Effect of short-term exposures to traffic air pollution and temperature on heart rate among healthy female students during commuting

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Submission date: 12-Apr-2023 09:25AM (UTC+0700)

Submission ID: 2062134157

File name: t-term_exposures_to_traffic_air_pollution_and_temperature_on.pdf (3.01M)

Word count: 7217

Character count: 34213



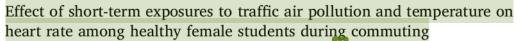
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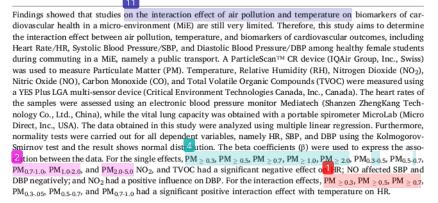
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ARTICLE INFO

Keywords: Exposures Traffic air pollution Temperature Interaction effect Heart rate





1. Introduction

For the past 30 years, scientists have been linking air pollution with health outcomes (Cao et al., 2018; Ezzati and Dockery, 2009; Miller et al., 2007; Roh et al., 2020; Stafoggia et al., 2022). However, most of the studies carried out focused on a macro-environment (MaE) scale, such as country (Cao et al., 2018; Roh et al., 2020) and long-term effects (Miller et al., 2007; Stafoggia et al., 2022), which led to difficulty in taking necessary corrective policy required at such scale (Reis et al., 2022). Previous reports also showed that studies on a micro-environment (MiE) scale, such as public transportation is still very limited (Peters et al., 2004; Setton et al., 2011) and the results are

still unclear due to differences in the methodology, samples, and dependent variable (Biel et al., 2020; Zhang et al., 2022). People can spend a lot of time in a particular MiE, including kitchen, office, and vehicle. A previous study revealed that exposure to specific MiE-related air pollution is two times greater than that of MaE (Peters et al., 2004). Furthermore, studies on exposure to MiE are urgently needed to implement the immediate necessary policy (Caplin et al., 2019; Heal et al., 2012). Biases that arise from a MiE-related study are fewer than from MaE. For example, the use of real-time personal exposure in a MiE-related study (Biel et al., 2020) versus the application of ambient exposure (Miller et al., 2007) and modeling (Roh et al., 2020) in others associated with MaE.

Peer review under responsibility of Turkish National Committee for Air Pollution Research and Control.

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Received 3 September 2022; Received in revised form 7 December 2022; Accepted 8 December 2022

Available online 9 December 2022

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One of the important MiE is public transportation, where people can spend hours traveling. In Indonesia, the most common medium of public transportation is angkot, a minibus with 9-14 seats. Most of the angkots in the country are obsolete since there is no strict regulation regarding their operating age limit. Previous studies revealed that the older the vehicle, the more pollution it emits (Tartakovsky et al., 2013; Wu et al., 2022). Furthermore, most angkots are also not equipped with air conditioning, hence, all the windows and doors are always open during commuting. Previous studies revealed that air pollution received by passengers is higher when vehicle windows are opened (Lim et al., 2021; Tartakovsky et al., 2013). Several studies showed that most of the pollution in MaE comes from the transportation MiE (Suarez-Bertoa et al., 2021; Walsh, 2008) and their number is expected to continue increasing rapidly in the future (Walsh, 2008). This is also exacerbated by long traffic jams that allow passengers to be exposed to more pollution for a longer period (Lipfert and Wyzga, 2008).

Apart from air pollution, passengers in angkot are also exposed to temperature (Grundstein et al., 2009; McLaren et al., 2005). Furthermore, literature studies show that the adverse effect of air pollutants on human health is dominated by cardiovascular outcomes (Biel et al., 2020; Miller et al., 2007; Peters et al., 2004; Stafoggia et al., 2022). Previous studies have also reported that temperature can affect cardiovascular health. Kupcikova et al. (2021) stated that temperature was associated with alterations in cardiac autonomic function in healthy adults affected by traffic-related air pollution (Kupcikova et al., 2021). Although air pollution and temperature have independent effect on cardiovascular health, studies investigating the interaction affect between both of them in a MiE is still very limited. Therefore, this study aims to investigate the interaction effect between air pollution, temperature, and biomarkers of cardiovascular outcomes, including Heart Rate/HR, Systolic Blood Pressure/SBP, and Diastolic Blood Pressure/DBP (Pangaribuan et al., 2019) in healthy female students. Females were selected as the samples because they are more susceptible to adverse effects of pollutants than males (Hong et al., 2005; Phung et al., 2016).

2. Material and methods

2.1. Study design and samples

An electronic invitation flyer was distributed at the Faculty of Public Health, Sam Ratulangi University, Indonesia to recruit samples. The inclusion criteria in this study were healthy female students with no history of cardiovascular and asthma disorder, not a smoker, and having received three doses of the Covid-19 vaccine. A total of 4 students who met the criteria were then selected to participate in the process. Before the start of this study, a 1-day training on all the instruments was carried out, as well as the distribution of informed consent. One *angkot* with a typical age of 30 years was used to transport all the samples and instruments. There were a total of 6 passengers in the *angkot*, namely 4 students, 1 researcher, and 1 driver. This study was carried out on August 4, 2022, from 08:00 to 16:00 (GMT+8). During the study, the passenger door and windows of the *angkot* were conditioned as usual (in the open state). A sample interval of 2 min was used for air pollution, temperature, RH, HR, SBP, and DBP.

Particulate Matter (PM) was measured using a Partical Scan™ CR device (IQAir Group, Inc., Swiss). ParticleScan™ CR uses a laser diode light source and collection optics for particle detection. Particles scatter the light from the laser diode beam in the direction of the collection optics. The collection optics focus the light onto a photodiode that

converts the bursts of light into electrical impulses. The pulse height is proportional to the particle size. Impulses are counted and their intensity is measured for particle sizing. The results are then displayed digitally for the specific size channel(s) and set measurement unit. ParticleScanTM CR has the following technical specification: minimum sensitivity of 0.3 μ m, flow rate of 0.0028 cubic meters per minute, and sample time of 1–600 s. ParticleScanTM CR can measure cumulative size of PM (PM \geq 0.3, PM \geq 0.5, PM \geq 0.7, PM \geq 1.0, Pt \geq 2.0, and PM \geq 5.0) and differential size of PM (PM_{0.30.5}, PM_{0.50.7}, PM_{0.7-1.0}, PM_{1.0-2.0}, and PM_{2.0-5.0}) at the same time. PM \geq 0.3 is defined as particulate matter with an aerodynamic diameter greater than and equal to 0.3 μ m, and so on. PM_{0.3-0.5} is defined as particulate matter with an aerodynamic diameter between 0.3 and 0.5 μ m, and so on.

Temperature, Relative Humidity (RH), Nitrogen Dioxide (NO₂), Nitric Oxide (NO), Carbon Monoxide (CO), and Total Volatile Organic Compounds (TVOC) were measured using a YES Plus LGA multi-sensor (Critical Environment Technologies Canada, Inc., Canada) device. The YES Plus LGA is a battery-powered, portable air quality monitor - information recording (data logging) instrument designed for intermittent or continuous operation. The standard range of its measurements is: temperature (–5.0 – 50 °C, 1 °C resolution), RH (0–100%, 1% resolution), NO₂ (0–5.0 ppm, 0.1 ppm resolution), NO (0–100 ppm, 0.1 ppm resolution), CO (0–50 ppm, 1 ppm resolution), TVOC (0–300 ppm, 1 ppb resolution), where ppm stands for parts per million and ppb stands for parts per billion.

2.2. Measurement of heart rate and vital lung capacity

The samples' heart rates were measured using an electronic blood pressure monitor Mediatech (Shanzen ZhengKang Technology Co., Ltd., China). It has the following technical specifications: measurement mode (oscillometric method), measurement range (40–195 times/min), and accuracy ($\pm 5\%$). The samples' vital lung capacities were measured using a portable spirometer MicroLab (Micro Direct, Inc., USA) which has measurement accuracy of $\pm 3\%$. Four vital capacity indices of the lungs were measured in the process including Forced Expired Volume in 1 s (FEV₁), Forced Vital Capacity (FVC), Peak Expiratory Flow Rate (PEF), and Forced expiratory flow at 50% of exhaled volume (FEF₅₀).

2.3. Quality assurance

Calibration and "zero count" tests for all the instruments were carried out before the start of the process. The protocols for calibration and "zero count" tests for all the instruments are explained in the device manual books and elsewhere.

2.4. Statistical analysis

The data obtained in this study were analyzed using multiple linear regression. Normality tests were carried out for all dependent variables, including HR, SBP, and DBP using kolmogorov-smirnov test, and result shows that the data are normally distributed. Subsequently, the beta coefficients (β) were used to express the association. All the statistical analyses were performed with IBM® SPSS® version 26 software. The following equation was then used to quantify the single effect of air pollution on HR, SBP, and DBP:

$$HR / SBP / DBP = \beta_0 + \beta_1 Aair Pollution + \varepsilon$$

The equation below was used to quantify the combined effect of air pollution and temperature on HR, SBP, and DBP:

Table 1 Characteristics of the samples.

Variables	$Mean \pm SD$
Age (years)	21 ± 0.82
Body Mass Index/BMI (kg/m ²)	20.65 ± 2.34
Heart Rate/HR (beats/minute)	84.34 ± 11.36
Systolic Blood Pressure/SBP (mmHg)	117.56 ± 20.68
Diastolic Blood Pressure/DBP (mmHg)	76.73 ± 22.53

Where β_0 denotes the regression model intercept; β_1 , β_2 , and β_3 denote beta coefficients for each covariate; and ϵ denotes a residual error. Increase in the interquartile range (IQR) of each covariate was used to express increment in HR, SBP, and DBP.

3. Results and discussion

3.1. Characteristics and vital lung capacity indices of samples

Table 1 shows the characteristics of the samples, namely four female students with a mean age and Body Mass Index (BMI) of 21 ± 0.82 years and 20.65 ± 2.34 , respectively. According to the World Health Organization (WHO) classification, they had normal weight (WHO, 2010). The samples used in this study were healthy with no history of cardio-vascular and asthma disorders as well as smoking to minimize biases. They had also received three doses of the Covid-19 vaccine and passed the coronavirus symptoms screening before the start of the process. The mean HR, SBP, and DBP of the samples were 84.34 ± 11.36 beats/minute, 117.56 ± 20.68 mmHg, and 76.73 ± 22.53 mmHg, respectively, and the box plot is presented in Fig. 13.

This study was carried out in one day on August 4, 2022, starting at 08:00 to 16:00 (GMT+8). A total of 8 h were used for measurement with a 1-h lunch break. Fig. 1 shows the study location and GPS tracking of the *angkot* travel routes from the beginning to the end of the experiment. The study began with the measurement of the samples' vital lung capacity. The process was then repeated shortly after the end of the study

at 16.00 (GMT+8). For each period, 2 measurements of vital lung capacity were taken and the best results were selected for further analysis.

Table 2 shows the results of measuring the vital lung capacity indices of the samples. Compared to the initial measurement, the indices obtained in the second round decreased by 6.32% (2.295 L–2.15 L), 8.03% (2.4275 L–2.2325 L), 11.72% (215.5 L/minute to 190.25 L/minute), and 2.64% (3.0325 L/second to 2.9525 L/second) for FEV $_1$, FVC, PEF, and FEF $_{50}$, respectively. This is in line with a previous study that found an increase in the interquartile range of PM $_{10}$ associated with a 5.1% decrease in FEV $_1$ from 95% CI 2.5%–7.7% as well as a 3.7% decrement in FVC from 95% CI 1.8%–5.5% among COPD women (Schikowski et al., 2005). Another study reported a 10 μ g/m 3 increase in acute PM $_{10}$ exposure, which was related to a -0.19 l/min change in PEF (Edginton et al., 2021).

3.2. Characteristics of air pollution and meteorological variables

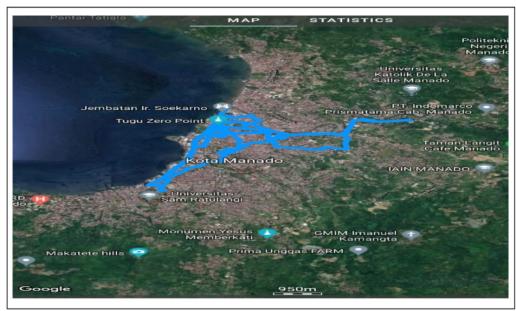
Figs. 2–12 show the plot of 2 min mean of air pollution and meteorological variables, while Table 3 presents the statistical description. The means of temperature and RH were 32.53 \pm 2.14 °C and 60.24 \pm 8.14%, respectively. The concentration means of PM $_{\geq}$ 0.3, PM $_{\geq}$ 0.5, PM

Table 2
Vital lung capacity indices of the samples.

Indices	Units	Min	Pred	Max	V_1	V_2
FEV ₁	Liter	2.0775	2.495	2.9425	2.295	2.15
FVC	Liter	2.265	2.7625	3.2625	2.4275	2.2325
PEF	Liter/minute	266	412	559	215.5	190.25
FEF ₅₀	Liter/second	NA	NA	NA	3.0325	2.9525

 FEV_1 : Forced Expired Volume in 1 s; FVC: Forced Vital Capacity; PEF: Peak Expiratory Flow Rate; FEF_{50} : Forced expiratory flow at 50% of exhaled volume. V_1 : First measurement of vital lung capa

Min: the minimum value of the vital lung capacity index of Asian women; Pred: the predictive value/mean value of vital lung capacity index of Asian women; Max: the maximum value of the lung vital capacity index of Asian women. NA: Not Available.



GPS tracking of the angkot travel routes

Fig. 1. The study location and the GPS tracking of the angkot travel routes.

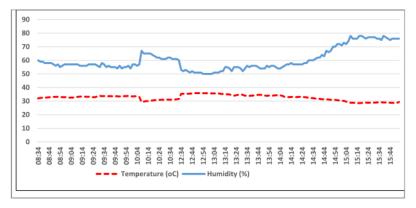


Fig. 2. Plot of 2-min mean temperature and RH.

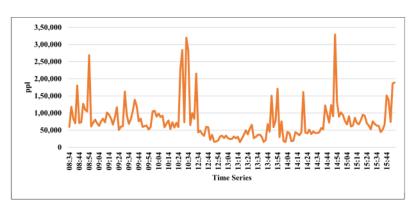


Fig. 3. Plot of 2-min mean PM $_{\geq\ 0.3}$ concentration.

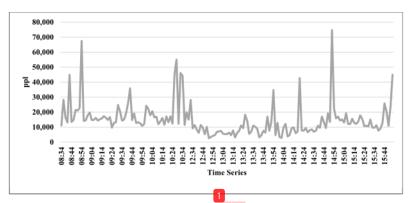


Fig. 4. Plot of 2-min mean PM > 0.5 concentration.

 $_{\geq}$ 0,7, PM $_{\geq}$ 1,0, PM $_{\geq}$ 2,0, and PM $_{\geq}$ 5,0 were 77,476.67 \pm 55,496.61, 14,943.66 \pm 10,972.04, 6252.03 \pm 4350.29, 2158.98 \pm 1689.12, 656.71 \pm 436.94, and 12.97 \pm 10.87, respectively. Furthermore, the average level of PM_{0.3-0.5}, PM_{0.5-0.7}, PM_{0.7-1.0}, PM_{1.0}, and PM_{2.0-5.0}, were 62,533.01 \pm 45,287.67, 8691.63 \pm 6688.05, 4093.05 \pm 2804.07, 1502.27 \pm 1283.97, and 643.7 \pm 430.62, respectively. All the PM air pollutants were measured in particles per liter/ppl. The mean concentration of NO₂, CO, NO, and TVOC were 0.07 \pm 0.04, 11.31 \pm 5.36, 0.48 \pm 0.23, and 42.00 \pm 6.85, respectively. All the gas air pollutants were measured in parts per million/ppm, excluding TVOC in parts per billion/

ppb

3.3. The single effect of air pollution on HR, SBP, and DBP

Table 4 shows the si 4 le effect of air pollution on HR, SBP, and DPB. For the PM pollutan $\stackrel{?}{\sim}$ PM $_{\geq}$ 0.3, PM $_{\geq}$ 0.5, PM $_{\geq}$ 0.7, PM $_{\geq}$ 1.0, PM $_{\geq}$ 2.0, PM_{0.3-0.5}, PM_{0.5-0.7}, PM_{0.7-1.0}, PM_{1.0-2.0}, and PM_{2.0-5.0} had a significant effect on HR, where an increase of 1 IQR in the parameters led to a decrease in HR by 1.08, 0.96, 0.94, 1.06, 1.05, 1.04, 0.94, 0.80, 1.04, 1.05 beats/minute, respectively. Furthermore, for the gaseous

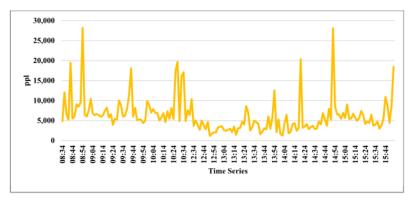


Fig. 5. Plot of 2-min mean PM $_{\geq\ 0.7}$ concentration.

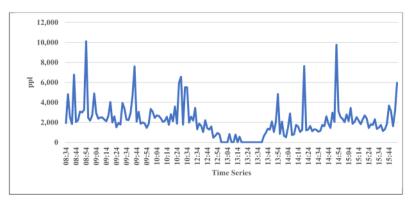


Fig. 6. Plot of 2-min mean PM $_{\geq~1.0}$ concentration.

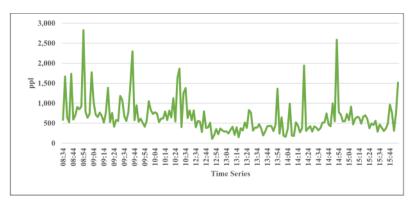


Fig. 7. Plot of 2-min mean PM $_{\geq\ 2.0}$ concentration.

pollutants, NO $_2$ and TVOC had a significant effect. An increase in NO $_2$ and TVOC concentration by 1 IQR led to a decrease in HR by 1.30 and 0.21 beats/minute, respectively.

This study results are inconsistent with previous studies that reported a positive effect of air pollution on HR (Dockery et al., 1999; Liu et al., 2014; Longo et al., 2008; Peters et al., 1999; Pope et al., 1999). However, this study is in line with several studies, where a negative influence was recorded (Angela et al., 2004; Gold et al., 2000). The small sample size used is likely not to detect a positive effect of air pollution on HR. It is also plausible that in certain vulnerable populations, particulate

pollution can lead to dysregulation of autonomic function, thereby reducing heart rate (Gold et al., 2000).

The results showed that SBP was only affected by the NO, where an increase of 1 IQR in NO concentration led to a decrease in SBP by 1.46 mmHg. This finding is inconsistent with previous studies, where a positive correlation was reported between them (Bruce et al., 2005; Kateryna et al., 2014). How ber, the results of this study are in line with Mette et al. (2012), where a doubling of NOx exposure during 1- and 5-year periods preceding enrollment was associated with a decrease of 0.53 mmHg in SBP (Mette et al., 2012).

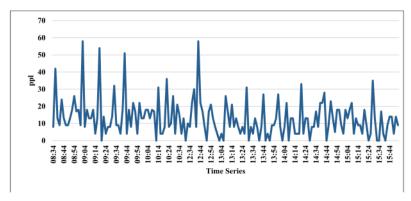


Fig. 8. Plot of 2-min mean PM $_{\geq 5.0}$ concentration.

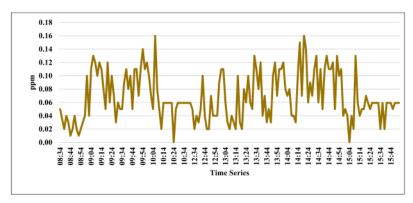


Fig. 9. Plot of 2-min mean NO2 concentration.

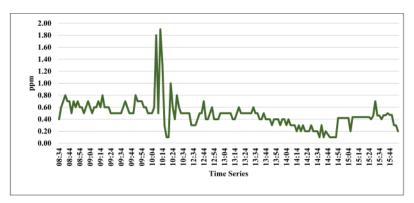


Fig. 10. Plot of 2-min mean NO concentration.

The observation revealed that NO had a negative effect on DBP, where a 1 IQR decrease in NO concentration led to a reduction of 1.43 mmHg in DBP. This finding is inconsistent with Chen et al. (2015) who reported that an increase in NO $_x$ by 20 μ g/m³ was associated with an increase in diastolic blood pressure by 0.34 mmHg (Chen et al., 2015 The negative effect of this pollutant on SBP and DBP is likely due to a shift in the sympathovagal balance due to an increase in vagal tone. Another explanation is related to the effect of NO as a potent vasodilator that diffuses freely across membranes (Ibald-Mulli et al., 2004; Mette et al., 2012). The results showed that NO $_2$ had a positive effect on DBP,

where an increase in NO_2 concentration by 1 IQR led to an increase in DBP by 2.90 mmHg. This finding is in line with Li et al. (2020), where an increase in the level of the pollutant by $10\,\mu\text{g/m}^3$ caused an increment in diastolic blood pressure by 0.797 mmHg (Li et al., 2020).

3.4. The interaction effect of air pollution and temperature on HR, SBP, and DBP

Table 5 shows the interaction effect of air bollution and temperature on HR, SBP, and DPB. For the PM pollutants, $PM \ge 0.3$, $PM \ge 0.5$, $PM \ge 0.7$,

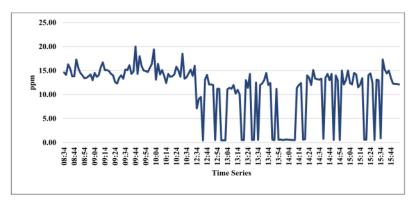


Fig. 11. Plot of 2-min mean CO concentration.

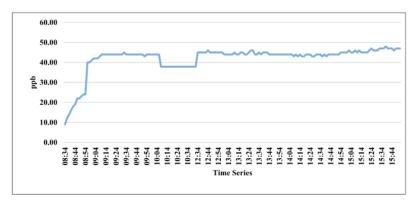


Fig. 12. Plot of 2-min mean TVOC concentration.

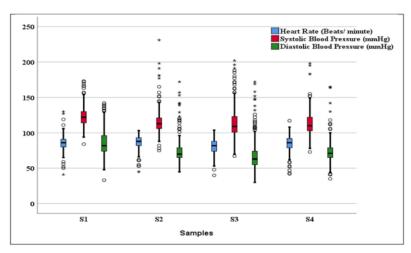


Fig. 13. The distribution of HR, SBP, DBP of the samples.

 $PM_{0.3-0.5}$, $PM_{0.5-0.7}$, and $PM_{0.7-1.0}$ had a signal ant interaction effect with temperature on heart rate. An increase in $PM \ge 0.3$, $PM \ge 0.5$, $PM \ge 0.7$, $PM_{0.3-0.5}$, $PM_{0.5-0.7}$, $PM_{0.7-1.0}$, and temperature by 1 IQR led to an increase in HR by 6.52, 4.52, 4.34, 6.75, 4.33, and 5.33 beats/minute, respectively. Previous studies have explored the single effect of temperature on heart rate. Madaniyazi et al. (2016) revealed that a 1 °C

increase in ambient temperature in a hot environment was associated with 0.133 beats/minute increment in HR compared to 0.063 beat/minute, which was obtained in a cold environme 7. (Madaniyazi et al., 2016). A previous study revealed that heart rate increased significantly (p < 0.05) from 66.5 bpm pre-exposure to 106.0 bpm during exposure to dry heat. It was then concluded that being exposed to high ambient

Table 3The statistical description of air pollutants and meteorological variables.

Variables	$\text{Mean} \pm \text{SD}$	Minimum	Maximum	IQR
PM ≥ 0.3 µm (ppl)	77,476.67 ±	15,164	329,071	47,118
	55,496.61			
$PM \ge 0.5 \mu m (ppl)$	$14,943.66 \pm$	2635	74,722	8066,00
	10,972.04			
$PM \ge 0.7 \mu m (ppl)$	6252.03 ± 4350.29	1187	28,189	3205
$PM \ge 1.0 \mu m (ppl)$	2158.98 ± 1689.12	1.06	10,106	1335
PM ≥ 2.0 µm (ppl)	656.71 ± 436.94	108	2828	375
$PM \ge 5.0 \mu m (ppl)$	12.97 ± 10.87	0	58	14
PM _{0.3-0.5} µm (ppl)	62,533.01 \pm	12,028	273,565	38,553
	45,287.67			
21 _{0.5-0.7} μm (ppl)	8691.63 ± 6688.05	1448	46,644	4867
PM _{0.7-1.0} μm (ppl)	4093.05 ± 2804.07	724	18,314	1918
PM _{1.0-2.0} μm (ppl)	1502.27 ± 1283.97	826	7278	998
PM _{2.0-5.0} μm (ppl)	643.7 ± 430.62	108	2802	365
NO ₂ (ppm)	0.07 ± 0.04	0	0.16	0.06
CO (ppm)	11.31 ± 5.36	0.43	20	3
NO (ppm)	0.48 ± 0.23	0.10	1.90	0.20
TVOC (ppb)	42.00 ± 6.85	9	48.00	2
Temperature (°C)	32.53 ± 2.14	28.50	35.90	3
RH (%)	60.24 ± 8.14	50	78	8

temperature produces a significant increase in HR through an increase in sympathetic and a decrease in parasympathetic drive (Bruce-Low et al.,

A previous study examined a modifying effect of PM_{2.5} on the relationship between temperature and HRV. The results showed that compared to the conditions of low PM_{2.5} (\leq 12.9 $\mu g/m^3$) levels, the effect of temperature on 19 V was greater when air pollution was high, namely >12.9 $\mu g/m^3$ (Ren et al., 2011). The findings in this study contribute to the literature that air pollution and temperature have a positive interaction effect on increasing heart rate. However, this study has several limitations, including the small number of samples, and the relatively short experimental period. This indicates that further studies with a large population and extended periods need to be carried out.

4. Conclusion

This study aims to determine the interaction effect of air pollution and temperature on cardiovascular health biomarker 4 such as HR, SBP, and DBP. For the single effects, it was observed tha 2 M $_2$ 0.3, PM $_2$ 0.5, PM $_2$ 0.7, PM $_2$ 1.0, PM $_2$ 2.0, PM0.3-0.5, PM0.5-0.7, PM0.7-1.0, PM1.0-2.0, PM2.0-5.0, NO2, and TVOC had a significant negative influence on HR; NO affected SBP and DBP negative $^{\rm II}_{\rm I}$ and NO2 had a positive influence on DBP. For the interaction effects, PM $_2$ 0.3, PM $_2$ 0.5, PM $_2$ 0.7, PM0.3-0.5, PM0.5-0.7, and PM0.7-1.0 had a significant positive interaction effect with

Table 4
The single effect of air pollutants and meteorological variables on HR, SBP, and DBP.

Variable	HR				SBP	DBP						
1	β_0	P-Value	β_1	P-Value	β_0	P- Value	β_1	P-Value	β_0	P- Value	β_1	P-Value
PM _{> 0.3} μm (ppl)	86.12	<0.001***	-1.08	0.004**	116.12	<0.001***	0.89	0.198	75.37	<0.001***	0.85	0.268
$PM \ge 0.5 \mu m (ppl)$	86.11	<0.001***	-0.96	0.003**	116.36	<0.001***	0.64	0.276	75.70	<0.001***	0.56	0.389
$PM \ge 0.7 \mu m (ppl)$	86.18	<0.001***	-0.94	0.004**	116.28	<0.001***	0.65	0.271	75.57	<0.001***	0.60	0.357
PM ≥ 1.0 µm (ppl)	86.05	<0.001***	-1.06	0.003**	116.31	<0.001***	0.77	0.229	75.52	<0.001***	0.75	0.282
PM > 2.0 µm (ppl)	86.18	<0.001***	-1.05	0.006**	116.48	<0.001***	0.61	0.376	75.70	<0.001***	0.59	0.432
PM ≥ 5.0 µm (ppl)	83.89	<0.001***	0.49	0.392	117.46	<0.001***	0.11	0.919	77.05	<0.001***	-0.34	0.763
PM _{0.3-0.5} μm (ppl)	86.06	<0.001***	-1.04	0.005**	116.10	<0.001***	0.89	0.190	75.34	<0.001***	0.85	0.252
21 _{0.5-0.7} μm (ppl)	86.03	<0.001***	-0.94	0.003**	116.44	<0.001***	0.63	0.284	75.80	<0.001***	0.52	0.416
PM _{0.7-1.0} μm (ppl)	86.06	<0.001***	-0.80	0.008**	116.41	<0.001***	0.54	0.328	75.73	<0.001***	0.47	0.437
PM _{1.0-2.0} μm (ppl)	85.91	<0.001***	-1.04	0.002**	116.35	<0.001***	0.801	0.201	75.56	<0.001***	0.78	0.252
PM _{2.0-5.0} μm (ppl)	86.20	<0.001***	-1.05	0.005**	116.48	<0.001***	0.601	0.371	75.68	<0.001***	0.60	0.421
NO ₂ (ppm)	85.83	<0.001***	-1.30	0.08°	115.32	<0.001***	1.97	0.146	73.43	<0.001***	2.90	0.05**
CO (ppm)	84.82	<0.001***	0.13	0.609	117.06	<0.001***	0.13	0.769	77.20	<0.001***	-0.12	0.80
NO (ppm)	83.60	<0.001***	0.31	0.415	121.04	<0.001***	-1.46	0.035**	80.14	<0.001***	-1.43	0.06*
TVOC (ppb)	88.84	<0.001***	-0.21	0.09°	124.20	<0.001***	-0.32	0.180	80.62	<0.001***	-0.19	0.47
Temperature (°C)	52.35	<0.001***	2.95	<0.001***	143.21	<0.001***	-2.37	0.037**	100.01	<0.001***	-2.15	0.08°
RH (%)	97.02	<0.001***	-1.68	<0.001***	105.42	< 0.001***	1.16	0.042**	65.86	<0.001***	1.44	0.09*

^{*:} p-value <0.10 **: p-value <0.05 ***: p-value <0.01.

Table 5
The interaction effect of air pollutants and temperature on HR.

Variable	HR							
4	β_0	P-Value	β_1	P-Value	β_2	P-Value	β_3	P-Value
PM ≥ 0.3*T	26.68	0.034**	21.01	0.005**	5.41	<0.001***	-19.90	0.004**
PM > 0.5*T	32.66	0.009**	15.04	0.025**	4.88	<0.001***	-15.40	0.020**
PM > 0.7 *T	33.30	0.011**	13.39	0.046**	4.81	<0.001***	-13.86	0.037**
PM ≥ 1.0*T	44.82	<0.001***	8.02	0.276	3.71	0.001***	-8.53	0.241
PM ≥ 2.0*T	43.90	0.002**	7.49	0.373	3.85	0.003**	-7.78	0.325
PM ≥ 5.0*T	61.15	<0.001***	-10.21	0.284	2.12	0.027**	10.14	0.268
PM 0 2 0 5 *T	26.46	0.033**	21.20	0.004**	5.42	<0.001***	-19.87	0.003**
2 _{0.5-0.7} *T	32.81	0.006**	15.87	0.017**	4.86	<0.001***	-16.40	0.014**
PM _{0.7-1.0} *T	31.56	0.015**	12.58	0.034**	4.98	<0.001***	-12.23	0.027**
PM _{1.0-2.0} *T	46.06	<0.001***	7.49	0.288	3.58	0.001***	-7.83	0.254
PM _{2.0-5.0} *T	43.34	0.002**	7.90	0.339	3.90	0.002**	-8.22	0.293
NO ₂ *T	42.39	0.006**	9.61	0.514	4.03	0.004**	-11.00	0.445
NO*T	34.20	0.065*	7.57	0.296	4.63	0.010**	-7.28	0.317
CO*T	40.63	0.012**	3.01	0.457	3.96	0.006**	-3.06	0.478
TVOC*T	123.32	0.266	-3.20	0.519	-3.20	0.755	8.07	0.548

^{*:} p-value <0.10 **: p-value <0.05 ***: p-value <0.01.

temperature on HR.

Credit author statement

Mandroy Pangaribuan: conception and design of study, acquisition of data, Formal analysis and interpretation of data, drafting the manuscript, and revising the manuscript critically for important intellectual content. Diana Vanda Daturara Doda: conception and design of study, Formal analysis and interpretation of data, and revising the manuscript critically for important intellectual content. Grace E.C. Korompis: conception and design of study and revising the manuscript critically for important intellectual content. Woodford B.S. Joseph: acquisition of data and revising the manuscript critically for important intellectual content. Ribka Wowor: acquisition of data and revising the manuscript critically for important intellectual content. Veronika Simangunsong: revising the manuscript critically for important intellectual content.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

This study was supported and funded by the Universitas Sam Ratulangi under research contract No: 157/UN12.13/LT/2022 and letter of assignment No: 662/UN12.13/LT/2022. The authors are grateful to Meivina Shalommita Putri Sumarauw, Annisa Puji Rahayu, and Yuniar Masloman for the technical support.

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