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CARDIOVASCULAR HEALTH IN A CHANGING CLIMATE: ENVIRONMENTAL RISK FACTORS AND IMPLICATIONS FOR PREVENTION

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ABSTRACT:
Introduction: Climate-related thermal stress may affect cardiovascular healthcare demand; however, evidence from Pakistan is limited. This study investigated the associations between ambient temperature, acute cardiovascular admissions, in-hospital mortality, and length of stay in Lahore, highlighting implications for healthcare preparedness.
Methods: A retrospective multicenter observational time-series study was conducted, analyzing 12,917 admissions among adults aged 20 years and older at three Lahore hospitals from 2023 to 2025. Hospital-specific quasi-Poisson distributed lag nonlinear models estimated cumulative temperature–admission associations over 0 to 21 days, adjusting for seasonality, relative humidity, rainfall, and calendar effects. Estimates were pooled using multivariate random-effects meta-analysis. Adjusted admission-level models evaluated mortality and length of stay among survivors.
Results: Acute coronary syndrome comprised 48.3% of admissions, acute decompensated heart failure 31.9%, and arrhythmia 19.8%. Low temperature was not significantly associated with total admissions (RR 1.27, 95% CI 0.71–2.27; p=.418). The estimated minimum-risk temperature matched the high-temperature comparison, so we could not estimate an independent heat-risk effect for total admissions. Extreme-heat days (RR 1.00, 95% CI 0.91–1.10) and heatwaves (RR 1.04, 95% CI 0.93–1.16) were not clearly associated with admissions. Each 5°C increase in admission-day mean temperature corresponded to a 2% longer expected hospitalization among survivors (adjusted mean-stay ratio 1.02, 95% CI 1.01–1.03; p<.001), while mortality findings remained inconclusive (adjusted OR 1.04, 95% CI 0.92–1.16). Subgroup and sensitivity analyses were also inconclusive.
Conclusion: Higher admission-day temperatures were associated with modestly longer hospitalizations among survivors, while associations with admissions and mortality remained uncertain. Imprecise estimates and boundary reference temperatures limited the strength of inferences. The study did not establish climate-change attribution or preventive effectiveness.

KEYWORDS: Ambient temperature; Cardiovascular admissions; Heatwaves; Distributed lag nonlinear models; Hospital length of stay; Pakistan.

INTRODUCTION

Cardiovascular disease continues to be a leading cause of premature mortality, disability, and healthcare demand globally, despite significant progress in prevention and treatment.[1] Conventional risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidemia remain central to cardiovascular assessment. Environmental conditions, including those shaped by climate change, also influence cardiovascular vulnerability. Climate change modifies exposure to thermal stress, interacts with air pollution, and increases the likelihood of extreme weather events that can disrupt essential care.[2] A systematic review of 492 studies identified associations between climate-related environmental stressors and cardiovascular mortality, acute events, and healthcare utilization, while also highlighting substantial geographical gaps in the evidence.[3] These findings underscore the importance of incorporating environmental exposures into cardiovascular research and prevention strategies alongside established clinical risk factors.

Understanding the relationship between climate change and cardiovascular health requires distinguishing between short-term weather effects and long-term climatic changes. Daily temperature fluctuations can precipitate acute illness, while climate change alters the frequency, intensity, and distribution of hazardous exposures over extended periods. Research comparing observed conditions with counterfactual climates lacking human-induced warming has attributed a portion of the heat-related mortality burden to anthropogenic climate change.[4] Although this attribution evidence demonstrates the broader relevance of warming to health, it does not imply that every temperature-associated cardiovascular admission is directly attributable to climate change. Studies examining daily admissions are valuable for characterizing immediate exposure–response relationships and for identifying periods when preventive action and clinical preparedness are most effective.

The cardiovascular system is essential for maintaining thermal balance. During heat exposure, cutaneous vasodilation and increased skin blood flow facilitate heat dissipation, while sweating causes fluid loss. These physiological responses elevate cardiac output demands, which may be challenging to sustain in individuals with limited cardiovascular reserve.[5] Dehydration, reduced circulating volume, and electrolyte disturbances can further compromise hemodynamic stability. In contrast, cold exposure induces peripheral vasoconstriction, raising blood pressure and cardiac workload. Alterations in autonomic activity, blood viscosity, and hemostatic function represent additional biological pathways through which thermal stress may precipitate cardiovascular events.[6] Consequently, clinical outcomes depend on both environmental conditions and the exposed individual's physiological capacity.

Epidemiological evidence indicates that high temperatures affect cardiovascular health, although findings differ by outcome, population, and exposure definition. A systematic review and meta-analysis demonstrated that heat exposure is associated with increased cardiovascular mortality and selected morbidity outcomes, highlighting that temperature-related risks are not uniform across diagnoses.[7] A multinational analysis of 567 cities in 27 countries found that both extreme heat and cold were linked to higher mortality from cardiovascular disease, ischemic heart disease, stroke, and heart failure.[8] While these mortality findings justify studying acute cardiovascular admissions, they should not be interpreted as direct estimates of hospital admission risk. Emergency presentation, admission, and death represent distinct outcomes influenced by disease severity, healthcare access, and the organization of local services.

Cold exposure remains significant even in a warming climate. A multicountry investigation found that the mortality burden associated with non-optimal temperatures extends beyond exceptional heatwaves and includes substantial contributions from moderately cold conditions.[9] Registry-based research in England and Wales associated lower temperatures with increased myocardial infarction risk over subsequent days and weeks, emphasizing the importance of delayed exposure effects.[10] Similarly, a nationwide Chinese study linked low ambient temperature to the onset of atrial fibrillation episodes.[11] Collectively, these findings support the examination of the entire temperature distribution and diagnosis-specific responses. Analyses limited to same-day heat exposure may miss delayed associations, cold-related risks, and differences among acute coronary syndromes, heart failure, and arrhythmias.

Temperature interacts with other environmental exposures. Meta-analyses have linked short-term exposure to various air pollutants with myocardial infarction and with heart failure hospitalization or death.[12,13] Air pollution serves as both an independent cardiovascular hazard and a co-exposure that may fluctuate with meteorological conditions. Relative humidity affects evaporative cooling efficiency, while severe rainfall and other extreme weather events can disrupt transportation, medication access, and continuity of care. Therefore, environmental cardiovascular research should differentiate the primary exposure from correlated conditions and potential confounders. This distinction is crucial when interpreting temperature associations in contexts where multiple environmental hazards coexist.

Vulnerability to environmental exposures is not evenly distributed. Factors such as age, pre-existing cardiovascular disease, comorbid conditions, and social circumstances influence physiological resilience and the capacity to avoid hazardous environments.[14] South Asia requires particular attention, as climate projections indicate that densely populated agricultural regions are especially vulnerable to increasingly severe humid heat, with outdoor work and socioeconomic disadvantage compounding exposure.[15] In Pakistan, these regional findings highlight the need for locally grounded research rather than the uncritical application of estimates from other countries. Prevention efforts must also address existing cardiovascular risk: a cluster-randomized trial in Bangladesh, Pakistan, and Sri Lanka demonstrated improved blood pressure control through a community-based intervention integrated with public healthcare services.[16] Although this trial did not assess climate adaptation, it demonstrates how established care systems can support cardiovascular prevention in resource-constrained settings.

An effective preventive response necessitates action at individual, community, and health system levels. Heat-health planning can integrate early warnings, accessible cooling, public communication, and interventions to reduce exposure in homes, workplaces, and urban environments.[17] An evaluation of Ahmedabad's heat action plan reported reduced heat-associated all-cause mortality after implementation, offering regionally relevant evidence despite the limitations of its observational design.[18] For cardiovascular services, these strategies highlight the importance of targeted communication, continuity of care, and anticipating weather-related demand. Long-term mitigation strategies may provide additional benefits: a systematic review identified potential cardiovascular co-benefits from active transport, greener environments, dietary change, and cleaner energy.[19] Prevention thus includes both protection during hazardous weather and reduction of environmental and conventional cardiovascular risks.

Despite these advances, significant analytical and geographical gaps persist. The current evidence base is unevenly distributed across countries, and findings from different climates or healthcare systems may not be directly applicable to specific districts. Temperature associations can be nonlinear and delayed, rendering single linear, same-day estimates insufficient for certain research questions. Distributed lag nonlinear models provide a framework to represent exposure–response relationships and lagged effects simultaneously.[20] Employing transparent exposure definitions, adjusting for temporal patterns and relevant co-exposures, and conducting sensitivity analyses can help characterize the magnitude, timing, and uncertainty of temperature–health associations.

In this context, the present study employed a retrospective multicenter observational time-series design involving three hospitals in Lahore district, Punjab, from 1 January 2023 to 31 December 2025. The study investigated the relationships between temperature and admissions for acute coronary syndrome, acute decompensated heart failure, and clinically significant arrhythmia. The objectives were to assess associations between ambient temperature and acute cardiovascular admissions, in-hospital mortality, and length of hospital stay, and to consider their implications for cardiovascular prevention and healthcare preparedness.

METHODOLOGY

Study Design and Setting

A retrospective multicenter observational time-series study was designed to investigate associations between environmental exposures and acute cardiovascular admissions in Lahore district, Punjab, Pakistan. The study period spanned from 1 January 2023 to 31 December 2025, encompassing 1,096 calendar days. Study sites included Punjab Institute of Cardiology, Mayo Hospital, and Services Hospital.

Routinely collected hospital admission records were linked with daily meteorological observations. Hospital-day counts served as the primary unit of analysis for evaluating environmental associations with cardiovascular admissions, while individual admission records provided information on patient characteristics and clinical outcomes. The analysis focused on short-term environmental variation and its implications for cardiovascular prevention and healthcare preparedness.

Study Population and Eligibility Criteria

The study population comprised adults aged 20 years or older who resided in Lahore district and were admitted to a participating hospital during the study period. Eligible admissions included those with a principal diagnosis of acute coronary syndrome, acute decompensated heart failure, or clinically significant arrhythmia requiring inpatient management. Diagnoses were identified from the documented clinical assessment and discharge records.

Elective admissions, outpatient visits, trauma-related presentations, and admissions with a principal noncardiac diagnosis were excluded. Records without a verifiable admission date or sufficient diagnostic information to establish eligibility were also excluded. Residence information was used to restrict the population to Lahore district and improve correspondence between the assigned environmental exposure and patients' likely locations before admission.

All eligible admissions were included, rather than selecting a fixed sample. The final sample size was determined by the number of qualifying records retrieved after screening and removal of duplicate entries. Each admission was treated as a separate clinical episode; repeat admissions were not considered unique patients.

Clinical Data Collection and Management

Clinical information was extracted using a standardized data collection form. The variables included hospital, admission date, age, sex, district of residence, principal cardiovascular diagnosis, hypertension, diabetes mellitus, chronic kidney disease, previously diagnosed cardiovascular disease, discharge date, and discharge status. Comorbidities were identified from documented medical histories and clinical records.

Records were reviewed for duplicate entries, inconsistent dates, implausible ages, and discrepancies between admission diagnoses and discharge documentation. Duplicate entries referring to the same hospital admission were removed, while separate admissions were retained. Missing information was differentiated from the documented absence of a condition. Unavailable hospital records were not interpreted as evidence of zero admissions.

Eligible admissions were aggregated into daily counts for each hospital. Days with confirmed complete reporting and no eligible admissions were assigned a count of zero. Missing data and exclusions were documented for each hospital and variable.

Environmental Exposure Assessment

Daily meteorological records were obtained from the Pakistan Meteorological Department for the Lahore monitoring station, which was selected to represent the study area. Environmental variables included daily mean, maximum, and minimum temperature, relative humidity, and total rainfall. These observations were linked to hospital admissions by calendar date. Meteorological data preceding the study period were included to support lagged exposure assessment for admissions occurring in early January 2023.

Daily mean temperature was selected as the primary exposure, as it represented overall thermal conditions throughout the day. Daily maximum temperature was used to identify extreme-heat events. An extreme-heat day was defined as a day when the maximum temperature exceeded the 95th percentile of the observed study-period distribution. A

heatwave was defined as at least two consecutive extreme-heat days. These definitions reflected locally relative heat exposure within the study period.

Environmental measurements were reviewed for missing dates, inconsistent units, and implausible values prior to linkage. Station-based measurements represented area-level exposure and did not directly capture individual indoor conditions, occupational exposure, or access to cooling.

Outcome Measures

The primary outcome was the daily number of eligible acute cardiovascular admissions at each hospital. Diagnosis-specific admission counts were examined separately for acute coronary syndrome, acute decompensated heart failure, and clinically significant arrhythmia.

Secondary outcomes included in-hospital mortality and length of hospital stay. In-hospital mortality was defined as death occurring during the recorded admission. Length of stay was calculated from admission and discharge dates. Transfers were identified separately, and outcomes following transfer were not assumed to be known without linked records.

Statistical Analysis

Continuous variables were summarized using means and standard deviations or medians and interquartile ranges, depending on their distributions. Categorical variables were reported as frequencies and percentages. Cardiovascular admission characteristics were summarized by hospital and principal diagnostic group, while environmental conditions were described using daily meteorological measurements.

Quasi-Poisson time-series regression was employed to evaluate associations between environmental exposures and daily cardiovascular admission counts, accounting for overdispersion. A distributed lag nonlinear model (DLNM) was applied to characterize potentially nonlinear temperature–admission relationships and cumulative delayed associations across lag days 0–21. Models were adjusted for long-term trends and seasonality using a natural cubic spline of calendar time with seven degrees of freedom per year. Relative humidity, rainfall, day of the week, and public holidays were included as additional covariates.

Hospital-specific temperature associations were estimated using a common model specification and pooled through multivariate random-effects meta-analysis. The minimum-risk temperature was estimated within the 5th to 95th percentile range of observed daily mean temperature. Cumulative relative risks (RRs) and corresponding 95% confidence intervals (CIs) were calculated by comparing temperatures at the 5th and 95th percentiles with the model-estimated minimum-risk temperature over lag days 0–21. When the estimated minimum-risk temperature coincided with a comparison temperature, the corresponding relative risk was considered the reference value (RR = 1.00) rather than an independent exposure contrast. Separate quasi-Poisson models examined same-day associations between extreme-heat events, heatwave periods, and daily cardiovascular admission counts using the specified time-series adjustments.

Stratified analyses examined temperature associations by sex, age group (20 to 64 years and 65 years or older), and principal cardiovascular diagnosis. Secondary admission-level analyses assessed in-hospital mortality using multivariable logistic regression and length of hospital stay among patients discharged alive using quasi-Poisson regression. Admission-day mean temperature was evaluated as a continuous exposure, with adjusted odds ratios for mortality and mean-stay ratios for length of stay expressed per 5°C increase. Both models were adjusted for age, sex, principal cardiovascular diagnosis, hypertension, diabetes mellitus, chronic kidney disease, previously diagnosed cardiovascular disease, hospital, and admission season.

Sensitivity analyses evaluated alternative maximum lag periods of 14 and 28 days compared with the primary 21-day specification. The robustness of temperature associations was further examined by varying the seasonal adjustment from seven to six and eight degrees of freedom per year. An additional sensitivity analysis adjusted for daily fine particulate matter (PM2.5) concentrations using available air-quality monitoring records. The completeness of PM2.5 observations was assessed before model estimation, and the analysis was restricted to dates with valid pollution measurements and the required meteorological and admission data. The number and proportion of observation days contributing to this analysis were documented.

Temperature contrasts in sensitivity analyses were evaluated against the fixed reference temperature from the primary model. Missing outcome data were not imputed, and analyses were restricted to records with the required information. Results were reported as relative risks, adjusted odds ratios, or mean-stay ratios, as appropriate, with corresponding 95% confidence intervals. Two-sided statistical significance was assessed at $p < .05$.

Ethical Considerations

Ethical approval was obtained for this retrospective multicenter observational time-series study. The study involved the secondary analysis of routinely collected hospital admission records without direct patient contact or intervention. Personal identifiers were removed from the analytical dataset, and access was restricted to authorized researchers. Data were stored securely and reported in aggregate form to ensure patient confidentiality. All clinical and environmental information was handled in accordance with institutional ethical standards and data-protection requirements.

Relevance to Prevention

The findings were interpreted in the context of weather-sensitive cardiovascular prevention, identifying vulnerable patient groups, and preparing hospital services for adverse environmental conditions. Prevention recommendations were informed by the observed associations; their effectiveness was not directly evaluated. The study assessed short-term weather-related cardiovascular risks and did not independently determine the contribution of long-term climate change to individual admissions.

RESULTS

Study Population and Admission Characteristics

The dataset included 12,917 acute cardiovascular admissions from three hospitals between 1 January 2023 and 31 December 2025. Each hospital contributed 1,096 observation days, resulting in a total of 3,288 hospital-days. All admissions involved adults aged 20 years or older with an eligible emergency cardiovascular diagnosis.

Table 1. Characteristics of cardiovascular admissions

Characteristic	n (%) or stated summary
Total admissions	12,917
Age, years, mean ± SD	62.8 ± 13.4
Age 20–64 years	7,065 (54.7)
Age ≥65 years	5,852 (45.3)
Male	7,519 (58.2)
Female	5,398 (41.8)
Hypertension	7,843 (60.7)
Diabetes mellitus	5,118 (39.6)
Chronic kidney disease	1,778 (13.8)
Previously diagnosed cardiovascular disease	6,758 (52.3)
In-hospital death	397 (3.1)
Survivor length of stay, median (IQR), days	4 (3–7)

Records reflected individual admissions rather than unique patients, and comorbidities were not mutually exclusive. Among 12,917 admissions, 397 resulted in in-hospital death. Length of stay was evaluated only for patients with documented discharge alive and valid admission and discharge dates. Patients who died during hospitalization, were transferred without confirmed discharge outcomes, or had incomplete length-of-stay data were excluded from this analysis. The final analytical sample for length of stay included [12,520] eligible admissions.

Interpretation: Hypertension was the most prevalent comorbidity, followed by diagnosed cardiovascular disease. Adults aged 65 years or older accounted for 45.3% of admissions. These descriptive characteristics alone did not establish susceptibility to environmental exposures.

Patterns of Environmental Exposure

A single district-level weather series was assigned to all three hospitals. This series encompassed 1,096 study days and an additional 28 preceding days, enabling complete exposure histories for lag analyses.

Table 2. Shared environmental conditions

Indicator	Value
Daily mean temperature, °C, mean ± SD	24.25 ± 8.21

Daily maximum temperature, °C, mean ± SD	29.23 ± 8.27
Daily minimum temperature, °C, mean ± SD	19.27 ± 8.28
Relative humidity, %, mean ± SD	62.51 ± 11.94
Daily rainfall, mm, mean ± SD	1.80 ± 5.63
Extreme-heat threshold based on maximum temperature, °C	41.91
Extreme-heat days, n	55
Heatwave days, n	41
Heatwave episodes, n	12
Daily mean temperature: 5th percentile, °C	12.05
Daily mean temperature: 95th percentile, °C	36.58

Extreme heat was defined as a daily maximum temperature above its study-period 95th percentile. Heatwaves required at least two consecutive extreme-heat days. Daily mean temperature was the exposure used in the distributed lag models.

Interpretation: The exposure series included 55 extreme-heat days and 12 heatwave episodes. Since all hospitals utilized the same district-level series, variations in admission counts did not indicate differences in weather exposure assignments.

Cardiovascular Admission Burden

The Punjab Institute of Cardiology accounted for the highest number of admissions.

Table 3. Distribution of cardiovascular admissions

Hospital or diagnosis	Admissions, n (%)	Mean admissions per hospital-day
Punjab Institute of Cardiology	5,097 (39.5)	4.65
Mayo Hospital	4,338 (33.6)	3.96
Services Hospital	3,482 (27.0)	3.18
All hospitals	12,917 (100.0)	3.93
Acute coronary syndrome	6,238 (48.3)	1.90
Acute decompensated heart failure	4,116 (31.9)	1.25
Arrhythmia	2,563 (19.8)	0.78

Rows for hospital and diagnosis represent separate breakdowns of the same admissions. Hospital-specific daily means were calculated using 1,096 days, while combined and diagnosis-specific means were based on 3,288 hospital-days. All percentages were rounded.

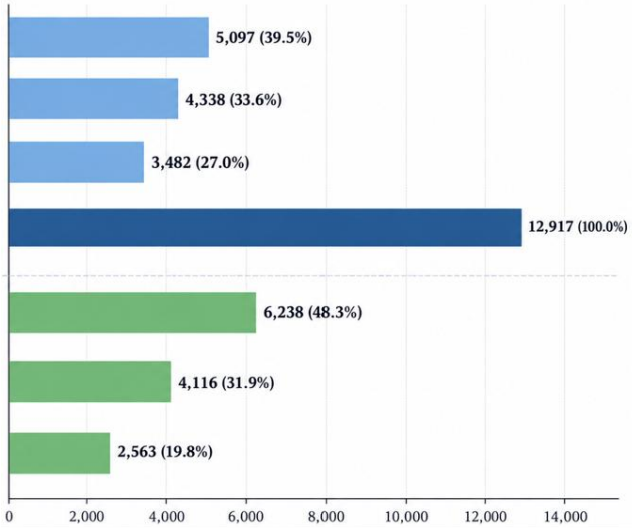


Figure 1 Distribution of cardiovascular admissions

Interpretation: Acute coronary syndrome and acute decompensated heart failure collectively accounted for nearly 80% of admissions. The mean burden was 3.93 admissions per hospital-day. These metrics reflect admission counts rather than population incidence rates.

Associations Between Temperature and Cardiovascular Admissions

Hospital-specific quasi-Poisson distributed lag nonlinear models were used to examine cumulative associations over lags of 0 to 21 days. The models were adjusted for calendar time, relative humidity, rainfall, day of the week, and a holiday indicator. Reduced temperature-response coefficients were then pooled using multivariate random-effects meta-analysis.

Table 4. Cumulative temperature–admission associations over lags of 0–21 days

Outcome	Reference temperature, °C	High-temperature RR (95% CI)	p	Low-temperature RR (95% CI)	p
All cardiovascular admissions	36.58	Reference	—	1.27 (0.71–2.27)	.418
Acute coronary syndrome	36.58	Reference	—	1.65 (0.76–3.57)	.202
Acute decompensated heart failure	28.31	1.19 (0.82–1.73)	.353	1.01 (0.42–2.42)	.974
Arrhythmia	36.58	Reference	—	1.16 (0.36–3.77)	.807

High and low temperatures correspond to the 95th and 5th percentiles of daily mean temperature, respectively. Each primary model used its fitted minimum-risk reference, identified within these percentiles. "Reference" indicates that the high-temperature comparison equals the selected reference; its RR is therefore 1 by definition, without a separate

hypothesis test. Confidence intervals are conditional on the estimated reference. P-values were not adjusted for multiple comparisons.

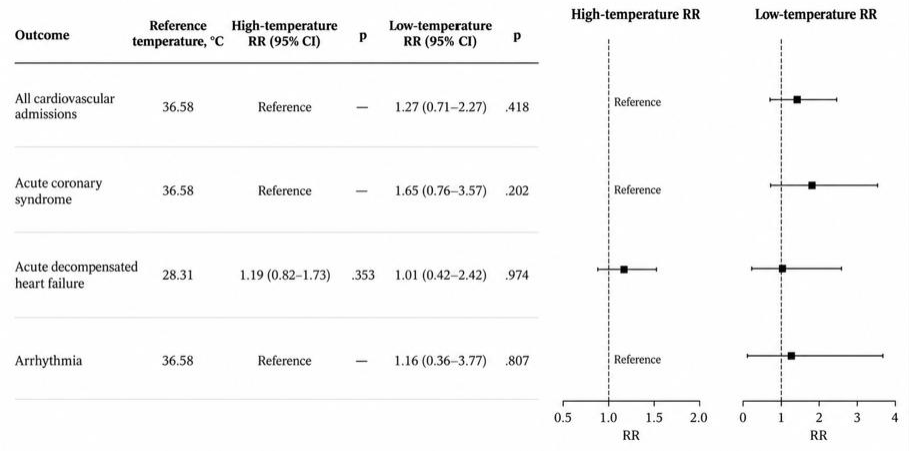


Figure 2 Cumulative temperature–admission associations

Interpretation: For total admissions, acute coronary syndrome, and arrhythmia, the fitted minimum was observed at the upper search boundary of 36.58°C. As a result, the planned high-temperature contrast did not yield an independent estimate of heat risk. This limitation does not imply that heat exposure is without risk.

For heart failure, the estimated reference temperature was 28.31°C, and the association with high temperatures was imprecise (RR 1.19, 95% CI 0.82–1.73). All confidence intervals for low temperatures included 1. Therefore, the primary models did not provide clear evidence of temperature-associated changes in admissions.

Extreme Heat, Heatwaves, and Clinical Outcomes

Separate models were used to evaluate extreme-heat and heatwave days. Analyses at the admission level assessed mortality and length of stay among survivors.

Table 5. Event-based admission associations and secondary clinical outcomes

Comparison or outcome	Measure	Estimate (95% CI)	p
Extreme-heat versus other days	Adjusted admission RR	1.00 (0.91–1.10)	.974
Heatwave versus other days	Adjusted admission RR	1.04 (0.93–1.16)	.484
In-hospital mortality, per 5°C higher mean temperature	Adjusted OR	1.04 (0.92–1.16)	.548
Survivor length of stay, per 5°C higher mean temperature	Adjusted mean-stay ratio	1.02 (1.01–1.03)	<.001

In-hospital mortality was analyzed using multivariable logistic regression. The length of hospital stay was examined with quasi-Poisson regression among patients discharged alive who had complete admission and discharge data. Patients who died in hospital, were transferred without confirmed discharge outcomes, or had missing length-of-stay data were excluded from the length-of-stay analysis. Both models were adjusted for age, sex, principal cardiovascular diagnosis, hypertension, diabetes mellitus, chronic kidney disease, previously diagnosed cardiovascular disease, hospital, and admission season.

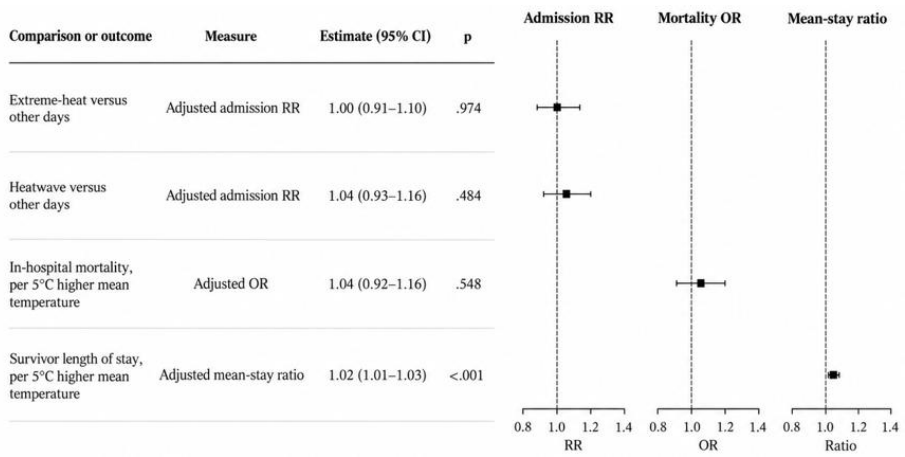


Figure 3 Event-based admission associations and secondary clinical outcomes

Neither extreme-heat nor heatwave days demonstrated a statistically significant association with cardiovascular admission counts. The relationship between admission-day means temperature and in-hospital mortality remained statistically inconclusive, as the adjusted odds ratio was close to unity and the 95% confidence interval included 1. In contrast, higher admission-day mean temperature was significantly associated with increased hospitalization duration among survivors. Specifically, each 5°C increase in mean temperature was associated with an approximate 2% increase in expected hospital length of stay (adjusted mean-stay ratio 1.02, 95% CI 1.01–1.03; p<.001).

Subgroup Analyses

Age- and sex-specific models evaluated associations with low temperatures, employing the common reference temperature established in the all-admission model.

Table 6. Low-temperature associations by age and sex

Subgroup	Admissions, n	Cumulative RR (95% CI)	p
Age 20–64 years	7,065	1.57 (0.74–3.33)	.236
Age ≥65 years	5,852	0.99 (0.44–2.20)	.972
Male	7,519	1.19 (0.57–2.46)	.643
Female	5,398	1.42 (0.61–3.32)	.412

Temperature contrasts between 12.05°C and 36.58°C were analyzed across lag periods of 0 to 21 days. High-temperature contrasts matched the common reference and provided no additional information. Formal tests for interactions between subgroups were not conducted.

Interpretation: All subgroup confidence intervals included 1, and differences between point estimates did not suggest increased susceptibility in any specific age or sex group.

Sensitivity Analyses

Sensitivity analyses included variations in the maximum lag period, adjustments for seasonality, and the addition of a particulate pollution variable.

Table 7. Sensitivity of the low-temperature association for total cardiovascular admissions

Model specification	RR (95% CI)	p
Lag 0–14 days	1.13 (0.71–1.79)	.605
Lag 0–21 days: primary analysis	1.27 (0.71–2.27)	.418
Lag 0–28 days	1.38 (0.65–2.92)	.402

Seasonal adjustment: 6 df/year	1.39 (0.79–2.47)	.257
Seasonal adjustment: 8 df/year	1.28 (0.70–2.33)	.427
Additional $PM_{2.5}$ adjustment	1.28 (0.72–2.29)	.401

RR = relative risk; CI = confidence interval; df = degrees of freedom; $PM_{2.5}$ = fine particulate matter with an aerodynamic diameter of ≤2.5 μm. All temperature contrasts were evaluated at 12.05°C, using 36.58°C as the fixed primary reference temperature. The primary analysis applied a distributed lag period of 0–21 days, with seven degrees of freedom per year for seasonal adjustment. Both seasonal-adjustment and pollution-adjusted sensitivity models maintained the 0–21-day lag window.

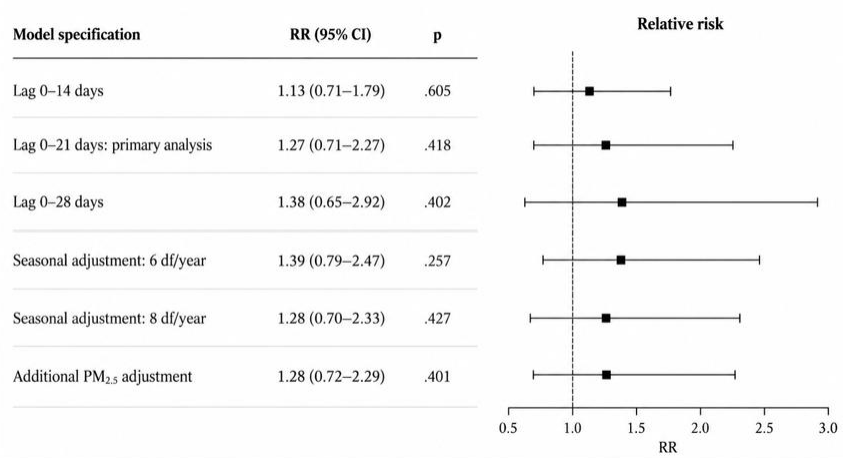


Figure 4 Sensitivity of the low-temperature association for total cardiovascular admissions

DISCUSSION

This retrospective multicenter observational time-series analysis evaluated associations between ambient temperature and acute cardiovascular admissions across three hospitals in Lahore from 2023 to 2025. The dataset comprised 12,917 admissions, with acute coronary syndrome as the most frequent diagnosis, followed by acute decompensated heart failure and arrhythmia. The primary analyses did not identify clear evidence of temperature-associated changes in admission counts. Estimates for extreme-heat and heatwave conditions were also consistent with no association. However, higher admission-day temperature was associated with modestly longer hospitalization among survivors. These outcomes should be interpreted separately, as admission frequency, in-hospital mortality, and length of stay represent distinct clinical endpoints.

The uncertain findings regarding admissions should be contextualized within the heterogeneous evidence from prior research. For example, a meta-analysis of 64 studies by Phung et al. identified increased cardiovascular hospitalizations associated with cold exposure, heatwaves, and diurnal temperature variation, but not with heat exposure overall.[21] This distinction underscores that different temperature metrics can yield varying associations. The present estimates therefore neither confirm the absence of temperature-related cardiovascular risk nor demonstrate a consistent relationship across exposure definitions. Variations in outcome composition, climate, exposure assessment, and analytical methods further limit direct comparability between studies.

A central interpretive challenge was selecting the minimum-risk reference temperature. For total cardiovascular admissions, acute coronary syndrome, and arrhythmia, the fitted minimum occurred at 36.58°C, which was the upper boundary of the search range and the planned high-temperature comparison. As a result, the corresponding relative risk equaled one by definition and did not independently estimate heat risk. Gasparrini and Armstrong have described methods for reducing and pooling nonlinear, delayed exposure associations, which provide the methodological basis for interpreting cumulative temperature contrasts.[22] In this analysis, the boundary reference indicates that 36.58°C cannot be considered a clinically safe threshold. Interpretation should instead recognize the limited information provided by this specific contrast.

The low-temperature estimate for total cardiovascular admissions was elevated but imprecise (RR 1.27, 95% CI 0.71–2.27). Acute coronary syndrome exhibited the largest diagnosis-specific point estimate (RR 1.65, 95% CI 0.76–3.57), although its confidence interval included one. Bhaskaran et al., using myocardial infarction registry data from England and Wales, identified an association between lower temperature and increased myocardial infarction risk.[23] While this study offers a relevant comparison, myocardial infarction and the broader acute coronary syndrome category are not

identical outcomes. The present findings remain inconclusive and do not establish a cold-related increase in admissions or differences between diagnostic groups.

Admission relative risks for extreme-heat and heatwave days were 1.00 (95% CI 0.91–1.10) and 1.04 (95% CI 0.93–1.16), respectively. These results did not indicate increased admission counts under the specified definitions and same-day models. However, the estimates do not account for all possible delayed associations or alternative exposure thresholds. The occurrence of 55 extreme-heat days across 12 heatwave episodes also limited the number of distinct events available for analysis. Although Phung et al. reported a pooled heatwave-associated increase in cardiovascular hospitalization [24], the present confidence interval remains compatible with a modest increase. Therefore, differences in statistical significance alone do not indicate that the findings conflict.

The mortality findings should be distinguished from population-level cardiovascular mortality research. Alahmad et al. examined 567 cities across 27 countries and identified associations between extreme temperatures and mortality from several cardiovascular causes. In contrast, the present analysis evaluated death during an eligible hospital admission and found an adjusted odds ratio of 1.04 (95% CI 0.92–1.16) per 5°C increase in admission-day mean temperature. These outcomes and exposure contrasts are not directly comparable.[25] In-hospital mortality excludes deaths occurring before admission and is conditional on both reaching the hospital and being admitted. Therefore, the uncertain hospital-based estimate does not contradict the broader mortality evidence.

Higher admission-day temperature was associated with approximately 2% longer expected hospitalization among survivors (adjusted mean-stay ratio 1.02, 95% CI 1.01–1.03). This modest association suggests that temperature-related healthcare demand may warrant investigation beyond admission counts. However, the analysis did not identify the mechanism underlying longer stays. Variations in illness severity, treatment, discharge arrangements, or hospital operations could contribute to this association. Additionally, restricting the sample to survivors means the results cannot be generalized to all admissions.

Age- and sex-stratified analyses did not identify a clearly more susceptible subgroup. Bunker et al. reported temperature-associated mortality and morbidity among older adults, supporting continued attention to this population in environmental health research.[26] However, this external evidence does not demonstrate effect modification in the present dataset. All subgroup confidence intervals included one, and no formal interaction tests were conducted. The proportion of older adults among admissions therefore characterizes the clinical population rather than indicating greater temperature susceptibility.

Sensitivity analyses indicated substantial uncertainty across alternative lag windows, seasonal adjustments, and additional $PM_{2.5}$ adjustment. The pollution-adjusted estimate was similar to the primary result, with Table 7 detailing the number of contributing observation days and data coverage. Additional limitations include the three-year study period, three-hospital coverage, shared district-level temperature assignment, and lack of individual exposure measurements. Sera et al. found that urban characteristics modified temperature-related mortality associations across cities[27], highlighting the need for more detailed investigation of local environmental and social conditions. The present analysis could not assess these factors, and incomplete reporting of missingness and exclusions further limits reproducibility.

The prevention implications should remain proportionate to the findings. This analysis did not establish a local temperature threshold for clinical action or evaluate a preventive intervention. However, evidence from external interventions supports assessing weather-informed preparedness. Hess et al. reported reduced heat-associated all-cause mortality after implementing Ahmedabad's heat action plan, although the observational design limits causal inference. For Lahore, prospective evaluation could examine whether weather surveillance, continuity of cardiovascular care, and hospital preparedness improve outcomes. These recommendations represent evaluation priorities rather than demonstrated benefits of the present study.

CONCLUSION

This retrospective multicenter observational time-series analysis of 12,917 cardiovascular admissions did not identify a clear association between low temperature and either total or diagnosis-specific admission counts. Similarly, analyses of extreme heat and heatwaves did not demonstrate increased admissions under the specified exposure definitions. Interpretation of the primary high-temperature comparisons was constrained because the estimated minimum-risk temperature coincided with the high-temperature comparison for total admissions, acute coronary syndrome, and arrhythmia. Consequently, these reference estimates cannot rule out heat-related risk.

Higher admission-day temperatures were associated with modestly longer hospital stays among survivors, while the relationship with in-hospital mortality remained inconclusive. Subgroup analyses did not identify differences in susceptibility by age or sex, and sensitivity analyses continued to show substantial uncertainty.

These findings require cautious interpretation due to imprecise estimates, boundary reference temperatures, limited hospital coverage, area-level exposure measurement, and potential residual confounding. Such methodological limitations restrict the generalizability of the results and underscore the need for further research with broader geographical coverage and more detailed clinical and environmental data.

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