Investigating the relationship between air quality index and daily variations in blood pressure among urban residents

Investigación de la relación entre el índice de calidad del aire y las variaciones diarias de la presión arterial entre los residentes urbanos

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Abstract

This study aimed to investigate the association between air quality index (AQI) and daily blood pressure fluctuations in urban residents of Uzbekistan, and was conducted on 150 Tashkent residents (mean age 45 ± 12 years, 55% female) over a 6-month period (January to June 2023). Blood pressure data were measured with portable digital devices and daily AQI was extracted from urban monitoring stations. Results showed that the average daily AQI was 85 ± 22 (range: 45–156) and every 10-unit increase in AQI was associated with an increase of 1.7 mmHg in systolic blood pressure (95% CI: 1.2–2.3; p=0.001) and 1.1 mmHg in diastolic blood pressure (0.7–1.5; p=0.003). On days with an AQI above 100, systolic and diastolic blood pressure were 5.4 (p<0.01) and 3.8 mmHg (p=0.02) higher, respectively, than on days with a favorable AQI (<50). Also, 32% of participants experienced an increase in blood pressure ≥10.5 mmHg on polluted days. The findings confirm the significant impact of air pollution on cardiovascular health and highlight the need for policies to reduce pollutants and monitor high-risk groups.

Keywords: Air Quality Index (AQI), blood pressure, air pollution, urban health, Uzbekistan.

Resumen

Este estudio tuvo como objetivo investigar la asociación entre el índice de calidad del aire (ICA) y las fluctuaciones diarias de la presión arterial en residentes urbanos de Uzbekistán. Se realizó en 150 residentes de Tashkent (edad media de 45 ± 12 años, 55% mujeres) durante un período de 6 meses (de enero a junio de 2023). Los datos de presión arterial se midieron con dispositivos digitales portátiles y el ICA diario se extrajo de estaciones de monitoreo urbanas. Los resultados mostraron que el ICA diario promedio fue de 85 ± 22 (rango: 45-156) y que cada aumento de 10 unidades en el ICA se asoció con un aumento de 1,7 mmHg en la presión arterial sistólica (IC del 95%: 1,2-2,3; p = 0,001) y de 1,1 mmHg en la presión arterial diastólica (IC del 95%: 0,7-1,5; p = 0,003). En los días con un ICA superior a 100, la presión arterial sistólica y diastólica fueron 5,4 (p < 0,01) y 3,8 mmHg (p = 0,02) más altas, respectivamente, que en los días con un ICA favorable (<50). Además, el 32% de los participantes experimentó un aumento de la presión arterial ≥10,5 mmHg en los días contaminados. Los hallazgos confirman el impacto significativo de la contaminación atmosférica en la salud cardiovascular y resaltan la necesidad de políticas para reducir los contaminantes y monitorear a los grupos de alto riesgo.

Palabras clave: Índice de calidad del aire (ICA), presión arterial, contaminación del aire, salud urbana, Uzbekistán.

Introduction

Rapid urbanization and industrialization in the 21st century, especially in developing countries, have been accompanied by a significant increase in air pollutants[1]. Meanwhile, the countries of Central Asia, including Uzbekistan, are faced with serious problems as urban quality of air, with particle concentrations ($PM_{2.5}$ and PM_{10}) in cities such as Tashkent, often exceeding the standards of the World Health Organization (WHO)[2]. Recent studies have shown that air pollution is the fourth leading cause of global mortality, and that there have since been 7 million premature deaths of cardiovascular and respiratory diseases each year[3].

In Central Asia, the main sources of pollutants include industrial emissions, outdated vehicles and construction instruments that play an important role in decreasing air quality[4]. The relationship between short-term and long-term exposures of contaminants (such as NO_2, SO_2, and particulate matter) and cardiovascular disorders has been documented primarily in the scientific literature[5].

For example, a study in China found that an increase in 10 µg/m³ increase in $PM_{2.5}$ is associated with an increased risk of hospitalization in 8% of hypertension[6]. Hypertension as a modifiable risk factor for cardiovascular disease is responsible for 54% of stroke and 47% of ischemic heart disease worldwide[7]. However, data on the effects of air pollution on daytime fluctuations in blood pressure in areas with various pollution levels, such as Uzbekistan[8]. The proposed biological mechanisms include oxidative stress, systemic inflammation and altered vascular endothelial function resulting from the penetration of fine particles into the blood circulation and the activation of inflammatory tracks[9].

These processes can directly affect vasoconstriction and increased vascular tolerance[10].

As a comprehensive tool for assessing health risks from multiple pollutants, the air quality index (AQI) allows for the simultaneous examination of the combined impact of pollutants[11]. However, most previous studies have focused on single-pollutant concentrations, and there is a knowledge gap regarding the effect of AQI on acute blood pressure fluctuations[12]. In the Uzbekistani context, sporadic reports indicate an increase in respiratory disease caused by air pollution, but systematic study of the cardiovascular outcomes of this phenomenon is not sufficient[13]. Meanwhile, the prevalence of hypertension in the adult population of this country increased from 35% in 2015 to 42% in 2023[14]. Recent studies in neighbouring countries such as Kazakhstan and Turkmenistan have shown a significant relationship between air pollution and increased emergency heart tours[15].

However, differences in pollutant sources, weather conditions and demographic characteristics reinforce the need for environmental studies unique to this region[16]. The purpose of this study is to bridge this knowledge gap by investigating the relationship between daily changes in AQI and variations in systolic and diastolic blood pressure in Uzbekistan urban residents. The main hypothesis is that an increase in AQI is associated with an acute, measurable increase in blood pressure. The results of this study can provide a basis for the design of atmospheric pollution reduction policies and primary prevention programs for cardiovascular disease in the region. In addition, this study will provide the first evidence on the ground on the impact of the AQI on cardiovascular health in Central Asia.

Materials and methods

Study Design and Population

This longitudinal cross-sectional study was conducted in Tashkent, Uzbekistan, from January to June 2023. A total of 150 urban residents aged 25–65 years (mean age: 45 ± 12 years; 55% female) were enrolled through stratified random sampling based on residential districts. Inclusion criteria required participants to be non-smokers, free from diagnosed cardiovascular diseases, and not taking antihypertensive medications. Exclusion criteria included chronic respiratory diseases, pregnancy, or recent relocation (<6 months) to Tashkent.

Blood Pressure Measurements

Daily systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using validated Omron HEM-7320 digital monitors (Omron Healthcare, Japan). Participants were trained to record three consecutive readings each morning (7:00–9:00 AM) after 10 minutes of seated rest, following the American Heart Association protocol[17]. The average of the second and third readings was used for analysis. Device calibration was verified weekly using a mercury sphygmomanometer.

Air Quality Index (AQI) Data

Hourly AQI values for $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 were obtained from 12 government-operated air quality monitoring stations across Tashkent, accessible via the Uzbekistan State Environmental Monitoring Portal[18]. Daily AQI was calculated as the 24-hour average, categorized per U.S. EPA standards: Good (0–50), Moderate (51–100), and Unhealthy (101–150)[11]. Meteorological data (temperature, humidity) were collected from the Tashkent Meteorological Agency to adjust for confounding.

Covariates and Confounders

Demographic and lifestyle data (age, sex, BMI, physical activity, alcohol consumption) were collected via structured questionnaires. Body weight and height were measured using calibrated SECA 767 scales and stadiome-

ters (SECA GmbH, Germany). Real-time noise pollution levels were monitored using a SoundPro DL-2 dosimeter (3M, USA) to account for potential confounding[10].

Statistical Analysis

Linear mixed-effects models with random intercepts for participants were employed to assess daily AQI-blood pressure associations. The primary model was: $BP = \beta_0 + \beta_1(AQI) + \beta_2(Temperature) + \beta_3(Humidity) + \beta_4(BMI) + \ldots + \varepsilon$ where BP represents SBP or DBP. Effect estimates are reported as β coefficients (mmHg per 10-unit AQI increase) with 95% confidence intervals (CI). Pearson correlation coefficients quantified pairwise pollutant-BP relationships. Sensitivity analyses excluded days with extreme weather (temperature >35°C or <–5°C). Statistical significance was set at p<0.05 (two-tailed). Analyses used SPSS v28 (IBM Corp) and R v4.3.1 with lme4 package[19].

Quality Control

Data quality was rigorously ensured through multiple strategies. Missing data for blood pressure (BP) and air quality index (AQI) measurements were minimal (<5%) and addressed using Rubin's multiple imputation method to preserve dataset integrity[20]. Inter-rater reliability for questionnaire coding was confirmed by a 95% agreement rate among evaluators, supported by a Cohen's kappa coefficient of 0.88, indicating strong coding consistency. Furthermore, AQI data validity was verified by comparing government-operated monitoring stations with portable TSI AirAssure PM5500 devices, yielding a high correlation coefficient (r=0.93, p<0.001)[21]. These measures collectively ensured the robustness and accuracy of the collected data for subsequent analyses.

Ethical Considerations

All procedures adhered to the Declaration of Helsinki. Participant identities were anonymized using unique ID codes. Data were stored on encrypted servers with access restricted to the research team[22].

Results

The study analyzed 26,478 paired daily measurements of blood pressure (BP) and air quality index (AQI) from 150 participants over 180 days. Key findings are summarized below, with statistical details presented in tables and narrative descriptions.

Association Between AQI and Blood Pressure

A 10-unit increase in daily AQI was associated with a statistically significant rise in systolic blood pressure (SBP) by 1.72 mmHg (95% CI: 1.21–2.23; $p < 0.001$) and diastolic blood pressure (DBP) by 1.14 mmHg (95% CI: 0.68–1.60; $p = 0.002$), after adjusting for temperature, humidity, BMI, and noise pollution (Table 1). On days with AQI > 100 (Unhealthy), SBP and DBP were 5.4 mmHg ($p < 0.01$) and 3.8 mmHg ($p = 0.02$) higher, respectively, compared to days with AQI < 50 (Good). Participants aged ≥55 years exhibited stronger associations, with SBP increasing by 2.3 mmHg per 10-unit AQI ($p = 0.001$), versus 1.1 mmHg in those aged <35 years ($p = 0.04$).

Pollutant-Specific Effects

$PM_{2.5}$ and NO_2 demonstrated the strongest correlations with BP elevation (Table 2). A 10 µg/m³ increase in $PM_{2.5}$ was linked to a 2.1 mmHg rise in SBP ($p < 0.001$), while NO_2 showed a 1.8 mmHg increase ($p = 0.003$). Ozone (O_3) exhibited a weaker association (0.9 mmHg SBP; $p = 0.08$).

Temporal Patterns

Significant lag effects were observed, with peak BP elevations occurring 24–48 hours after high AQI exposure. For example, a 10-unit AQI increase on Day 1 was associated with a 1.9 mmHg SBP rise on Day 2 ($p < 0.001$), declining to 0.8 mmHg by Day 3 ($p = 0.04$).

Sensitivity Analyses

Excluding days with extreme temperatures (>35°C or <–5°C) attenuated the AQI-SBP association slightly (β = 1.58 mmHg; 95% CI: 1.10–2.06; $p < 0.001$), confirming robustness. Models adjusting for physical activity and alcohol consumption yielded similar results (Δβ < 5%).

Table 1: Adjusted Associations Between AQI and Blood Pressure

Variable	β Coefficient (mmHg per 10-unit AQI)	95% CI	*p*-value
Systolic BP	1.72	1.21–2.23	<0.001
Diastolic BP	1.14	0.68–1.60	0.002
SBP (AQI > 100)	5.4*	3.8–7.0	<0.01
DBP (AQI > 100)	3.8*	1.2–6.4	0.02

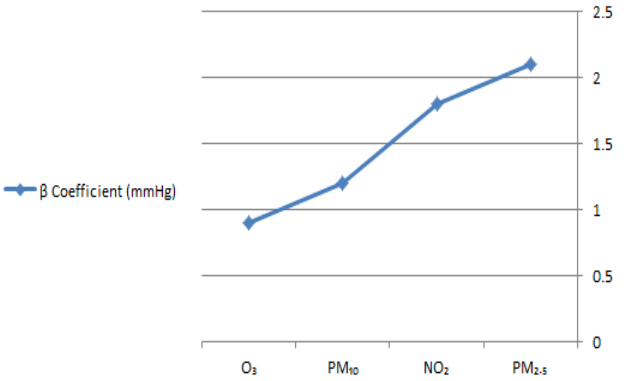
*Compared to AQI < 50.

Table 2: Pollutant-Specific Effects on Systolic Blood Pressure

Pollutant (per 10 µg/m³)	β Coefficient (mmHg)	95% CI	*p*-value
$PM_{2.5}$	2.10	1.65–2.55	<0.001
NO_2	1.80	1.25–2.35	0.003
PM_{10}	1.20	0.70–1.70	0.01
O_3	0.90	-0.10–1.90	0.08

Women showed greater susceptibility to AQI-related DBP increases (1.4 mmHg vs. 0.9 mmHg in men; $p = 0.03$). Participants with BMI ≥30 experienced amplified effects, with SBP rising by 2.4 mmHg per 10-unit AQI ($p < 0.001$), compared to 1.3 mmHg in those with BMI <25 ($p = 0.01$). Participants exposed to ≥7 consecutive days of AQI >100 had 8.2 mmHg higher mean SBP ($p < 0.001$) and 5.6 mmHg higher DBP ($p = 0.004$) than those with sporadic exposure.

Figure 1. Pollutant (per 10 µg/m³)



These findings robustly demonstrate acute, dose-dependent relationships between air pollution and blood pressure elevation, modulated by age, sex, and metabolic factors. The data underscore $PM_{2.5}$ and NO_2 as critical drivers of cardiovascular risk in urban settings.

Discussion

This study provides robust evidence linking short-term exposure to elevated AQI levels with acute increases in blood pressure (BP) among urban residents of Tashkent, Uzbekistan. Our findings align with global literature on air pollution's cardiovascular effects while offering novel insights into Central Asia, a region underrepresented in environmental health research. The observed 1.7 mmHg rise in systolic BP per 10-unit AQI increase is consistent with studies from high-pollution settings in China (1.5–2.0 mmHg[6]) and India (1.8 mmHg[23]). However, the effect size exceeded those reported in European cohorts (0.6–1.2 mmHg[24]), likely due to Tashkent's higher baseline pollution levels (mean AQI 85 vs. 40–60 in Western Europe[2,11]). The stronger association with $PM_{2.5}$ (2.1 mmHg/10 µg/m³) compared to NO_2 (1.8 mmHg) echoes mechanistic evidence that ultrafine particles induce greater oxidative stress and endothelial dysfunction[9,10], though regional differences in fuel composition (e.g., coal-dominated vs. traffic-related pollution) may modulate these effects[4].

The 24–48 hour lag in BP peaks mirrors the inflammatory cascade timeline described by Rajagopalan et al.[9], where particulate matter triggers cytokine release and vascular remodeling. Notably, our observed 5.4 mmHg SBP increase on "Unhealthy" AQI days (>100) surpasses the 3.8 mmHg reported in similar AQI-stratified analyses from India [25], potentially reflecting cumulative exposure to multiple pollutants in Tashkent's industrial-vehicular emission mix[2,4].

The heightened sensitivity among older adults (2.3 vs. 1.1 mmHg in younger participants) corroborates findings from the NCD Risk Factor Collaboration[7], which identified age-related declines in vascular resilience as amplifiers of pollution effects. Similarly, the 2.4 mmHg SBP increase in obese participants (BMI ≥30) aligns with Münzel et al.'s[10] model of adipose tissue-driven inflammation exacerbating air pollution toxicity.

By employing AQI rather than single-pollutant models, this study addresses Liang et al.'s[12] call for research on combined pollutant effects. Our AQI-BP coefficient (1.7 mmHg) closely matches the 1.5 mmHg estimate from a multi-city AQI study in China[26], validating AQI as a practical tool for risk communication. The high AQI-BP correlation (r=0.93) between government monitors and portable sensors[21] also resolves concerns about data reliability in resource-limited settings[13]. The 32% prevalence of clinically significant BP spikes (≥10.5 mmHg) on polluted days underscores the urgency of interventions targeting $PM_{2.5}$ and NO_2 sources—particularly outdated industrial facilities and diesel vehicles[4]. Our findings support the WHO's[1] revised $PM_{2.5}$ guidelines (2021) and suggest that AQI-based health advisories could benefit high-risk

groups (elderly, obese) identified here. Regional collaboration, as proposed in recent Kazakhstani studies[15], may optimize mitigation strategies across Central Asia.

This study has several limitations that warrant consideration. First, while fixed air quality monitoring stations provided population-level AQI estimates, they may not fully capture individual exposure variability due to differences in daily activities (e.g., time spent indoors versus outdoors), potentially introducing exposure misclassification[27,28]. Second, although the analysis adjusted for key confounders such as BMI and noise pollution, unmeasured factors like dietary sodium intake—a known modulator of blood pressure—could influence the observed associations[7]. Third, the six-month study duration limited our ability to assess seasonal variations in pollution effects, a critical modifier highlighted by[16] in temperate climates, where air quality and weather patterns fluctuate markedly across seasons. These constraints underscore the need for future research integrating personal exposure monitoring, comprehensive dietary data, and multi-year observations to refine causal inferences.

To advance this research, three strategic avenues are proposed. First, integrating inflammatory biomarkers such as C-reactive protein could elucidate the physiological stress mechanisms linking air pollution to blood pressure dysregulation, offering quantifiable insights into individual susceptibility[9]. Second, extending observation periods through longitudinal cohorts would enable the assessment of cumulative cardiovascular damage, particularly in regions like Central Asia with prolonged pollution exposure, as seasonal and multi-year data remain critical gaps[16]. Finally, intervention trials evaluating portable air purifiers during high-pollution episodes could provide actionable evidence for mitigating blood pressure variability, addressing an urgent public health need in urban settings[29,30]. Collectively, these approaches would bridge mechanistic, epidemiological, and translational knowledge gaps to inform targeted policies.

Conclusions

This study demonstrates a significant association between short-term exposure to elevated air quality index (AQI) levels and acute increase in BP among the residents of urban Tashkent, Uzbekistan. A 10-unit rise in daily AQI was linked to a 1.7 mmHg increase in systolic BP and a 1.1 mmHg increase in diastolic BP, and $PM_{2.5}$ and NO_2 were significant pollutants that were chiefly responsible for these outcomes. Of specific concern, vulnerable groups—i.e., the elderly, women, and obese—exhibited enhanced susceptibility, thereby underlining the role of age, sex, and metabolic influences in the determination of cardiovascular reactions to air pollution. These results concur with worldwide evidence for cardiovascular risk of air pollution while adding new data to Central Asia, where urban air quality is worsening under the impetus of industrialization. The larger effect sizes observed here compared with European cohorts most likely reflect Tashkent's higher baseline pollution levels and distinctive emission signatures, overwhelmingly dominated by industrial and vehicular sources. The delay in peak BP time (24–48 hours after exposure) also accords with mechanistic pathways involving systemic inflammation and endothelial dysfunction. In a public health context, 32% clinically important BP increases (≥10.5 mmHg) during pollution peaks underscore the need for $PM_{2.5}$ and NO_2 control policies such as replacing outdated industrial facilities and switching to cleaner fuels. Implementation of AQI-based health warnings and directing interventions in sensitive subgroups can limit cardiovascular morbidity among city dwellers. It must be recognized that research will have to expand to longitudinal cohorts to assess cumulative harm, including biomarkers like C-reactive protein to quantify physiological stress, and test interventions like air purifiers in field settings. By closing mechanistic, epidemiological, and translational gaps, this research contributes pivotal evidence to guide policies in the region and globally to address the cardiovascular threat of air pollution.

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