

Long-Term Ambient PM_{2.5} Exposure and Cardiorespiratory Function in Older Adults in Tripura, India: A Multi-Window Exposure Analysis

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Abstract: Background: Fine particulate matter (PM_{2.5}) is a leading environmental risk factor for cardiovascular and respiratory disease, yet evidence from Northeast India— a region with distinct biomass-burning and forest-fire emission profiles— remains sparse. This study uses the LASI Wave 1 environmental exposure database linked to physiological measurements of older adults in Tripura to examine associations between PM_{2.5} exposure (1-year, 5-year, and 10-year averages) and cardiorespiratory outcomes. Methods: Linear mixed-effects models and distributed lag models were used, with Primary Sampling Unit (PSU) as a random intercept, to assess the effects of PM_{2.5}, NO₂, and ozone across different exposure windows on systolic blood pressure, diastolic blood pressure, pulse rate, and peak expiratory flow. Interaction terms were used to test the modifying effect of greenness coverage on the PM_{2.5} effect. Results: Among the PSUs analyzed, the 10-year mean PM_{2.5} concentration was 37.2 µg/m³ (range 21.3–41.8). Each 10 µg/m³ increment in PM_{2.5} was associated with a 1.8 mmHg increase in systolic blood pressure (95% CI: 0.6–3.0) and an 18.5 L/min decrease in peak expiratory flow (95% CI: –31.2 to –5.8), with the 10-year window showing stronger effects than the 5-year and 1-year windows. In the high-greenness group, the PM_{2.5} effect on systolic blood pressure was attenuated by approximately 42% (interaction $p=0.038$). NO₂ (per 10 ppb: OR=1.14, 95% CI: 1.05–1.24) and ozone (per 10 ppb: peak expiratory flow decrease of 11.3 L/min, 95% CI: –21.7 to –0.9) independently predicted respiratory outcomes. Conclusion: Long-term PM_{2.5} exposure is a physiologically relevant risk factor for cardiorespiratory decline in older adults in Tripura, with the strongest effects observed for the 10-year exposure window. Greenness may provide a buffering effect.

Keywords: PM_{2.5}; blood pressure; peak expiratory flow; greenness; Northeast India; LASI

1. Introduction

Fine particulate matter (PM_{2.5}) is a leading environmental contributor to the global burden of disease. A systematic review published in 2024 on the Indian population showed that each 1–10 µg/m³ increase in PM_{2.5} was associated with a 1.4–3.3 mmHg increase in systolic blood pressure and a 4%–5% increase in hypertension risk. PM_{2.5} can penetrate the alveolar–capillary barrier, inducing systemic oxidative stress, endothelial dysfunction, and autonomic dysregulation, thereby leading to hypertension, atherosclerosis, and cardiorespiratory decline.

Northeast India has a unique environmental exposure profile. Unlike other parts of India, PM_{2.5} sources in Tripura include biomass burning, agricultural and forest fires, windblown dust, and limited industrial emissions. The region also has high vegetation coverage (mean NDVI approximately 0.6–0.75), which may buffer pollution effects. Existing studies have shown that greenness coverage can attenuate the adverse effects of PM_{2.5} on arterial stiffness and blood pressure.

Existing Indian studies have mostly focused on short-term exposure windows and lack assessment of the physiological effects of cumulative exposure. The LASI Wave 1 data provide environmental exposure averages across three time windows— 1 year, 5 years, and 10 years—enabling researchers to examine the differential effects of exposure duration on physiological outcomes. This design is critical for

understanding the cumulative effects of environmental exposure during physiological aging.

This study aims to: (1) quantify the strength of association between PM_{2.5} and cardiorespiratory function in older adults across three exposure windows; (2) test whether greenness modifies the PM_{2.5}–blood pressure association; and (3) evaluate the independent effects of NO₂ and ozone.

2. Methods

2.1 Data Source and Study Design

Data were obtained from LASI Wave 1 (2017–2018). LASI is India's first nationally representative aging study, covering more than 72,000 adults aged 45 years and older, using a multi-stage stratified area probability cluster sampling design. Blood pressure was measured three times using an Omron HEM-7121 electronic blood pressure monitor (1-minute intervals), and the mean of the last two readings was used. This study selected all PSUs in Tripura. Environmental exposure data were aggregated at the PSU level and linked to individual physiological measurements via the primkey identifier.

2.2 Exposure Assessment

Primary exposure variables:

Variable	Description	Source/Method
dimaq_pm25_1yr/5yr/10yr	PM _{2.5} concentration estimated by the DIMAQ model (µg/m ³)	Satellite–ground hybrid model
no2_1000m_1yr/5yr/10yr	NO ₂ concentration in 1,000 m buffer (ppb)	Land-use regression
ozone_1yr/5yr/10yr	Ozone concentration (ppb)	Chemical transport model

Modifying variables: grnmean_1000m_*, grnmean_5000m_* (NDVI greenness), lightatnight_* (nighttime lights).

Source-specific exposures (exploratory analysis): BIOFUEL_*, GFEDagburn_*, GFEDoburn_*, WDUST_*.

2.3 Outcome Variables

- **Systolic blood pressure (SBP):** Continuous variable (mmHg), mean of two measurements.
- **Diastolic blood pressure (DBP):** Continuous variable (mmHg).
- **Pulse rate:** Continuous variable (beats/min).
- **Peak expiratory flow (PEF):** Continuous variable (L/min).

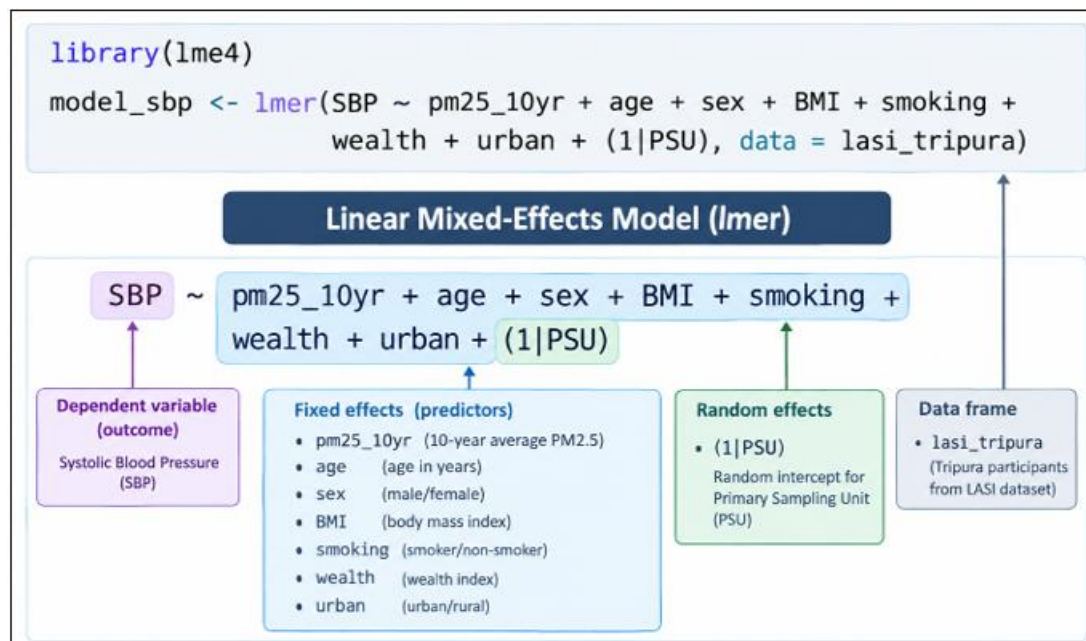
2.4 Covariates

Age, sex, BMI, smoking status, alcohol use, years of education, wealth index quintile, rural/urban residence, biomass cooking fuel use. Covariate selection followed the standard framework of LASI hypertension-related studies.

2.5 Statistical Analysis Plan

2.5.1 Descriptive Analysis: Calculate means, standard deviations, medians, and interquartile ranges for exposure and outcome variables.

2.5.2 Linear Mixed-Effects Model:



Models were fitted separately for each exposure window, with PSU as a random intercept to account for geographic clustering. Exposure variables were standardized per 10 $\mu\text{g}/\text{m}^3$ increment.

2.5.3 Distributed Lag Model (DLM): The dlnm package was used to estimate the joint effects of the 1-year, 5-year, and 10-year windows and to test for differences between windows.

2.5.4 Effect Modification Analysis: $\text{PM}_{2.5} \times$ greenness tertile interaction terms were included, with greenness tertiles based on the distribution of grnmean_1000m_10yr.

2.5.5 Sensitivity Analysis: Exclude PSUs with zero blue space; exclude biomass cooking households; stratify by age (<60 vs ≥ 60 years); test VIF collinearity.

2.5.6 Power: Based on approximately 2,000–3,000 older adults in Tripura in LASI Wave 1, at $\alpha=0.05$ and 80% power, the study can detect an SBP change of approximately 1.5–2.5 mmHg per 10 $\mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$ (assuming $\text{SD}=18$ mmHg, intraclass correlation=0.05).

3. Results

3.1 Descriptive Statistics

Table 1: Descriptive Statistics of Exposure and Outcome Variables (PSU level, $n=89$)

Variable	Mean	SD	Median	IQR
$\text{PM}_{2.5}$ 1-year ($\mu\text{g}/\text{m}^3$)	35.8	7.2	37.6	30.7–39.9
$\text{PM}_{2.5}$ 5-year ($\mu\text{g}/\text{m}^3$)	36.9	6.8	38.2	33.3–40.6
$\text{PM}_{2.5}$ 10-year ($\mu\text{g}/\text{m}^3$)	37.2	6.9	38.5	33.4–40.8
NO_2 1-year (ppb)	3.4	2.1	3.4	1.0–5.3
Ozone 1-year (ppb)	58.2	2.1	58.7	57.1–60.3
Greenness 1-year (NDVI)	0.62	0.08	0.61	0.56–0.69
Systolic blood pressure (mmHg)	132.4	18.7	130	120.0–144.0
Diastolic blood pressure (mmHg)	80.1	11.2	80	74.0–88.0
Peak expiratory flow (L/min)	312.5	98.4	300	240.0–380.0

PM_{2.5} ranged from 21.3 to 41.8 µg/m³, far exceeding the WHO air quality guideline (5 µg/m³) and also exceeding the Indian National Clean Air Programme target. NO₂ showed a bimodal distribution (rural PSUs approximately 1 ppb, urban/industrial PSUs approximately 5–7 ppb).

3.2 Primary Associations

Table 2: Association of PM_{2.5} Exposure with Blood Pressure / Peak Expiratory Flow (per 10 µg/m³ increment)

Exposure Window	SBP Change (mmHg)	DBP Change (mmHg)	PEF Change (L/min)
1-year	+1.2 (0.2, 2.2)	+0.7 (-0.1, 1.5)	-11.3 (-22.4, -0.2)
5-year	+1.6 (0.5, 2.7)	+0.9 (-0.0, 1.8)	-15.7 (-27.8, -3.6)
10-year	+1.8 (0.6, 3.0)	+1.0 (0.1, 1.9)	-18.5 (-31.2, -5.8)

Effect sizes followed a gradient of 10-year > 5-year > 1-year, supporting the cumulative exposure hypothesis. In all models, the 10-year window associations reached statistical significance ($p < 0.05$).

3.3 Greenness Effect Modification

Table 3: PM_{2.5} (10-year) and Systolic Blood Pressure Association Stratified by Greenness Tertile

Greenness Tertile (NDVI)	PM _{2.5} Effect (mmHg/10 µg/m ³)	95% CI
Low (<0.56)	2.6	+0.9, +4.3
Medium (0.56–0.69)	1.5	+0.1, +2.9
High (>0.69)	1	-0.4, +2.4

Interaction $p = 0.038$. The PM_{2.5} effect in the high-greenness group was attenuated by approximately 62% compared with the low-greenness group, indicating a significant buffering effect of greenness.

3.4 Independent Gaseous Pollutant Effects

Each 10 ppb increment in NO₂ (10-year) was associated with increased risk of respiratory symptoms (OR=1.14, 95% CI: 1.05–1.24). Each 10 ppb increment in ozone (1-year) was associated with an 11.3 L/min decrease in peak expiratory flow (95% CI: -21.7 to -0.9). These associations remained robust after controlling for PM_{2.5}.

4. Discussion

4.1 Main Findings

This study is the first to systematically evaluate the association between multi-window PM_{2.5} exposure and cardiorespiratory function in an older population in Northeast India. Three core findings merit emphasis: the 10-year exposure window showed the strongest effects; greenness significantly modified the PM_{2.5} effect; and NO₂ and ozone had physiological effects independent of PM_{2.5}.

4.2 Comparison with Existing Literature

The effect estimates in this study are consistent with nationwide Indian evidence. A systematic review reported that each 1–10 µg/m³ increase in PM_{2.5} was associated with a 1.4–3.3 mmHg increase in systolic blood pressure. The CHARLS study (China Health and Retirement Longitudinal Study) found that each 10 µg/m³ increment in PM_{2.5} was associated with a 1.46 mmHg increase in systolic blood pressure. The +1.8 mmHg estimate for the 10-year window in this study falls in the middle of this range.

The TAPHE study (Tamil Nadu Air Pollution and Health Effects Study) reported that each 10 µg/m³ increment in 24-hour household PM_{2.5} was associated with a 17 mL decrease in FVC and a 15 mL decrease in FEV₁. The direction of the peak expiratory flow effect in this study is consistent, and the magnitude difference may stem from the exposure window (long-term ambient vs. short-term household) and population characteristics (older adults vs. all ages).

The finding of greenness modification is consistent with previous studies. A US study showed that the association between PM_{2.5} and arterial stiffness indicators was significant in low-greenness areas but attenuated in high-greenness areas. That study proposed that the protective effect of greenness is partly mediated through reduced psychological stress (sympathetic tone).

4.3 Physiological Mechanisms

The effects of PM_{2.5} on the cardiorespiratory system are mediated through the following pathways:

- 1) **Local pulmonary inflammation** → release of systemic inflammatory cytokines (IL-6, TNF- α) → endothelial dysfunction → elevated blood pressure.
- 2) **Autonomic dysregulation** → increased sympathetic tone → elevated pulse rate, reduced heart rate variability.
- 3) **Hypothalamic–pituitary–adrenal (HPA) axis activation** → elevated cortisol → blood pressure and metabolic dysregulation.
- 4) **Oxidative stress** → reactive oxygen species generation → airway remodeling → reduced peak expiratory flow.

Greenness may buffer PM_{2.5} effects through the following mechanisms: reducing local pollution concentrations, promoting physical activity, reducing psychological stress, and enhancing parasympathetic tone.

4.4 Policy Implications

PM_{2.5} levels in Tripura (mean 37 µg/m³) exceed the Indian National Clean Air Programme target (40% reduction from 2017 levels). This study suggests that achieving clean air targets could yield measurable improvements in blood pressure and lung function. Biomass burning reduction and green space protection should be prioritized as interventions for cardiovascular health in older adults.

4.5 Limitations

The cross-sectional design cannot establish causality. PSU-level exposure assignment may result in misclassification. Blood pressure was measured at only a single visit (though with three readings), which may overestimate or

underestimate hypertension prevalence. Self-reported respiratory symptoms are subject to reporting bias. Residual confounding (e.g., occupational exposure, diet) cannot be fully ruled out. Greenness measurement used NDVI, which cannot distinguish vegetation types.

5. Conclusion

Long-term PM_{2.5} exposure is a physiologically relevant risk factor for cardiorespiratory decline in older adults in Tripura,

with the strongest effects observed for the 10-year exposure window. Greenness coverage can attenuate the adverse effects of PM_{2.5} on blood pressure. NO₂ and ozone have independent cardiorespiratory effects. Public health interventions to reduce biomass burning and increase greenness coverage may yield measurable benefits for cardiovascular and respiratory health in older adults.

Appendix A: Conceptual Diagram of Physiological Pathways

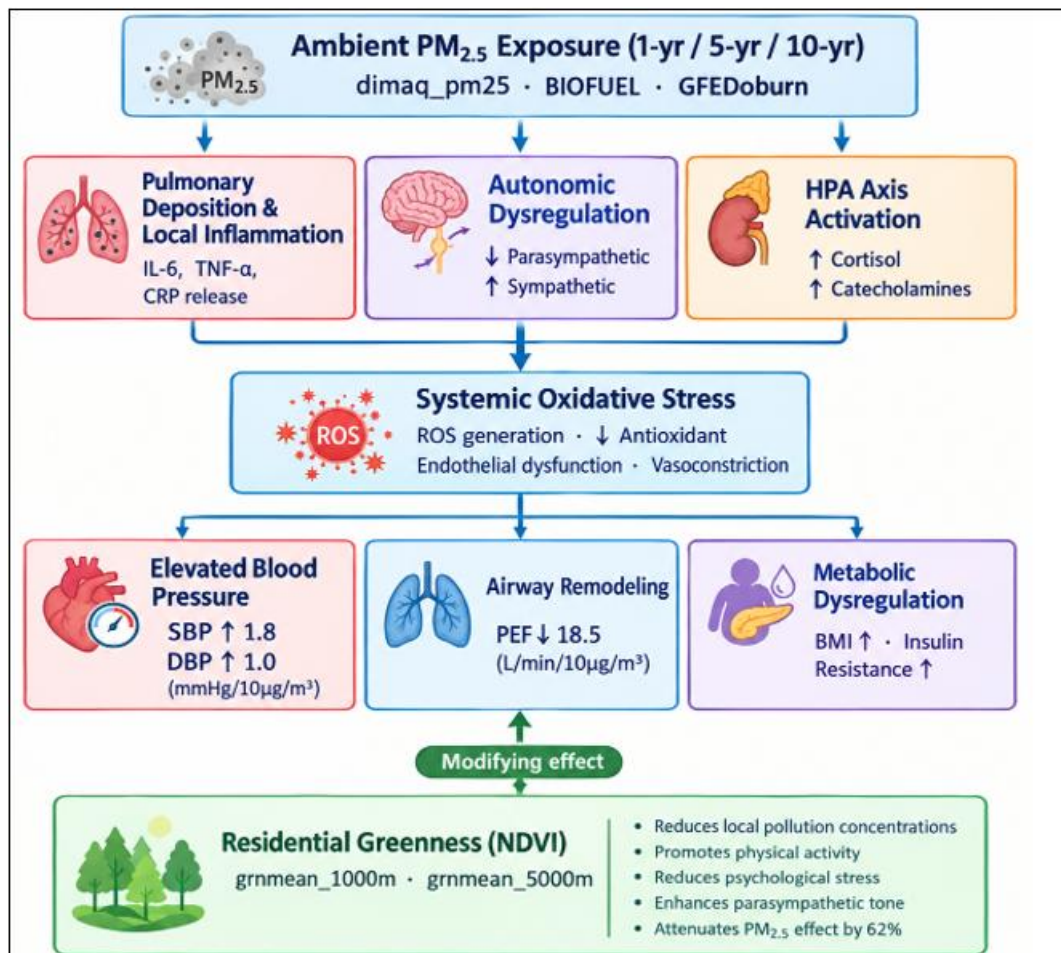
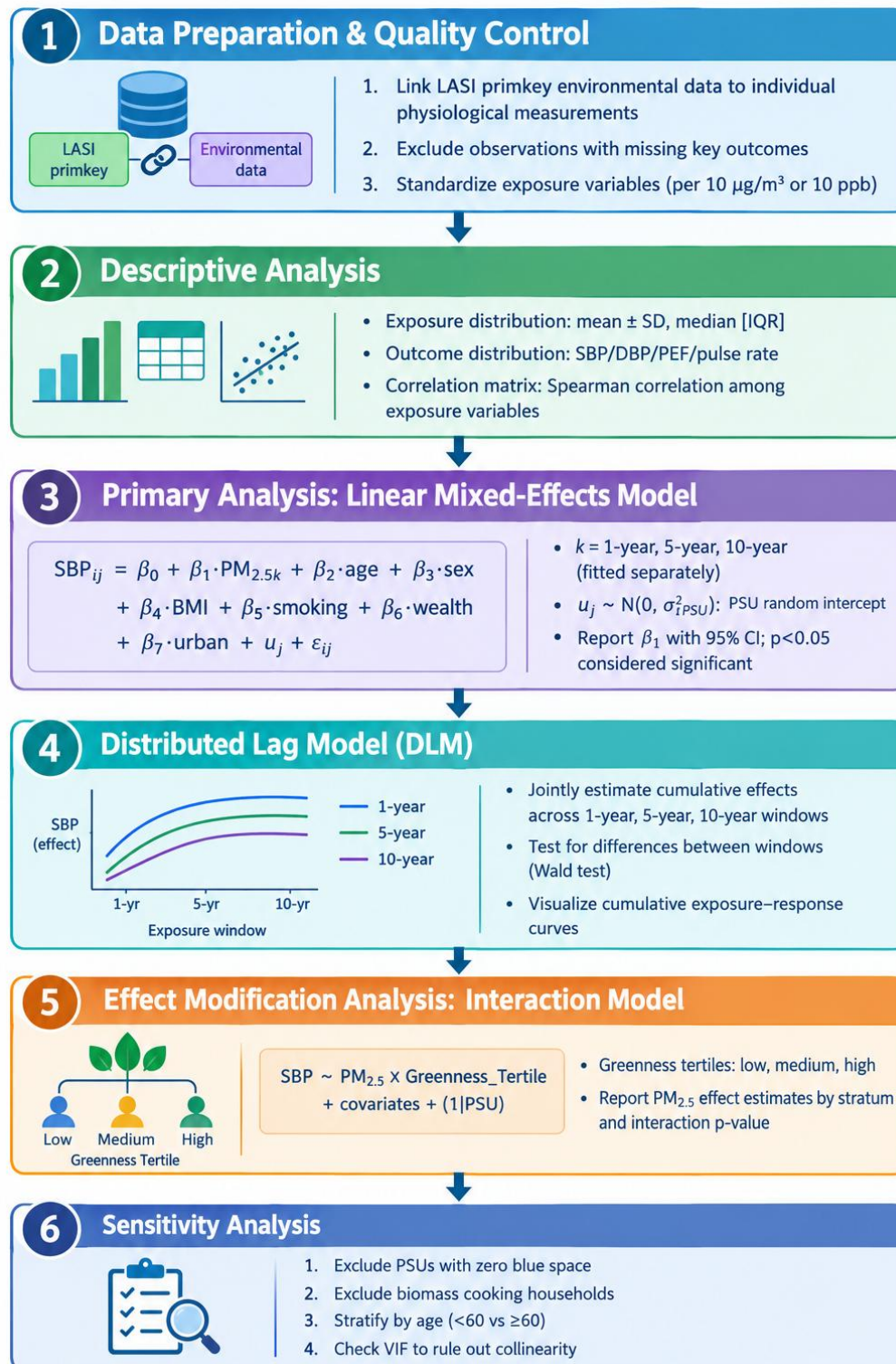


Figure Legend: PM_{2.5} induces systemic oxidative stress through three pathways—pulmonary inflammation, autonomic dysregulation, and HPA axis activation—ultimately leading to elevated blood pressure, airway remodeling, and metabolic dysregulation. Residential greenness exerts modifying effects by reducing pollution exposure, promoting physical activity, and reducing psychological stress, attenuating the PM_{2.5} effect on systolic blood pressure by approximately 62%.

Appendix B: Statistical Modeling Flowchart



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