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Ambient Particulate Matter with Blood Pressure in Adult Women Living in Urban City

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Abstract

Ambient air pollution, especially from fine particles, contributes to human mortality from cardiovascular and respiratory diseases, for which high blood pressure (BP) is a major modifiable risk factor. This study aimed to analyze the influence of ambient particulate matter ($PM_{2.5}$) on the risk of high BP leading to hypertension. This study used a cross-sectional design on 118 adult women living in Central Jakarta, Indonesia. Participants were selected from a 1-km radius of the nearest air quality monitoring station with available data $PM_{2.5}$. Linear regression was analyzed to examine the relationship between $PM_{2.5}$ and systolic and diastolic BPs adjusted for potential confounders. The annual means of $PM_{2.5}$ concentration was 36±5.74 μg/m^3. The linear regression model showed that $PM_{2.5}$ exposure was associated with systolic BP after controlling with age and body mass index ($r = 0.408$; $R^2 = 0.167$). The second model showed that exposure to $PM_{2.5}$ concentration could explain about 10.9% variation of diastolic BP after controlling with age, length of stay, body mass index, smoking status, and diabetes mellitus record. Ambient air $PM_{2.5}$ has a risk of BP and hypertension among adult women in Central Jakarta.

Keywords: ambient air pollution, blood pressure, hypertension, $PM_{2.5}$, women

Introduction

High blood pressure (BP) is the leading risk factor for death and disability-adjusted life-years (DALYs).[1] High BP leading to hypertension is influenced by various elements, such as genetic, environmental, adaptive, nervous, endocrine, and hemodynamic factors.[2] Hypertension is a significant risk factor for the incidence of cardiovascular disease and a leading cause of premature deaths in the world. About 1.13 billion population in the world suffer from hypertension, and most of them live in low-middle-income countries.[3] According to the 2018 Indonesian Basic Health Research, the prevalence of hypertension in the population aged 18 years and older based on a doctor's diagnosis was 8.4%, while the prevalence based on measurement was 34.1%. Central Jakarta is one of the cities with a high prevalence of hypertension in Indonesia. The prevalence of hypertension based on a doctor's diagnosis is about 12.16%, while the prevalence based on measurement is slightly higher than the national figure, about 39.05%. However, Central Jakarta is the municipality with the highest prevalence of hypertension in the Special Capital Region of Jakarta Province.[4]

High blood pressure is also influenced by environmental factors, one of which is air pollution.[5,6] Ambient air pollution increases mortality and morbidity, shortens life expectancy, and is a major contributor to the world's disease burden.[7] Particulate matter (PM), especially $PM_{2.5}$, is a pollutant resulting in public health problems the world concerns.[8,9] The primary sources of PM from human activities include motor vehicle exhaust gases, industrial activities, agriculture, biomass combustion, and construction activities.[7,9] These airborne particles can generate health problems depending on where a person lives.[10] The microscopic size of $PM_{2.5}$ allows these particles to be absorbed into the lower bloodstream when someone breathes, resulting in health effects such as asthma, lung cancer, and heart disease.[9] In 2013, PM was classified as the cause of lung cancer by the International Agency for Research on Cancer.[8] In 2016, $PM_{2.5}$ contributed to the deaths of approximately 105.7 million DALYs and 4.1 million deaths in the world.[11] The increase in hypertension prevalence, along with the decrease in environmental quality, is characterized by an increase in the concentration of air pollutants in the environment.

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Over the past decade, several studies have found that exposure to air pollutants can affect BP. A previous study shows that long-term exposure to air pollutants is associated with increased BP and a high prevalence of hypertension in children and adults.[12] Short-term exposure to $PM_{2.5}$ within 24 hours is associated with increased systolic blood pressure (SBP), and an increase in $PM_{2.5}$ concentration is associated with increased BP.[13,14] Long-term exposure to $PM_{2.5}$ is statistically associated with prevalence of hypertension.[15] Several previous studies have also examined the relationship between long-term $PM_{2.5}$ exposure and the incidence of hypertension in a correlation analysis in urban populations, in which the results showed that increasing $PM_{2.5}$ concentrations in ambient air were associated with changes in the incidence of hypertension and increasing SBP.[7,11,12,16]

Adult women were selected as participants in this study based on the intensity of staying at home compared to men. A previous study on ambient particulate air pollution in peri-urban India shows that women spend most of their time near home compared to men at 83% of the daytime vs. 57% for men.[5] Results of the study show a positive association between long-term exposure to ambient $PM_{2.5}$ with BP and hypertension, with a stronger association found for SBP than diastolic blood pressure (DBP).[5] A previous study in Indonesia shows that women aged over 60 years were more likely to have hypertension compared to the ones under 60 years.[17]

A previous study shows that every 5 μg/m^3 change in exposure to $PM_{2.5}$ was associated with a 16% change in the incidence of hypertension (hazard ratio, 0.84; 95% CI, 0.82–0.86).[5] Short-term exposures of SO_2, $PM_{2.5}$, and PM_{10} are significantly associated with the incidence of hypertension.[18] Air pollution due to exposures to $PM_{2.5}$, ultrafine ($PM_{0.1}$), ozone, nitrogen oxides, and transition metals potentially generate health effects through oxidative stress.[19] Oxidative stress acts as a mediator against cell damage and inflammation to several systems that eventually affect BP.[20]

There were various findings to understand the role of ambient air pollution, especially from PM exposure to cardiovascular disease and BP. However, the relationship in adult women is still limited. The difference from other studies is that, in this study, the sampling area of the subject used mapping from the nearest Air Quality Monitoring Station (AQMS) to the residence. This study aims to determine the effect of ambient air particulate pollutants on SBP and DBP to predict hypertension.

Method

An epidemiological study with a cross-sectional design was carried out among residents in Central Jakarta. This study was conducted from May to June 2023. A multistage cluster random sampling method was adopted as a representative sampling approach to minimize selection bias in large areas. Initially, mapping of the study location based on the three nearest AQMS in Central Jakarta as the first cluster area to the residence with a proximity of ≤1,000 meters was conducted (Figure 1). From the mapping, about five subdistricts and nine villages in Central Jakarta were around the ring and selected as the second cluster. The nine villages were Bendungan Hilir, Kebon Sirih, Senen, Kwitang, Gunung Sahari Selatan, Kemayoran, Gunung Sahari Utara, Kartini, and Pasar Baru. Of nine villages, about 21 community units were selected as sampling areas for this study.

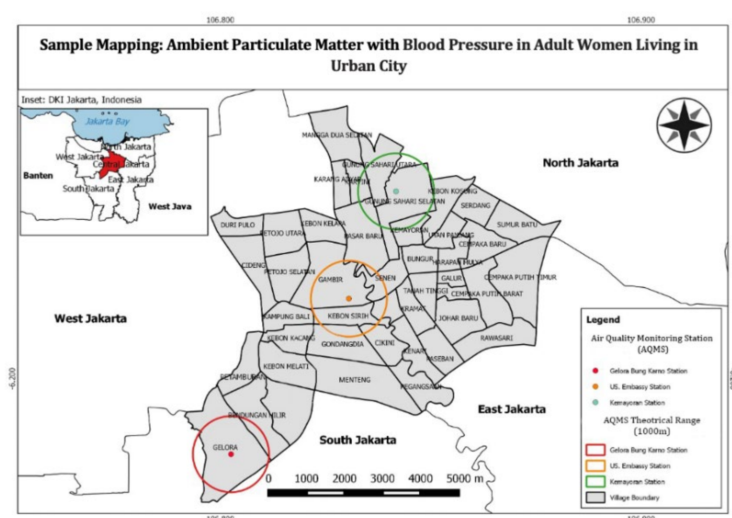

Figure 1. Mapping of the Study Location Based on the Three Nearest AQMS in Central Jakarta

The participants were selected using simple random sampling from the name list available based on the inclusion and exclusion criteria. Of the total population of 203,428 people, about 118 participants were obtained as the minimum sample, calculated using a hypothesis test comparing two means.[14] The inclusion criteria for participants were adult women residents aged 18-65 years living or working at those nine villages in Central Jakarta for at least one year. Exclusion criteria were pregnant women and those who were rejected as study subjects.

There were three data collections in this study. First, $PM_{2.5}$ concentrations were collected from three AQMS in Central Jakarta. Second, participant data measurements consist of measuring blood pressure to diagnose cases of hypertension and measuring body height (centimeters) and weight (kilograms) to calculate the human body mass index (BMI). Third, sociodemographic and health information data were collected by asking for a structural questionnaire from each participant. Prior to data collection, all participants were explained about the study by field staff and signed a consent form.

Ambient air pollution exposure in this study was defined as $PM_{2.5}$-micron categorization in micrograms per cubic meter ($\mu g/m^3$). The data on $PM_{2.5}$ concentration in 2022 was collected from three AQMS in Central Jakarta and had received permission from the relevant agency. The three sources of air quality monitoring stations are stations belonging to the U.S. Embassy (open access data), Kemayoran Station belongs to Indonesia's Meteorological, Climatological, and Geophysical Agency, and Gelora Bung Karno Station belongs to the Indonesian Ministry of Environment and Forestry. The AQMS was a network of continuous ground-based air quality monitoring stations. From three AQMS data, the daily concentration data was obtained and calculated to get the annual concentration of $PM_{2.5}$ in 2022. The average number of $PM_{2.5}$ concentrations used in this study was obtained from the annual $PM_{2.5}$ concentration from the three AQMS.

Each participant's BP was measured at least twice by a professional nurse using a digital sphygmomanometer (Omron Automatic Blood Pressure Monitor HEM 7156, Kyoto, Japan). BP was measured in the sitting position, and the cuff was placed around 2 cm above the right or left arm elbow. Participants quietly sat in a reclined chair for at least five minutes with both feet flat on the floor and arms supported at heart level. At least 30 minutes before the measurement, participants avoided smoking, drank coffee or tea, and did exercise.[16] The SBP and DBP were obtained from the average of at least two measurements. If the mean of two measurements were more than 10 mmHg, the third measurement was carried out. From the BP measurement, it was classified as hypertension cases if the SBP was 140 mmHg or higher or the DBP was 90 mmHg or higher.[11,15,16]

To adjust for potential confounders, the following covariates were considered in the statistical analysis: age (continuous), duration of stay in the study area (continuous), employment status (employed, unemployed), BMI (continuous), smoking status (yes, no), family records of hypertension (yes, no) and diabetes mellitus (yes, no). The participants were also asked about current hypertension medication use and records of diagnosed high BP by a doctor.

A descriptive analysis was taken to illustrate the sample distribution based on various characteristics. Pearson's correlation and linear regression were used to determine the independent variable as a continuous variable. In contrast, t-test analysis was used to determine the independent variable as a categorical variable. Multivariate linear regression models were employed to estimate the association between the exposure of $PM_{2.5}$ with SBP and DBP by considering variables as potential confounders. In multivariate linear regression analysis, all potential confounders were carried out to the full model. Variables with a p-value of >0.05 were excluded one by one from the highest and got the final model. Effect estimates are presented as mmHg change for BP measurements per 1 $\mu g/m^3$ increase in $PM_{2.5}$ concentration.

Results

From the three AQMS in Central Jakarta, the average $PM_{2.5}$ concentration was 36.0 $\mu g/m^3$. Among 118 adult women, the mean SBP and DBP from direct measurement were 120.1 mmHg and 80.3 mmHg. The BP was also categorized and revealed that about 23.7% of adult women suffered from hypertension phase 1. The participants were adult women with an average age of 46 years who had lived in the study area for about 38 years. The height and weight measurement showed that adult women had an average BMI of about 27.4 (overweight). From the structural interview, about 12.7% of adult women were taking antihypertensive medicine, and about 27.1% were diagnosed with hypertension by a doctor in the past. About 16.9% of adult women were smokers, and almost all of them started smoking when they were adolescents. Regarding genetic factors, about 42.4% of adult women had a family record of hypertension (from both parents), and about 9.3% had a record of diabetes mellitus (Table 1).

Table 1. Characteristics and Health Information of the Participants

Variable	Category	n=118	%
$PM_{2.5}$ (mean±SD, μg/m³)		36±5.74	
SBP (mean±SD, mmHg)		120.1±19.6	
DBP (mean±SD, mmHg)		80.3±11.5	
Age (mean±SD, years)		46 .1±10.6	
Length of stay (mean±SD, years)		38.2±15.5	
BMI (mean±SD, kg/m²)		27.4±5.6	
Hypertension Category	Normal	42	35.6
	Pre-Hypertension	44	37.3
	Phase 1 Hypertension	28	23.7
	Phase 2 Hypertension	3	2.5
	Phase 3 Hypertension	1	0.8
Taking antihypertensive medicine	Yes	15	12.7
	No	103	87.3
Hypertension diagnosed by a doctor	Yes	32	27.1
	No	86	72.9
Employment status	Employed	36	30.5
	Unemployed	82	69.5
Smoking status	Yes	20	16.9
	No	98	83.1
Family history of hypertension	Yes	50	42.4
	No	68	57.6
History of diabetes mellitus	Yes	11	9.3
	No	107	90.7

Note: SD = standard deviation

There was a moderate correlation between age and length of stay in the study area with SBP ($r = 0.341$, $r = 0.303$), and it had a positive linear relationship. It suggested that increasing age or length of stay was accompanied by increasing SBP ($R^2 = 0.116$, $R^2 = 0.092$). The BMI had a weak relationship with SBP ($r = 0.215$), while $PM_{2.5}$ concentration had a very weak relationship with SBP ($r = 0.034$). Both of them had a positive linear relationship with SBP. The statistical correlation between $PM_{2.5}$ concentration, age, length of stay, and BMI with DBP had a low power ($r = 0.011$, $r = 0.025$. $r = 0.129$, $r = 0.233$, respectively) (Table 2).

Table 2. The Association of $PM_{2.5}$ and other Covariates with Blood Pressure (continuous variable)

Variable	R	R^2	p-value
SBP			
$PM_{2.5}$ concentration (μg/m³)	0.034	0.001	0.715
Age (years)	0.341	0.116	0.001
Length of stay (years)	0.303	0.092	0.001
BMI (kg/m²)	0.215	0.046	0.019
DBP			
$PM_{2.5}$ concentration (μg/m³)	0.011	0.000	0.903
Age (years)	0.025	0.001	0.791
Length of stay (years)	0.129	0.017	0.165
BMI (kg/m²)	0.233	0.054	0.011

Notes: SBP = systolic blood pressure, BMI = body mass index, DBP = diastolic blood pressure

Independent t-test analysis showed there was no significant relationship between employment status, smoking status, family record of hypertension, and record of diabetes mellitus with SBP (p-values = 1.000; 0.192; 0.685; 0.356). When analyzed with DBP, the result was the same: there was no statistically significant relationship between employment status, smoking status, family record of hypertension, and record of diabetes mellitus with SBP (p-values = 0.403; 0.068; 0.664; 0.786) (Table 3).

Results showed that after controlling with age and BMI, the exposure of $PM_{2.5}$ concentration in a multivariate linear regression model could explain about 16.7% variation of SBP. It showed that the higher the $PM_{2.5}$ concentration, the lower the SBP level. With every increased $PM_{2.5}$ concentration of about 1 μg/m³, the SBP level would decrease by about 0.45 mmHg after controlling with age and BMI.

Another model showed that exposure to $PM_{2.5}$ concentration could explain about 10.9% variation of DBP after controlling with age, length of stay, BMI, smoking status, and record of diabetes mellitus. Every increased $PM_{2.5}$ concentration of about 1 μg/m³, the DBP level would decrease by about 0.049 mmHg. The higher the $PM_{2.5}$ concentration, the lower the DBP level. (Table 4).

Table 3. The Association of Other Covariates with Blood Pressure (categorical variable)

Variable	Category	Mean±SD	T (t-test)	p-value
SBP				
Employment status	Employed	120.19	0.001	1.000
	Unemployed	120.19		
Smoking Status	Yes	114.98	1.311	0.192
	No	121.26		
Family record of hypertension	Yes	121.05	-0.407	0.685
	No	119.57		
Record of diabetes mellitus	Yes	125.41	-0.927	0.356
	No	119.66		
DBP				
Employment status	Employed	78.92	-0.839	0.403
	Unemployed	90.86		
Smoking status	Yes	75.98	1.842	0.068
	No	81.14		
Family record of hypertension	Yes	80.83	-0.436	0.664
	No	79.86		
Record of diabetes mellitus	Yes	79.36	0.272	0.786
	No	80.36		

Notes: SD = standard deviation, SBP = systolic blood pressure, DBP = diastolic blood pressure

Table 4. Final Linear Regression Model of $PM_{2.5}$ with Blood Pressure

Variable	Coeff. β	p-value	r	R^2
SBP				
$PM_{2.5}$ concentration (μg/m³)	-0.453	0.135	0.408	0.167
Age (years)	0.655	0.001		
BMI (kg/m²)	0.668	0.029		
DBP				
$PM_{2.5}$ concentration (μg/m³)	-0.049	0.793	0.330	0.109
Age (years)	-0.095	0.450		
Length of stay (years)	0.149	0.080		
BMI (kg/m²)	0.479	0.013		
Smoking status	-5.422	0.054		
Record of diabetes mellitus	-2.260	0.541		

Notes: SBP = systolic blood pressure, BMI = body mass index, DBP = diastolic blood pressure

Discussion

Air pollution has been an environmental health problem in Indonesia, especially in the metropolitan city of Jakarta. This study found that the annual mean concentration of $PM_{2.5}$ in 2022 around Central Jakarta was 36 μg/m³, greater than the national (15 μg/m³) and the WHO's standard (5 μg/m³).[21,22] In urban areas, motor vehicles are the major source of PM in ambient air, especially from gasoline and diesel vehicle emissions.[23] Central Jakarta is the location of the central government buildings, as well as being the center for finance and business with high traffic activity. In this study, the high concentration of $PM_{2.5}$ in participants' residences was revealed to be motor vehicle density. Data shows that the number of motorized vehicles in Jakarta is likely to increase from 2020 to 2022, dominated by two-wheeled vehicles.[24] In Jakarta, vehicle exhaust gas is the major source of air pollution, especially $PM_{2.5}$ in the dry and rainy seasons, contributing around 32-57%, followed by industrial burning activities around 43%.[25]

The SBP and DBP results from the direct measurement categorized as hypertension category showed that adult women in Jakarta mostly had pre-hypertension and hypertension phase 1. The prevalence of all the hypertension phases in this study was 27.0%, lower than the national data in 2018 at 34.1% and slightly higher than in Central Jakarta at 39.5%.[26] Of the total hypertension cases in adult women, only 12.7% declared that they were taking antihypertensive medicine. This finding indicated that there was low awareness of the disease and low adherence to taking medication as they suffered from high BP.

In bivariate analysis, $PM_{2.5}$ concentration was not significantly associated with SBP or DBP, but other variables showed a significant relationship. Age and length of stay in the study area were positively associated with SBP, whereas only BMI status was significant with both SBP and DBP levels. In this study, most participants were at the age of 46 years on average. A previous study in Bekasi City, Indonesia, showed that there was a relationship between age and the occurrence of high BP. The participants aged ≥40 years had a 9.3 times higher risk of hypertension than those aged <40 years.[27] Another study in Tokyo shows that the prevalence of hypertension grade 1 increased with age.[28] BP likely increases with age, in which the high prevalence of hypertension is influenced by various health risks, and the incidence of the disease mainly occurs in older people.[29,30] Aging is a continual process that decreases physiologic function across all organ systems and increases vulnerability to infection and disease. Transformation in arterial and arteriolar stiffness are primary factors contributing to hypertension in the elderly.[30,31]

According to this study, high SBP occurred more often in people staying longer at the study location, indicating a greater possibility of $PM_{2.5}$ living in the body. The longer someone lives in a residence, the longer they get exposed to air pollutants. A previous study in five rural regions in China shows that the length of stay is related to long-term exposure to air pollutants ($PM_{2.5}$, PM_{10}, and NO_2), which increases BP and hypertension.[32] This study also revealed that the participants living in the study area for 38 years were associated with SBP. However, these results differ from a previous double-blind crossover study stating that healthy adults and non-smokers aged 18-50 years who were exposed to $PM_{2.5}$ tended to increase DBP, not SBP.[33]

In this study, BMI was associated with SBP and DBP. These results are similar to a previous study in two urban cities in Germany, stating that BMI and high BP had a significant relationship.[31] The other study shows that the hypertension risk increases by about two to three times in overweight people compared with someone with a normal BMI.[32] People with higher BMI status reflect increased body fat mass. Fat cells are characterized by being sensitive to lipolysis and producing high quantities of inflammatory cytokines. As the inflammatory process has an important role in the pathogenesis of hypertension, these processes could respond to BP elevation and end-organ damage.[34,35]

Although the relationship between $PM_{2.5}$ and hypertension in the bivariate analysis was not significantly related, but in the multivariate linear regression model, there was a risk of $PM_{2.5}$ concentration with SBP and DBP after controlling with another confounding variable. This finding was consistent with the previous study among reproductive-age adults in China, which found that exposure to $PM_{2.5}$ in a long-term period was associated with an increase in both SBP and DBP.[36] The mechanism of increased blood pressure from short-term exposure to $PM_{2.5}$ concentrations is related to autonomic nervous system imbalance, generation, or release of endogenous biological mediators such as cytokines.

This study had several limitations. First, the research design was cross-sectional, and causal relationships on individual variables could not be attempted. A causal association was unlikely to be established after someone was exposed to $PM_{2.5}$. They had to have high BP. The inability to measure individual exposure to $PM_{2.5}$ and the use of secondary data led to incomplete or unavailable data. The second limitation was that there was less information on indoor air pollutants. Some potential influences on indoor $PM_{2.5}$ concentration, such as type of cooking fuel and home ventilation, could not be excluded. However, the study tried to include the effect of smoking as an important source of household air pollutants. Third, the number of samples in this study was insufficient to build a statistical association between each variable with SBP and DBP.

Despite those limitations, this study had several strengths. First, environmental health studies conducted regarding the influence of air pollutants on cardiovascular risk factors are still limited, especially in Indonesia. Second, this study tried to predict the influence of $PM_{2.5}$ on traffic activities with a simple spatial model based on the distance from the nearest AQMS with available data of $PM_{2.5}$ concentration to the study location. Future studies need to predict the spatiotemporal model of $PM_{2.5}$ or other air pollutants' exposure to estimate the exposure at each participant's residence.

Conclusion

Air pollution in Jakarta, Indonesia, is still an environmental problem faced by people living near sources of air pollution. Exposure to $PM_{2.5}$ in ambient air is associated with SBP after controlling with age and BMI. $PM_{2.5}$ concentrations explain variations in DBP after controlling for age, length of stay, BMI, smoking status, and record of diabetes mellitus. $PM_{2.5}$ ambient air has a risk of developing BP and hypertension in adult women in Central Jakarta. Further studies need to predict biological markers of exposure or effects that will be measured on the human body as an early predictor of high BP, which leads to hypertension and cardiovascular disease.

Abbreviations

DALYs: Disability-Adjusted Life-Years; BP: blood pressure; SBP: systolic blood pressure; DBP: diastolic blood pressure; AQMS: Air Quality Monitoring Station; BMI: body mass index.

Ethics Approval and Consent to Participate

This study was approved and reviewed by the Research and Ethics Committee of the Faculty of Public Health, Universitas Indonesia (letter number Ket- 36/UN2.F10. D11/PPM.00.02/2023).

Competing Interest

The authors declare no conflict of interest.

Availability of Data and Materials

The generated dataset is available to share with the corresponding author upon reasonable request.

Authors' Contribution

Conceptualization: NA, HK. Data curation: LF, BW. Formal Analysis: NA, LF. Funding acquisition: HK. Methodology: HK, BW. Project administration: NA. Visualization: NA, LF. Writing-original draft: NA, HK, BW, LF. Writing review & editing: NA, LF.

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References

1. Stanaway JD, Afshin A, Gakidou E, et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: A systematic analysis for The Global Burden of Disease Study 2017. Lancet. 2018; 392 (10159): 1923-1994. DOI: 10.1016/S0140-6736(18)32225-6.
2. Oparil S, Acelajado MC, Bakris GL, et al. Hypertension. Nature Rev Dis Primers. 2018; 4: 1-21. DOI: 10.1038/nrdp.2018.14.
3. World Health Organization. Hypertension. Geneva: World Health Organization; 2021.
4. Badan Penelitian dan Pengembangan Kesehatan. Hasil Utama Riskesdas 2018. Jakarta: Kementerian Kesehatan Republik Indonesia; 2018.
5. Curto A, Wellenius GA, Milà C, et al. Ambient particulate air pollution and blood pressure in peri-urban India. Epidemiol. 2019; 30 (4): 492-500. DOI: 10.1097/EDE.0000000000001027.
6. Bo Y, Guo C, Lin C, et al. Dynamic changes in long-term exposure to ambient particulate matter and incidence of hypertension in adults: A natural experiment. Hypertension. 2019; 74 (3): 669-677. DOI: 10.1161/HYPERTENSIONAHA.119.13212.
7. Centers for Disease Control and Prevention. Air quality: Air pollutants. Atlanta, GA: Centers for Disease Control and Prevention; 2021.
8. Li N, Chen G, Liu F, et al. Associations between long-term exposure to air pollution and blood pressure and effect modifications by behavioral factors. Environ Res. 2020; 182: 109109. DOI: 10.1016/j.envres.2019.109109.
9. IQ Air. 2020 World Air Quality Report Region & City PM2.5 Ranking. Goldach: IQ Air; 2020.
10. Zhang Z, Dong B, Li S, et al. Exposure to ambient particulate matter air pollution, blood pressure and hypertension in children and adolescents: A national cross-sectional study in China. Environ Int. 2019; 128: 103-108. DOI: 10.1016/j.envint.2019.04.036.
11. Liang F, Liu F, Chen J, et al. Long-term exposure to fine particulate matter and hypertension incidence in China: The China-PAR cohort study. Hypertension. 2019; 73: 1195-1201. DOI: 10.1161/HYPERTENSIONAHA.119.12666.
12. Verma MK, Jaiswal A, Sharma P, et al. Oxidative stress and biomarker of TNF-α, MDA and FRAP in hypertension. J Med Life. 2019; 12 (3): 253-259. DOI: 10.25122/jml-2019-0031.
13. Yekti R, Bukhari A, Jafar N, et al. Measurement of malondialdehyde (MDA) as a good indicator of lipid peroxidation. Int J Allied Med Sci Clin Res. 2020; 6 (4): 838-840. DOI: 10.61096/ijamscr.v6.iss4.2018.838-840.
14. Lemeshow S, Hosmer DW Jr, Klar J, et al. Adequacy of sample size in health studies. Chichester: John Wiley & Sons; 1990. Published on behalf of the World Health Organization. ISBN: 0-471-92517-9.
15. National High Blood Pressure Education Program. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Bethesda (MD): National Heart, Lung, and Blood Institute (US); 2004.
16. Cao H, Li B, Liu K, et al. Association of long-term exposure to ambient particulate pollution with stage 1 hypertension defined by the 2017 ACC/AHA Hypertension Guideline and cardiovascular disease: The CHCN-BTH cohort study. Environ Res. 2021; 199: 111356. DOI: 10.1016/j.envres.2021.111356.
17. Bantas K, Gayatri D. Gender and hypertension (data analysis of the Indonesia Basic Health Research 2007). J Epidemiol Kesehat Indonesia. 2019; 3 (1): 7-17. DOI: 10.7454/epidkes.v3i1.3142.
18. Gkaliagkousi E, Gavriilaki E, Triantafyllou A, et al. Asymmetric dimethylarginine levels are associated with augmentation index across naïve untreated patients with different hypertension phenotypes. J Clin Hypertens. 2018; 20 (4): 680-685. DOI: 10.1111/jch.13237.
19. Li C, Shang S. Relationship between sleep and hypertension: Findings from the NHANES (2007–2014). Int J Environ Res Public Health. 2021; 18 (15): 7867. DOI: 10.3390/ijerph18157867.
20. Li N, Chen G, Liu F, et al. Associations of long-term exposure to ambient PM 1 with hypertension and blood pressure in rural Chinese population: The Henan rural cohort study. Environ Int. 2019; 128: 95-102. DOI: 10.1016/j.envint.2019.04.037.
21. Pemerintah Pusat. Peraturan Pemerintah Nomor 22 Tahun 2021 tentang Pedoman Perlindungan dan Pengelolaan Lingkungan Hidup. Jakarta: Kementerian Sekretariat Negara Republik Indonesia; 2021.
22. World Health Organization. Ambient (outdoor) air pollution. Geneva: World Health Organization; 2022.
23. United States Environmental Protection Agency. Integrated Science Assessment (ISA) for Particulate Matter. Washington, DC: United States Environmental Protection Agency; 2019.
24. Badan Pusat Statistik Provinsi DKI Jakarta. Jumlah kendaraan bermotor menurut jenis kendaraan (unit) di Provinsi DKI Jakarta 2020-2022. Jakarta: Badan Pusat Statistik Provinsi DKI Jakarta; 2023.
25. Vital Strategies. Identifying the main sources of air pollution in Jakarta: A source apportionment study. New York, Vital Strategies; 2022.
26. Badan Penelitian dan Pengembangan Kesehatan. Hasil Utama Riskesdas 2018. Jakarta: Kementerian Kesehatan Republik Indonesia; 2018.

27. Maulidina F, Harmani N, Suraya I. Faktor-Faktor yang Berhubungan dengan Kejadian Hipertensi di Wilayah Kerja Puskesmas Jati Luhur Bekasi Tahun 2018. ARKESMAS. 2019; 4 (1): 149–155. DOI: 10.22236/arkesmas.v4i1.3141.
28. Suzuki Y, Kaneko H, Yano Y, et al. Age-Dependent Relationship of Hypertension Subtypes with Incident Heart Failure. J Am Heart Assoc. 2022; 11 (9): e025406. DOI: 10.1161/JAHA.121.025406.
29. Centers for Disease Control and Prevention. High Blood Pressure Risk Factors. Atlanta, GA: Centers for Disease Control and Prevention; 2024.
30. Buford TW. Hypertension and aging. Ageing Res Rev. 2016; 26: 96–111. DOI: 10.1016/j.arr.2016.01.007.
31. Singh A, Rastogi N. Airborne Particles in Indoor and Outdoor Environments. In: Sonwani, S., Shukla, A. (eds) Airborne Particulate Matter. Singapore: Springer; 2022. DOI: 10.1007/978-981-16-5387-2_4.
32. Azzubaidi SBS, Rachman ME, Muchsin AH, et al. Hubungan tekanan darah dengan IMT (Indeks Massa Tubuh) pada mahasiswa angkatan 2020 Fakultas Kedokteran Universitas Muslim Indonesia. J Mhs Kedokt. 2023; 3 (1): 54-61.
33. Franklin BA, Brook R, Arden Pope C 3rd. Air pollution and cardiovascular disease. Curr Probl Cardiol. 2015; 40 (5): 207-238. DOI: 10.1016/j.cpcardiol.2015.01.003.
34. Caillon A, Paradis P, Schiffrin EL. Role of immune cells in hypertension. Br J Pharmacol. 2019; 176 (12): 1818-1828. DOI: 10.1111/bph.14427.
35. Aatola H, Hutri-Kähönen N, Juonala M, et al. Prospective relationship of change in ideal cardiovascular health status and arterial stiffness: The Cardiovascular risk in Young Finns Study. J Am Heart Assoc. 2014; 3 (2): e000532. DOI: 10.1161/JAHA.113.000532.
36. Xie X, Wang Y, Yang Y, et al. Long-term effects of ambient particulate matter (with an aerodynamic diameter ≤2.5 µm) on hypertension and blood pressure and attributable risk among reproductive-age adults in China. J Am Heart Assoc. 2018; 7 (9): e008553. DOI: 10.1161/JAHA.118.008553.