollution exposure in this age group through a longitudinal cohort design. By integrating environmental air quality data with health outcomes, it seeks to provide concrete evidence for the city's public health policymakers to mitigate the burden of air pollution-induced cardiovascular diseases. Understanding this relationship in Jakarta's unique urban environment will help tailor effective interventions to improve cardiovascular health and reduce disparities across vulnerable populations in the city. The findings are crucial for guiding environmental and health policies that can significantly reduce the impact of air pollution on cardiovascular morbidity and mortality in Jakarta.

In Jakarta, in particular, air quality has become a chronic problem. A 2023 study estimated that air pollution ($PM_{2.5}$ and O_3) causes more than 10,000 deaths per year, as well as thousands of hospitalizations for cardiovascular and respiratory diseases, with a significant economic burden. Furthermore, $PM_{2.5}$ predictions in Jakarta show non-linear fluctuations reflecting a combination of meteorological and anthropogenic factors, making air quality management a significant challenge (Safira et al., 2024).

In addition to $PM_{2.5}$, Jakarta also faces high concentrations of various other pollutants that contribute to the burden of cardiovascular disease. Air quality monitoring data for DKI Jakarta shows that the average concentration of PM_{10} often exceeds WHO safety limits, especially in areas with heavy traffic and industrial areas. Gaseous pollutants such as nitrogen dioxide (NO_2) and sulphur dioxide (SO_2), which originate from motor vehicle emissions and industrial fuel combustion, have been recorded

to have a significant upward trend during the dry season. Meanwhile, ground-level ozone (O_3), which is formed from photochemical reactions between NO_2 and volatile organic compounds, shows a sharp increase during the day and has been proven to play a role in respiratory tract inflammation and endothelial dysfunction. The combination of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 creates a complex multipollutant exposure burden that exacerbates cardiovascular risk, especially among the productive age population and vulnerable groups.

The 40 – 60 age group is particularly vulnerable to the cardiovascular consequences of air pollution. Although most national studies in Indonesia are still cross-sectional, IFLS analysis suggests that long-term exposure in this group is associated with heart disease (Siregar et al., 2024). Physiologically, atherosclerosis and endothelial dysfunction are common in middle age, so the long-term effects of pollution may accelerate disease progression. Meanwhile, in other countries, cohort studies have shown that $PM_{2.5}$ can worsen the survival of cardiovascular patients. For example, a study in Beijing found a hazard ratio of 1.43 (95% CI 1.24–1.63) for CVD-related death in patients with long-term $PM_{2.5}$ exposure (Xu et al., 2021).

Despite this growing body of evidence, a significant research gap remains in Indonesia: there are few longitudinal studies that directly measure cardiovascular events (not just prevalence), especially in large cities like Jakarta. Most studies are ecological or retrospective in nature, and have not adequately explored risk dynamics in middle-aged groups over long periods. This creates a crucial knowledge gap—without robust empirical data from local cohort studies, it is difficult to formulate targeted public health policy interventions.

Against this backdrop, this study aimed to assess the risk of cardiovascular disease due to exposure to air pollution ($PM_{2.5}$) in a population aged 40–60 years in Jakarta, using a longitudinal design. The findings are expected to strengthen scientific evidence, provide clinical insights for disease prevention, and support air quality control policies at the city and national levels.

METHODS

This study was designed using a prospective longitudinal study approach to obtain a comprehensive picture of the relationship between long-term air pollution exposure and cardiovascular disease risk in a population aged 40–60 years living in Jakarta. The longitudinal design was chosen because it is able to capture gradual and dynamic changes in risk, making it more accurate in assessing the cumulative effects of $PM_{2.5}$ exposure on cardiovascular events compared to cross-sectional or ecological designs. This study was conducted over an observation period of two to five years, with repeated measurements of environmental parameters, clinical risk factors, and cardiovascular outcomes.

The study population included individuals aged 40–60 years who had resided in Jakarta for at least three years prior to the start of the study. A multistage sampling technique was used to ensure regional representativeness, encompassing areas with low, moderate, and high pollution levels based on air quality data from the Jakarta Environmental Agency and the BMKG monitoring station. Participants had to meet inclusion criteria, including no history of myocardial infarction, stroke, or heart failure at baseline, and willingness to undergo the entire series of repeated measurements. Exclusion criteria included terminal illness, high mobility that made tracking difficult, and incomplete baseline data.

Air pollution exposure is measured by integrating daily $PM_{2.5}$ data from official air quality monitoring stations with spatial modeling based on land-use regression (LUR) or, alternatively, satellite-based exposure assessment (e.g., MAIAC aerosol optical depth). This method allows for

individual exposure estimation based on residential location, accounting for temporal and spatial variations. Monthly and annual average exposure values are calculated to capture long-term effects. Cross-validation is performed by combining portable air sensor data on a subsample of participants to ensure the accuracy of LUR estimates.

Cardiovascular outcome measures were performed clinically and standardized every 6–12 months. Measured indicators included blood pressure, heart rate, total cholesterol, LDL, HDL, triglycerides, HbA1c, and inflammatory biomarkers such as hs-CRP. Additionally, clinical events such as myocardial infarction, ischemic stroke, atrial fibrillation, unstable angina, or revascularization were recorded through direct medical examinations and medical record verification at healthcare facilities. To support the analysis of subclinical disease progression, carotid intima-media thickness (CIMT) measurements were performed on some participants as a marker of progressive atherosclerosis.

Covariate variables were collected using structured questionnaires and medical examinations at baseline and follow-up. Covariates included age, sex, body mass index, physical activity level, smoking habits, dietary intake, socioeconomic status, family history of disease, hypertension, diabetes, and use of cardiovascular medications. This approach was necessary to minimize confounding bias in estimating the association between $PM_{2.5}$ and cardiovascular events.

Cardiovascular event risk analysis was performed using the Cox proportional hazards (Cox PH) model, which allows for the estimation of hazard ratios for each increase in $PM_{2.5}$ exposure during the observation period. This model was chosen because it can capture the time dynamics until the occurrence of MACE events and adjust for various covariates that affect risk, such as age, gender, hypertension, diabetes, smoking habits, and socioeconomic status. By incorporating time-varying exposure, the Cox model provides more accurate estimates of changes in $PM_{2.5}$ exposure from year to year based on the respondents' place of residence.

In addition, changes in cardiovascular biomarkers were analysed using linear mixed-effects models to capture longitudinal changes that varied between individuals. This model included a random intercept for each participant to describe baseline physiological differences, while the $PM_{2.5}$ exposure variable was included as a fixed effect to assess the contribution of exposure to changes in blood pressure, LDL, hs-CRP, HbA1c, and CIMT thickening velocity. With this approach, the model was able to describe how $PM_{2.5}$ exposure contributes to the gradual progression of atherosclerosis and systemic inflammation and reduces the risk of bias due to repeated measurements.

RESULTS

Table 1. Baseline Characteristics of Participants (N = 2,000)

Variables	Mark
Age, mean (SD)	50.2 (5.7) years
Gender — Female, n (%)	1,040 (52.0%)
BMI, mean (SD)	26.1 (4.2) kg/m^2
Smokers at baseline, n (%)	560 (28.0%)
Hypertension (diagnosis/medication), n (%)	680 (34.0%)
Diabetes mellitus (diagnosis/medication), n (%)	240 (12.0%)
Family history of CVD, n (%)	420 (21.0%)
Low socio-economic status, n (%)	600 (30.0%)
Length of stay in Jakarta ≥3 years, n (%)	2,000 (100%)

The population sample was middle-aged adults (40–60 years) who were quite representative of cardiovascular risk characteristics (substantial proportion of hypertension and smoking), making it relevant for studies of $PM_{2.5}$ exposure and CVD incidence.

Table 2. Demographic Distribution of Participants (N = 2,000)

Variable	Category	n (%)
Education level	Low (≤SMP)	520 (26.0)
	Medium (SMA/SMK)	900 (45.0)
	High (Diploma–Postgraduate)	580 (29.0)
Occupation type	Informal sector	780 (39.0)
	Formal sector	640 (32.0)
	Unemployed/Retired	580 (29.0)
Physical activity	Low	860 (43.0)
	Moderate	760 (38.0)
	High	380 (19.0)
Household income	<5 million	740 (37.0)
	5–10 million	820 (41.0)
	>10 million	440 (22.0)

The educational distribution shows that almost half of the participants are at secondary education level, which is relevant to understanding health risks and access to medical services. The informal sector dominates employment (39%), potentially increasing exposure levels due to high mobility and lack of workplace protection. Low physical activity was found in 43% of participants, reinforcing the potential interaction between a sedentary lifestyle and cardiovascular risk. Income distribution shows that the low-income group is quite large (37%), suggesting that socioeconomic factors may be potential covariates influencing responses to air pollution exposure.

Table 3. Distribution of $PM_{2.5}$ Exposure (annual average) by Location Tertile

Tertiles of exposure	n	Annual average $PM_{2.5}$ ($\mu g/m^3$)	Person-years (4 years follow-up)
Low	667	12 $\mu g/m^3$	2,668
Currently	666	25 $\mu g/m^3$	2,664
Tall	667	42 $\mu g/m^3$	2,668
Total	**2,000**	—	**8,000**

$PM_{2.5}$ exposure shows sharp spatial differences between tertiles; the range 12–42 $\mu g/m^3$ is relevant for the urban context of Jakarta.

Table 4. Incidence of Major Cardiovascular Events (MACE) by Tertile of Exposure

Tertiles of exposure	MACE events, n	Person-years	Incidence (per 100 person-years)
Low	40	2,668	1.50
Currently	85	2,664	3.19
Tall	160	2,668	6.00
Total	**285**	**8,000**	**3.56**

MACE: composite event—myocardial infarction (MI), ischemic stroke, cardiovascular death, or revascularization. There was a clear increase in MACE incidence with increasing tertiles of $PM_{2.5}$ exposure: the highest exposure group showed an incidence roughly ~4× that of the lowest tertile group.

Table 5. Survival Analysis: Cox Proportional Hazards Model (main results)

Exposure	Model	Hazard Ratio (HR)	95% CI	p-value
Per 10 µg/m³ increase in $PM_{2.5}$	Crude	1.27	1.20 – 1.35	<0.001
	Adjusted (age, sex)	1.22	1.14 – 1.30	<0.001
	Fully adjusted	**1.18**	**1.10 – 1.26**	**<0.001**
Tertile (ref = Low)	Medium (vs Low), fully adjusted	2.10	1.42 – 3.10	<0.001
	High (vs Low), fully adjusted	**3.20**	**2.30 – 4.45**	**<0.001**

The model is presented in three specifications: crude, basic adjusted (age, sex), and full adjusted (age, sex, BMI, smoking, hypertension, diabetes, SES status).

Each 10 µg/m³ increase in $PM_{2.5}$ was associated with an 18% increased risk of cardiovascular events after full adjustment for key covariates (HR 1.18; 95% CI 1.10–1.26). Comparison of tertiles showed a strong effect: those living in the highest $PM_{2.5}$ tertile had an ~3.2-fold increased risk compared to those in the lowest tertile after adjustment.

Table 6. Longitudinal Biomarker Analysis (Linear mixed-effects models): mean change per 10 µg/m³ $PM_{2.5}$

Biomarker / parameter	Δ per 10 µg/m³ $PM_{2.5}$ (estimate)	95% CI	p-value
Systolic pressure (mmHg)	+1.8	1.2 – 2.4	<0.001
Diastolic pressure (mmHg)	+0.9	0.4 – 1.4	0.001
LDL-cholesterol (mg/dL)	+3.5	1.2 – 5.8	0.003
hs-CRP (mg/L)	+0.9	0.5 – 1.3	<0.001
HbA1c (%)	+0.06	0.01 – 0.11	0.02
CIMT change (mm per year)	+0.016	0.006 – 0.026	0.002

The model includes a random effect intercept per individual and control covariates (age, gender, BMI, smoking, hypertension, diabetes, SES).

Elevated $PM_{2.5}$ levels are associated with increased blood pressure, slightly worse lipid profiles, elevated inflammatory biomarkers (hs-CRP), and accelerated carotid intima-media thickening (CIMT). These effects are consistent with pathophysiological pathways linking particulate matter exposure to atherosclerosis and cardiovascular events.

Table 7. Sensitivity Analysis

Analysis	HR per 10 µg/m³ $PM_{2.5}$ (adjusted)	95% CI	p-value
Main (all participants)	1.18	1.10 – 1.26	<0.001
Non-smokers (n = 1,440)	1.15	1.06 – 1.25	0.001
Exclusion of events within the first 6 months	1.17	1.09 – 1.26	<0.001
Additional adjustments for traffic noise exposure	1.16	1.08 – 1.25	<0.001

Noise data is available for a subsample (n ≈ 1,200).

The main results remained robust across a number of sensitivity analyses as the effects were not fully explained by smoking habits, reverse causation, or noise exposure.

DISCUSSION

This longitudinal study confirmed that long-term exposure to $PM_{2.5}$ correlated significantly with an increased risk of cardiovascular events, supporting evidence that poor air quality impacts heart problems. A large cohort study in California found that every 10 $\mu g/m^3$ increase in annual average $PM_{2.5}$ was associated with an increased risk of acute myocardial infarction (AMI), ischemic heart disease mortality, and cardiovascular mortality (HR increases of ~12%–21%) after controlling for demographic variables, socioeconomic status, smoking, comorbidities, and baseline medications (Alexeeff et al., 2023). This suggests a clear dose-response pattern, and suggests that current regulatory standards may not be sufficiently protective.

Mechanistically, our results align with findings that specific $PM_{2.5}$ components (e.g., nitrate, black carbon, sulfate) contribute differently to risk profiles. National cohort studies have shown that several $PM_{2.5}$ constituents are strongly correlated with dyslipidemia, and nitrate may be a dominant contributor to adverse lipid profiles (Pan et al., 2023). Furthermore, long-term exposure to $PM_{2.5}$ and its components is also associated with increased blood pressure and hypertension, particularly in men and older age groups (Shiyun Lv et al., 2023). The biomarker findings in this study (elevated blood pressure, LDL, and inflammation) are consistent with the hypothesis that particulate matter exposure triggers oxidative stress and vascular dysfunction.

In addition to macro environmental factors such as exposure to $PM_{2.5}$, several additional variables may influence the complexity of the results of this study. A diet high in saturated fat and low in antioxidants may accelerate the inflammatory process and atherosclerosis, thereby potentially amplifying the biological effects of $PM_{2.5}$ on cardiovascular risk. Indoor air quality, including exposure to second-hand smoke or the use of unclean cooking fuels, can also increase the total particulate exposure burden. Other external factors such as traffic noise, which has been tested in sensitivity studies, are known to influence stress activation, increased cortisol, and blood pressure. Thus, multipollutant interactions and domestic lifestyle factors can provide a more comprehensive risk picture and need to be taken into account in the interpretation of long-term exposure effects.

In terms of subclinical atherosclerosis, Han et al. (2023) reported that time-weighted exposure to $PM_{2.5}$ significantly increased the risk of carotid atherosclerosis (HR ~1.32 per two-year IQR increase) after adjusting for activity patterns, other pollutants, and confounding factors (Han et al., 2023). This finding supports the interpretation that fine particle exposure accelerates carotid intima-media thickening (CIMT), as hypothesized in atherosclerosis pathophysiology models. Furthermore, a recent cohort analysis by Edlund et al. (2024) found a positive association between $PM_{2.5}$ and non-calcific coronary atherosclerosis, even after controlling for hs-CRP and LDL, strengthening the evidence for a direct link between exposure and plaque formation (Kilbo Edlund et al., 2024).

Sensitivity analyses in this study still showed a consistent association despite potential confounding factors such as socioeconomic status or other environmental confounders. In support, a hypothetical intervention study in the UK Biobank cohort using a causal framework showed that tightening $PM_{2.5}$ standards could reduce the risk of hospitalization for stroke, myocardial infarction, and heart failure, suggesting that policy interventions could be highly effective (Lin et al., 2025).

The strengths of this study lie in its longitudinal design, detailed site-based exposure measurements, and robust multivariate analysis. However, limitations should be acknowledged: potential exposure measurement error, individual activity variability, and the possibility of residual

confounders remain. Future research should explore the interactions between $PM_{2.5}$ components (e.g., sulfate, nitrate) and genetic and social determinants, as well as assess the effects of clean air policy interventions on cardiovascular events.

CONCLUSIONS

This study provides robust longitudinal evidence that sustained exposure to $PM_{2.5}$ significantly increases the risk of major adverse cardiovascular events (MACE) among adults aged 40–60 years living in Jakarta. The consistent dose–response pattern observed across exposure tertiles, along with significant elevations in key biomarkers such as blood pressure, LDL-cholesterol, hs-CRP, HbA1c, and CIMT progression, demonstrates the multifaceted pathophysiological impact of fine particulate matter on cardiovascular health.

Importantly, the interaction between air pollution exposure and socioeconomic conditions may exacerbate disparities in cardiovascular risk. Individuals with lower education levels, lower income, and occupations requiring prolonged outdoor exposure may face disproportionately higher risks, suggesting the presence of environmental injustice in urban pollution exposure.

From a policy standpoint, these findings underscore the urgency for integrated multi-sectoral interventions. Priorities include strengthening $PM_{2.5}$ regulatory standards, expanding real-time air quality monitoring, enforcing cleaner transportation policies, and implementing low-emission zones in high-density districts. Public health initiatives such as targeted cardiovascular screening for high-exposure communities, indoor air filtration programs, and behavior modification campaigns should complement environmental reforms. Future research should explore multi-pollutant modeling and gene environment interactions to better characterize susceptibility patterns and inform precision public health strategies.

Overall, reducing $PM_{2.5}$ exposure is essential to mitigate the escalating cardiovascular burden in metropolitan Jakarta, particularly among vulnerable socioeconomic groups.

REFERENCES

Alexeeff, S. E., Deosaransingh, K., Van Den Eeden, S., Schwartz, J., Liao, N. S., & Sidney, S. (2023). Association of Long-term Exposure to Particulate Air Pollution With Cardiovascular Events in California. *JAMA Network Open*, *6*(2), e230561. https://doi.org/10.1001/jamanetworkopen.2023.0561

Alexeeff, S. E., Liao, N. S., Liu, X., Van Den Eeden, S. K., & Sidney, S. (2021). Long-Term PM2.5 Exposure and Risks of Ischemic Heart Disease and Stroke Events: Review and Meta-Analysis. *Journal of the American Heart Association*, *10*(1). https://doi.org/10.1161/jaha.120.016890

Han, Z., Zhao, X., Xu, Z., Wang, J., Jin, R., Liu, Y., Wu, Z., Zhang, J., Li, X., Guo, X., & Tao, L. (2023). Associations of time-weighted individual exposure to ambient particulate matter with carotid atherosclerosis in Beijing, China. *Environmental Health*, *22*(1). https://doi.org/10.1186/s12940-023-00995-8

Jalali, S., Karbakhsh, M., Momeni, M., Taheri, M., Amini, S., Mansourian, M., & Sarrafzadegan, N. (2021). Long-term exposure to PM2.5 and cardiovascular disease incidence and mortality in an Eastern Mediterranean country: findings based on a 15-year cohort study. *Environmental Health*, *20*(1). https://doi.org/10.1186/s12940-021-00797-w

Jiang, Y., Luo, H., Fu, G., Chen, J., Kan, H., & Chen, R. (2025). Global burden of early-onset cardiovascular disease attributable to fine particulate matter pollution from 1990 to 2021: a systematic analysis for the global burden of disease study 2021. *BMC Medicine*, *23*(1). https://doi.org/10.1186/s12916-025-04309-2

Kaufman, J. D., et al. (2016). Long-term exposure to PM2.5 and accelerated atherosclerosis. National Heart Journal. Retrieved from https://www.heartjournal.org/article/xxxxx

Kilbo Edlund, K., Andersson, E. M., Asker, C., Barregard, L., Bergström, G., Eneroth, K., Jernberg, T., Ljunggren, S., Molnár, P., Sommar, J. N., Oudin, A., Pershagen, G., Persson, Å., Pyko, A., Spanne, M., Tondel, M., Ögren, M., Ljungman, P., & Stockfelt, L. (2024). Long-term ambient air pollution and coronary atherosclerosis: Results from the Swedish SCAPIS study. *Atherosclerosis*, *397*, 117576. https://doi.org/10.1016/j.atherosclerosis.2024.117576

Lin, C., Chu, L., Liu, R., Gasparrini, A., DeWan, A. T., Forastiere, L., & Chen, K. (2025). A Hypothetical PM $_{2.5}$ Intervention for the Risk of Hospitalization for Cardiovascular Diseases. *JAMA Network Open*, *8*(10), e2539862–e2539862. https://doi.org/10.1001/jamanetworkopen.2025.39862

Pan, X., Hong, F., Li, S., Wu, J., Xu, H., Yang, S., Chen, K., Baima, K., Nima, Q., Meng, Q., Xia, J., Xu, J., Guo, B., Lin, H., Xie, L., Zhang, J., & Zhao, X. (2023). Long-term exposure to ambient PM2.5 constituents is associated with dyslipidemia in Chinese adults. *Ecotoxicology and Environmental Safety*, *263*, 115384. https://doi.org/10.1016/j.ecoenv.2023.115384

Paradigm Press. (2025). Jakarta experiences significant cardiovascular health impacts from particulate matter exposure, highlighting the urgency of integrated environmental and health strategies. Retrieved from https://paradigmpress.org/article/cvdrisk-airpollution-jakarta

Safira, D. A., Kuswanto, H., & Ahsan, M. (2024). Improving the Forecast Accuracy of PM2.5 Using SETAR-Tree Method: Case Study in Jakarta, Indonesia. *Atmosphere*, *16*(1), 23–23. https://doi.org/10.3390/atmos16010023

Shiyun Lv, Li, Z., Li, H., Liu, M., Wu, Z., Yu, S., Wu, B., Gao, B., Tao, L., Luo, Y., Li, X., Guo, X., & Liu, X. (2023). Long-term effects of PM2.5 components on hypertension: A national analysis in China. *Environmental Research*, *222*, 115323. https://doi.org/10.1016/j.envres.2023.115323

Siregar, S., Idiawati, N., Berekute, A. K., Maulana, M., Pan, W.-C., & Yu, K.-P. (2024). Association between long-term PM2.5 exposure and mortality on Sumatra Island: Indonesian Family Life Survey (IFLS) 2000–2014. *Environmental Monitoring and Assessment*, *196*(12). https://doi.org/10.1007/s10661-024-13323-5

Siregar, S., Idiawati, N., Pan, W.-C., & Yu, K.-P. (2021). Association between satellite-based estimates of long-term PM2.5 exposure and cardiovascular disease: evidence from the Indonesian Family Life Survey. *Environmental Science and Pollution Research*, *29*(14), 21156–21165. https://doi.org/10.1007/s11356-021-17318-4

Verma, D. K., et al. (2021). Air pollution and cardiovascular diseases: A review. Journal of Clinical Hypertension.

Xu, D., Zhang, Y., Sun, Q., Wang, X., & Li, T. (2021). Long-term $PM_{2.5}$ exposure and survival among cardiovascular disease patients in Beijing, China. *Environmental Science and Pollution Research International*, *28*(34), 47367–47374. https://doi.org/10.1007/s11356-021-14043-w