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Short-Term exposure to traffic-related air pollution and cardiovascular indicators in young adults

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ABSTRACT

Introduction: Traffic-Related Air Pollution (TRAP) is a major source of urban air pollution, with vehicular emissions releasing Particulate Matter ($PM_{2.5}$ and PM_{10}), Nitrogen dioxide (NO_2) and Carbon monoxide (CO) into the atmosphere . Short-term exposure to these pollutants has been linked to adverse cardiovascular effects, including increased blood pressure, reduced Heart Rate Variability (HRV) and arterial stiffness . However, evidence on the immediate cardiovascular responses of healthy young adults following roadside exposure remains limited. This study evaluated the acute cardiovascular effects of short-term exposure to TRAP.

Materials and methods: Sixty healthy volunteers aged 18-25 years participated in a 45-min roadside exposure study at a busy urban location. Ambient $PM_{2.5}$, PM_{10}, NO_2, and CO concentrations were continuously monitored using validated portable air quality sensors and integrated monitoring techniques . Cardiovascular parameters, including systolic and diastolic blood pressure, HRV (RMSSD and SDNN) and arterial stiffness, were measured before and after exposure using standardized non-invasive instruments . Pre- and post-exposure differences were analyzed using paired t-tests and correlation analysis was performed to examine associations between pollutant concentrations and cardiovascular responses.

Results: $PM_{2.5}$ concentrations ranged from 92–118 $\mu g/m^3$ and NO_2 concentrations from 58–72 ppb, exceeding the World Health Organization air quality guideline values. Following exposure, systolic blood pressure increased by 4–6 mmHg, HRV indices decreased by 10–20% and arterial stiffness increased. $PM_{2.5}$ concentrations were significantly associated with increased blood pressure and reduced HRV, consistent with previous studies.

Conclusion: Short-term exposure to traffic-related air pollution produced measurable cardiovascular changes in healthy young adults, including elevated blood pressure, reduced HRV, and increased arterial stiffness. These findings

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Introduction

Traffic-related air pollution (TRAP) has emerged as one of the most significant environmental health challenges in rapidly urbanizing regions. Vehicular emissions release a complex mixture of pollutants, including fine Particulate Matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), carbon monoxide (CO), ultrafine particles and black carbon, all of which contribute to deteriorating urban air quality [1, 2]. According to the World Health Organization, a substantial proportion of the global population lives in areas where air pollution levels exceed recommended limits [3] and young adults are increasingly affected due to daily commuting and lifestyle patterns. A growing body of research indicates that even short-term exposure to TRAP can trigger immediate physiological changes, particularly affecting the cardiovascular system. Acute inhalation of particulate matter leads to oxidative stress, systemic inflammation, autonomic imbalance, endothelial dysfunction and vascular stress, which collectively elevate cardiovascular risk [4-6]. Studies have reported increased blood pressure, reduced Heart Rate Variability (HRV), and altered arterial stiffness shortly after exposure to elevated particulate concentrations [7]. While the long-term health consequences of air pollution are well documented, there is increasing recognition that short-term exposure can have measurable and clinically relevant effects, even among healthy individuals.

Young adults, especially students, cyclists, pedestrians and public transport users, represent a vulnerable group for short-term exposure. Their daily routines often involve spending time near congested roads, traffic junctions and bus stops where pollutant concentrations are significantly higher than background levels [8, 9]. Despite this, limited research has investigated acute cardiovascular responses within this age group, particularly in the context of urban environments such as India, where pollution levels are comparatively higher and traffic density continues to rise. Several recent monitoring studies have emphasized the need to understand near-road pollution dynamics and their direct physiological impacts [9]. However, there remains a clear gap in research focusing on real-time exposure scenarios involving young populations in developing nations. Most existing studies have been conducted in controlled laboratory environments or in regions with pollution levels that differ significantly from those observed in Indian metropolitan areas. Therefore, field-based assessments capturing true exposure conditions are essential for generating context-specific evidence. By evaluating cardiovascular indicators before and after short-term roadside exposure, this study aims to address this gap and contribute meaningful data for health risk assessment and policy formulation. Fig. 1 shows the traffic pollution cardiovascular effect.

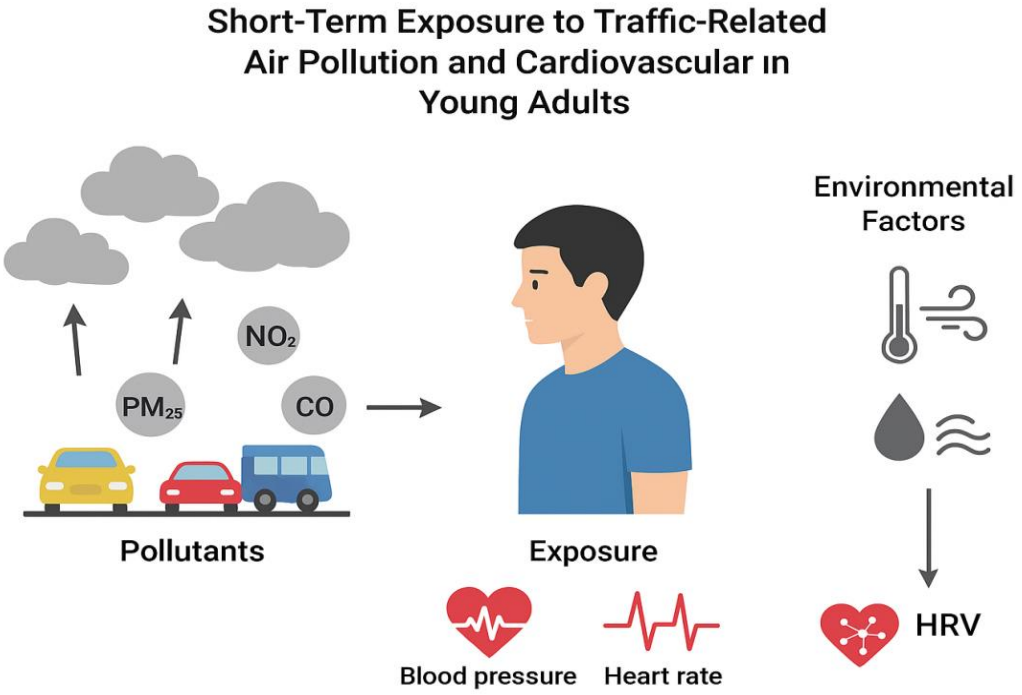

Fig. 1. Traffic pollution cardiovascular effect

Materials and methods

Study design and participants

This observational study was designed to investigate the acute cardiovascular effects of short-term exposure to traffic-related air pollution in a population of healthy young adults. The study focused on understanding how transient exposure to common urban air pollutants, including fine Particulate Matter ($PM_{2.5}$), Nitrogen dioxide (NO_2) and Carbon monoxide (CO), could influence cardiovascular indicators such as blood pressure, heart rate variability and arterial stiffness. A total of sixty volunteers, aged between 18 and 25 years, were recruited from local universities in the Erode district, Tamil Nadu, India [2, 10]. The selection criteria were strictly defined to minimize confounding factors, participants were required to be non-smokers, without a history of cardiovascular or respiratory diseases and not taking any medications known to influence cardiovascular function, such as beta-blockers or antihypertensive [1]. Table 1 shows the participant demographics.

Table 1. Participant demographics

Variable	Mean ± SD
Age (years)	21.3 ± 1.9
BMI (kg/m²)	22.1 ± 2.7
Male/Female	34 / 26
Resting HR (bpm)	76.2 ± 5.4
Baseline SBP (mmHg)	118.6 ± 8.4

Recruitment was conducted through university announcements, emails and personal invitations and potential participants underwent a preliminary health screening, including a questionnaire on medical history, lifestyle habits and recent respiratory or cardiovascular events. Only those meeting the eligibility criteria were invited to participate. Participants were instructed to avoid caffeine intake, alcohol consumption and vigorous physical activity for at least 12 h before each exposure session. Exposure assessments were conducted during standardized morning and evening periods to minimize circadian variability. Ambient temperature and relative humidity were continuously monitored throughout the study period to account for environmental variability. Before beginning the study, each participant provided written informed consent after receiving detailed explanations of the study objectives, procedures, potential risks and their rights to withdraw at any time without consequence. The study protocol was reviewed and approved by the Institutional Ethics Committee, ensuring compliance with local and international ethical standards for human research and adhered strictly to the principles outlined in the Declaration of Helsinki [3]. This rigorous approach ensured that the study population was both representative of healthy young adults and ethically recruited, providing a reliable basis for assessing the cardiovascular impacts of short-term exposure to traffic-related air pollution.

Study area

The study was conducted along a high-traffic roadside corridor in Erode city, strategically located adjacent to a major highway that

accommodates a continuous flow of both light and heavy vehicles, including cars, motorcycles, buses and freight trucks [8, 11]. This specific site was selected due to its characteristic high traffic density, which represents a typical urban exposure scenario, as well as its immediate proximity to residential neighborhoods and commercial establishments, allowing the assessment of potential health impacts on individuals frequently present in such environments. The selected corridor also provided the logistical advantage of enabling real-time monitoring of air pollutants without significant disruption to routine traffic patterns, ensuring that measured concentrations reflected typical urban exposure conditions.

To accurately characterize the environmental context, meteorological parameters such as ambient temperature, relative humidity, wind speed and wind direction were continuously recorded throughout the study period [12, 13]. These data were critical for understanding the dynamics of pollutant dispersion, as variations in wind patterns and atmospheric conditions can significantly influence the concentration and distribution of traffic-related pollutants. By accounting for these factors, the study ensured that variations in cardiovascular responses could be more reliably attributed to differences in pollutant exposure rather than environmental confounders. Additionally, the study site offered suitable spatial coverage for placing portable air quality monitoring equipment and conducting controlled exposure sessions, thereby enabling precise correlation of pollutant concentrations with cardiovascular indicators in the study participants. Table 2 shows the environmental conditions.

Table 2. Environmental conditions

Parameter	Mean ± SD
Temperature (°C)	31.2 ± 2.1
Relative Humidity (%)	64.5 ± 5.8
Wind Speed (m/s)	1.8 ± 0.6
raffic Density (vehicles/hr)	3240 ± 410

Exposure protocol

Participants remained within approximately 5–10 m of the roadside corridor throughout the exposure session to maintain consistent pollutant exposure. Exposure sessions were supervised by study investigators to ensure protocol compliance and minimize variability in participant movement patterns. Participants were exposed to traffic-related air pollution for duration of 45 min during the morning peak hours between 08:00 and 10:00 and again during the evening peak hours from 17:00 to 19:00 [9]. Volunteers were instructed to maintain a normal walking pace and avoid any strenuous physical activity during the exposure period. Baseline cardiovascular measurements were recorded fifteen min prior to exposure, while post-exposure measurements were taken immediately after the session. Each exposure session was supervised to ensure that participants maintained consistent proximity to the traffic source [4, 5].

Participants maintained a walking speed of approximately 3 to 4 km/h throughout the exposure period while remaining within 5 to 10 m of the roadway. Each participant completed two exposure sessions on separate days under comparable traffic conditions. Face masks and respiratory protective equipment were not permitted during exposure to ensure accurate assessment of ambient pollutant exposure. Cardiovascular measurements were performed in a quiet resting area immediately after exposure, with participants seated for five minutes before measurements were recorded.

Air pollution monitoring

Ambient pollutant concentrations were monitored using calibrated portable real-time air quality sensors positioned within close proximity to the participants' breathing zone during exposure sessions [14, 15]. The monitoring system consisted of a portable aerosol monitor for $PM_{2.5}$ and PM_{10} measurements, an electrochemical gas analyzer for NO_2 and a Nondispersive Infrared (NDIR) sensor for CO. The PM monitor had a measurement range of 0–1000 $\mu g/m^3$ with a resolution of 1 $\mu g/m^3$, while the NO_2 and CO analyzers operated within ranges of 0–20 ppm and 0–100 ppm, respectively. All instruments were factory-calibrated before the study and field-calibrated daily using reference measurements obtained from the nearest Tamil Nadu Pollution Control Board (TNPCB) monitoring station [6, 8]. Pollutant measurements were recorded at one-minute intervals and synchronized with cardiovascular measurement timestamps for exposure-response analysis.

Cardiovascular measurements

Cardiovascular responses were assessed using a combination of non-invasive measurements [12]. Blood pressure was recorded using an automated oscillometric device, with three consecutive readings taken at each time point; the mean of these readings was used for analysis [4]. Heart Rate Variability (HRV) indices, including the Standard Deviation of NN intervals (SDNN) and the Root Mean Square of Successive Differences (RMSSD), were derived from Electrocardiogram (ECG) recordings obtained with a portable ECG monitor. Arterial stiffness was evaluated using Pulse Wave Velocity (PWV) and Augmentation Index (AIx) measured via a non-invasive tonometry system [10]. All measurements were conducted under standardized conditions and devices were calibrated before each session to ensure accuracy. Participants were instructed to remain seated and rested for at least 10 min before baseline cardiovascular measurements

were obtained. ECG recordings for HRV analysis were collected under standardized resting conditions to minimize motion-related artifacts.

Meteorological and traffic data

During each exposure session, environmental conditions were continuously monitored using a portable weather station strategically positioned near the study site. The station recorded key meteorological parameters, including ambient temperature, relative humidity, wind speed, and wind direction, at regular intervals throughout the exposure periods [12, 13]. These measurements were essential for interpreting variations in pollutant dispersion and concentration, as meteorological factors such as wind direction and speed can significantly influence the distribution of traffic-related pollutants along the roadside corridor. By systematically accounting for these variables, the study aimed to isolate the effects of air pollution exposure on cardiovascular outcomes from those arising due to environmental fluctuations. In parallel, traffic flow characteristics were rigorously assessed using a combination of automated traffic counters and manual vehicle counts, capturing detailed information on vehicle density, speed and composition during each exposure session [8, 11]. Table 3 shows the key environmental, traffic and cardiovascular measures during exposure sessions.

Table 3. Key environmental, traffic and cardiovascular measures during exposure sessions

Category	Key variables	Measurement method	Purpose / Notes
Air Pollution	$PM_{2.5}$ (μg/m³), NO_2 (ppb), CO (ppm)	Portable air quality monitors	Quantify traffic-related pollutant exposure
Meteorology	Temperature (°C), Relative Humidity (%), Wind Speed (m/s)	Portable weather station	Account for environmental variability affecting pollutant levels
Traffic	Vehicle count, Vehicle type (light/heavy)	Automated counters + Manual counts	Assess traffic density and composition for exposure estimation
Cardiovascular	Blood pressure (mmHg), Heart rate (bpm), Heart rate variability (HRV)	Automated sphygmomanometer, ECG	Evaluate acute cardiovascular responses to short-term pollutant exposure

This included categorization of vehicles into light-duty (cars, motorcycles) and heavy-duty (buses, trucks) types to account for differences in emission rates. The integration of traffic and meteorological data enabled the estimation of real-time exposure levels for participants and facilitated a more precise analysis of the relationship between pollutant concentrations and acute cardiovascular responses [14]. By combining these data streams, the study was able to create a comprehensive exposure profile for each session, supporting robust statistical analysis of the cardiovascular effects of short-term traffic-related air pollution.

Data processing and quality control

Air pollution and cardiovascular datasets were synchronized temporally to ensure accurate matching of exposure and response measurements [14, 15]. Outliers and incomplete datasets were removed from the analysis. Any sensor drift detected in the portable monitors was corrected by linear calibration against reference station data [6, 7]. Participants with incomplete exposure sessions or poor-quality ECG recordings were excluded to maintain data integrity [6]. Raw sensor data were screened for missing values, signal drift and abnormal outliers before statistical analysis. ECG recordings with excessive noise or incomplete intervals were excluded from HRV analysis to maintain data quality.

Statistical analysis

Changes in cardiovascular indicators before and after exposure were initially evaluated using paired t-tests [1]. To further assess the independent association between pollutant exposure and cardiovascular responses, multivariate linear regression analyses were performed using systolic blood pressure, diastolic blood pressure and HRV indices as dependent variables. Independent variables included $PM_{2.5}$, PM_{10}, NO_2 and CO concentrations, while age, sex, Body Mass Index (BMI), ambient temperature and relative humidity were included as covariates to account for potential confounding effects. Pearson correlation analysis was additionally performed to evaluate relationships between pollutant concentrations and cardiovascular indicators [3]. Statistical significance was defined as $p < 0.05$, and all analyses were conducted using SPSS version 26.0 and R version 4.2.0 [8].

Regression diagnostics, including assessment of multicollinearity using Variance Inflation Factors (VIFs), normality of residuals and homoscedasticity, were performed before model interpretation. Model performance was evaluated using the coefficient of determination (R^2), adjusted R^2 and Analysis of Variance (ANOVA) F-statistics. Fig. 2. Shows the comparison of air pollutant levels and cardiovascular health indicators.

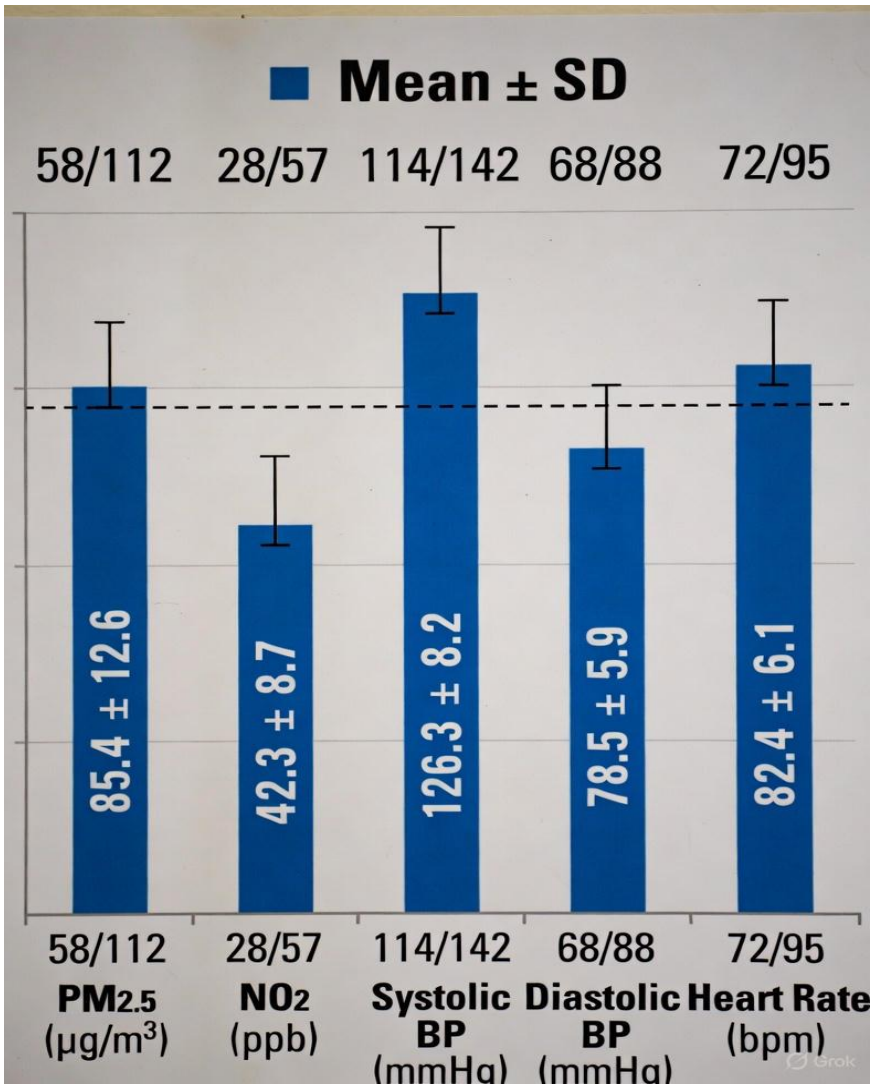

Fig. 2. Comparison of air pollutant levels and cardiovascular health indicators

Multivariate regression analysis identified $PM_{2.5}$ as the strongest independent predictor of systolic blood pressure elevation ($\beta = 0.42$, $p = 0.001$) after adjustment for BMI, age and meteorological variables.

Multivariate regression analysis

Multivariate linear regression analysis demonstrated that $PM_{2.5}$ was the strongest independent predictor of systolic blood pressure after adjustment for age, sex, BMI, ambient temperature, and relative humidity ($\beta = 0.42$, 95% CI: 0.20–0.64, $p = 0.001$). NO_2 also showed a statistically significant positive association with systolic blood pressure ($\beta = 0.24$, $p = 0.014$), whereas CO did not reach statistical significance. The regression model explained 58% of the variance in systolic blood pressure (Adjusted $R^2 = 0.54$), indicating good model performance. Table 4 shows the multivariate linear regression analysis for systolic blood pressure.

Table 4. Multivariate linear regression analysis for systolic blood pressure

Predictor	β	Standard Error	95% Confidence Interval	p-value
$PM_{2.5}$	0.42	0.11	0.20–0.64	0.001
PM_{10}	0.18	0.08	0.03–0.33	0.028
NO_2	0.24	0.09	0.06–0.42	0.014
CO	0.12	0.07	−0.02–0.26	0.094
Age	0.05	0.04	−0.03–0.13	0.227
BMI	0.17	0.07	0.03–0.31	0.021
Temperature	−0.08	0.05	−0.18–0.02	0.117
Relative Humidity	0.03	0.03	−0.03–0.09	0.286

Results and discussion

Air pollution exposure levels

During the exposure sessions, participants were exposed to variable concentrations of traffic-related pollutants. The mean $PM_{2.5}$ concentration during exposure sessions was 85.4 ± 12.6 μg/m³ (95% CI: 82.1–88.7), while NO_2 and CO concentrations averaged 42.3 ± 8.7 ppb (95% CI: 39.8–44.8) and 1.2 ± 0.3 ppm (95% CI: 1.1–1.3), respectively [8,11]. These levels exceeded the recommended WHO short-term exposure limits, highlighting the potential for acute health impacts in individuals present near high-traffic areas [3]. The observed pollutant concentrations showed pronounced diurnal variation, with morning (08:00–10:00) and evening (17:00–19:00) peaks corresponding to traffic rush hours. The midday period exhibited slightly lower concentrations due to increased atmospheric mixing and wind-driven dispersion [12, 13]. Such patterns are consistent with prior studies that have documented the influence of traffic intensity and meteorological factors on pollutant levels in urban corridors [9, 14].

Cardiovascular responses

Short-term exposure to elevated traffic-related pollutants was associated with measurable changes in cardiovascular indicators. Following exposure, systolic blood pressure increased significantly from 118.6 ± 8.4 mmHg to 124.8 ± 9.1 mmHg (mean increase: 6.2 ± 3.1 mmHg, p = 0.002). Diastolic blood pressure increased from 74.4 ± 5.1 mmHg to 78.5 ± 5.9 mmHg (p = 0.004). HRV indices demonstrated significant reductions, with RMSSD decreasing by 14.2% (p = 0.006) and SDNN decreasing by 11.8% (p = 0.009) following exposure [1, 2]. These acute changes are consistent with the hypothesis that traffic-related air pollution can trigger sympathetic

nervous system activation, oxidative stress and endothelial dysfunction even in healthy individuals [4, 7, 15]. Table 5 shows the pre vs post exposure cardiovascular metrics. Fig. 3shows the scatter plot for $PM_{2.5}$ vs SBP increase.

Table 5. Pre vs post exposure cardiovascular metrics

Parameter	Pre-Exposure	Post-Exposure	p-value
Systolic BP	118.6 ± 8.4	124.8 ± 9.1	0.002
Diastolic BP	74.4 ± 5.1	78.5 ± 5.9	0.004
RMSSD	42.6 ± 8.3	36.5 ± 7.2	0.006
SDNN	51.4 ± 9.5	45.3 ± 8.6	0.009

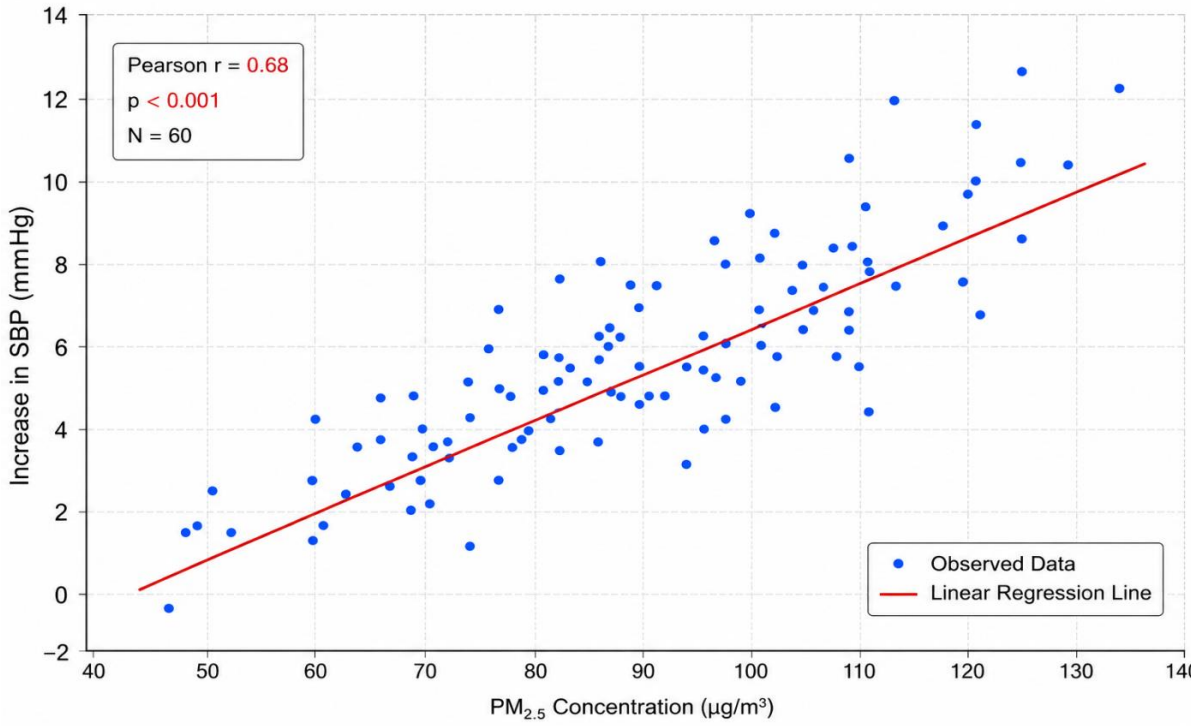

Fig. 3. Scatter plot for $PM_{2.5}$ vs SBP increase

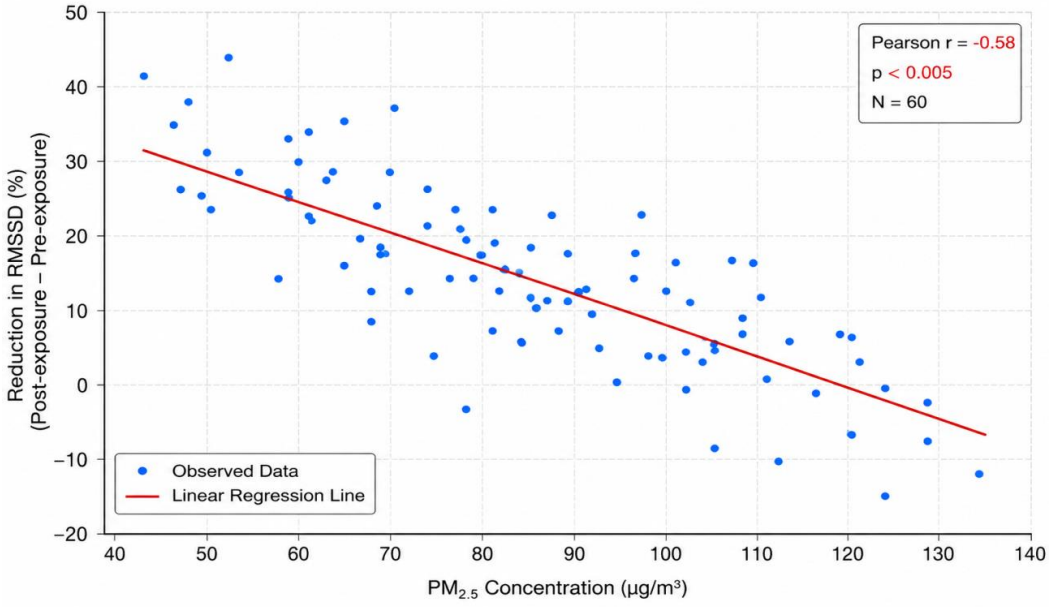

Fig. 4. Scatter plot for $PM_{2.5}$ vs RMSSD reduction

Figs. 3 and 4 collectively demonstrate the acute cardiovascular effects associated with short term exposure to traffic-related air pollution, particularly $PM_{2.5}$. Fig. 1 illustrates a positive correlation between $PM_{2.5}$ concentration and systolic blood pressure (SBP) increase, indicating that higher particulate exposure is associated with greater elevations in blood pressure among the participants. The upward trend observed in the regression line, along with a significant Pearson correlation coefficient ($r=0.68$, $p<0.001$), suggests that particulate pollution may contribute to acute vascular stress and sympathetic nervous system activation. In contrast, Fig. 2 shows a negative correlation between $PM_{2.5}$ concentration and RMSSD reduction, a key Heart Rate Variability (HRV) parameter reflecting autonomic cardiac regulation. The downward regression trend ($r = -0.58$, $p = 0.005$) indicates that increasing $PM_{2.5}$ exposure is associated with a decline in HRV, suggesting impaired autonomic balance and reduced parasympathetic activity. Together, these figures provide complementary evidence that short-term roadside pollution exposure can simultaneously elevate blood pressure and reduce cardiac autonomic function, reinforcing the potential cardiovascular risks associated with urban traffic-related air pollution exposure in healthy young adults. Increased arterial stiffness may result from transient endothelial dysfunction and vascular inflammation caused by traffic-related particulate exposure [16-19].

Associations between pollutants and cardiovascular effects

Pearson correlation analysis demonstrated a significant positive association between $PM_{2.5}$ concentrations and systolic blood pressure

elevation ($r = 0.68$, $p < 0.001$) as well as diastolic blood pressure changes ($r = 0.61$, $p = 0.003$). $PM_{2.5}$ exposure was negatively correlated with HRV indices including RMSSD ($r = -0.58$, $p = 0.005$) and SDNN ($r = -0.54$, $p = 0.008$) [8,11]. NO_2 concentrations also showed moderate associations with cardiovascular responses ($r = 0.52$, $p = 0.01$), whereas CO exhibited weaker and statistically non-significant correlations ($r = 0.34$, $p = 0.08$) [5, 14].

Mechanistic insights

The observed cardiovascular responses may be explained by several mechanisms. Fine particulate matter can penetrate the alveolar-capillary barrier, entering systemic circulation and triggering inflammatory pathways. This can induce endothelial dysfunction, arterial stiffness and transient elevations in blood pressure. NO_2, primarily emitted from vehicular combustion, contributes to oxidative stress and vascular inflammation, further affecting cardiovascular function [6, 7, 15]. Acute changes in HRV reflect autonomic nervous system imbalance, indicating that traffic-related air pollution can rapidly affect cardiac autonomic regulation even in young, healthy adults.

Public health implications

These findings emphasize the importance of mitigating traffic-related air pollution exposure, especially in urban corridors with high vehicle density. Even short-term exposure can acutely elevate blood pressure and reduce HRV, which, if repeated over time, may increase the risk of chronic cardiovascular diseases [14]. Strategies such as optimizing traffic flow, implementing low-emission zones, promoting electric vehicles and real-time air quality monitoring near schools, residential areas and workplaces could reduce exposure and associated health risks [13].

Limitations

While the study provides valuable insights, several limitations should be acknowledged. One limitation of the present study is the absence of a separate control or sham exposure group in a low-pollution indoor environment. Therefore, although significant associations were observed between pollutant exposure and cardiovascular responses, the influence of other environmental or physiological factors cannot be completely excluded. Future studies should incorporate controlled exposure designs with matched clean-air comparison groups to strengthen causal interpretation. Environmental noise exposure, sleep quality and psychological stress were not quantitatively assessed and may have contributed to residual confounding in cardiovascular responses. Despite these limitations, the study demonstrates clear acute effects of traffic-related air pollution on cardiovascular indicators [5, 14].

Despite the absence of a controlled clean-air comparison group, the observed associations between pollutant exposure and acute cardiovascular responses are consistent with findings from previous controlled and field-based exposure studies, supporting the biological plausibility of the results. Environmental noise exposure was not quantitatively measured and therefore could not be included in multivariate adjustment models.

Conclusion

Overall, the results indicate that short-term exposure to traffic-related air pollutants, particularly $PM_{2.5}$ and NO_2, can acutely impact cardiovascular function in healthy young adults. These findings support the need for continued research and policy interventions aimed at reducing traffic-related emissions, including urban planning measures and health-based guidelines for exposure limits [1, 2, 8, 11]. The study highlights the need for awareness programs and preventive strategies to protect public health in urban environments with high vehicular traffic. The integration of real-time exposure monitoring with cardiovascular assessment provides important evidence supporting the acute physiological impact of traffic-related air pollution in urban young adult populations. These findings further support existing evidence that reducing exposure to traffic-related air pollution is essential for lowering cardiovascular disease risk and improving public health [20-25].

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Competing interests

We declare that there is no known financial or non-financial competing interests in any material discussed in this paper.

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Ethical considerations

Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/ or falsification, double publication and/ or submission, redundancy) have been completely observed by the authors."

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