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Air Pollution and Blood Pressure: Evidence From Indonesia

Jaime Madrigano[1,2], Daisy Yan[3], Tianjia Liu[4,5,6], Eimy Bonilla[4,7,8], Nina Yulianti[9], Loretta J. Mickley[7], and Miriam E. Marlier[2,3]

[1]Department of Environmental Health and Engineering, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD, USA, [2]RAND Corporation, Santa Monica, CA, USA, [3]Department of Environmental Health Sciences, University of California Los Angeles, Los Angeles, CA, USA, [4]School of Engineering and Applied Sciences, Harvard University, Cambridge, MA, USA, [5]Department of Earth and Planetary Sciences, Harvard University, Cambridge, MA, USA, [6]Now at Department of Earth System Science, University of California, Irvine, Irvine, CA, USA, [7]John A. Paulson School of Engineering and Applied Sciences, Harvard University, Cambridge, MA, USA, [8]Now at Department of Earth, Environment, and Equity, Howard University, Washington, DC, USA, [9]Graduate Program Study of Environmental Science/Department of Agrotechnology, University of Palangka Raya, Palangka Raya, Indonesia

Key Points:

- Exposure to $PM_{2.5}$ in the preceding month was associated with higher diastolic blood pressure (DBP) in this Indonesian cohort
- Associations between $PM_{2.5}$ and blood pressure were more pronounced in elderly participants
- There was no association between fire-specific $PM_{2.5}$ and blood pressure; however, this may be due to the timing of health data collection

Supporting Information:

Supporting Information may be found in the online version of this article.

Correspondence to:

J. Madrigano,
jmadrig4@jhu.edu

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Author Contributions:

Conceptualization: Jaime Madrigano, Miriam E. Marlier
Data curation: Loretta J. Mickley, Miriam E. Marlier
Formal analysis: Jaime Madrigano, Eimy Bonilla, Miriam E. Marlier
Funding acquisition: Jaime Madrigano, Miriam E. Marlier
Methodology: Jaime Madrigano, Loretta J. Mickley, Miriam E. Marlier
Project administration: Jaime Madrigano, Miriam E. Marlier
Validation: Daisy Yan, Nina Yulianti
Visualization: Jaime Madrigano, Daisy Yan, Miriam E. Marlier

Abstract Indonesia faces significant air quality issues due to multiple emissions sources, including rapid urbanization and peatland fires associated with agricultural land management. Limited prior research has estimated the episodic shock of intense fires on morbidity and mortality in Indonesia but has largely ignored the impact of poor air quality throughout the year on biomarkers of cardiovascular disease risk. We conducted a cross-sectional study of the association between particulate matter less than 2.5 microns in diameter ($PM_{2.5}$) and blood pressure. Blood pressure measurements were obtained from the fifth wave of the Indonesian Family Life Survey (IFLS5), an ongoing population-based socioeconomic and health survey. We used the GEOS-Chem chemical transport model to simulate daily $PM_{2.5}$ concentrations at $0.5° \times 0.625°$ resolution across the IFLS domain. We assessed the association between $PM_{2.5}$ and diastolic and systolic blood pressure, using mixed effects models with random intercepts for regency/municipality and household and adjusted for individual covariates. An interquartile range increase in monthly $PM_{2.5}$ exposure was associated with a 0.234 (95% CI: 0.003, 0.464) higher diastolic blood pressure, with a greater association seen in participants age 65 and over (1.16 [95% CI: 0.24, 2.08]). For the same exposure metric, there was a 1.90 (95% CI: 0.43, 3.37) higher systolic blood pressure in participants 65 and older. Our assessment of fire-specific $PM_{2.5}$ yielded null results, potentially due to the timing and locations of health data collection. To our knowledge, this is the first study to provide evidence for an association between $PM_{2.5}$ and blood pressure in Indonesia.

Plain Language Summary Although the association between air pollutants and cardiovascular disease risk has been widely reported, there have been few studies conducted in Indonesia, a country with both high population and air pollution burden. In this study of over 25,000 Indonesian residents, we found that participants with higher exposure to air pollution in the month before a clinical exam had higher blood pressure than participants with lower exposure to air pollution. This association was stronger in participants ages 65 and older.

1. Introduction

1.1. Air Pollution Is a Risk Factor for Cardiovascular Disease Worldwide

Air pollution is increasingly recognized as a critical driver of global health. In 2019, it was estimated that air pollution was responsible for 6.67 million deaths worldwide, making it the fourth leading risk factor for death globally (Health Effects Institute, 2020). Cardiovascular disease (CVD) is the leading cause of death worldwide and it is estimated that 19% of all CVD deaths are attributable to air pollution (Hadley et al., 2018). While time series studies to estimate the impacts of short-term exposure to air pollution on mortality have been conducted in cities throughout the world (Liu et al., 2019), cohort studies, frequently used to estimate exposure-response curves and allow for assessment of biomarkers to explain pathways of disease occurrence, have primarily been conducted in North America and Europe. Direct extrapolation of exposure-response functions from high income countries to low and middle income countries (LMICs) is problematic because exposure to ambient air pollution is substantially greater in LMICs than in high income countries and the health response to air pollution may not be linear (Hystad, Yusuf, & Brauer, 2020). Thus, cohort studies of the health effects of air pollution are critically needed in LMICs where the public health burden is far greater (Tonne, 2017).

Writing – original draft: Jaime Madrigano
Writing – review & editing: Jaime Madrigano, Daisy Yan, Eimy Bonilla, Nina Yulianti, Loretta J. Mickley, Miriam E. Marlier

1.2. Air Pollution in Indonesia

While there is a growing body of evidence for the association between ambient air pollution and CVD risk in cohort studies conducted in China and India (Hystad, Larkin, et al., 2020; Tonne et al., 2017), there remains a gap in other LMICs. Indonesia is the fourth most populous country in the world, behind China, India, and the United States, and has ranked within the top 20 most polluted countries in the world ("IQAir | First in Air Quality," 2023). The burden of air pollution in Indonesia has been estimated to cut the average resident's life expectancy by 1.2 years, though in some regions of the country that estimate is far greater, resulting in a total of 309 million life-years lost due to particulate air pollution (Greenstone & Fan, Qing, 2019). Fires in Indonesia, which typically correspond with the dry season running from June into December, are a devastating source of severe episodic particulate air pollution, particularly in intensive burning vegetation and peatland regions such as Sumatra, Kalimantan, and Papua (Hayasaka, 2023; Usup & Hayasaka, 2023; Yulianti & Hayasaka, 2023). The 2015 season produced the highest levels of fire emissions in a decade (Field et al., 2016). In addition, the country has seen a substantial increase in electricity generation from coal-fired power plants and vehicular burning of gasoline and diesel over the last decade, with vehicle exhaust and coal combustion being two of the largest drivers of particulate pollution in the capital city of Jakarta (Vital Strategies, 2020).

1.3. Studies of Air Pollution and Health Outcomes in Indonesia

Existing studies on the health impacts of air pollution exposure in Indonesia have been limited. Several studies have investigated the health impacts of high-intensity episodic fire events through either convenience samples, electronic medical records, or administrative data (Aditama, 2000; Kunii et al., 2002; Ming et al., 2018; Mott et al., 2005; Phung et al., 2022; Sheldon & Sankaran, 2017) for populations in Indonesia and neighboring countries. Other cohort-based studies within Indonesia have demonstrated that fire-induced, higher levels of air pollution exposure are associated with early life mortality, respiratory morbidities, and poor general health (Frankenberg et al., 2005; Jayachandran, 2009; Rosales-Rueda & Triyana, 2019). However, cardiovascular outcomes and their relation to environmental pollution have only been minimally explored within Indonesia.

Hypertension is an important risk factor for CVD with a high prevalence worldwide. It has been estimated that one-third of adults in Southeast Asia have hypertension and the prevalence within Indonesia is 34% (Mohammed Nawi et al., 2021; Turana et al., 2019). Evidence also indicates that hypertension is not well-controlled within the Indonesian population and in a survey of adults with hypertension, less than a third were aware of their condition (Hussain et al., 2016). Therefore, efforts to reduce population-wide exposures could play an important role in decreasing the public health burden within the country. In this study, we aim to assess the association between particulate air pollution and systolic blood pressure (SBP) and disastolic blood pressure (DBP). In earlier work, we observed an association between annual exposure to particulate matter less than 2.5 microns in diameter ($PM_{2.5}$) and DBP; however, due to the coarse temporal nature (annual average) of our exposure metric we were unable to disentangle days on which extreme fire emissions were present from days when other sources were primary drivers of pollution (Marlier et al., 2021). In the present work, we examine the association between daily, weekly, and monthly exposure to $PM_{2.5}$ and BP.

2. Data and Methods

2.1. $PM_{2.5}$ Exposure Estimates

We used a global chemical transport model, GEOS-Chem version 12.8.2; Yantosca et al., 2023), to simulate $PM_{2.5}$ concentrations across Equatorial Asia. GEOS-Chem is a three-dimensional global atmospheric composition model driven by meteorological observations from the Modern-Era Retrospective analysis for Research and Applications, version 2 (Merra-2; https://gmao.gsfc.nasa.gov/reanalysis/MERRA-2/) (Gelaro et al., 2017). We performed two sets of aerosol-only modeling simulations at $0.5° \times 0.625°$ resolution across Asia, once with and once without fire emissions included as an emissions source, to produce daily surface total and fire-specific $PM_{2.5}$ concentrations across the Indonesian Family Life Survey (IFLS) domain. Boundary conditions were taken from global tropospheric simulations at $4° \times 5°$ resolution for the same years. Fire emissions were from the Global Fire Assimilation System (GFAS) inventory, version 1.2 (Kaiser et al., 2012). Due to non-linearities in the modeling process, where any net daily fire-only modeled concentrations were negative, we set those values to zero. Total $PM_{2.5}$ for each simulation was calculated based on aerosol mass concentrations of ammonium, sulfate, nitrate, black carbon, organic carbon, sea salt, and dust with a diameter of less than 2.5 microns. We validated simulated

$PM_{2.5}$ concentrations with ground station observations of $PM_{2.5}$ and PM_{10} collected in Indonesia, Singapore, and Malaysia. We computed the linear correlation (r), fractional bias (FB), and normalized mean bias fraction (NMBF) between model and ground-based observations.

Fractional bias (FB) is calculated by,

$$\mathrm{FB} = \frac{1}{N}\sum \frac{(M_i - O_i)}{(M_i + O_i)/2}$$

and normalized mean bias factor (NMBF) is calculated by,

$$\mathrm{NMBF} = \frac{\sum(M_i - O_i)}{\sum O_i}, if\ \overline{M} \geq \overline{O}\ and\ \mathrm{NMBF} = \frac{\sum(M_i - O_i)}{\sum M_i}, if\ \overline{M} < \overline{O},$$

where M_i and O_i are modeled (GEOS-Chem) and observed (station) daily mean pairs.

2.2. The Indonesian Family Life Survey (IFLS)

The IFLS is an ongoing longitudinal survey that collects indicators of health and well-being at the individual, household, and community levels. It uses a sample of households which represented about 83% of the Indonesian population at the time of its initiation in 1993. The survey collects data on individual respondents, their families, their households, the communities in which they live, and the health and education facilities they use. The fifth wave of the IFLS (IFLS5) is the most recent survey and was fielded in late 2014 and early 2015.

2.2.1. Blood Pressure Measurements

IFLS5 interviewers were trained in taking physical health measurements. Blood pressure measurements were taken on participants (aged 15 years and older) three times on alternate arms in a seated position, using an Omron meter, HEM-7203. Most of the time a normal sized cuff was used; large cuffs were available as needed (Strauss et al., 2016). We calculated the mean value for both systolic and DBP for each participant from the three measurements.

2.2.2. Covariates

Sociodemographic factors were assessed through interview questions on age, sex, education, and subjective economic status. As done in a prior analysis, we categorized education into those with no or only elementary education versus those with higher than elementary education (Peltzer & Pengpid, 2018). Subjective economic status was assessed by the question "Please imagine a six step ladder where on the bottom (the first step), stand the poorest people, and on the highest step (the sixth step), stand the richest people (Peltzer & Pengpid, 2018). On which [economic] step are you today?" The answers ranged from (1) poorest to (6) richest. As in previous analysis, we collapsed this into three categories (poor, medium, rich). Heights were measured to the nearest millimeter using a Seca plastic height board (model 213). Weights were taken to the nearest tenth of a kilogram using a Camrymodel EB1003 scale (Strauss et al., 2016). Body mass index (BMI) was calculated as weight in kg divided by the square of height in meters. Tobacco use was assessed with two questions: (a) "Have you ever chewed tobacco, smoked a pipe, smoked self rolled cigarettes, or smoked cigarettes/cigars?" (Yes, No) and (b) "Do you still have the habit or have you totally quit?" (Still have, Quit) (Strauss et al., 2016). Responses were categorized into never, quitters, and current tobacco users. We computed daily average surface temperatures (at 2 m height) for each kabupaten from the European Center for Medium-Range Weather Forecasts (ECMWF) ERA5 Fifth Generation atmospheric reanalysis of the global climate, which combines modeled data and observations ("Copernicus Climate Change Service (C3S) (2017): ERA5: Fifth generation of ECMWF atmospheric reanalyzes of the global climate.," 2017).

2.3. Exposure and IFLS Data Merging

We estimated daily $PM_{2.5}$ concentrations for each kabupaten (municipality/regency) or kota (city) of residence in our study area by matching to geospatial maps of kabupaten and kota boundaries ("Indonesia—Subnational Administrative Boundaries—Humanitarian Data Exchange," 2020). Because blood pressure has been associated

with short and long-term air pollution (de Bont et al., 2022), in addition to assessing $PM_{2.5}$ exposure on the day of the clinical assessment, we calculated multiple moving averages of exposure (1 week, 2 weeks, and 1 month). The moving average is the mean exposure for the time period before each clinical assessment.

2.4. Statistical Analysis

Descriptive statistics were computed to describe the study population and exposures. Both DBP and SBP were right-skewed. In order to preserve interpretability, we removed outliers instead of log-transforming these variables for use in the analysis. We estimated the cross-sectional association between $PM_{2.5}$ exposures and DBP and SBP using linear mixed-effects models. To account for spatial clustering of individuals within households and households within kabupatens/kotas, we used nested random effects for each household within each kabupaten/kota. All models were adjusted for temperature, age, sex, education, subjective economic status, BMI, tobacco use, and hypertensive medication use. Because both air pollution and blood pressure can vary seasonally, we also included sine and cosine terms in our analysis (Stolwijk et al., 1999). We conducted several sensitivity analyses to determine if usage of hypertensive medication or missing data influenced the observed association by adjusting the BP values of those who reported taking antihypertensive medication (Tobin et al., 2005), restricting the analysis to those who did not take antihypertensive medication, and running the analysis with a missing indicator for subjective economic status (the covariate which had the greatest amount of missing data).

Since the association between $PM_{2.5}$ and blood pressure may vary by age, sex, and smoking status (Curto et al., 2019; Künzli et al., 2005; Rajagopalan et al., 2018; Turner et al., 2017), we ran models for the overall adult (ages 18 and older) population and then separately for middle-age (ages 45–64) and elderly (65+) participants. We also ran models stratified by sex and smoking status. Although the time period of health data collection only minimally overlapped with an intense fire season, we performed a primary and secondary analysis to determine if any association observed between $PM_{2.5}$ and BP was driven primarily by fire-specific emissions or not. Our primary analysis used the daily surface $PM_{2.5}$ concentrations with fire emissions included (total $PM_{2.5}$ concentrations) and our secondary analysis used the net daily fire concentrations (daily surface $PM_{2.5}$ concentrations with fire emissions–daily surface $PM_{2.5}$ concentrations without fire emissions) as our exposure metric. All mixed-effects models were conducted using the PROC MIXED procedure in SAS (version 9.4; SAS Institute Inc., Cary, NC, USA).

3. Results

3.1. Study Sample

IFLS5 respondents included children and adults ($n = 52{,}587$). After restricting our sample to participants 18 years and older and those with complete measures on air pollution and blood pressure, a total of 30,237 participants remained. After dropping outliers and observations with missing covariate data, our analytic sample consisted of 26,878 participants in the DBP analysis and 26,283 participants in the SBP analysis (Figure S1 in Supporting Information S1). The mean age of participants was 40 years, approximately 1/3 were current tobacco users, and the mean BMI was 23.4 (Table 1). Just over a quarter of the population was hypertensive and less than 3% of participants used medication for hypertension.

3.2. $PM_{2.5}$ Exposure

Simulated $PM_{2.5}$ from the GEOS-Chem model shows the influence of fires on total pollution and variations between 2014 and 2015 (Figure 1). In certain cities/regencies, annual average total $PM_{2.5}$ concentrations reached >100 $\mu g/m^3$ in 2015 but were less than 50 $\mu g/m^3$ in 2014. When fire emissions were removed from the model simulations (right column), annual average $PM_{2.5}$ concentrations were below 50 $\mu g/m^3$ and highest on the island of Java. When fires were included, we see different spatial patterns, with the highest concentrations in Sumatra and Kalimantan, with higher peak concentrations. We compared simulated all-source $PM_{2.5}$ to ground station observations from six sites in Indonesia and six sites in Malaysia and Singapore (Tables S1 and S2 in Supporting Information S1; Figures S2–S8 in Supporting Information S1).

The linear correlation coefficient (r) was moderate. We found better model performance in 2015 ($r = 0.6$, $p = <0.0001$) than 2014 ($r = 0.3$, $p = <0.0001$). The FB, which ranges from −2 to 2, was within 0.5, with an average of 0.2 in 2014 and −0.01 in 2015. The average NMBF across all sites was 0.3 in 2014 and 0.5 in 2015.

Table 1
Participant Characteristics[a]

	N = 30,237
Age, mean (SD)	40.0 (16.2)
Female (%)	53.5
Tobacco use	
Current (%)	32.6
Former (%)	5.1
Never (%)	62.3
BMI, mean (SD)	23.4 (4.5)
Education	
None/elementary (%)	37.3
Subjective economic status	
Poor (%)	25.4
Medium (%)	70.9
Rich (%)	3.7
Hypertensive (%)[b]	26.0
Hypertension medication (%)	2.8
Systolic blood pressure, mean (SD)[c]	125.7 (16.0)
Diastolic blood pressure, mean (SD)[d]	78.4 (10.2)

[a]Missing data were reported for age (n = 6, <0.1%), BMI (n = 203, 0.7%), tobacco use (n = 151, 0.5%), education (n = 137, 0.5%), subjective economic status (n = 1,636, 5.4%). [b]Hypertension was defined as SBP ≥140 mm Hg and/or DBP ≥90 mm Hg and/or current use of antihypertensive medication. (Chobanian et al., 2003). [c]Among n = 28,677 participants, after removing outliers. [d]Among n = 29,492 participants, after removing outliers.

Table S3 in Supporting Information S1 presents the fire-only $PM_{2.5}$ exposure metrics used in the secondary analysis. Due to the episodic nature of the fires and their regional occurrence, the mean and interquartile range (IQR) is lower for this exposure metric than the total $PM_{2.5}$, even though the maximum value is similar to that seen for the overall $PM_{2.5}$ exposure metric used in the primary analysis.

On the day of the clinical assessment, mean $PM_{2.5}$ exposure was 27.3 μg/m^3, though this varied greatly with a minimum same-day exposure of 0.8 μg/m^3 (considered "good," according to the Air Quality Index, AQI) and a maximum of 696.9 μg/m^3 (considered "Hazardous according to the AQI) (Table 2). Much of this variation was due to the contributions of fire emissions.

3.3. Association Between $PM_{2.5}$ and Blood Pressure

The mean SBP in the population was 126.0 with a standard deviation (SD) of 16.0. The mean DBP in the population was 78.4 with a SD of 10.2. In the overall population, our findings show a small but significant association between $PM_{2.5}$ and DBP. An IQR increase in monthly $PM_{2.5}$ exposure was associated with a 0.234 (95% CI: 0.003, 0.464) higher DBP (Table 3). Exposures over other averaging periods were positive but included the null. There was no significant association between $PM_{2.5}$ and SBP in the overall population. In sensitivity analyses, we saw results of similar magnitude after adjusting the BP values of those who reported taking antihypertensive medication, restricting the analysis to those who did not take antihypertensive medication, and running the analysis using a missing indicator (Tables S4–S6 in Supporting Information S1)

In age-stratified models, our results showed stronger associations between $PM_{2.5}$ and both DBP and SBP in older participants. In participants 65 and older, an IQR increase in $PM_{2.5}$ exposure in the preceding two weeks and month, respectively, was associated with 0.89 (95% CI: 0.13, 1.65) and 1.16 mm Hg (95% CI: 0.24, 2.08) higher DBP (Figure 2). In this population, we observed consistent results with SBP. An IQR increase in $PM_{2.5}$ exposure in the preceding two weeks and month, respectively, was associated with a 1.24 (95% CI: 0.0, 2.47) and 1.90 (95% CI: 0.43, 3.37) mm Hg higher SBP for participants ages 65 and older (Figure 2). For DBP, we observed a stronger association in females than in males and in never-smokers and current smokers than in former smokers (Figures 3 and 4). Our secondary analysis using fire emissions only as the exposure metric produced null results (Table S7 in Supporting Information S1).

4. Discussion

Our results provide evidence of an association between medium-term (weekly and monthly) ambient particulate matter air pollution exposure and higher SBP and DBP within a population-based cohort in Indonesia. If this association is causal, the results have important implications for public health, since the CVD burden in Indonesia is high and air pollution is a modifiable at the population level. Several studies in Southeast Asia have reported associations between particulate matter air pollution and BP or other CVD risk factors. In India, short- and long-term exposure to $PM_{2.5}$ was associated with higher SBP in a prospective cohort study in Delhi and long-term exposure to $PM_{2.5}$ was associated with higher SBP and a higher odds of hypertension in women in Andhra Pradesh (Curto et al., 2019; Prabhakaran et al., 2020). In the same cohort, $PM_{2.5}$ was also associated with vascular damage and carotid intima-media thickness (Ranzani et al., 2020a, 2020b). In Delhi, the reported difference in SBP associated with an IQR difference in $PM_{2.5}$ for the overall population was similar in magnitude to what we found in the 65 and older population, potentially due to much higher levels of pollution reported in Delhi than in the present study. In Indonesia, a recent study of residents of Sumatra Island, using data from the IFLS, observed an association between long-term $PM_{2.5}$ exposure and the odds of having doctor-diagnosed CVD (Siregar et al., 2022). Although this study used a more restricted sample of participants than our study, our findings support

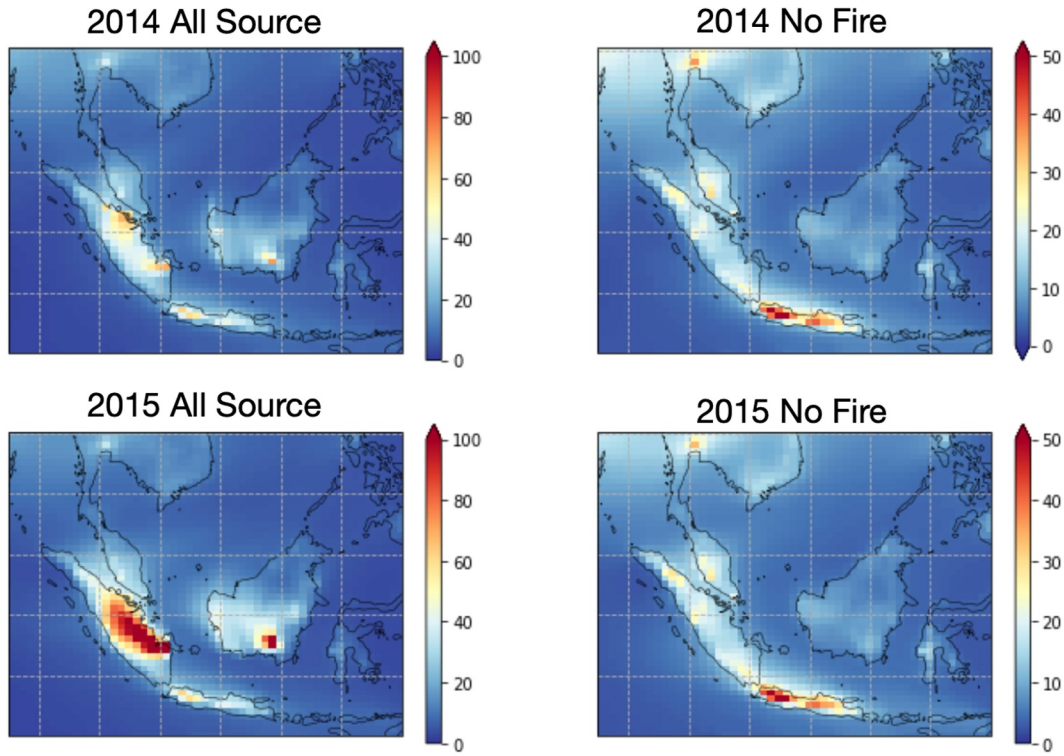

Figure 1. Simulated annual mean surface $PM_{2.5}$ concentrations ($\mu g/m^3$) in 2014 (top row) and 2015 (bottom) with (left) and without (right) fires. Note change in scale.

blood pressure as one possible mechanism by which $PM_{2.5}$ is increasing the risk of CVD in this population. Other studies using the IFLS have reported associations between smoke pollution and a variety of health outcomes, including early life mortality, respiratory health, and difficulties with activities of daily living (Frankenberg et al., 2005; Jayachandran, 2009; Rosales-Rueda & Triyana, 2019). Smoke from major fire events has also been associated with morbidity and mortality, including CVD morbidity, in the neighboring countries of Malaysia and Singapore (Mott et al., 2005; Sahani et al., 2001; Sheldon & Sankaran, 2017).

Worldwide, there is extensive evidence that exposure to particulate air pollution affects blood pressure, both from epidemiologic studies and through controlled human exposure studies where changes in blood pressure have been observed in response to air pollution (Rajagopalan et al., 2018; Yang et al., 2018). This may happen through a variety of mechanisms, including oxidative stress and inflammation, autonomic nervous system imbalance triggering the sympathetic response, and an increase in arterial vasoconstriction (Giorgini et al., 2016). Exposure to particulate matter can also lead to endothelial disfunction and altered system hemodynamics (Brook et al., 2010).

We found a stronger association between ambient exposure to $PM_{2.5}$ and BP among elderly participants in our cohort. Across all exposure periods, we observed stronger associations among participants 65 years of age and older than in the cohort, overall. The elderly represent a population with increased cardiovascular risk, and thus, may be more susceptible to the effects of air pollution. Our findings correspond to other studies which have also demonstrated an association between $PM_{2.5}$ and blood pressure response in the elderly (Wellenius et al., 2012) and a larger body of evidence that generally finds that the elderly are more susceptible to the CVD risks posed by air pollution exposure (Brook et al., 2010; Samoli et al., 2008). Previous studies have also reported sex differences in the association between air pollution and blood pressure. Similar to our results, a study in over 5,000 residents of Andhra Pradesh, India found a stronger association between $PM_{2.5}$ and BP in women than in men (Curto et al., 2019) and a large Taiwanese cohort study also found a stronger association between

Table 2
Summary of Exposure Metrics, Concentration of $PM_{2.5}$ in $\mu g/m^3$

Duration	Mean	Minimum	IQR	Maximum
Same day	27.3	0.8	12.2–35.6	696.9
1 week	27.4	1.5	13.5–35.6	462.5
2 weeks	27.2	1.5	13.9–35.6	383.1
1 month	26.9	1.5	14.1–36.4	285.1

Table 3
Increase in Blood Pressure per IQR[a] $PM_{2.5}$

	Diastolic blood pressure (n = 26,878)				Systolic blood pressure (n = 26,283)			
Exposure metric	Estimate	95% CI		p-value	Estimate	95% CI		p-value
Same day	0.020	−0.095	0.134	0.735	−0.098	−0.260	0.065	0.239
1 week	0.099	−0.046	0.244	0.180	0.044	−0.162	0.250	0.673
2 weeks	0.133	−0.051	0.317	0.157	0.089	−0.172	0.350	0.503
1 month	0.234	0.003	0.464	0.047	0.059	−0.269	0.386	0.726

[a]IQRs: 23.4 μg/m^3 (same day); 22.1 μg/m^3 (1 week); 21.7 μg/m^3 (2 weeks); 22.3 μg/m^3 (1 month).

$PM_{2.5}$ and SBP in women than in men (Zhang et al., 2018). However, other studies have found the opposite holds true, where associations are stronger in men than in women (Song et al., 2021). In part, these discrepancies may be explained by the consideration of sex and age together. It has been hypothesized that estrogen may play a protective role in this relationship; thus, putting men and postmenopausal women at higher risk (Liao et al., 2023). These results may also be explained by differential exposure error in these groups, as women and the elderly may spend more time in or around the home, compared with men and younger individuals (Künzli et al., 2005). Several studies have also examined effect modification by smoking. In the present study, we found stronger associations in current and never smokers compared to former smokers. The literature is inconsistent on this subject with some studies showing stronger associations between air pollution and CVD risk factors and outcomes in smokers (Li et al., 2020; Turner et al., 2017) and others showing stronger associations in non-smokers (Künzli et al., 2005; Zhang et al., 2018). While the mechanisms by which air pollution and smoking impact CVD overlap (inflammation, oxidative stress, thrombus formation, and sympathetic nervous system effects), pathways may be differentially activated by each exposure potentially leading to synergy (Turner et al., 2017), or in some, not leading to any additional response when the same pathway is activated (Li et al., 2020).

Previous studies have shown significant health impacts from episodic fire events in Indonesia (Frankenberg et al., 2005; Jayachandran, 2009; Rosales-Rueda & Triyana, 2019) and studies in other locations, primarily North America, have demonstrated CVD impacts of wildfire smoke (Chen et al., 2021). In contrast, in the present study, we found no association between fire-only $PM_{2.5}$ concentrations and blood pressure. This null result may be driven by a limitation in the cohort data collection in that it was not designed to study smoke from fires, specifically. Although there was some temporal overlap between the IFLS5 data collection period and the 2015 fire season, more than 85% of health data collection measurements took place prior to June 2015, the beginning of the major fire season. Consequently, the fire-specific $PM_{2.5}$ estimates were highly skewed with over 90% of estimates below 5 μg/m^3 which likely did not provide sufficient statistical power to detect associations with BP. Spatially, while the IFLS was designed to include participants from 13 provinces of Indonesia, the study population in more heavily weighted to urban centers such as Jakarta that are less influenced by fire-specific air pollution than other regions. Therefore, the associations observed in the present analysis are likely driven by vehicular and industrial

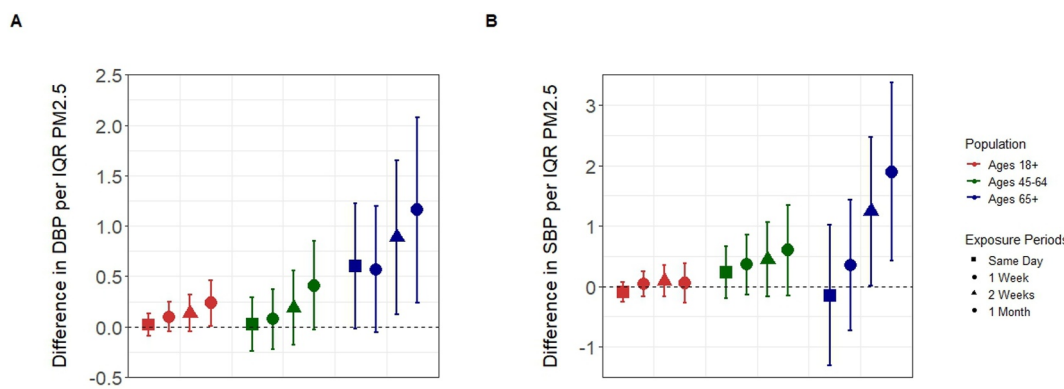

Figure 2. Difference in (a) diastolic blood pressure and (b) systolic blood pressure per interquartile range $PM_{2.5}$, by age and time period of exposure.

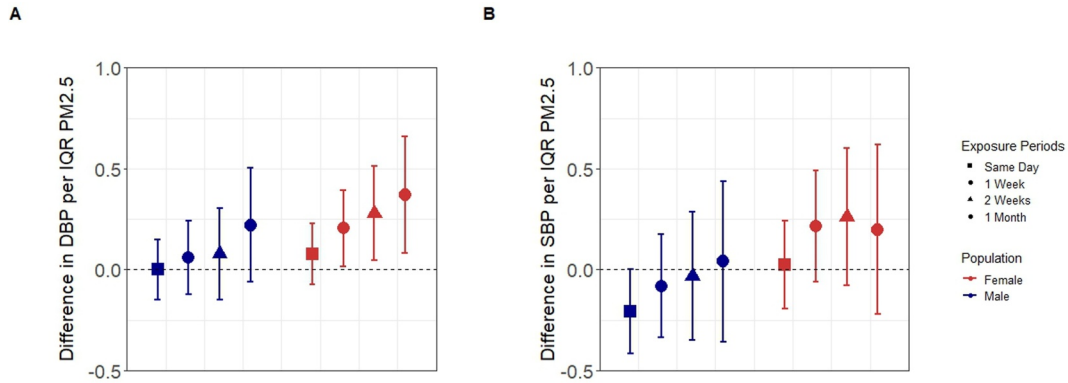

Figure 3. Difference in (a) diastolic blood pressure and (b) systolic blood pressure per interquartile range $PM_{2.5}$, by sex and time period of exposure.

emission sources which the present study was more suited to analyze than fire emissions. However, future efforts to measure health outcomes during Indonesian fire events and analyze them in the context of fire-produced pollution are urgently needed.

To our knowledge, this is the first population-based study to assess the association between short- and medium-term exposure to $PM_{2.5}$ and CVD risk factors in Indonesia. While many studies have projected health impacts from air pollution in Indonesia, they have relied on underlying exposure response functions developed in other locations. Here, we provide evidence to support this association using Indonesia-specific data. Our study supports the hypothesis that exposure to ambient air pollution is associated with higher systolic and DBP among Indonesian residents, particularly the elderly. However, we must acknowledge several limitations to contextualize these findings. The results presented here are based on a cross-sectional analysis using one wave of the IFLS, and therefore, can not be used to assess causality. Although the IFLS is a longitudinal study, our analysis focused on IFLS5 because data on both blood pressure measurements in IFLS participants and global fire emissions were available, and the data collection period partially overlapped with an intense fire season. However, future work can assess the burden of overall $PM_{2.5}$ on blood pressure longitudinally. Our study controlled for potential confounders that may be related to blood pressure and exposure (based on residential location) but undoubtedly the potential for unmeasured and residual confounding exists, due to self-report of some covariates. Further, it should be noted that bias due to selection (resulting from both attrition and conduct of a complete case analysis) may have impacted our findings. However, we note that attrition in the IFLS is very low (Dartanto et al., 2020; Thomas et al., 2012) and a sensitivity analysis using a missing indicator produced results of similar magnitude. We used estimates of ambient $PM_{2.5}$ at the municipality/city level (based on predictions from the GEOS-Chem chemical transport model) as a proxy for personal exposure. Like many air pollution studies of large population cohorts, this likely resulted in some error in our exposure metric, as the coarse spatial resolution of GEOS-Chem

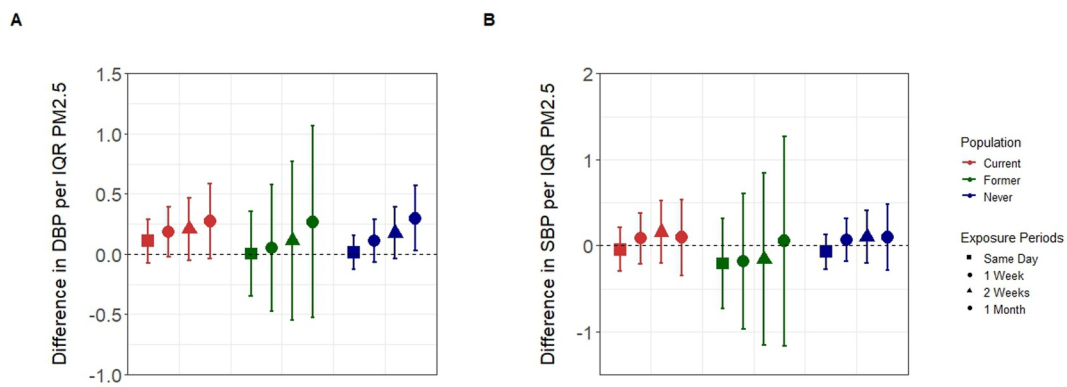

Figure 4. Difference in (a) diastolic blood pressure and (b) systolic blood pressure per interquartile range $PM_{2.5}$, by tobacco status and time period of exposure.

is unable to resolve fine spatial gradients in air pollution exposure. We found moderate correlations between simulated $PM_{2.5}$ and ground station observations, although we note uncertainties in comparing relatively coarse grid cells to point station observations. The advantages of our modeling approach are that it provides spatially and temporally continuous daily $PM_{2.5}$ exposure estimates across the study domain, filling in gaps between sparse monitoring stations and permits us to test the contribution of individual emissions sources. Given the gaps in knowledge on exposure-response relationships in many parts of Southeast Asia and the extremely high burden of air pollution within Indonesia, we believe these results fill an important need.

Indonesia is one of the most populous countries in the world and has a significant air pollution burden from both episodic fire activity and industrial sources. This study provides evidence that poor air quality may contribute to the high prevalence of CVD risk in the Indonesian population. Policies to decrease this population-wide exposure could play an important role in lowering the public health burden within the country.

Conflict of Interest

The authors declare no conflicts of interest relevant to this study.

Data Availability Statement

Public Use Data from the Indonesian Family Life Survey used in this research is available upon data usage agreement and registration. Detailed information can be found at Strauss et al. (2016).

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