



# Environmental health risks and impacts of PM<sub>2.5</sub> exposure on human health in residential areas, Bantul, Yogyakarta, Indonesia

Azham Umar Abidin<sup>a,\*</sup>, Anisful Lailil Munawaroh<sup>b</sup>, Aulia Rosinta<sup>c</sup>, Arvi Tri Sulistiyan<sup>d</sup>, Iwan Ardianta<sup>e</sup>, Fajri Mulya Iresha<sup>f</sup>

<sup>a</sup> Department of Environmental Engineering, Faculty of Civil Engineering and Planning, Universitas Islam Indonesia, Indonesia

<sup>b</sup> Department of Information and Medical Service, Vocational School, Applied Master's Program in Occupational Health and Safety, Universitas Gadjah Mada, Indonesia

<sup>c</sup> Department of Community, Family, and Occupational Medicine, Faculty of Medicine, Khon Kaen University, Thailand

<sup>d</sup> Faculty of Medicine, Universitas Islam Indonesia, Indonesia

<sup>e</sup> Laboratory of Air Quality, Department of Environmental Engineering, Universitas Islam Indonesia, Indonesia

<sup>f</sup> Laboratory of Solid and Hazardous Wastes, Department of Environmental Engineering, Universitas Islam Indonesia, Indonesia

## ARTICLE INFO

Handling Editor: Prof. L.H. Lash

### Keywords:

environmental health  
health impact  
health risk assessment  
PM<sub>2.5</sub> exposure

## ABSTRACT

Air pollution, particularly PM<sub>2.5</sub>, significantly impacts public health in developing areas. This study evaluates PM<sub>2.5</sub> exposure among residents and conducts a health risk assessment within the human community in Bantul Regency, Indonesia, utilizing a high-volume air sampler (HVAS) over 24 h in a residential area and interviewing 36 respondents. The findings of this study show that PM<sub>2.5</sub> concentrations varied from 50.7 to 61.9 µg/m<sup>3</sup>, exceeding the national ambient air quality standards (NAAQS) of 35 µg/m<sup>3</sup>. The risk hazard quotient (RQ) values of PM<sub>2.5</sub> were greater than 1, signifying considerable health risk. Epidemiological statistical analysis indicates a significant correlation (p-value < 0.05) between PM<sub>2.5</sub> exposure, health complaints, and respondent characteristics. Residents report health issues including cough, headache, eye irritation, breathlessness, and wheezing. The findings emphasize the imperative for more rigorous air quality standards and regulations, enhanced public awareness and education regarding preventive practices, and urban planning development strategies incorporating green infrastructure. These measures are crucial for alleviating health hazards and enhancing air quality in impacted areas.

## 1. Introduction

Global air pollution, particularly fine particulate matter (PM<sub>2.5</sub>), is a public health concern. Hazardous PM<sub>2.5</sub> is experienced by approximately 7.3 billion individuals, with 80 % of this demographic residing in low-income and middle-income countries [1]. Exposure to PM<sub>2.5</sub> leads to severe health impacts, including approximately 4.11 million premature deaths in 2018 [2]. Regional disparities in PM<sub>2.5</sub> pollution are pronounced, with urbanization contributing to elevated levels in developing countries [3,4]. Even though numerous industrialized countries have successfully reduced PM<sub>2.5</sub> concentrations in urban areas [3], the primary determinants of health hazards are still associated with socioeconomic factors, including industrial settings and rates of urbanization [5]. The elevated PM<sub>2.5</sub> concentrations are linked to population growth in rapidly urbanizing regions like India and sub-Saharan Africa, and these conditions are further exacerbated by rising temperatures [6].

PM<sub>2.5</sub> pollution is a major concern in Indonesia, especially in urban areas such as Jakarta and Yogyakarta, as well as other major cities where PM<sub>2.5</sub> concentrations frequently exceed world health organization (WHO) standards, posing significant health hazards without a safe limit. This has raised concerns regarding public health and diabetes, as exposure to PM<sub>2.5</sub> has been linked to elevated coronavirus disease (COVID-19) mortality rates and elevated plasma glucose levels in non-diabetic adolescents [7,8]. Among the most significant sources of PM<sub>2.5</sub> pollution are biomass burning, vehicular emissions, and industrial activities [9]. Meteorological conditions and seasonal fluctuations elevate PM<sub>2.5</sub> levels, increasing the risk for vulnerable populations, including informal landfill workers. This highlights the urgent need for targeted measures to mitigate health and environmental impacts [10, 11].

PM<sub>2.5</sub> has significant health impacts, including dementia, cardiovascular disease, and respiratory disorders [12,13]. It is evident that a

\* Corresponding author.

E-mail address: [azham.abidin@uii.ac.id](mailto:azham.abidin@uii.ac.id) (A.U. Abidin).

<https://doi.org/10.1016/j.toxrep.2025.101949>

Received 9 December 2024; Received in revised form 20 January 2025; Accepted 2 February 2025

Available online 5 February 2025

2214-7500/© 2025 The Author(s). Published by Elsevier B.V. This is an open access article under the CC BY-NC license (<http://creativecommons.org/licenses/by-nc/4.0/>).

global concern persists, as only 8 % of major cities adhere to WHO's annual PM<sub>2.5</sub> limits [14] despite efforts to mitigate the issue. Remote sensing, geospatial data, and high-resolution population metrics have improved exposure assessment, leading to more accurate health risk estimates [15,16]. These evaluations have been improved by integrating microenvironmental exposure and using bioassays for toxicity measurements [17,18]. Furthermore, regional vulnerabilities have been identified and emission sources have been delineated through data fusion and source apportionment [19,20]. These insights are essential for developing effective environmental policies, including emission-reducing urban designs and public health interventions, emphasizing Indonesia.

Demographic disparities in PM<sub>2.5</sub> exposure are significant, with low-income populations disproportionately affected [21,22]. In certain areas, racial and ethnic minorities face greater exposure, while in developing countries, even non-disadvantaged populations may be considerably affected [23,24]. Both the youth and the elderly populations are particularly vulnerable [25]. The necessity for equitable health interventions is underscored by the fact that in Indonesia, PM<sub>2.5</sub> levels that fall below WHO standards are associated with a higher prevalence of diabetes [26]. Increased insulin resistance and altered glucose metabolism have been linked to prolonged exposure to PM<sub>2.5</sub> in urban populations. Several studies have demonstrated that long-term exposure to PM<sub>2.5</sub> is associated with elevated fasting blood glucose levels and insulin resistance indices [27]. The mechanisms that underlie these effects are systemic inflammation, oxidative stress, and alterations in sphingolipid metabolism [28,29]. Exposure to PM<sub>2.5</sub> can disrupt insulin signaling pathways, with a particular emphasis on the IRS-1/AKT pathway [29]. Furthermore, glucose homeostasis has been demonstrated to be influenced by short-term exposure to PM<sub>2.5</sub>, as evidenced by increases in fasting insulin and observed HOMA-IR [30,31]. The NLRP3 inflammasome is pivotal in insulin resistance exacerbated by PM<sub>2.5</sub> [32]. The metabolic effects induced by PM<sub>2.5</sub> may be more prevalent in specific subgroups, including females, overweight individuals, and older adults [31,33].

Due to its distinctive semi-urban characteristics and susceptibility to elevated PM<sub>2.5</sub> levels, Sitimulyo, Bantul Regency, was chosen as the study site. The area is affected by various emission sources, including residential biomass combustion, vehicular emissions, and its proximity to volcanic activity, i.e., Mount Merapi. Furthermore, the demographic profile of this area, which includes a combination of urban and rural populations and long-term residents, renders it an ideal case study for assessing health risks associated with air pollution in semi-urban settings in Indonesia.

This study utilized a real-time health risk assessment model tailored for semi-urban areas in Indonesia, unlike prior research that often focused on static exposure assessments in developed countries. This study addresses significant gaps in understanding non-carcinogenic health hazards by integrating respondent characteristics, meteorological data, real-time PM<sub>2.5</sub> exposure, and associated health symptoms in humans. Despite Indonesia's national ambient air quality standards (Government Regulation (PP) No. 22 of 2021) establishing the PM<sub>2.5</sub> limit at 55 µg/m<sup>3</sup> [34], this study referenced the U.S. NAAQS standard of 35 µg/m<sup>3</sup> to conform to international benchmarks. This approach enables a comparative analysis of the health risks associated with PM<sub>2.5</sub> exposure in Indonesia against more stringent global standards, emphasizing areas where local regulations could be enhanced to mitigate health risks.

The findings will provide practical insights for public health policies, with a particular emphasis on vulnerable populations, including women, elderly adults, and long-term residents. Furthermore, this study aligns with the sustainable development goals (SDGs) by simultaneously enhancing public health outcomes (SDG 3), promoting sustainable urban planning (SDG 11), and addressing environmental health disparities (SDG 10) [35].

## 2. Material and methods

### 2.1. Study area

The study was conducted at a residence in Sitimulyo, Bantul Regency, the Special Region of Yogyakarta, Indonesia. The locations were selected based on high PM<sub>2.5</sub> exposure levels and particular demographic characteristics. Fig. 1 shows a map of the study area. The PM<sub>2.5</sub> measurement was performed at three sampling points over 24 h, with a total sample size of 36 respondents. The three sampling points were deliberately selected to represent various distances from potential PM<sub>2.5</sub> sources, i.e., high-traffic areas, residential biomass combustion areas, and sites adjacent to landfill activities. The selected points were determined by accessibility and population density to ensure a representative air quality assessment in Sitimulyo, Bantul Regency.

Participants meeting the established inclusion criteria were selected for this study. Participants aged 18 years or older had lived in the study area for nearly a decade and were willing to engage in all phases of the research. The questionnaire encompassed personal information, respondent characteristics, symptoms associated with dust exposure, and health-related complaints. This study excluded pregnant women to adhere to ethical standards and to minimize potential health risks. Exposure studies that involve this demographic often necessitate tailored risk assessment models and additional ethical considerations, which are beyond the scope of this research.

### 2.2. Measurement PM<sub>2.5</sub>

Particulate matter was collected in accordance with SNI 7119.14:2016 standard for PM<sub>2.5</sub> measurement. This study utilized a high-volume air sampler (HVAS) equipped with Whatman No. 1 (CAT 1001–125) with a porosity of less than 0.3 µm for 24 h in a residential area. The HVAS was installed with an airflow rate of 1000–1200 L/min. The filter paper was held in a desiccator for at least 24 h before and after sample collection. The silica gel was positioned at the base of the desiccator. Gravimetric methods were employed to analyze the filter paper utilized to capture particulate matter to estimate the weight of the particles accumulated, as described in Eq. 1:

$$C = \frac{(W_2 - W_1) \times 10^6}{V} \quad (1)$$

C = particulate concentration (µg/m<sup>3</sup>)

W<sub>1</sub> = initial filter weight (g)

W<sub>2</sub> = final filter weight (g)

V = sample air volume (m<sup>3</sup>)

This study used the U.S. NAAQS standard for PM<sub>2.5</sub> (35 µg/m<sup>3</sup>) was utilized in this study for its stringency and global applicability in health risk assessments. The U.S. benchmark was chosen in this study to emphasize the potential gaps in local air quality guidelines and their implications for public health, despite the fact that Indonesia's standard (55 µg/m<sup>3</sup>) had been established.

### 2.3. Data analysis

Under the analysis guidelines of the Ministry of Health, Indonesia, the analyzed data was used to determine the environmental health risk. Meteorological data, i.e., temperature, humidity, and air pressure, were collected at each sampling location to better interpret the results. A statistical analysis was carried out to determine the correlations between health complaints and PM<sub>2.5</sub>. Regression analyses were performed to evaluate variables education level, gender, age, and duration of residency, with a significance threshold set at a p-value < 0.05. The spatial distribution of PM<sub>2.5</sub> was analyzed using ArcGIS (v.10.8).

The application of regression analysis may be deficient in statistical robustness due to the limited sample size (n = 36) and the dependence on 3 PM<sub>2.5</sub> concentration measurements. Therefore, the data are





Fig. 1. The map of the location sampling.

distributed to provide a descriptive overview of PM<sub>2.5</sub> exposure levels, health complaints, and demographic characteristics. This approach enables a clearer visualization of the observed trends and their potential implications for public health.

The risk assessments were conducted in accordance with the environmental health risk analysis guidelines (EHRAG) issued by the Directorate General of Public Health, Ministry of Health, Indonesia (2021). These revised guidelines provide a comprehensive framework encompassing hazard identification, dose-response assessment, and exposure assessment. The hazard identification phase focuses on identifying potential routes of exposure to risk agents and understanding their effects at various concentrations. The dose-response assessment includes calculating reference values, such as RfD, RfC, and SF, to evaluate the potential health impacts of exposure. By incorporating recent advancements, the 2021 guidelines enable a more accurate and standardized approach for assessing environmental health risks. This study employed predetermined default values for exposure time, frequency, duration, and body weight in the HRA calculations; however, direct measurements were not performed due to logistical and resource constraints. These default values align with the environmental health risk analysis guidelines (EHRAG) of the Indonesian Ministry of Health.

However, future studies should incorporate detailed questionnaires and direct assessments to improve the specificity and accuracy of risk characterization.

The health risk assessment (HRA) methodology was used to assess the health risks linked to PM<sub>2.5</sub> exposure. The calculation was performed utilizing Eq. 2:

$$I = \frac{C \times R \times tE \times fE \times Dt}{Wb \times tavg} \quad (2)$$

I (Intake)= intake (mg/kg/day)

C (Concentration)= concentration of the agent on the air medium (mg/m<sup>3</sup>)

R (Rate)= inhalation rate or volume of incoming air per hour(m<sup>3</sup>/h)

tE (time of exposure)= duration of exposure each day (hours/day)

fE (frequency of exposure)= number of days of exposure per year (days/year). To reflect real-time exposure conditions in this study, fE was set at 350 days per year, considering days when exposure was impossible because of interruptions or holidays. In accordance with accepted health risk assessment procedures, a value of 360 days per year was utilized for time-averaged calculations, guaranteeing comparability with international guidelines.

Dt (duration time)= number of years of exposure (real-time exposure) (years)

Wb (weight of body)= exposed human weight (kg)

tavg (time average)= average time period (30 years x 365 days/year for non-carcinogen effects)

While this study focused on tropical conditions, the methodology can be adapted for other regions by adjusting key parameters, such as exposure duration and frequency, to reflect local environmental and demographic characteristics. For example, relative humidity levels in subtropical climates could be incorporated into the calculation of PM<sub>2.5</sub> concentrations and their health impacts. Eq. 3 was used to determine the risk level for risk characterization based on non-carcinogenic effects.

$$RQ = \frac{I}{RfC} \tag{3}$$

I = intake (mg/kg.day)  
RQ = risk hazard quotient  
RfC = reference concentration (mg/kg.day)

3. Result and discussion

3.1. Particulate matter concentration and meteorological data

The PM<sub>2.5</sub> concentrations observed in Sitimulyo ranged from 50.7 to 61.9 µg/m<sup>3</sup>. The PM<sub>2.5</sub> measurements surpassed the national ambient air quality standards (NAAQS) set by the US-EPA, which is 35 µg/m<sup>3</sup>.

The observed value can be attributed to a combination of anthropogenic and natural factors. Significant factors comprise the prevalent utilization of biomass fuels for cooking, vehicular emissions in semi-urban areas, and the proximity to mount Merapi, which regularly releases fine particulate matter during volcanic activity. Furthermore, meteorological conditions, including elevated humidity and stable atmospheric pressure, may intensify the accumulation of particulate matter, as noted in previous studies on air quality in similar settings.

The study highlights the significant health risks associated with elevated PM<sub>2.5</sub> concentrations in residential settings, as all measured locations exceeded the 35 µg/m<sup>3</sup> US-EPA national ambient air quality standards (NAAQS). Table 1 shows this information in detail. The average concentration of PM<sub>2.5</sub> is 57.4 µg/m<sup>3</sup>. This study offers evidence-based mitigation strategies and detailed insights into the effects of PM<sub>2.5</sub> pollution on vulnerable groups by combining demographic, meteorological, and real-time exposure data.

Meteorological conditions with high humidity (68.23–75.94 %) and stable air pressure (746.4–747.7 mmHg) significantly impacted the dispersion and accumulation of PM<sub>2.5</sub>. While utilized in tropical environments characterized by high humidity, this method can be modified for subtropical regions by integrating region-specific factors, including differing temperatures, lower humidity, and seasonal fluctuations. These modifications would enhance the model’s applicability, ensuring accurate health risk assessments tailored to different climatic conditions.

High humidity promotes particle growth through hygroscopic processes, and stable atmospheric conditions trap pollutants near the ground level. These findings align with research conducted in China, demonstrating that meteorological factors significantly influence PM<sub>2.5</sub> concentrations. A study found a 30–50 % reduction in annual mean

Table 1  
PM<sub>2.5</sub> concentration and meteorological parameters.

Sampling Point	PM <sub>2.5</sub> (µg/m <sup>3</sup> )	Temperature (°C)	Humidity (%)	Air Pressure (mmHg)
1	50.7	30.71	68.23	746.7
2	61.9	29.92	69.65	746.4
3	59.6	28.12	75.94	747.6
Mean	57.4	29.58	71.27	746.9

PM<sub>2.5</sub> levels from 2013 to 2018, with around 12 % of the reduction attributed to favorable meteorological conditions [36].

Vertical stratification of PM<sub>2.5</sub> adds another layer of complexity, as concentrations typically decrease with altitude and show more pronounced stratification in the morning compared to the afternoon. The distribution of PM<sub>2.5</sub> is further influenced by seasonal variations, with winter typically exhibiting the highest concentrations due to increased heating and stagnant atmospheric conditions. Nevertheless, dust peaks in the spring are associated with coarse particulate matter in northern regions [37]. These variations align with the findings of this study, where meteorological conditions, such as stable air pressure and high humidity, likely contributed to pollutant persistence.

Furthermore, PM<sub>2.5</sub> concentrations generally correlate negatively with precipitation, relative humidity, air temperature, and wind speed, while demonstrating a positive correlation with surface pressure [37]. These relationships highlight the necessity of incorporating meteorological variables into air quality models to enhance predictive precision. Enhanced forecasting can enable authorities to anticipate high-exposure periods and implement temporary measures, i.e., restricting high-emission activities during unfavorable conditions [17].

The PM<sub>2.5</sub> Reference Concentration (RfC) value in this study was adopted from the NAAQS, which is 35 µg/m<sup>3</sup>. This value is not yet included in the integrated risk level information system (IRIS) list or the agency for toxic substances and disease registry’s (ATSDR) table. The RfC value for assessing risk exposure to PM<sub>2.5</sub>, as determined by Eq. 2, is 0.01 mg/kg/day. Table 2 shows the health risk assessment of PM<sub>2.5</sub> based on the exposure factor for dose-response evaluation.

The findings of this study highlight the disparity between Indonesia’s PM<sub>2.5</sub> threshold and more stringent international standards such as the U.S. NAAQS. The elevated PM<sub>2.5</sub> concentrations measured in Bantul Regency, which exceed both benchmarks, underscore the need for revisiting national air quality standards. Implementing more stringent regulations could improve public health safeguards, especially for at-risk groups, and align Indonesia’s regulatory framework with international best practices.

The calculated frequency of exposure (fE) values used in this study differ depending on the analysis context. For real-time exposure, fE was set to 350 days/year, reflecting realistic conditions that account for non-exposure days, such as holidays or personal interruptions. The time-averaged value of 360 days per year corresponds with standard assumptions in health risk assessments, facilitating comparability with other studies. This dual approach balances the need for practical applicability and methodological consistency in long-term health risk modelling. The application of standard assumptions for exposure time, frequency, duration, and body weight in this study provides a general

Table 2  
Exposure factors for dose-response assessment.

Exposure Factors	Unit	Realtime	Reference
R (Rate)	m <sup>3</sup> /h	0.83	Guidelines for Assessing Human Health Risks from Environmental Hazards, Environmental Health Risk Analysis Guidelines and U.S. Environmental Protection Agency [38–40]
tE (Time of Exposure)	hours/day	24	EHRAG [40]
fE (Frequency of Exposure)	days/year	350	EHRAG [40]
Dt (Duration Time)	Years	30	EHRAG [40]
Wb (Weight of Body)	Kg	55	EHRAG [40]
tavg (Time Average)	30 years x 365 days/year for non-carcinogen	10,950	EHRAG [40]



estimation of health risks but may not fully capture individual variability among residents in the studied area. The direct collection of data via questionnaires and body weight evaluations would improve the accuracy of risk assessment. Integrating these methodologies in future research will be essential for customizing public health interventions to meet specific population requirements.

3.2. Health risk assessment

Health risk assessments related to PM<sub>2.5</sub> were carried out using the risk hazard quotient (RQ), shown in Table 3. An RQ value > 1 indicates a significant non-carcinogenic health risk. All sampling points showed RQ > 1, indicating that long-term exposure to PM<sub>2.5</sub> can pose a serious health threat.

The calculated RQ values > 1 confirm that PM<sub>2.5</sub> concentrations in the study area pose significant non-carcinogenic health risks. This finding is consistent with global studies linking particulate matter exposure to respiratory, cardiovascular, and metabolic disorders [38, 39]. Both short-term and long-term exposure to PM<sub>2.5</sub> have been associated with increased mortality, reduced life expectancy, and heightened risks of chronic diseases [38].

Implementing 350 days per year for real-time exposure guarantees that the health risk assessment accurately represents actual exposure patterns, whereas the 360 days per year utilized for time average calculations comply with established guidelines for long-term risk assessment. These methodological considerations are vital for accurate risk characterization, particularly when comparing findings with global studies.

Health risk assessments estimate that over 4 million deaths globally are attributable to PM<sub>2.5</sub> exposure annually, emphasizing the magnitude of its public health burden [39]. Non-carcinogenic health effects are commonly evaluated using hazard quotients (HQ) and hazard indices (HI), with values exceeding 1 indicating a significant risk [40,41]. The findings of this study, which showed an RQ > 1 for all sampled locations, corroborate these risk thresholds and highlight the potential for adverse health outcomes in the affected population.

Furthermore, PM<sub>2.5</sub> particles exhibit distinct deposition patterns within the respiratory tract, with the highest deposition fractions occurring in the head region, thereby heightening vulnerability to respiratory diseases [40]. These risks are particularly concerning given the high prevalence of health complaints (88.9 %) among residents, further underscoring the urgent need for public health interventions.

The results also align with studies showing a dose-response relationship between PM<sub>2.5</sub> exposure and adverse health outcomes [12,41]. As implemented in this study, integrating real-time exposure data enhances the accuracy of health risk assessments and offers practical insights for policy implementation.

PM<sub>2.5</sub> levels in occupational environments frequently exceed safety limits, presenting considerable health hazards for workers [10,42]. Exposure to PM<sub>2.5</sub> might induce immediate respiratory symptoms and chronic health consequences, potentially impacting many organs [43]. The respiratory system is especially susceptible, since PM<sub>2.5</sub> exposure is related to increased susceptibility to infections and compromised host immune responses [2].

3.3. Characteristic respondent and health complaint

Health complaints were reported by a substantial number of

**Table 3**  
Health risk assessment of PM<sub>2.5</sub>.

Sampling Point	Intake (mg/kg/day)	Risk hazard Quotient (RQ)
L1	0.0176	1.76
L2	0.0215	2.14
L3	0.0206	2.06

respondents (88.9 %). The demographic data revealed that most respondents were female (72.2 %) and had lived in the area for more than 10 years (86.1 %), with a notable percentage being over 50 years of age (55.6 %). Data collected from 36 residents provided insights into health complaints and demographic factors, as summarized in Table 5.

Fig. 2 shows a clustered bar that categorizes the respondent data by gender, age group, education level, and years of residence to further illustrate these demographic factors' distribution. This comprehensive visualization highlights the concentration of health complaints among specific demographic groups. For instance, respondents over 50 years of age and those with extended residence in the area (>10 years) were the most prominent in the dataset.

Despite the fact that this study is preliminary, as it includes only 36 respondents and three sampling areas, it provides essential baseline data on the health effects of PM<sub>2.5</sub> exposure in a semi-urban environment. Future studies should expand the sample size and include additional geographic regions to improve generalizability and provide more robust data for policymaking.

In consultation with a biostatistician, the sample size for this study (n = 36) was determined to be adequate for exploratory analysis due to logistical constraints. While the sample size limits the generalizability of the findings, it provides preliminary insights into the correlations between PM<sub>2.5</sub>, health symptoms, and demographic characteristics in the study area. In order to identify significant associations, regression analysis was implemented as an initial measure, recognizing the study's exploratory nature.

3.4. Statistical analysis of exposure PM2.5 with health complaints and characteristic respondent

The statistical analysis indicated a strong correlation between PM<sub>2.5</sub> exposure and many independent variables, such as health complaints, educational attainment, gender, length of residence, and age (Table 1). All independent variables had p-values below the significance threshold of 0.05, indicating statistically significant correlations.

The stacked bar chart (Fig. 2) clearly represents the p-values for each independent variable concerning the significance threshold (reference line set at 0.05). Among the variables, health complaints demonstrated the strongest correlation with PM<sub>2.5</sub> exposure, as indicated by the lowest p-value of 0.001. Educational attainment, gender, and length of residence also exhibited highly significant relationships, with p-values of 0.000 and 0.01, respectively. Furthermore, a p-value of 0.043 was obtained, indicating a significant association between age and PM<sub>2.5</sub> exposure.

Including the reference line in Fig. 2 emphasizes the significance threshold, illustrating that all variables analyzed fall below this critical value. These findings highlight the potential impact of PM<sub>2.5</sub> exposure on both health outcomes and sociodemographic characteristics, highlighting the necessity for targeted interventions to alleviate air pollution in impacted communities.

This study acknowledges the limitations posed by the small sample size and restricted number of PM<sub>2.5</sub>. These factors may have affected the statistical power of the regression analysis and the robustness of the observed associations. These preliminary findings should be validated

**Table 4**  
Correlation exposure PM<sub>2.5</sub> with health complaints and characteristic respondent.

Dependent Variable	Independent Variable	p-value
PM <sub>2.5</sub> exposure	Health complaints	0.001*
	Education level	0.000*
	Gender	0.000*
	Duration of residence	0.01*
	Age	0.043*

\* significant correlation p-value < 0.05

**Table 5**  
Demographics and health complaints of respondents.

Variable	Category	Frequency	Percentage (%)
Health Complaints	No	4	11.1
	Yes	32	88.9
Education Level	No School	10	27.8
	Elementary School	10	27.8
	Middle School	8	22.2
	High School	8	22.2
Gender	Female	26	72.2
	Male	10	27.8
Age Group	20–30 Years	3	8.3
	31–40 Years	5	13.9
	41–50 Years	8	22.2
	> 50 Years	20	55.6
Years of Residence	< 5 Years	1	2.8
	5–10 Years	4	11.1
	> 10 Years	31	86.1

and expanded through the use of larger sample sizes and more comprehensive data collection in future studies.

The application of regression analysis with statistical rigor was difficult due to the small sample size and limited PM<sub>2.5</sub> measurements. The descriptive nature of the findings is further demonstrated by the presentation of the data as distributions, which provides a fundamental understanding of PM<sub>2.5</sub> exposure and its health implications. Future studies with larger sample sizes and more comprehensive measurements to facilitate more robust inferential statistical analysis.

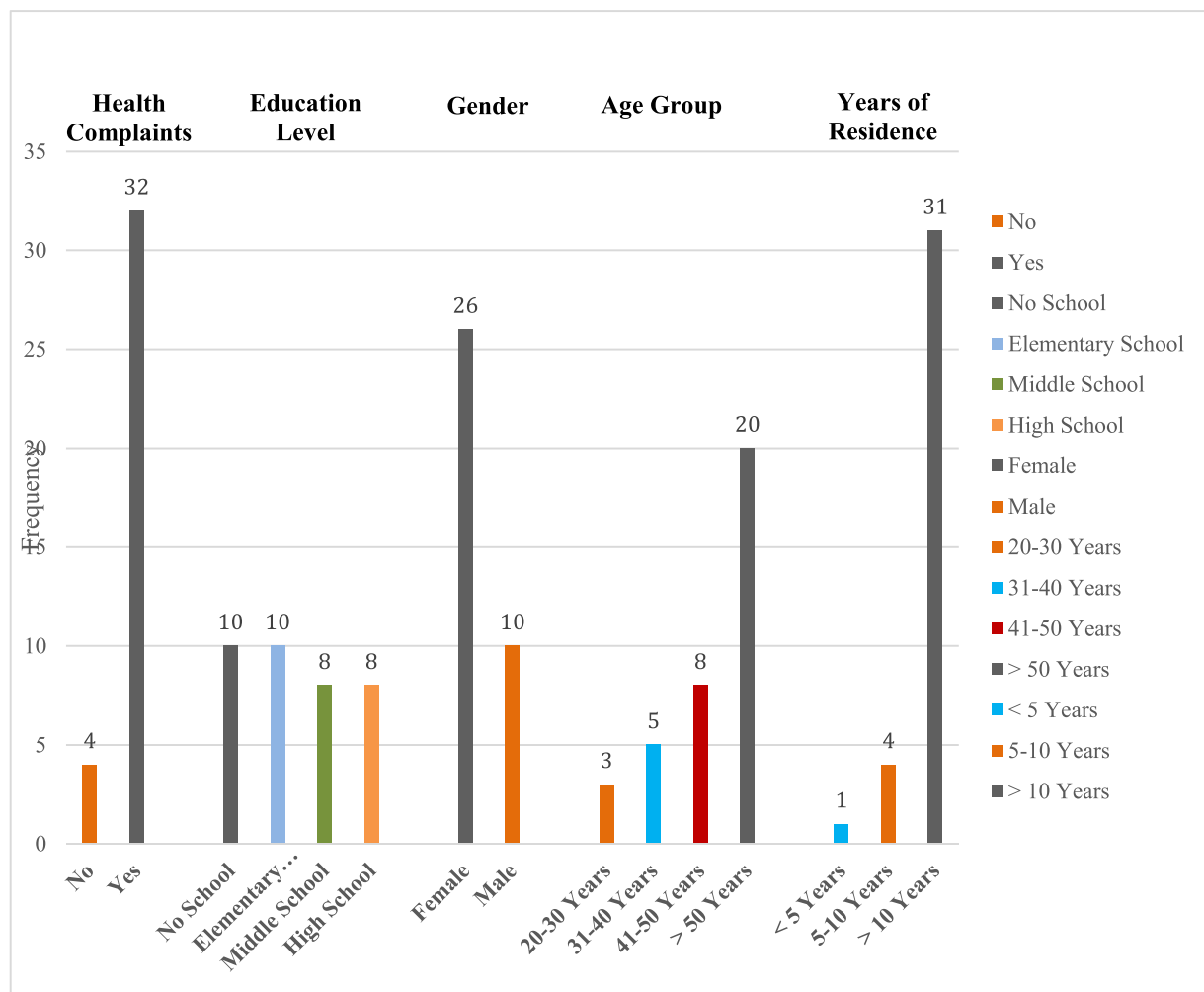
### 3.4.1. Health complaints

Health complaints demonstrated a p-value of 0.001, indicating a strong correlation between PM<sub>2.5</sub> exposure and increased reported health difficulties among respondents. This finding supports the notion that PM<sub>2.5</sub> can adversely affect respiratory health and overall well-being. Common health complaints residents report includes coughing, headaches, eye irritation, breathlessness, and wheezing.

Multiple studies have shown that exposure to fine particulate matter is significantly linked to various negative health effects. Individuals exposed to elevated levels of PM<sub>2.5</sub> frequently report respiratory symptoms, including coughing, dyspnea, wheezing, and chest constriction. Moreover, extended exposure has been associated with a heightened risk of chronic respiratory ailments such as asthma and chronic obstructive pulmonary disease (COPD). Affected populations frequently experience a reduced quality of life and an elevated incidence of hospitalizations due to such circumstances [44]. Individuals can reduce their exposure by refraining from engaging in outdoor activities during periods of elevated pollution, using masks, and optimizing ventilation.

In addition to respiratory repercussions, PM<sub>2.5</sub> exposure systemically influences general health. Headaches and eye irritation are commonly observed, particularly among those employed or living in highly polluted surroundings, such as congested urban areas or waste sites. Furthermore, data indicate that PM<sub>2.5</sub> can inhibit immune system function, increasing an individual's vulnerability to infections and exacerbating pre-existing health disorders [10,45].

PM<sub>2.5</sub> exposure significantly impacts respiratory health through



**Fig. 2.** Distribution of respondents by demographic variables.

multiple mechanisms. It induces oxidative stress, leading to cellular damage and exacerbating conditions such as asthma [44,46]. Inflammatory responses are also induced by PM<sub>2.5</sub>, which increases the likelihood of respiratory infections [2,47]. These mechanisms highlight the complex pathways through which PM<sub>2.5</sub> exposure adversely affects health, emphasizing the urgent need for targeted public health interventions and regulatory actions.

Recent studies have emphasized the importance of implementing various strategies to mitigate the health risks associated with PM<sub>2.5</sub> exposure to reduce the health complaints associated with it. Indoor interventions, such as high-efficiency particulate air (HEPA) filter purifiers, have significantly lower indoor PM<sub>2.5</sub> and improve cardiopulmonary health outcomes [48]. The use of well-fitting N95 respirators has proven effective in reducing individual PM<sub>2.5</sub> exposure and improving cardiovascular health markers [49]. On a broader scale, public health interventions targeting reductions in residential solid fuel use and agricultural emissions can substantially decrease PM<sub>2.5</sub> exposure and related mortality [50].

For long-term effects, it is imperative to take policy-level actions, such as increasing public awareness of pollution hazards and implementing air pollution prevention measures [51]. Significant reductions in attributable mortality and the promotion of environmental justice may result from an effort to align PM<sub>2.5</sub> levels with WHO guidelines [52]. Addressing the economic and behavioral barriers to clean fuel adoption is imperative to guarantee sustainable risk mitigation in vulnerable populations [49,53]. These combined strategies highlight the importance of a multifaceted approach address PM<sub>2.5</sub>-related health risks effectively.

#### 3.4.2. Education level

A strong correlation was observed between PM<sub>2.5</sub> exposure and educational level ( $p = 0.000$ ). The health concerns associated with air pollution may be more well-understood by individuals with higher educational attainment, and they may have better access to preventive strategies to reduce exposure. Recent studies have highlighted the influence of education on understanding and mitigating air pollution risks. Higher educational levels are associated with greater access to air quality information and adopting protective behaviors [54].

However, individuals with lower levels of education may be more likely to contribute (pay) to pollution mitigation [55] and are more vulnerable to health risks associated with PM<sub>2.5</sub> [56]. Particulate matter education can improve self-care knowledge and indoor air quality among COPD patients [57]. Individual efforts, such as using HEPA air purifiers and well-fitting respirators, can effectively reduce PM<sub>2.5</sub> exposure and improve cardiovascular health [48]. The Theory of Planned Behavior reveals differences in attitudes and behavioral intentions between undergraduate and postgraduate parents regarding PM<sub>2.5</sub> protection [58]. As air quality improves, individual behavioral changes become increasingly important in reducing exposure risks [59].

Education plays a pivotal role in shaping individuals' understanding, attitudes, and behaviors towards mitigating air pollution risk. While higher education levels are linked to greater access to air quality information and increased adoption of protective measures, individuals with lower education levels may exhibit a willingness to invest in pollution mitigation and are disproportionately vulnerable to PM<sub>2.5</sub>-related health impacts. Educational interventions, such as particulate matter education programs, can enhance self-care knowledge and improve indoor air quality, particularly for at-risk populations, such as COPD patients. As air quality improves, fostering behavior changes across educational levels becomes critical to reducing PM<sub>2.5</sub> exposure and enhancing public health.

#### 3.4.3. Gender

The findings of this study reveal a significant correlation between gender and the effects of PM<sub>2.5</sub> exposure ( $p$ -value = 0.000), underscoring the importance of addressing biological and behavioral

distinctions in air pollution research. Prior studies support these results, demonstrating that women face heightened long-term health risks from PM<sub>2.5</sub>. For instance, women exhibit a greater risk of ischemic heart disease than men [60], whereas rural females are disproportionately affected by PM<sub>2.5</sub>-related lung cancer and leukemia [4]. In addition, elderly women have also been identified as being more vulnerable to cognitive decline associated with air pollution exposure [61].

Experimental evidence corroborates these findings, highlighting the biological vulnerabilities among females. Female mice show increased susceptibility to M<sub>2.5</sub>-induced insulin resistance, hepatic lipid accumulation [62], and depletion of ovarian follicles, indicating potential reproductive health implications [63]. Male-specific effects include reduced circulating endothelial progenitor cells and heightened sensitivity to maternal PM<sub>2.5</sub> exposure-induced bronchopulmonary dysplasia [64,65]. However, contradictory evidence persists concerning cardiovascular risks, with certain studies indicating no substantial gender disparities [66].

These findings highlight the necessity of implementing targeted interventions to mitigate PM<sub>2.5</sub>-related health effects. HEPA air purifiers have been shown to reduce indoor PM<sub>2.5</sub> concentrations and improve cardiopulmonary health outcomes significantly. In the same way, well-fitting N95 respirators have demonstrated effectiveness in reducing PM<sub>2.5</sub> exposure and providing cardiovascular benefits [48]. On a broader scale, gender-specific public health campaigns and behavioral interventions could enhance the effectiveness of national air quality management strategies [59,67]. These findings emphasize the critical need to integrate gender-sensitive approaches into air pollution policies and public health frameworks to ensure the equitable and effective mitigation of exposure risks.

#### 3.4.4. Duration of residence

The analysis highlights a substantial association between prolonged PM<sub>2.5</sub> exposure and adverse health outcomes, illustrating the cumulative health risks of extended exposure durations. Medium- to long-term exposure (> 30 days) is strongly linked to an increased incidence of cardiovascular diseases [68,69], whereas short- to long-term exposure (0–360 days) is significantly associated with cerebrovascular and respiratory conditions [68]. Long-term exposure (11–18 years) further exacerbates the risk of frailty and multimorbidity [56]. Furthermore, the cumulative effects of sustained exposure are further demonstrated by the significant correlation between adverse health effects and the duration of residency in areas with high PM<sub>2.5</sub> levels ( $p$ -value = 0.01).

These findings collectively emphasize the progressive health risks attributable to PM<sub>2.5</sub> exposure, spanning acute effects on cardiovascular and respiratory health to chronic diseases and increased mortality rates. This emphasizes the urgent need for sustainable environmental management and focused intervention strategies to mitigate PM<sub>2.5</sub> pollution, safeguard public health, and diminish exposure-related long-term health impacts.

#### 3.4.5. Age

Age is a critical factor influencing susceptibility to the health impacts of PM<sub>2.5</sub> exposure, as demonstrated by a significant correlation ( $p$ -value = 0.043). Evidence indicates that certain demographic groups, particularly children and older adults, are more vulnerable to physiological and lifestyle factors. Children are notably at risk of respiratory complications, developmental delays, and potential neurotoxic effects [70]. Moreover, exposure to PM<sub>2.5</sub> in children is associated increased respiratory-related hospitalizations, particularly during warmer seasons [71,72]. Middle-aged and elderly individuals are similarly prone to adverse health outcomes, including a higher likelihood of developing cardiovascular diseases, metabolic disorders, and cognitive impairments [68,73,74]. Prolonged exposure to PM<sub>2.5</sub> in older adults is associated with elevated rates of cardiovascular hospitalizations [71,75], as well as an increased risk of Alzheimer's disease and age-related cerebral damage, possibly mediated by oxidative stress mechanisms [76].

Changes further influence age-related susceptibility in physiological functions and lifestyle behaviors. For instance, handgrip strength and visual contrast sensitivity are factors that influence gait variability, which increases with age [77]. Moreover, consistent health behaviors and diverse social activities are associated with improved cognitive function and reduced mortality risk in older adults [78]. Aging also leads to greater variability in physiological functions, such as renal capacity and heart rate variability [79]. Other contributing factors include age-related changes in the skin microbiome and rest-activity rhythms, shaped by demographic and physiological characteristics [80,81]. Long-term fluctuations in physiological measures, including body weight and blood pressure, have also been associated with increased mortality risk and accelerated epigenetic aging [77].

These findings highlight the necessity of developing public health strategies that specifically address age-related vulnerabilities to PM<sub>2.5</sub>. To mitigate the long-term health risks associated with air pollution, it is imperative to implement targeted interventions that prioritize the protection of children and older adults. By integrating physiological and lifestyle factors into environmental health policies, it is feasible to mitigate the cumulative effects of PM<sub>2.5</sub> exposure and improve the well-being of vulnerable populations.

The effects of PM<sub>2.5</sub> on public health are mediated through multifaceted mechanisms that integrate biological, behavioral, and social dimensions. This study highlights the critical necessity for comprehensive mitigation strategies that prioritize public education, implement evidence-based environmental regulations, and deliver targeted interventions to at-risk populations. These coordinated initiatives could significantly reduce the health burden and promote enduring enhancements in global quality of life.

### 3.5. Integrated approach to mitigation

A multifaceted approach that considers both environmental and socioeconomic factors is necessary to mitigate the health impacts of PM<sub>2.5</sub>. Industrial emissions, population density, and urbanization have been demonstrated to have a substantial impact on PM<sub>2.5</sub> concentrations in various study [5,82]. Economic losses from PM<sub>2.5</sub> exposure are substantial, with short-term exposure causing higher costs than long-term exposure in some cases [83]. Furthermore, the health risks associated with PM<sub>2.5</sub> are significantly influenced by socioeconomic factors, including the rate of urbanization, energy intensity, and industrial structure, which exhibit regional differences [5]. The relationship between PM<sub>2.5</sub> concentration and related mortality has been significantly influenced by socioeconomic indicators, health risk factors, and governance variables, as demonstrated by advanced econometric techniques [84].

Strategies to reduce exposure include improving indoor air quality through advanced filtration systems and encouraging individuals to minimize outdoor activity during periods of high pollution [85–87]. Localized interventions, such as establishing community air quality monitoring networks, can empower residents with real-time data to make informed decisions [88,89]. Addressing the root causes of PM<sub>2.5</sub> pollution requires stricter enforcement of emission standards for industrial and vehicular sources, particularly in urbanizing regions like Indonesia. Green infrastructure should be incorporated into urban planning policies to promote sustainable urban development and mitigate pollution [90]. Furthermore, policies must consider equity by targeting socioeconomic disparities that exacerbate vulnerability to pollution. For example, providing healthcare subsidies for low-income populations and investing in public health infrastructure could significantly reduce the health burden of PM<sub>2.5</sub> pollution [91].

These actions align with sustainable development goals (SDGs), particularly SDG 3 (good health and well-being) and SDG 10 (reduced inequalities). In order to effectively mitigate the health consequences of PM<sub>2.5</sub> concentrations and their environmental and socioeconomic determinants, it is imperative to implement comprehensive strategies

[84–86].

This study provides a descriptive analysis of PM<sub>2.5</sub> exposure and associated health impacts, utilizing distributions to present observed patterns. This study offers significant preliminary insights into the health risks linked to PM<sub>2.5</sub> exposure, yet several limitations should be acknowledged. Increasing the sample size and the number of sampling points is essential for future research to improve the reliability and generalizability of the findings. While the limited number of sampling areas and respondents limits this study to a preliminary scope, it underscores the urgent need for targeted interventions to mitigate PM<sub>2.5</sub>-related health risks. Expanding future research is crucial for shaping comprehensive public health strategies and environmental policies.

Health complaints were self-reported, and the focus on non-carcinogenic risks does not account for the potential long-term carcinogenic effects of PM<sub>2.5</sub>. Furthermore, the study did not address synergistic effects with other pollutants, which could further elucidate health risk assessments. Future studies should investigate the synergistic effects of PM<sub>2.5</sub> and other pollutants to enhance risk assessment models and fortify policy frameworks, thus assuring more effective public health interventions. The exclusion of pregnant women from the study population represents a limitation, as this group is particularly vulnerable to PM<sub>2.5</sub>. Future studies should incorporate this demographic to provide a more comprehensive risk assessment.

This study demonstrates a robust approach to assessing health risks associated with PM<sub>2.5</sub> exposure in tropical regions. The adaptability of this method to other climatic conditions, such as subtropical areas with varying humidity, underscores its potential for broader applications. Further studies should emphasize region-specific calibrations to improve the precision and applicability of global health risk evaluations. This study highlights significant health risks associated with PM<sub>2.5</sub> but also underscores the need for methodological enhancements, including direct measurements of individual exposure patterns and body weight, to refine risk estimates and inform targeted mitigation strategies.

## 4. Conclusion

This study highlights the significant public health hazards associated with increased PM<sub>2.5</sub> concentrations in residential areas. The measured concentration of PM<sub>2.5</sub> ranges from 50.7 to 61.9 µg/m<sup>3</sup>, indicating that all sampling points exceeded the US-EPA NAAQS of 35 µg/m<sup>3</sup>, with risk hazard quotient (RQ) values consistently above 1, signifying considerable non-carcinogenic health hazards. Epidemiological statistical analysis indicates a significant correlation between PM<sub>2.5</sub> exposure and health complaints, with a p-value of less than 0.05. Residents' health complaints include cough, headache, eye irritation, breathlessness, and wheezing. These findings underscore the need for comprehensive mitigation strategies, including stringent emission regulations, sustainable urban development, and equitable public health initiatives. Furthermore, it promotes the achievement of sustainable development goals (SDGs) related to good health and well-being, as well as sustainable cities and communities.

### CRedit authorship contribution statement

**Ardianta Iwan:** Project administration, Methodology, Data curation. **Iresha Fajri Mulya:** Writing – review & editing, Supervision. **Rosinta Aulia:** Writing – original draft, Formal analysis. **Sulistiyani Arvi Tri:** Writing – review & editing, Resources, Conceptualization. **Abidin Azham Umar:** Writing – original draft, Investigation, Formal analysis, Data curation, Conceptualization. **Munawaroh Anisful Lailil:** Writing – original draft, Formal analysis.

### Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:



No If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## Acknowledgements

We would like to convey our sincere appreciation to the Department of Environmental Engineering at Universitas Islam Indonesia for their support. We express our profound gratitude to the Respondent for this study

## Data Availability

The data that has been used is confidential.

## References

- [1] J. Rentschler, N. Leonova, Global air pollution exposure and poverty, *Nat. Commun.* 14 (1) (2023) 4432.
- [2] X. Yang, Y. Wang, C. Zhao, H. Fan, Y. Yang, Y. Chi, et al., Health risk and disease burden attributable to long-term global fine-mode particles, *Chemosphere* 287 (2022) 132435.
- [3] C.H. Lim, J. Ryu, Y. Choi, S.W. Jeon, W.K. Lee, Understanding global PM<sub>2.5</sub> concentrations and their drivers in recent decades (1998–2016), *Environ. Int.* 144 (2020) 106011.
- [4] Q. Wang, M.P. Kwan, K. Zhou, J. Fan, Y. Wang, D. Zhan, The impacts of urbanization on fine particulate matter (PM<sub>2.5</sub>) concentrations: empirical evidence from 135 countries worldwide, *Environ. Pollut.* 247 (2019) 989–998.
- [5] Z. Zhang, C. Shao, Y. Guan, C. Xue, Socioeconomic factors and regional differences of PM<sub>2.5</sub> health risks in China, *J. Environ. Manag.* 251 (2019) 109564.
- [6] X. Lu, D. Yuan, Y. Chen, J.C.H. Fung, Impacts of urbanization and long-term meteorological variations on global PM<sub>2.5</sub> and its associated health burden, *Environ. Pollut.* 270 (2021) 116003.
- [7] B. Haryanto, I. Trihandini, F. Nugraha, F. Kurniasari, Indirect effects of PM<sub>2.5</sub> exposure on COVID-19 mortality in Greater Jakarta, Indonesia: an ecological study, *agh* 90 (1) (2024) 34.
- [8] W. Yu, D.C. Sulistyoningrum, D. Gasevic, R. Xu, M. Julia, I.K. Murni, et al., Long-term exposure to PM<sub>2.5</sub> and fasting plasma glucose in non-diabetic adolescents in Yogyakarta, Indonesia, *Environ. Pollut.* 257 (2020) 113423.
- [9] S. Siregar, N. Idiawati, P. Lestari, A.K. Berekute, W.C. Pan, K.P. Yu, Chemical composition, source appointment and health risk of PM<sub>2.5</sub> and PM<sub>2.5-10</sub> during forest and Peatland fires in Riau, Indonesia, *Aerosol Air Qual. Res.* 22 (9) (2022) 220015.
- [10] A.U. Abidin, F.B. Maziya, S.H. Susetyo, M. Yoneda, Y. Matsui, Exposure particulate matter (PM<sub>2.5</sub>) and health risk assessment on informal workers in landfill site, Indonesia, *Environ. Chall.* 13 (2023) 100795.
- [11] N. Cholianawati, T. Sinatra, G.A. Nugroho, D. Agustian Permadi, A.-H. Indrawati, et al., Diurnal and daily variations of PM<sub>2.5</sub> and its multiple-wavelet coherence with meteorological variables in Indonesia, *Aerosol Air Qual. Res.* 24 (3) (2024) 230158.
- [12] J. Lelieveld, A. Pozzer, U. Pöschl, M. Fnais, A. Haines, T. Münzel, Loss of life expectancy from air pollution compared to other risk factors: a worldwide perspective, *Cardiovasc. Res.* 116 (11) (2020) 1910–1917.
- [13] M. Ru, M. Brauer, J. Lamarque, D. Shindell, Exploration of the global burden of dementia attributable to PM<sub>2.5</sub>: what do we know based on current evidence? *GeoHealth* 5 (5) (2021) e2020GH000356.
- [14] S.C. Anenberg, P. Achakulwisut, M. Brauer, D. Moran, J.S. Apte, D.K. Henze, Particulate matter-attributable mortality and relationships with carbon dioxide in 250 urban areas worldwide, *Sci. Rep.* 9 (1) (2019) 11552, 9.
- [15] Y. Song, B. Huang, Q. He, B. Chen, J. Wei, R. Mahmood, Dynamic assessment of PM<sub>2.5</sub> exposure and health risk using remote sensing and geo-spatial big data, *Environ. Pollut.* 253 (2019) 288–296.
- [16] X. Xu, K. Shi, Z. Huang, J. Shen, What Factors dominate the change of PM<sub>2.5</sub> in the world from 2000 to 2019? A study from multi-source data, *IJERPH* 20 (3) (2023) 2282.
- [17] S. Chen, D. Li, X. Wu, L. Chen, B. Zhang, Y. Tan, et al., Application of cell-based biological bioassays for health risk assessment of PM<sub>2.5</sub> exposure in three megacities, China, *Environ. Int.* 139 (2020) 105703.
- [18] V. Kazakos, Z. Luo, I. Ewart, Quantifying the health burden misclassification from the use of different PM<sub>2.5</sub> exposure tier models: a case study of London, *IJERPH* 17 (3) (2020) 1099.
- [19] C.P. Kuo, J.S. Fu, P.C. Wu, T.J. Cheng, T.Y. Chiu, C.S. Huang, et al., Quantifying spatial heterogeneity of vulnerability to short-term PM<sub>2.5</sub> exposure with data fusion framework, *Environ. Pollut.* 285 (2021) 117266.
- [20] J. Xie, L. Jin, J. Cui, X. Luo, J. Li, G. Zhang, et al., Health risk-oriented source apportionment of PM<sub>2.5</sub>-associated trace metals, *Environ. Pollut.* 262 (2020) 114655.
- [21] A. Jbaily, X. Zhou, J. Liu, T.H. Lee, L. Kamareddine, S. Verguet, et al., Air pollution exposure disparities across US population and income groups, *Nature* 601 (7892) (2022) 228–233.
- [22] S. Murtyas, N.T. Toosty, A. Hagishima, N.H. Kusumaningdyah, Relation between occupants' health problems, demographic and indoor environment subjective evaluations: a cross-sectional questionnaire survey study in Java Island, Indonesia, Atiqul Haq SM, Editor. *PLoS ONE* 16 (7) (2021) e0254460.
- [23] N. Cooper, D. Green, L.D. Knibbs, Inequalities in exposure to the air pollutants PM<sub>2.5</sub> and NO<sub>2</sub> in Australia, *Environ. Res. Lett.* 14 (11) (2019) 115005.
- [24] J. Huang, X. Li, Y. Zhang, S. Zhai, W. Wang, T. Zhang, et al., Socio-demographic characteristics and inequality in exposure to PM<sub>2.5</sub>: a case study in the Sichuan Basin, China, *Environ. Pollut.* 316 (2023) 120630.
- [25] A. Horton, S.J. Jones, H. Brunt, Air pollution and public health vulnerabilities, susceptibilities and inequalities in Wales, UK, *J. Public Health* 45 (2) (2023) 432–441.
- [26] M.A.H. Suryadhi, P.A.R. Suryadhi, K. Abudureyimu, I.M.W. Ruma, A.S. Calliope, D. N. Wirawan, et al., Exposure to particulate matter (PM<sub>2.5</sub>) and prevalence of diabetes mellitus in Indonesia, *Environ. Int.* 140 (2020) 105603.
- [27] X. Gong, S. Wang, X. Wang, S. Zhong, J. Yuan, Y. Zhong, et al., Long-term exposure to air pollution and risk of insulin resistance: a systematic review and meta-analysis, *Ecotoxicol. Environ. Saf.* 271 (2024) 115909.
- [28] L. Zhao, J. Fang, S. Tang, F. Deng, X. Liu, Y. Shen, et al., PM<sub>2.5</sub> and serum metabolome and insulin resistance, potential mediation by the gut microbiome: a population-based panel study of older adults in China, *Environ. Health Perspect.* 130 (2) (2022) 027007.
- [29] X. Duan, X. Zhang, J. Chen, M. Xiao, W. Zhao, S. Liu, et al., Association of PM<sub>2.5</sub> with insulin resistance signaling pathways on a microfluidic liver–kidney microphysiological system (LK-MPS) device, *Anal. Chem.* 93 (28) (2021) 9835–9844.
- [30] S. Peng, J. Sun, F. Liu, Z. Li, C. Wu, H. Xiang, The effect of short-term fine particulate matter exposure on glucose homeostasis: a panel study in healthy adults, *Atmos. Environ.* 268 (2022) 118769.
- [31] M. Zhan, Z. Li, X. Li, B. Tao, Q. Zhang, J. Wang, Effect of short-term ambient PM<sub>2.5</sub> exposure on fasting blood glucose levels: a longitudinal study among 47,471 people in eastern China, *Environ. Pollut.* 290 (2021) 117983.
- [32] J. Zhong, G. Zhao, S. Edwards, J. Tran, S. Rajagopalan, X. Rao, Particulate air pollution exaggerates diet-induced insulin resistance through NLRP3 inflammasome in mice, *Environ. Pollut.* 328 (2023) 121603.
- [33] J. Qin, W. Xia, G. Liang, S. Xu, X. Zhao, D. Wang, et al., Association of fine particulate matter with glucose and lipid metabolism: a longitudinal study in young adults, *Occup. Environ. Med.* 78 (6) (2021) 448–453.
- [34] Government of the Republic of Indonesia, Appendix VII of government regulation number 22 of 2021 concerning the implementation of environmental protection and management: ambient air quality standards, Gov. Repub. Indones. (2021). Available from: (<https://kku.world/5d5xle>).
- [35] United Nations. The Sustainable Development Goals Report. [cited 2024 Nov 19]. Available from: (<https://unstats.un.org/sdgs/report/2024/>).
- [36] S. Zhai, D.J. Jacob, X. Wang, L. Shen, K. Li, Y. Zhang, et al., Fine particulate matter (PM<sub>2.5</sub>) trends in China, 2013–2018: separating contributions from anthropogenic emissions and meteorology, *Atmos. Chem. Phys.* 19 (16) (2019) 11031–11041. Aug 29.
- [37] S.J. Lu, D. Wang, Z. Wang, B. Li, Z.R. Peng, X.B. Li, et al., Investigating the role of meteorological factors in the vertical variation in PM<sub>2.5</sub> by unmanned aerial vehicle measurement, *Aerosol Air Qual. Res.* 19 (7) (2019) 1493–1507.
- [38] S. Basith, B. Manavalan, T.H. Shin, C.B. Park, W.S. Lee, J. Kim, et al., The impact of fine particulate matter 2.5 on the cardiovascular system: a review of the invisible killer, *Nanomaterials* 12 (15) (2022) 2656.
- [39] T. Sukuman, K. Ueda, S. Sujaritpong, H. Praekunatham, K. Punnasiri, T. Wimuktayon, et al., Health impacts from PM<sub>2.5</sub> exposure using environmental epidemiology and health risk assessment: a review, *Appl. Environ. Res.* (2023) 1–14.
- [40] S.J. Mbazima, Health risk assessment of particulate matter 2.5 in an academic metallurgy workshop, *Indoor Air* 32 (9) (2022). Available from: (<https://onlinelibrary.wiley.com/doi/10.1111/ina.13111>).
- [41] D. Roy, G. Singh, Y.C. Seo, Carcinogenic and non-carcinogenic risks from PM<sub>10</sub>- and PM<sub>2.5</sub>-Bound metals in a critically polluted coal mining area, *Atmos. Pollut. Res.* 10 (6) (2019) 1964–1975.
- [42] S. Shojaei Barjoei, H. Azimzadeh, A. Mosleh Arani, M. Kuchakzadeh Occupational monitoring and health risks assessment of respiratory exposure to dust in an industrial unit of producing China Clay. TKJ [Internet]. 2020Apr 4 [cited 2024 Nov 24]; Available from: (<https://publish.kne-publishing.com/index.php/TKJ/article/view/2584>).
- [43] Schraufnagel DE, J.R. Balmes, C.T. Cowl, S. De Matteis, S.H. Jung, K. Mortimer, et al., Air pollution and noncommunicable diseases, *Chest* 155 (2) (2019) 409–416.
- [44] Y. Liu, Q. Yuan, X. Zhang, Z. Chen, X. Jia, M. Wang, et al., Fine particulate matter (PM<sub>2.5</sub>) induces inhibitory memory alveolar macrophages through the AhR/IL-33 pathway, *Cell. Immunol.* 386 (2023) 104694.
- [45] Zaręba, K. Piszczatowska, K. Dżaman, K. Soroczynska, P. Motamed, M. Szczepański, et al., The Relationship between fine particle matter (PM<sub>2.5</sub>) exposure and upper respiratory tract diseases, *JPM* 14 (1) (2024) 98.
- [46] B. Sun, J. Song, Y. Wang, J. Jiang, Z. An, J. Li, et al., Associations of short-term PM<sub>2.5</sub> exposures with nasal oxidative stress, inflammation and lung function impairment and modification by GSTT1-null genotype: a panel study of the retired adults, *Environ. Pollut.* 285 (2021) 117215.
- [47] C. Guo, S. Lv, Y. Liu, Y. Li, Biomarkers for the adverse effects on respiratory system health associated with atmospheric particulate matter exposure, *J. Hazard. Mater.* 421 (2022) 126760.

- [48] R.W. Allen, P. Barn, Individual- and household-level interventions to reduce air pollution exposures and health risks: a review of the recent literature, *Curr. Environ. Health Rpt.* 7 (4) (2020) 424–440.
- [49] J.D. Newman, D.L. Bhatt, S. Rajagopalan, J.R. Balmes, M. Brauer, P.N. Breyse, et al., Cardiopulmonary impact of particulate air pollution in high-risk populations, *J. Am. Coll. Cardiol.* 76 (24) (2020) 2878–2894.
- [50] L. Conibear, C.L. Reddington, B.J. Silver, C. Knote, S.R. Arnold, D.V. Spracklen, Regional policies targeting residential solid fuel and agricultural emissions can improve air quality and public health in the Greater Bay Area and Across China, *GeoHealth* 5 (4) (2021) e2020GH000341.
- [51] E. Consensus Task Force, X. Shi, G. Duan, China CDC Key Laboratory of Environment and Population Health, National Institute of Environmental Health, Chinese Center for Disease Control and Prevention, Beijing, China, School of Public Health, Zhengzhou University, Zhengzhou, Henan, China. Recommendations of Controlling and Preventing Acute Health Risks of Fine Particulate Matter Pollution — China, 2021, *China CDC Wkly.* 4 (16) (2022) 329–341.
- [52] X. Morelli, S. Gabet, C. Rieux, H. Bouscasce, S. Mathy, R. Slama, Which decreases in air pollution should be targeted to bring health and economic benefits and improve environmental justice? *Environ. Int.* 129 (2019) 538–550.
- [53] F.J. Kelly, J.C. Fussell, Global nature of airborne particle toxicity and health effects: a focus on megacities, wildfires, dust storms and residential biomass burning, *Toxicol. Res.* 9 (4) (2020) 331–345.
- [54] P.A. Schulte, A. Bhattacharya, C.R. Butler, H.K. Chun, B. Jacklitsch, T. Jacobs, et al., Advancing the framework for considering the effects of climate change on worker safety and health, *J. Occup. Environ. Hyg.* 13 (11) (2016) 847–865.
- [55] B. Zhang, B. Wu, J. Liu, PM<sub>2.5</sub> pollution-related health effects and willingness to pay for improved air quality: evidence from China's prefecture-level cities, *J. Clean. Prod.* 273 (2020) 122876.
- [56] J. Lin, H. Zheng, P. Xia, X. Cheng, W. Wu, Y. Li, et al., Long-term ambient PM<sub>2.5</sub> exposure associated with cardiovascular risk factors in Chinese less educated population, *BMC Public Health* 21 (1) (2021) 2241.
- [57] S.E. Guo, M.C. Chi, S.L. Hwang, C.M. Lin, Y.C. Lin, Effects of particulate matter education on self-care knowledge regarding air pollution, symptom changes, and indoor air quality among patients with chronic obstructive pulmonary disease, *IJERPH* 17 (11) (2020) 4103.
- [58] S.K. Woo, B. LePage, Y.T. Chiang, W.T. Fang, Predicting the protective behavioral intentions for parents with young children that possess different levels of education in Hong Kong using the theory of planned behavior for air polluted with PM<sub>2.5</sub>, *BMC Public Health* 22 (1) (2022) 761.
- [59] B. Zou, S. Li, Y. Lin, B. Wang, S. Cao, X. Zhao, et al., Efforts in reducing air pollution exposure risk in China: state versus individuals, *Environ. Int.* 137 (2020) 105504.
- [60] J. Zhang, X. Wang, M. Yan, A. Shan, C. Wang, X. Yang, et al., Sex Differences in cardiovascular risk associated with long-term PM<sub>2.5</sub> exposure: a systematic review and meta-analysis of cohort studies, *Front Public Health* 10 (2022) 802167.
- [61] H. Kim, J. Noh, Y. Noh, S.S. Oh, S.B. Koh, C. Kim, Gender difference in the effects of outdoor air pollution on cognitive function among elderly in Korea, *Front. Public Health* 7 (2019) 375.
- [62] R. Li, Q. Sun, S.M. Lam, R. Chen, J. Zhu, W. Gu, et al., Sex-dependent effects of ambient PM<sub>2.5</sub> pollution on insulin sensitivity and hepatic lipid metabolism in mice, *Part Fibre Toxicol.* 17 (1) (2020) 14.
- [63] U. Luderer, J. Lim, L. Ortiz, J.D. Nguyen, J.H. Shin, B.D. Allen, et al., Exposure to environmentally relevant concentrations of ambient fine particulate matter (PM<sub>2.5</sub>) depletes the ovarian follicle reserve and causes sex-dependent cardiovascular changes in apolipoprotein E null mice, *Part Fibre Toxicol.* 19 (1) (2022) 5.
- [64] X. Liu, Y. Xiao, Q. Zhu, Y. Cui, H. Hao, M. Wang, et al., Circulating endothelial progenitor cells are preserved in female mice exposed to ambient fine particulate matter independent of estrogen, *LJMS* 22 (13) (2021) 7200.
- [65] H. Yue, X. Ji, T. Ku, G. Li, N. Sang, Sex difference in bronchopulmonary dysplasia of offspring in response to maternal PM<sub>2.5</sub> exposure, *J. Hazard. Mater.* 389 (2020) 122033.
- [66] S. Heo, J.Y. Son, C.C. Lim, K.C. Fong, H.M. Choi, R.U. Hernandez-Ramirez, et al., Effect modification by sex for associations of fine particulate matter (PM<sub>2.5</sub>) with cardiovascular mortality, hospitalization, and emergency room visits: systematic review and meta-analysis, *Environ. Res. Lett.* 17 (5) (2022) 053006.
- [67] S. Maji, S. Ahmed, M. Kaur-Sidhu, S. Mor, K. Ravindra, Health risks of major air pollutants, their drivers and mitigation strategies: a review, *Air Soil Water Res.* 16 (2023) 11786221231154659.
- [68] J. Hu, F. Wang, H. Shen, The influence of PM<sub>2.5</sub> exposure duration and concentration on outpatient visits of urban hospital in a typical heavy industrial city, *Environ. Sci. Pollut. Res.* 30 (54) (2023) 115098–115110.
- [69] C. Liu, K.H. Chan, J. Lv, H. Lam, K. Newell, X. Meng, et al., Long-term exposure to ambient fine particulate matter and incidence of major cardiovascular diseases: a prospective study of 0.5 million adults in China, *Environ. Sci. Technol.* 56 (18) (2022) 13200–13211.
- [70] T. Amnuaylojaroen, N. Parasin, Pathogenesis of PM<sub>2.5</sub>-related disorders in different age groups: children, adults, and the elderly, *Epigenomes* 8 (2) (2024) 13.
- [71] K. Ebisu, B. Malig, S. Hasheminassab, C. Sioutas, Age-specific seasonal associations between acute exposure to PM<sub>2.5</sub> sources and cardiorespiratory hospital admissions in California, *Atmos. Environ.* 218 (2019) 117029.
- [72] H.M. Strosnider, H.H. Chang, L.A. Darrow, Y. Liu, A. Vaidyanathan, M. J. Strickland, Age-specific associations of ozone and fine particulate matter with respiratory emergency department visits in the United States, *Am. J. Respir. Crit. Care Med.* 199 (7) (2019) 882–890.
- [73] R. Yan, T. Ku, H. Yue, G. Li, N. Sang, PM<sub>2.5</sub> exposure induces age-dependent hepatic lipid metabolism disorder in female mice, *J. Environ. Sci.* 89 (2020) 227–237.
- [74] Y. Zhang, X. Ji, T. Ku, B. Li, G. Li, N. Sang, Ambient fine particulate matter exposure induces cardiac functional injury and metabolite alterations in middle-aged female mice, *Environ. Pollut.* 248 (2019) 121–132.
- [75] S. DeFlorio-Barker, J. Crooks, J. Reyes, A.G. Rappold, Cardiopulmonary effects of fine particulate matter exposure among older adults, during wildfire and non-wildfire periods, in the United States 2008–2010, *Environ. Health Perspect.* 127 (3) (2019) 037006.
- [76] L. Wang, L.Y. Wei, R. Ding, Y. Feng, D. Li, C. Li, et al., Predisposition to Alzheimer's and age-related brain pathologies by PM<sub>2.5</sub> exposure: perspective on the roles of oxidative stress and TRPM2 channel, *Front Physiol.* 11 (2020) 155.
- [77] L.K. Lau, K.B. Abdul Jabbar, W.J.B. Pang, K.K. Chen, W.T. Seah, J. Ullal Mallya, et al., Physiological and cognitive determinants of gait variability of asian population: the Yishun Study, *Gerontology* 69 (3) (2023) 301–311.
- [78] R. Koffer, S. Lee, S. Charles, Intraindividual variability across adulthood: diversity, variety, and adherence as key indicators of daily life, *Innov. Aging* 7 (ement\_1) (2023) 277–278.
- [79] G. Kuchel, Clinical considerations, *Innov. Aging* 4 (2020) 855, 855.
- [80] P.A. Dimitriu, B. Iker, K. Malik, H. Leung, W.W. Mohn, G.G. Hillebrand, New insights into the intrinsic and extrinsic factors that shape the human skin microbiome, in: D.S. Guttman (Ed.), *mBio*, 10, 2019.
- [81] D.A. Wallace, D.A. Johnson, S. Redline, T. Sofer, J. Kossowsky, Rest-activity rhythms across the lifespan: cross-sectional findings from the US representative National Health and Nutrition Examination Survey, *SLEEP* 46 (11) (2023) zsad220.
- [82] Y. Wang, C. Liu, Q. Wang, Q. Qin, H. Ren, J. Cao, Impacts of natural and socioeconomic factors on PM<sub>2.5</sub> from 2014 to 2017, *J. Environ. Manag.* 284 (2021) 112071.
- [83] L.T. Bui, N.H.T. Nguyen, P.H. Nguyen, Chronic and acute health effects of PM<sub>2.5</sub> exposure and the basis of pollution control targets, *Environ. Sci. Pollut. Res.* 30 (33) (2023) 79937–79959.
- [84] D. Lončar, N.B. Tyack, V. Krstić, J. Paunković, Methods for assessing the impact of PM<sub>2.5</sub> concentration on mortality while controlling for socio-economic factors, *Heliyon* 8 (10) (2022) e10729.
- [85] C. Carlsten, S. Salvi, G.W.K. Wong, K.F. Chung, Personal strategies to minimise effects of air pollution on respiratory health: advice for providers, patients and the public, *Eur. Respir. J.* 55 (6) (2020) 1902056.
- [86] R.J. Laumbach, K.R. Cromar, G. Adamkiewicz, C. Carlsten, D. Charpin, W.R. Chan, et al., Personal interventions for reducing exposure and risk for outdoor air pollution: an official American Thoracic Society Workshop Report, *Ann. ATS* 18 (9) (2021) 1435–1443.
- [87] M.M. Maestas, R.D. Brook, R.A. Ziemba, F. Li, R.C. Crane, Z.M. Klaver, et al., Reduction of personal PM<sub>2.5</sub> exposure via indoor air filtration systems in Detroit: an intervention study, *J. Expo. Sci. Environ. Epidemiol.* 29 (4) (2019) 484–490.
- [88] P.B. English, L. Olmedo, E. Bejarano, H. Lugo, E. Murillo, E. Seto, et al., The Imperial county community air monitoring network: a model for community-based environmental monitoring for public health action, *Environ. Health Perspect.* 125 (7) (2017) 074501.
- [89] Hsu Y.C., Dille P., Cross J., Dias B., Sargent R., Nourbakhsh I. Community-Empowered Air Quality Monitoring System. In: *Proceedings of the 2017 CHI Conference on Human Factors in Computing Systems* [Internet]. Denver Colorado USA: ACM, 2017 [cited 2024 Dec 2]. p. 1607–1619. Available from: (<https://dl.acm.org/doi/10.1145/3025453.3025853>).
- [90] Abraham Adesina Abayomi, Chukwudi Okwundu Azubuike, Queen Sikhakhane Nwokediegwu Zamathula, Towards sustainable urban development: conceptualizing green infrastructure and its impact on urban planning, *Int J. Appl. Res. Soc. Sci.* 6 (7) (2024) 1274–1296.
- [91] J.M. Samet, Urban air pollution, health, and equity, *J. Epidemiol. Commun. Health* 58 (1) (2004) 3–5.