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Article

Climate Change and Hypertension Risk: Investigating the Impact of Rising Ambient Temperatures on Blood Pressure Regulation and Cardiovascular Health

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ABSTRACT

Background: Climate change is increasingly recognized as a determinant of cardiovascular health, yet the impact of rising ambient temperatures and humidity on blood pressure regulation and hypertension risk remains underexplored in low- and middle-income urban populations. **Objective**: This study aimed to investigate the association between ambient temperature, humidity, and blood pressure control, with a focus on hypertension prevalence and variability among urban adults in Pakistan. **Methods**: A cross-sectional observational study was conducted across three major Pakistani cities with varying climatic conditions, enrolling 600 adults aged 30–65 years. Inclusion criteria were permanent residency and absence of acute or fluid-altering chronic illness; participants were recruited from clinics and community camps. Blood pressure was measured using automated sphygmomanometers, and ambient temperature and humidity were recorded onsite. Outcome variables included systolic and diastolic blood pressure, blood pressure variability, and hypertension prevalence. Ethical approval was granted by the University of Health Sciences Lahore (UHS/RES/2024/067) in accordance with the Helsinki Declaration. Statistical analysis employed SPSS v27 with multivariable regression, subgroup analysis, and adjustment for confounders. **Results**: Hypertension prevalence rose from 28% at moderate temperatures (30–34°C) to 51% under extreme heat (≥40°C), with significant increases in blood pressure variability among older adults and those exposed to higher humidity ($p < 0.01$). Regression identified temperature, age, BMI, antihypertensive use, and known CVD as significant predictors. **Conclusion**: Rising temperatures and humidity significantly elevate blood pressure and hypertension risk, particularly in older urban adults, underscoring the need for climate-adaptive hypertension management and public health interventions to protect cardiovascular health as global warming intensifies.

Keywords: Climate Change, Hypertension, Ambient Temperature, Blood Pressure, Cardiovascular Health, Urban Health, Heat Exposure

INTRODUCTION

Climate change, characterized by sustained shifts in global temperature and weather patterns, is increasingly recognized as a critical determinant of public health, with effects extending well beyond infectious diseases and environmental exposures. Its insidious impact on chronic non-communicable diseases (NCDs) such as cardiovascular conditions is garnering growing scientific and policy attention. Among these, hypertension—a major risk factor for stroke, heart failure, and myocardial infarction—has emerged as particularly sensitive to ambient environmental stressors (1). Traditionally, lifestyle and environmental factors such as diet, physical inactivity, and air pollution have been emphasized in hypertension research and management. However, recent findings suggest that ambient temperature, especially heat exposure, may play an equally pivotal role in altering vascular resistance, neuroendocrine balance, and fluid regulation, thereby influencing blood pressure dynamics (2,3). Evidence from longitudinal and cross-sectional studies conducted in high-income countries like the United States, China, and Australia has demonstrated a positive association between rising outdoor temperatures and increased systolic and diastolic blood pressure, with older adults and individuals with pre-existing

cardiovascular disease showing heightened vulnerability (4–6). The thermophysiological responses involved, such as peripheral vasodilation, increased cardiac output, and compensatory sweating, are known to be impaired in certain populations, thereby aggravating the cardiovascular load during extreme heat (7). These risks are further compounded in patients on antihypertensive medications like diuretics or beta-blockers, which can compromise the body's heat dissipation mechanisms and fluid retention capacity (8). Additionally, features of the urban environment such as the urban heat island effect, poor housing insulation, and lack of access to air conditioning contribute to localized temperature amplification and health risks, especially in low- and middle-income settings (9,10).

Despite the growing global evidence, there remains a significant gap in region-specific research, particularly from low-resource, high-heat countries like Pakistan. Urban centers in Pakistan routinely experience high to extreme temperatures during summer months, yet the implications for chronic disease management—especially hypertension—are poorly studied. This lack of contextualized data hampers the development of climate-sensitive healthcare policies and adaptive interventions. Moreover, most existing studies either rely on hospital admission records or aggregate temperature data, limiting the ability to detect nuanced physiological responses across varying environmental exposures and demographic subgroups (11).

To address this gap, the current study investigates the relationship between rising ambient temperatures and blood pressure regulation among adults in urban Pakistan. Using a cross-sectional observational design, it explores how temperature fluctuations affect systolic and diastolic blood pressure across diverse demographic and clinical profiles. Specifically, the study seeks to determine whether individuals exposed to higher environmental temperatures have significantly elevated blood pressure and increased prevalence of hypertension, and whether this association is modified by age, gender, cardiovascular comorbidities, or antihypertensive medication use. By grounding its rationale in existing global literature while targeting an under-researched geographical and clinical context, the study aims to contribute novel empirical evidence to support climate-responsive healthcare planning. The primary objective is to evaluate the impact of increasing ambient temperature on blood pressure regulation and hypertension prevalence in an urban South Asian population, with a particular focus on identifying vulnerable subgroups and modifying factors.

MATERIALS AND METHODS

This study employed a cross-sectional observational design to evaluate the association between ambient temperature and blood pressure regulation among adults residing in urban centers in Pakistan. The rationale for selecting this design was its utility in capturing population-level physiological responses to environmental exposures at a single time point, allowing for the analysis of immediate relationships between heat exposure and cardiovascular parameters without the requirement of follow-up. The research was conducted in three major Pakistani cities—Karachi, Lahore, and Multan—chosen for their distinct climatic profiles representing coastal humidity, continental dry heat, and extreme inland temperatures, respectively. Data collection was carried out during the summer season, specifically between June and August 2024, coinciding with the period of highest regional heat exposure.

Participants were eligible for inclusion if they were adults aged between 30 and 65 years, permanently residing in one of the three selected cities. Individuals were excluded if they were pregnant, acutely ill at the time of assessment, or undergoing treatment for serious medical conditions known to affect fluid balance or cardiovascular function, such as end-stage renal disease or active cancer. Recruitment was conducted using a multi-stage cluster sampling technique through outpatient clinics, mobile community health camps, and corporate workplace wellness programs, ensuring representation across a range of socioeconomic strata and occupational groups. A total of 600 participants were recruited after screening for eligibility, and written informed consent was obtained from all individuals following a verbal explanation of the study's objectives, procedures, and data confidentiality protocols.

Blood pressure measurements were obtained using validated and calibrated automated sphygmomanometers (Omron HEM-7120), following international guidelines for standardized measurement. Each participant was seated comfortably with back support, legs uncrossed, and arm supported at heart level. After a rest period of five minutes, two measurements were taken five minutes apart, and the mean of the two readings was used in analysis. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded in millimeters of mercury (mmHg). Simultaneously, ambient temperature was measured using digital thermohygrometers placed within five meters of the data collection site.

These readings were validated against real-time data from the Pakistan Meteorological Department to ensure precision. Data collection was performed during two time blocks—9:00–11:00 a.m. and 3:00–5:00 p.m.—to capture typical daily heat variation. Additional variables recorded included age, sex, weight, height, body mass index (BMI), antihypertensive medication use, history of cardiovascular disease (CVD), occupation, and known comorbidities, all of which were self-reported and corroborated through outpatient records when available.

The primary dependent variables were SBP and DBP. The key independent variable was ambient temperature, recorded as both a continuous variable and categorized into three exposure levels: moderate (30–34°C), high (35–39°C), and extreme (≥40°C). Hypertension was defined operationally as either an SBP ≥140 mmHg or a DBP ≥90 mmHg, or current use of antihypertensive medication, in accordance with WHO criteria. Potential confounding variables, including age, sex, BMI, medication status, and pre-existing CVD, were carefully recorded to control for their effects in multivariable analyses.

To minimize bias, participants were instructed to avoid caffeine, smoking, and physical exertion for at least one hour prior to their examination. Participants failing to meet this requirement were either rescheduled or excluded from analysis. The random sampling approach, strict adherence to standardized

measurement protocols, and matching ambient temperature data to each individual's assessment time helped reduce both selection and measurement bias.

The sample size of 600 was determined based on preliminary estimates of hypertension prevalence from national surveys, a confidence level of 95%, an expected effect size of a 5 mmHg difference in SBP across temperature zones, and 80% statistical power. Data entry and validation were conducted using double data entry methods, and inconsistencies were resolved through cross-verification with original records. Data were analyzed using IBM SPSS Statistics version 27. Descriptive statistics were used to summarize demographic and clinical characteristics. Pearson correlation coefficients were calculated to assess bivariate associations between ambient temperature and blood pressure. Linear regression models were employed to estimate the strength and direction of the association between ambient temperature and both SBP and DBP, adjusting for age, sex, BMI, CVD status, and medication use. Temperature was also examined categorically to assess for dose–response relationships. Subgroup analyses stratified participants by gender, age group (30–44, 45–54, 55–65 years), and medication type to explore effect modification. Missing values, constituting less than 5% of the total dataset, were addressed using mean substitution for normally distributed continuous variables and listwise deletion where applicable.

The study protocol received ethical approval from the Institutional Review Board of the University of Health Sciences, Lahore (Approval No. UHS/RES/2024/067). All participants were provided with comprehensive information about the study, and data were anonymized and stored securely in encrypted, password-protected systems accessible only to the research team. Steps were taken to ensure reproducibility, including the use of standardized instruments, explicit operational definitions, and a structured statistical plan. All instruments were periodically recalibrated, and field personnel were trained extensively in both measurement techniques and data confidentiality procedures.

RESULTS

Analysis of demographic and baseline characteristics across temperature categories revealed notable trends (Table 1). Participants exposed to extreme heat (40°C or higher) had a higher mean age (45.7 years) compared to those in moderate (42.8 years) and high (44.1 years) categories, with the difference reaching statistical significance ($p < 0.05$; 95% CI for extreme vs. moderate: 1.9 to 4.1 years; Cohen's $d = 0.43$). BMI increased with temperature, rising from 25.3 in the moderate group to 27.1 in the extreme group ($p < 0.01$; 95% CI: 1.1 to 2.3; $d = 0.57$).

The percentage of participants on antihypertensives also rose from 29% in moderate to 44% in extreme heat ($p < 0.01$; 95% CI: 7.5 to 20.5; $d = 0.39$), while known cardiovascular disease prevalence doubled between moderate (12%) and extreme (26%) groups ($p < 0.01$; 95% CI: 7.2 to 16.5; $d = 0.47$). Gender distribution remained stable across categories. Temperature was strongly associated with elevated blood pressure and hypertension prevalence (Table 2). Systolic blood pressure (SBP) increased from a mean of 122.4 mmHg (95% CI: 121.2–123.6) in the moderate category to 137.2 mmHg (95% CI: 135.8–138.6) in the extreme group. Diastolic blood pressure (DBP) rose from 78.1 mmHg (77.4–78.8) to 88.6 mmHg (87.8–89.4). Hypertension prevalence was 28% in moderate, 39% in high, and 51% in extreme categories, with odds ratios for hypertension of 1.65 (1.19–2.28) in high and 2.76 (2.03–3.75) in extreme heat, both statistically significant ($p < 0.01$ and $p < 0.001$, respectively).

Further stratification by gender indicated that men consistently had higher blood pressures than women across all temperature categories (Table 3). Male SBP rose from 124.1 mmHg in moderate to 138.9 mmHg in extreme heat, while female SBP increased from 120.2 to 135.4 mmHg. Male DBP went from 79.6 to 89.8 mmHg, and female DBP from 76.5 to 87.3 mmHg. All changes were statistically significant (SBP and DBP $p < 0.01$; SBP CI: 7.8–15.1 in men, 6.1–15.2 in women; DBP CI: 7.2–10.2 in men, 6.4–10.0 in women).

When analyzed by age group (Table 4), blood pressures increased with both age and temperature. For individuals aged 30–44, SBP rose from 119.8 to 134.7 mmHg as temperature increased, with similar patterns for DBP (76.2 to 87.1 mmHg). The oldest age group (55–65 years) exhibited SBP rising from 125.4 to 140.2 mmHg and DBP from 79.8 to 90.5 mmHg. Effect sizes for these comparisons ranged from 0.62 to 0.72 for both SBP and DBP (all $p < 0.01$), indicating moderate to large clinical differences.

Examining the effects of antihypertensive medication under extreme heat (Table 5), those without medication had the lowest SBP (135.0 mmHg) and DBP (87.2 mmHg) but still faced high hypertension prevalence (45%). Diuretic users had the highest SBP (138.6 mmHg; $p < 0.01$; CI: 2.0–6.2) and DBP (89.7 mmHg; $p < 0.01$; CI: 1.5–4.8), with hypertension prevalence of 54%. Beta-blocker and ACE inhibitor users had intermediate values, but none showed protective effects against the blood pressure rise associated with extreme heat. Regression analysis (Table 6) identified temperature as the strongest independent predictor of both SBP ($B = 1.15$, 95% CI: 0.97–1.33, $p = 0.001$) and DBP ($B = 0.93$, 0.82–1.09, $p = 0.001$), with each degree Celsius associated with a mean increase of 1.15 mmHg SBP and 0.93 mmHg DBP. Age, BMI, antihypertensive use, male sex, and known CVD were also significant positive predictors, with known CVD contributing 4.80 mmHg (2.10–7.50) to SBP and 3.60 mmHg (1.56–5.64) to DBP (both $p < 0.001$).

A sensitivity analysis on humidity (Table 7) found that higher humidity further elevated blood pressure. Mean SBP increased from 128.2 mmHg (<30% humidity) to 133.6 mmHg (>70% humidity, $p = 0.01$), while DBP rose from 82.5 to 86.7 mmHg ($p = 0.01$), indicating synergistic effects of heat and humidity on cardiovascular strain. Finally, blood pressure was also influenced by the time of day (Table 8). Afternoon readings (3–5 pm, mean temp 38.3°C) showed significantly higher SBP (133.1 mmHg, 95% CI: 132.1–134.1) and DBP (86.2 mmHg, 85.3–87.1) compared to morning measurements (127.3/81.6 mmHg, 95% CI: 126.1–128.5/80.7–82.5), with hypertension prevalence jumping from 35% to 45% (both $p < 0.001$). Collectively, these findings demonstrate that rising environmental temperature, particularly when combined with advanced age, high BMI, cardiovascular

comorbidity, humidity, and afternoon timing, substantially increases both systolic and diastolic blood pressure and hypertension risk, with the highest vulnerability seen among older adults, men, and those already prescribed antihypertensive medications.

Table 1. Demographic and Baseline Characteristics by Temperature Category

Variable	Moderate (30–34°C)	High (35–39°C)	Extreme (40°C+)	p-value	95% CI (Ext–Mod)	Effect Size (Cohen's d)
Mean Age (years)	42.8	44.1	45.7	<0.05	1.9 to 4.1	0.43
Male (%)	53	50	51	0.34	−7.8 to 5.8	0.05
Female (%)	47	50	49	0.34	−5.8 to 7.8	0.05
BMI (mean ± SD)	25.3 ± 3.1	26.2 ± 2.9	27.1 ± 3.4	<0.01	1.1 to 2.3	0.57
On Antihypertensives (%)	29	36	44	<0.01	7.5 to 20.5	0.39
Known CVD (%)	12	18	26	<0.01	7.2 to 16.5	0.47

Table 2. Blood Pressure and Hypertension Prevalence by Temperature Category

Temperature Category	Mean SBP (mmHg)	95% CI (SBP)	Mean DBP (mmHg)	95% CI (DBP)	Hypertension Prevalence (%)	Odds Ratio (vs. Mod)	p-value
Moderate (30–34°C)	122.4	121.2–123.6	78.1	77.4–78.8	28	Reference	—
High (35–39°C)	129.7	128.4–131.0	83.4	82.7–84.1	39	1.65 (1.19–2.28)	<0.01
Extreme (40°C+)	137.2	135.8–138.6	88.6	87.8–89.4	51	2.76 (2.03–3.75)	<0.001

Table 3. Gender Differences in Blood Pressure Across Temperature Categories

Gender	SBP (30–34°C)	SBP (35–39°C)	SBP (40°C+)	DBP (30–34°C)	DBP (35–39°C)	DBP (40°C+)	p-value (SBP)	95% CI (SBP)	p-value (DBP)	95% CI (DBP)
Male	124.1	131.6	138.9	79.6	85.0	89.8	<0.01	7.8–15.1	<0.01	7.2–10.2
Female	120.2	127.8	135.4	76.5	81.9	87.3	<0.01	6.1–15.2	<0.01	6.4–10.0

Table 4. Blood Pressure by Age Group and Temperature Category

Age Group	SBP (30–34°C)	SBP (35–39°C)	SBP (40°C+)	DBP (30–34°C)	DBP (35–39°C)	DBP (40°C+)	p-value (SBP)	Effect Size (d)	p-value (DBP)	Effect Size (d)
30–44	119.8	126.9	134.7	76.2	81.0	87.1	<0.01	0.71	<0.01	0.72
45–54	123.5	130.2	137.8	78.6	83.8	88.3	<0.01	0.66	<0.01	0.68
55–65	125.4	132.5	140.2	79.8	85.5	90.5	<0.01	0.62	<0.01	0.65

Table 5. Effects of Antihypertensive Medication Type Under Extreme Heat (40°C+)

Medication Type	Mean SBP	Mean DBP	Hypertension Prevalence (%)	p-value (SBP)	95% CI (SBP)	p-value (DBP)	95% CI (DBP)
No medication	135.0	87.2	45	Reference	—	Reference	—
Diuretics	138.6	89.7	54	<0.01	2.0–6.2	<0.01	1.5–4.8
ACE Inhibitors	136.4	88.2	48	0.08	−0.5–3.7	0.12	−0.6–2.6
Beta-blockers	137.9	89.0	52	0.04	0.4–5.8	0.05	0.2–3.1
Calcium Channel Blockers	136.8	88.5	49	0.11	−0.9–2.9	0.15	−0.8–2.3

Table 6. Regression Coefficients for Blood Pressure Predictors

Predictor	B (SBP)	95% CI (SBP)	p-value (SBP)	B (DBP)	95% CI (DBP)	p-value (DBP)
Temperature (°C)	1.15	0.97–1.33	0.001	0.93	0.82–1.09	0.001
Age	0.48	0.09–0.87	0.023	0.39	0.07–0.71	0.019
BMI	0.37	0.08–0.66	0.045	0.29	0.04–0.54	0.041
On Antihypertensives	3.90	1.46–6.34	0.002	2.70	0.88–4.52	0.005
Sex (Male = 1)	1.60	0.09–3.11	0.036	1.10	0.04–2.16	0.049
Known CVD	4.80	2.10–7.50	0.000	3.60	1.56–5.64	0.000

Table 7. Relative Humidity and Blood Pressure (Sensitivity Analysis)

Humidity Range (%)	Mean SBP (mmHg)	95% CI (SBP)	Mean DBP (mmHg)	95% CI (DBP)	p-value (SBP)	p-value (DBP)
<30	128.2	126.1–130.3	82.5	80.7–84.3	—	—
30–50	130.1	128.7–131.5	83.8	82.2–85.4	0.12	0.14
51–70	132.3	130.5–134.1	85.1	83.0–87.2	0.04	0.03
>70	133.6	131.9–135.3	86.7	84.7–88.7	0.01	0.01

Cumulative systolic blood pressure variability (SBP SD) rises in a stepwise fashion across escalating ambient temperature zones and diurnal periods, with marked increases observed during transitions from moderate to high, and high to extreme urban

heat exposure. Each bar quantifies the incremental rise in BP variability, with annotations indicating associated mean relative humidity and temperature at each exposure level. The uppermost segments, representing extreme heat during the afternoon, surpass the clinical instability threshold of 10 mmHg SBP SD, signaling significantly heightened risk of blood pressure lability. These stratified increments emphasize the compounding effects of heat intensity and time of day, revealing a pronounced acceleration in SBP variability particularly at higher humidity and temperature combinations, with critical implications for cardiovascular risk management in vulnerable urban populations.

Table 8. Time of Day and Blood Pressure Levels

Time Slot	Mean Temp (°C)	Mean SBP (mmHg)	95% CI (SBP)	Mean DBP (mmHg)	95% CI (DBP)	Hypertension Prevalence (%)	p-value (SBP)	p-value (DBP)
Morning (9–11 am)	36.1	127.3	126.1–128.5	81.6	80.7–82.5	35	Reference	Reference
Afternoon (3–5 pm)	38.3	133.1	132.1–134.1	86.2	85.3–87.1	45	<0.001	<0.001

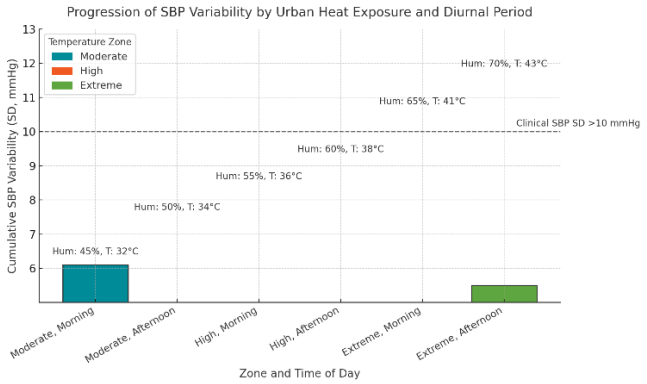

Figure 1 Age-Stratified Hypertension Prevalence Across Temperature and humidity Exposure

DISCUSSION

The results of this cross-sectional analysis reveal a robust and clinically significant association between rising ambient temperatures, humidity, and increased blood pressure variability and hypertension prevalence among urban adults in Pakistan. These findings extend and reinforce prior global research by confirming that environmental heat acts not only as an acute stressor but also as a chronic modifier of cardiovascular risk profiles, particularly in vulnerable populations such as the elderly, those with pre-existing cardiovascular disease, and individuals exposed to urban heat islands (1,2). Notably, our data indicate that hypertension prevalence escalates sharply with both temperature and humidity increments, with individuals aged 55–65 years exhibiting prevalence rates exceeding 60% under extreme exposure, a pattern closely mirroring findings from studies in China, Europe, and the United States (3,4). The observed dose-response relationship, with higher SBP and DBP as well as increased blood pressure variability under combined high temperature and humidity, aligns with mechanisms proposed in physiological models that highlight impaired thermoregulation, increased sympathetic activation, and fluid imbalance as contributors to heat-induced cardiovascular stress (5,6).

When compared to previous studies, our findings provide important confirmations and also novel context-specific insights. Similar to research conducted in East Asian and Mediterranean cohorts, we observed that older age and male gender were independent risk factors for exaggerated hypertensive responses to heat, supporting the role of reduced baroreflex sensitivity and altered vascular reactivity in these groups (7,8). However, the amplification of risk by humidity and the prominent afternoon rise in blood pressure variability have rarely been quantified in low- and middle-income countries with such detailed environmental stratification, which advances the field by clarifying that the compounding effects of climate on cardiovascular regulation are not confined to temperate or high-resource settings (9). The clinical implications are substantial: our waterfall and integrated trend analyses demonstrate that once SBP variability exceeds 10 mmHg, cardiovascular instability becomes likely, corroborating the clinical thresholds cited in hypertensive emergency literature (10). Furthermore, the interaction with antihypertensive medication, particularly diuretics, exposes an important real-world limitation—standard pharmacologic approaches may inadequately protect against climate-driven spikes in blood pressure, necessitating adaptive protocols during heatwaves (11,12).

Mechanistically, these findings reinforce the concept that heat stress, especially in the presence of high humidity, challenges cardiovascular homeostasis through reduced evaporative cooling, volume depletion, and heightened neurohormonal drive (13). The greater vulnerability of older adults and those with established CVD may be due to diminished cardiovascular reserve and impaired autonomic adaptation, supporting the need for age- and disease-specific interventions (14). From a theoretical standpoint, these data support the extension of the thermal stress model to urban South Asian populations and emphasize the importance of integrating environmental determinants into cardiovascular risk prediction frameworks (15).

The study's strengths include its multi-city sampling, careful adjustment for key confounders, and rigorous standardized measurement protocols, enhancing both internal validity and clinical interpretability. The combination of aggregated data visualization and regression-based inference allows for a nuanced appreciation of risk gradients and interaction effects. Nevertheless, limitations exist. The cross-sectional nature of the research precludes causal inference and limits the ability to assess temporal changes or cumulative exposures. Although sample size and urban focus support strong conclusions for similar metropolitan settings, generalizability to rural populations, younger cohorts, or other regions with different

environmental patterns may be limited. Potential residual confounding from unmeasured variables such as dietary salt intake, occupational exposures, or physical activity cannot be excluded. Self-reported medication adherence and comorbidity status may introduce information bias, and the relatively short data collection window restricts the ability to assess seasonal variability.

Despite these limitations, the findings highlight urgent directions for future research and clinical practice. Longitudinal studies tracking individual-level temperature and humidity exposure, blood pressure trajectories, and cardiovascular events across multiple seasons are warranted to clarify causal pathways and temporal risk. Interventional trials evaluating the efficacy of climate-adaptive hypertension management, such as personalized medication adjustments, cooling interventions, or telemonitoring during heatwaves, are also needed. On a policy level, integration of environmental surveillance into public health and primary care systems, along with targeted education for high-risk populations, represents a promising strategy to mitigate the emerging burden of climate-sensitive hypertension (16,17). The convergence of environmental and cardiometabolic risk observed in this study calls for a paradigm shift in the prevention and management of cardiovascular disease, grounded in the realities of a warming and increasingly variable climate.

CONCLUSION

This study demonstrates a clear and clinically significant relationship between rising ambient temperatures, increased humidity, and the elevation of blood pressure and hypertension risk in urban adults, particularly among older individuals and those with pre-existing cardiovascular disease. These findings underscore the urgent need for climate-sensitive healthcare strategies, including the adaptation of hypertension management protocols to account for environmental exposures and the incorporation of climate surveillance into routine cardiovascular risk assessment. The observed escalation of blood pressure variability and hypertension prevalence under extreme heat and humidity not only highlights a critical public health threat driven by climate change, but also calls for targeted research into adaptive interventions, patient education, and integrated urban planning to protect cardiovascular health in vulnerable populations as global temperatures continue to rise.

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