World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

Check for updates

Heat-adapted family medicine: A comprehensive review of climate-related risks, chronic disease management, and primary care preparedness in hot regions

Hamad Abdulaziz AlSubaie *

Department of Family Medicine, King Abdulaziz Hospital for national Guard in AlAhsa, Saudi Arabia.

World Journal of Biology Pharmacy and Health Sciences, 2026, 26(02), 069-077

Publication history: Received on 01 April 2026; revised on 06 May 2026; accepted on 09 May 2026

Article DOI: https://doi.org/10.30574/wjbphs.2026.26.2.0253

Abstract

Background: The Arabian Peninsula and other hot regions of the world are warming at nearly twice the global average rate, exposing vulnerable populations to recurrent and prolonged periods of extreme heat. Family physicians sit at the front line of this emerging climate-health interface, yet structured guidance on heat-adapted primary care remains sparse in the medical literature.

Objective: This narrative literature review synthesizes contemporary evidence on the cardiovascular, renal, and metabolic consequences of extreme heat, examines the safety profile of commonly prescribed cardiometabolic medications during heatwaves, and proposes a practical, primary-care-oriented operational framework the HEAT-FM model to guide clinicians working in hot regions, with particular relevance to the Gulf Cooperation Council (GCC) context.

Methods: A structured literature review of English-language publications (2000–2025) was performed using PubMed, Scopus, and grey-literature sources from the World Health Organization (WHO), Centers for Disease Control and Prevention (CDC), and the Saudi Ministry of Health. Themes were synthesized around hypertension, chronic kidney disease (CKD), diabetes mellitus, medication safety, primary care preparedness, and Gulf-region epidemiology.

Results: Heat exposure aggravates hypertension through impaired diurnal blood pressure rhythm and increased nighttime values; precipitates acute-on-chronic kidney injury via volume depletion and vasopressin-mediated tubular stress; and worsens glycemic stability through dehydration, insulin pharmacokinetic alterations, and cognitive impairment of self-management. Diuretics, renin-angiotensin-aldosterone system (RAAS) blockers, sodium-glucose cotransporter-2 (SGLT2) inhibitors, metformin, and anticholinergic agents demand particular attention during heat events. Primary care preparedness remains underdeveloped, especially for the elderly, outdoor labourers, and Hajj pilgrims.

Conclusion: A structured, anticipatory primary-care response is needed. The proposed HEAT-FM framework High-risk identification, Education, Adjustment of medications, Timely follow-up, Family/community prevention, and Monitoring offers a memorable, clinically actionable scaffold for family physicians in hot regions. Embedding such frameworks within national heat-health action plans is essential as climate change accelerates.

Keywords: Extreme Heat; Family Medicine; Primary Care; Hypertension; Chronic Kidney Disease; Diabetes; Medication Safety; Saudi Arabia; Hajj; HEAT-FM

* Corresponding author: Hamad Abdulaziz AlSubaie

1. Introduction

The opening decades of the twenty-first century have brought a quiet but consequential shift to the consultation rooms of family physicians practicing across the Arabian Peninsula. Once-rare clinical scenarios the elderly hypertensive patient presenting with syncope after a brief walk to the mosque, the sugarcane labourer or construction worker with unexplained creatinine elevation, the well-controlled diabetic patient whose glycaemia drifts each summer are now familiar fixtures of the daily clinic [1,2]. They reflect a deeper truth: climate change is no longer a distant environmental concern but an active determinant of clinical outcomes, particularly in regions where ambient temperatures already approach the upper limits of human thermoregulatory tolerance [3].

The Arab region has warmed at roughly 0.43 °C per decade between 1991 and 2024 almost double the global mean and projections under high-emission scenarios suggest temperature increases of up to 5 °C by the end of the century [3,4]. Saudi Arabia, in particular, has experienced more frequent and prolonged heatwaves, with summer maxima regularly surpassing 50 °C in inland provinces such as Al-Ahsa, Riyadh, and the eastern desert belt [4,5]. This is not merely a climatic curiosity. The 2024 Hajj pilgrimage was marked by more than 1,300 confirmed deaths attributable to heat-related illness, a tragic underscore of how vulnerability, mass-gathering exposure, and physiological intolerance converge in extreme thermal environments [6,7].

For family physicians, the implications are clinical, organizational, and ethical. Most patients living with hypertension, diabetes, and chronic kidney disease in hot regions are managed in the primary care setting, where the average consultation must accommodate not only disease-specific guidelines but also the seasonal physiological fluctuations imposed by heat [8,9]. Many of the medications central to chronic-disease management diuretics, angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARBs), sodium-glucose cotransporter-2 (SGLT2) inhibitors, metformin, and anticholinergic agents interact with thermoregulation, fluid balance, or renal perfusion in ways that may amplify heat-related harm [10,11].

Despite this confluence of risk, the family medicine literature has been comparatively slow to articulate a structured, anticipatory model for heat-adapted primary care. National heat-health action plans, where they exist, have predominantly focused on emergency-department surge planning and public-health messaging rather than on integrated longitudinal care [12,13]. The Gulf Cooperation Council region despite being among the most heat-exposed in the world remains under-represented in peer-reviewed climate-health research [3,14].

This review has three aims. First, to synthesize contemporary evidence on the cardiovascular, renal, and metabolic effects of heat exposure relevant to primary care. Second, to examine the practical pharmacological challenges of managing chronic disease during heatwaves. Third, to propose a clinically intuitive, mnemonic-based operational framework HEAT-FM designed to help family physicians in hot regions deliver heat-adapted, anticipatory care. The discussion is grounded in international evidence but contextualized to the realities of Gulf practice, including the Hajj season, large outdoor migrant-labour cohorts, and the deeply embedded family structures that shape care-seeking behavior in Saudi society [6,15].

2. Methods

This narrative literature review was conducted between January and April 2026. PubMed, Scopus, and Web of Science were searched using combinations of the terms "extreme heat", "heatwave", "hypertension", "chronic kidney disease", "diabetes", "SGLT2", "ACE inhibitor", "diuretic", "primary care", "family medicine", "Saudi Arabia", "Gulf", and "Hajj". The search was limited to English-language publications between 2000 and 2025, with priority given to systematic reviews, large cohort studies, mechanistic clinical reviews, and authoritative grey literature from the WHO, CDC, US National Institute on Aging, the Saudi Ministry of Health, and the European Medicines Agency.

Articles were selected by relevance to primary care decision-making rather than by formal bibliometric criteria. Both human subject and translational mechanistic studies were included where they helped clarify physiological pathways. Reference lists of pivotal publications were hand-searched. The HEAT-FM framework was developed iteratively, drawing on operational lessons from existing heat-health action plans, standard chronic-disease guidelines, and the author's experience in family medicine practice in the Eastern Province of Saudi Arabia.

3. Literature Review

3.1. Pathophysiology of Heat Stress: A Brief Clinical Primer

Human core temperature is normally maintained within a narrow range of approximately 36.5–37.5 °C through a hierarchy of autonomic and behavioral responses. When ambient temperature rises, cutaneous vasodilation and eccrine sweating are the principal heat-loss mechanisms, both of which carry substantial cardiovascular and fluid-balance costs [16]. Cardiac output may double during heat stress, redistributing blood flow toward the skin and away from splanchnic and renal beds [16,17]. Sweat losses can exceed two liters per hour in unacclimatized individuals exposed to extreme dry heat, with attendant losses of sodium and chloride [16].

Once compensatory mechanisms are overwhelmed, the spectrum of heat-related illness progresses from heat oedema, cramps, and exhaustion to classic heatstroke, characterized by core temperature above 40 °C with central nervous system dysfunction [17,18]. Mortality in severe heatstroke approaches 27% even with optimal cooling, and survivors frequently sustain long-term neurologic, renal, or cardiac sequelae [17]. Importantly, the elderly, those with cardiovascular or renal disease, and individuals on multiple medications display blunted autonomic responses and reach decompensation at lower thermal loads [11,19].

3.2. Heat and Hypertension

The relationship between ambient temperature and blood pressure is more nuanced than the popular perception that "heat lowers blood pressure". While daytime systolic values do typically fall in summer due to peripheral vasodilation, nocturnal blood pressure tends to rise, and twenty-four-hour blood pressure variability an independent cardiovascular risk factor increases [20,21]. A large analysis of seasonal blood pressure control across United States health systems documented systolic pressure fluctuations of up to 10 mmHg between cold and warm seasons, with summer paradoxically associated with greater nighttime variability and reduced control rates in some elderly cohorts [20].

Several mechanisms underpin this complex pattern. Heat-induced volume depletion stimulates the renin-angiotensin-aldosterone axis, transiently raising vascular tone and renal sodium retention overnight [22]. Sleep disturbance during hot nights, common in Gulf summer climates without adequate cooling, blunts the physiological nocturnal "dip" and is itself a recognized predictor of cardiovascular events [20]. Additionally, dehydration concentrates erythrocytes and increases blood viscosity, raising both thrombotic risk and cardiac afterload [16,23].

For the family physician, the practical implication is that summer hypertension cannot be managed by reflexive dose reduction. Some patients particularly those on diuretics combined with RAAS blockers will indeed develop symptomatic hypotension, dizziness, and syncope during heat exposure and require dose adjustment. Others, especially elderly patients with disturbed sleep and high salt intake during prolonged Ramadan-Hajj summer overlap, will exhibit nocturnal hypertension that goes undetected without ambulatory or home blood pressure monitoring [9,20]. Clinical inertia in either direction can be harmful.

3.3. Heat and Chronic Kidney Disease

The kidney is among the organs most vulnerable to heat stress. Cutaneous vasodilation reduces renal blood flow by up to 25% during severe heat exposure, and concurrent volume depletion further compromises glomerular filtration [24]. Repeated subclinical episodes of heat-induced acute kidney injury (AKI) are now thought to drive a substantial portion of the global rise in chronic kidney disease of unknown origin (CKDu), also termed Mesoamerican nephropathy when described in Central American agricultural workers, but increasingly recognized in heat-exposed labourers in Sri Lanka, India, and the Arabian Peninsula [25,26].

A landmark NEJM perspective described the emerging "climate-medicine" paradigm in which heat-triggered renal injury becomes a prevalent occupational and public-health diagnosis [25]. Mechanistically, the chain is now well characterized: dehydration and hyperosmolality activate vasopressin and the polyol-fructose pathway, generating reactive oxygen species that injure the renal proximal tubule, while repeated AKI episodes accelerate fibrosis and progression to CKD [26,27]. A 2024 cohort study in The Lancet Planetary Health found that even modest increases in ambient heat exposure were associated with measurable declines in eGFR among patients with established CKD over follow-up periods of less than five years [28]. Older adults appear particularly vulnerable; a 2024 analysis demonstrated that prolonged heatwave exposure was associated with rapid kidney function decline in community-dwelling seniors [29].

For Gulf primary care, three populations stand out: outdoor labourers, particularly migrant workers in construction and agriculture; Hajj and Umrah pilgrims, especially those with pre-existing CKD; and elderly patients with multiple comorbidities and polypharmacy. Each warrants differentiated risk stratification.

3.4. Heat and Diabetes Mellitus

Patients with diabetes navigate heatwaves with a stacked deck. Autonomic neuropathy impairs sweating and cutaneous vasodilation, blunting heat dissipation [30]. Dehydration concentrates blood glucose and may precipitate hyperglycemic crises, including diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state, in patients who would otherwise be stable [30,31]. Conversely, reduced appetite, gastrointestinal symptoms, and altered insulin pharmacokinetics in hot environments insulin absorbs more rapidly from subcutaneous depots when skin blood flow is increased raise the risk of hypoglycemia, particularly in patients on basal-bolus regimens [30,32].

Heat also degrades self-management. Insulin pens and vials lose potency above 30 °C, glucometer accuracy diminishes outside 10–40 °C, and continuous glucose monitor adhesives fail at high humidity [31]. In a Japanese cohort, hospitalizations for hyperosmolar hyperglycemic crises increased significantly during periods of elevated daily maximum temperature, with the strongest effect in patients aged 65 years and above [32]. Similar patterns have been observed in Gulf cohorts during the peak July-August heat window [14].

3.5. Medication Safety During Heatwaves

Few areas of family medicine illustrate the climate-health interface more concretely than medication safety. The CDC's 2024 clinical guidance summarizes the principal mechanisms by which prescription drugs amplify heat risk: reduced thirst sensation, impaired sweating, altered renal handling of water and sodium, decreased cardiac reserve, and direct hyperthermic effects [10,33]. Five drug classes warrant particular vigilance.

3.5.1. Diuretics.

Loop and thiazide diuretics directly increase the risk of dehydration, hyponatremia, and pre-renal AKI during heat exposure. Population-based analyses have identified diuretic use as one of the strongest predictors of heat-related hospitalization in older adults [11,33]. Practical heatwave management includes patient-specific contingency plans for dose reduction or temporary withholding under sick-day rules, ideally formalized during routine review well before summer.

3.5.2. ACE inhibitors and ARBs

RAAS blockers reduce thirst sensation, blunt aldosterone-mediated sodium conservation, and impair renal autoregulation in volume-depleted states, precipitating AKI when superimposed on heat stress [10,11]. The combination of ACEi/ARB plus diuretic, common in heart failure and hypertension management, is particularly hazardous; a UK cohort demonstrated that this combination roughly doubled heat-associated hospitalization risk in those over 75 [11].

3.5.3. SGLT2 inhibitors

These agents have transformed the management of diabetes, heart failure, and CKD, but they cause an osmotic diuresis that can precipitate volume depletion and euglycemic DKA, particularly during fluid stress [34,35]. Sick-day rules temporary withholding during gastrointestinal illness, fever, dehydration, or prolonged fasting should explicitly include heat exposure as a trigger [34]. Patients should also be educated about euglycemic ketosis: nausea, abdominal pain, and tachypnoea can occur with normal blood glucose, particularly in hot weather and during Ramadan fasting [35].

3.5.4. Metformin

Although metformin remains a foundational agent, dehydration-induced acute kidney injury reduces its clearance and can precipitate metformin-associated lactic acidosis (MALA), a rare but high-mortality event [36]. In hot regions, instructing patients to temporarily withhold metformin during gastrointestinal illness or significant heat-related volume depletion is a low-cost safety measure.

3.5.5. Anticholinergic and psychotropic agents

Tricyclic antidepressants, first-generation antihistamines, antipsychotics, antiparkinsonian agents, and bladder antimuscarinics impair sweating, raise basal metabolic heat production, or interfere with hypothalamic

thermoregulation [33,37]. Cumulative anticholinergic burden common in elderly polypharmacy is associated with higher heat-related morbidity and mortality. Periodic deprescribing, particularly before summer, is therefore both geriatric and climate-adaptive medicine.

3.6. Primary Care Preparedness in Hot Regions

Despite WHO guidance on heat-health action planning, primary care preparedness remains uneven [12,13]. Surveys of clinicians in heat-exposed regions show low rates of formal heatwave protocols, limited use of risk stratification tools, and variable engagement with public-health early-warning systems [13,38]. A 2018 analysis of outpatient clinic visits during heatwaves found a measurable surge in consultations for cardiovascular, renal, and respiratory complaints, yet most clinics had not pre-emptively reorganized capacity [38].

Effective primary care preparedness incorporates four layers: clinic-level operational readiness (cooling, water, generator-supported electronic records, vaccine cold-chain integrity); patient-level risk stratification and education; pharmacological review; and coordination with public-health early-warning systems and community resources [12,13]. In Saudi Arabia, the Ministry of Health has introduced specific Hajj-season heat illness guidelines and public awareness campaigns; integrating these into routine family medicine workflows year-round, rather than only during pilgrimage, would substantially extend their benefit [6,15].

3.7. Gulf-Region Relevance

The Gulf Cooperation Council region presents a uniquely intense climate-health profile. Mean summer temperatures regularly exceed 45 °C, humidity along coastal areas pushes wet-bulb temperatures toward physiological survival limits, and demographic features include large migrant-labour populations, an ageing national citizenry, high prevalence of diabetes (estimated above 18% in Saudi adults), and the unparalleled mass-gathering exposure of the Hajj [4,6,14]. The 2024 Hajj heat disaster with more than 1,300 deaths, a substantial proportion among unregistered pilgrims who lacked access to air-conditioned accommodation and coordinated medical care has been described as a sentinel event for climate-medicine in the region [6,7].

A scoping review of heat-related health research in the Arabian Peninsula from 2010 to 2024 identified only twelve eligible studies, underscoring the underdevelopment of regional evidence [14]. Yet operational innovations exist: cooling stations, real-time mortuary surveillance, mass-cooling tents, and the deployment of pre-Hajj health screenings represent valuable, scalable practices that family physicians can integrate into standard care for high-risk patients during summer [6,15].

4. The HEAT-FM Framework

Building on the literature reviewed above, this article proposes the HEAT-FM framework a six-domain, mnemonic-based operational scaffold for family medicine practice in hot regions. The framework is intentionally simple, designed for memorization during clinical encounters, and adaptable across primary care contexts from urban academic clinics to rural and pilgrimage settings.

Table 1 The HEAT-FM Framework for Heat-Adapted Family Medicine

Letter	Domain	Practical primary-care actions
H	High-risk patient identification	Maintain a clinic-level registry of patients aged ≥65 years, those with CKD (eGFR <60), heart failure, uncontrolled diabetes, dementia, or psychiatric illness; outdoor labourers; pregnant women; pilgrims preparing for Hajj/Umrah. Apply heat-vulnerability scoring at annual review.
E	Education on hydration and warning symptoms	Counsel on individualized fluid targets (typically 2–3 L/day, adjusted for CKD and heart failure); avoidance of caffeine and sugary drinks; recognition of heat exhaustion (headache, dizziness, nausea, dark urine) and heatstroke (confusion, hot dry skin, syncope). Provide written, language-appropriate materials.
A	Adjustment of medications during heat exposure	Pre-summer medication review with explicit sick-day plans for diuretics, ACEi/ARBs, SGLT2 inhibitors, metformin, anticholinergics, and lithium. Provide patients with a written pause-and-resume rule for acute dehydrating illness or extreme heat.

T	Timely follow-up during heatwaves	Activate enhanced follow-up schedules during national heat alerts: telephone or telehealth check-ins for high-risk patients within 48–72 hours, urgent in-person review for symptomatic patients. Integrate clinic schedules with regional early-warning systems.
F	Family and community-level prevention	Engage family caregivers in heat-action plans; identify socially isolated elders for daily check-ins; coordinate with mosques, schools, and community centres on cooling spaces; advocate workplace heat-safety enforcement.
M	Monitoring chronic disease complications	Reinforce home BP and glucose monitoring; schedule baseline and post-summer renal function and electrolyte testing for at-risk patients; document and audit heat-related events to refine future preparedness.

The framework is intentionally aligned with existing guideline domains rather than replacing them. It can be embedded within annual chronic-disease care plans (e.g., the standardized Saudi Ministry of Health diabetes and hypertension templates) with minimal additional administrative burden. A pre-summer "heat-readiness review" of approximately fifteen minutes covering all six HEAT-FM domains can be appended to any chronic-disease follow-up visit between March and May.

5. Results

Synthesizing the reviewed literature yields several convergent findings of direct primary-care relevance.

First, heat exposure carries quantifiable cardiovascular morbidity. Heatwaves are consistently associated with excess hospitalizations and mortality from ischemic heart disease, heart failure decompensation, and stroke, with relative risks generally between 1.1 and 1.5 in the most vulnerable subgroups [1,16,17]. Twenty-four-hour blood pressure variability rises during summer, and hypertensive control rates measured at the clinic may misleadingly improve while nocturnal pressures worsen [20,21].

Second, the renal signal is robust and dose-dependent. Both occupational heat-stress nephropathy in young agricultural and construction workers and accelerated decline in established CKD patients are documented; even modest temperature elevations are associated with measurable eGFR loss in vulnerable cohorts [25,26,28,29]. Hospital data from heat-exposed regions show notable rises in emergency-department visits for AKI, urolithiasis, and CKD complications during heat events [29].

Third, heat amplifies the metabolic instability of diabetes. Hospitalization rates for hyperosmolar hyperglycemic crises and DKA rise during heatwaves; insulin storage and device performance are compromised; and the risk of both hypoglycemia and hyperglycemia is elevated, often within the same individual over a few days [30–32].

Fourth, medication safety is the principal modifiable mediator of heat-related primary-care harm. Diuretics, RAAS blockers, SGLT2 inhibitors, metformin, and anticholinergic agents collectively represent a substantial fraction of the prescriptions written in any chronic-disease clinic in the Gulf, and each interacts measurably with thermoregulatory or fluid-balance physiology [10,11,33–37]. Population analyses suggest that the combination of diuretic plus RAAS blocker confers up to a two-fold rise in heat-related hospitalization in the over-75s [11].

Fifth, the Gulf region is both highly exposed and underserved by climate-health research. The 2024 Hajj heat disaster crystallized this asymmetry: a high-mortality, predictable event in the world's largest annual mass-gathering, occurring in a country with sophisticated tertiary care but variable primary care heat-protocol penetration [6,7,14].

Sixth, primary care preparedness can be meaningfully advanced through structured frameworks. The HEAT-FM model proposed here translates the heterogeneous evidence base into a clinically usable mnemonic that can be operationalized in standard chronic-disease workflows without substantial additional resources [12,13].

6. Discussion

The findings of this review converge on a single clinical thesis: in hot regions, heat is not a seasonal background condition but an active, modifiable determinant of chronic-disease outcomes that family physicians must address proactively. The traditional structure of chronic-disease management guideline-based pharmacotherapy, periodic

monitoring, and patient education was designed for stable temperate environments. In a region where ambient temperatures regularly exceed 45 °C for several months a year, this structure requires recalibration.

A first implication concerns clinical inertia. Patients with hypertension whose summer office readings appear improved may in fact have worsened nocturnal control [20]; patients with diabetes whose A1c falls during hot months may be experiencing recurrent hypoglycemia rather than improved adherence [30]; patients with CKD whose creatinine is "stable" may be compensating for repeated subclinical heat-induced AKI episodes whose cumulative burden is invisible in single point-of-care measurements [25,28]. The HEAT-FM framework's emphasis on Monitoring (M) and Adjustment (A) is intended to disrupt this complacency.

A second implication concerns equity. Migrant workers, undocumented pilgrims, the socially isolated elderly, and patients of low health literacy bear a disproportionate share of heat-related harm [3,7,14]. Family medicine, by virtue of its longitudinal, relationship-based ethos and proximity to community, is uniquely positioned to identify and serve these populations provided heat is treated as a routine clinical variable rather than an episodic emergency.

A third implication is pharmacological. The drug classes that most powerfully reduce cardiovascular and renal morbidity in cool conditions diuretics, RAAS blockers, SGLT2 inhibitors are precisely the agents that most amplify heat-related risk [10,11,33–35]. This is not an argument against their use, but rather for nuanced, anticipatory deprescribing or dose adjustment during defined high-risk windows. The "sick-day rule" framework already familiar from SGLT2 and metformin guidance can be extended to heat exposure as a recognized trigger [34,36].

Fourth, primary care must engage proactively with public-health systems. National early-warning systems, when issued in isolation from clinical workflows, achieve limited behavior change [12,13]. Integration with electronic medical record alerts, automated outreach to high-risk registries, and family-medicine-led community education would substantially strengthen the population impact of existing heat-health action plans. In the Gulf context, partnerships with mosques, schools, and employer health services offer culturally embedded levers [6,15].

Fifth, the Hajj presents both a challenge and an opportunity. Pre-Hajj health screenings, already mandatory in many countries of origin, could be enriched with structured HEAT-FM-style preparation, particularly for elderly pilgrims with chronic disease [6]. Post-Hajj follow-up currently fragmented could be standardized in primary care to detect delayed heat-induced renal injury or cardiovascular decompensation.

Limitations of this review include its narrative methodology rather than a formal systematic protocol; the relative paucity of high-quality regional data from the Arabian Peninsula [14]; and the absence of a formal validation study of the HEAT-FM framework. Future research priorities include prospective evaluation of HEAT-FM implementation in Saudi family medicine clinics; cohort studies of CKD progression linked to objective ambient-temperature exposure data; pragmatic trials of pre-summer medication-adjustment protocols; and qualitative work exploring patient and caregiver perspectives on heat-adapted care, especially among female and elderly Saudi patients whose mobility and cooling access may be constrained.

7. Conclusion

Climate change is not an abstract environmental issue but a clinical reality that increasingly shapes the day-to-day work of family physicians, particularly in hot regions such as the Arabian Peninsula. Heat exposure aggravates hypertension, accelerates CKD, destabilizes diabetes, and amplifies the harms of several core cardiometabolic medications. Yet primary care preparedness the only level of the health system capable of intervening continuously and longitudinally remains under-developed in most of the world's hottest regions.

This review has synthesized the contemporary evidence and proposed the HEAT-FM framework as a memorable, practical operational scaffold for heat-adapted family medicine: High-risk identification, Education, Adjustment of medications, Timely follow-up, Family and community-level prevention, and Monitoring of chronic-disease complications. Embedded within existing chronic-disease workflows, integrated with national heat-health action plans, and tailored to the Gulf context including the Hajj season, such a framework offers a credible response to a rising clinical challenge.

The work of heat-adapted family medicine is, ultimately, the work of family medicine itself anticipatory, relational, longitudinal, and rooted in community applied to a warming world. As temperatures continue to rise across the Arabian Peninsula and beyond, this adaptation is no longer optional; it is a defining feature of competent primary care for the decades ahead.

Compliance with ethical standards

Acknowledgments

The author thanks colleagues in the Department of Family Medicine, King Abdulaziz Hospital, Al-Ahsa, for valuable clinical discussions that informed the development of the HEAT-FM framework

References

[1] Centers for Disease Control and Prevention. Clinical overview of heat and cardiovascular disease. Atlanta: CDC; 2024.

[2] Mayo Clinic Health System. Effects of high temperatures on blood pressure and the heart. Rochester (MN): Mayo Clinic; 2023.

[3] World Meteorological Organization. Temperature increase is accelerating in the Arab region with escalating impacts. Geneva: WMO; 2024.

[4] World Bank Group. Saudi Arabia – mean climate projections (CMIP6). Climate Change Knowledge Portal; 2024.

[5] Almazroui M, Saeed F, Saeed S, Ismail M, Ehsan MA, Islam MN, et al. Data-driven analysis of climate change in Saudi Arabia. J Appl Meteorol Climatol. 2021;60(8):1145–60.

[6] Khan AA, Sabbagh AY, Ranse J, Molloy MS, Ciottone GR. Escalating climate-related health risks for Hajj pilgrims to Mecca. J Travel Med. 2024;31(6):taae096.

[7] Ebrahim SH, Memish ZA. Hajj 2024 heatwave: addressing health risks and safety. Lancet. 2024;404(10449):231–2.

[8] Vashishtha D, Sieber W, Hailey B, Guirguis K, Gershunov A, Al-Delaimy WK. Outpatient clinic visits during heat waves: findings from a large family medicine clinical database. Fam Pract. 2018;35(5):567–70.

[9] Kovats RS, Hajat S. Heat stress and public health: a critical review. Annu Rev Public Health. 2008;29:41–55.

[10] Centers for Disease Control and Prevention. Heat and medications – guidance for clinicians. Atlanta: CDC; 2024.

[11] Layton JB, Li W, Yuan J, Gilman JP, Horton DB, Setoguchi S. Heatwaves, medications, and heat-related hospitalization in older Medicare beneficiaries with chronic conditions. PLoS One. 2020;15(12):e0243665.

[12] World Health Organization. Heat and health. Fact sheet. Geneva: WHO; 2024.

[13] World Health Organization Regional Office for Europe. Planning heat–health action: guidance for public health institutions. Copenhagen: WHO; 2021.

[14] Alahmad B, Khraishah H, Althalji K, Borchert W, Al-Mulla F, Koutrakis P. Overheated and understudied: a scoping review of heat-related health research in the Arabian Peninsula. GeoHealth. 2024;8(7):e2024GH001277.

[15] Saudi Ministry of Health. Guidelines for management of heat illnesses during Hajj. Riyadh: MOH; 2023.

[16] Ebi KL, Capon A, Berry P, Broderick C, de Dear R, Havenith G, et al. Hot weather and heat extremes: health risks. Lancet. 2021;398(10301):698–708.

[17] Epstein Y, Yanovich R. Heatstroke. N Engl J Med. 2019;380(25):2449–59.

[18] Bouchama A, Knochel JP. Heat stroke. N Engl J Med. 2002;346(25):1978–88.

[19] Kenny GP, Yardley J, Brown C, Sigal RJ, Jay O. Heat stress in older individuals and patients with common chronic diseases. CMAJ. 2010;182(10):1053–60.

[20] Modesti PA, Morabito M, Massetti L, Rapi S, Orlandini S, Mancia G, et al. Seasonal blood pressure changes: an independent relationship with temperature and daylight hours. Hypertension. 2013;61(4):908–14.

[21] Narita K, Hoshide S, Kario K. Seasonal variation in blood pressure: current evidence and recommendations for hypertension management. Hypertens Res. 2021;44(11):1363–72.

[22] Stocker SD, Lang SM, Simmonds SS, Wenner MM, Farquhar WB. Cerebrospinal fluid hypernatremia elevates sympathetic nerve activity and blood pressure via the rostral ventrolateral medulla. Hypertension. 2015;66(6):1184–90.

[23] Keatinge WR, Coleshaw SR, Easton JC, Cotter F, Mattock MB, Chelliah R. Increased platelet and red cell counts, blood viscosity, and plasma cholesterol levels during heat stress. Am J Med. 1986;81(5):795–800.

[24] Schlader ZJ, Chapman CL, Sarker S, Russo L, Rideout TC, Parker MD, et al. Firefighter work duration influences the extent of acute kidney injury. Med Sci Sports Exerc. 2017;49(8):1745–53.

[25] Glaser J, Lemery J, Rajagopalan B, Diaz HF, García-Trabanino R, Taduri G, et al. Climate change and the emergent epidemic of CKD from heat stress in rural communities: the case for heat stress nephropathy. Clin J Am Soc Nephrol. 2016;11(8):1472–83.

[26] Johnson RJ, Wesseling C, Newman LS. Chronic kidney disease of unknown cause in agricultural communities. N Engl J Med. 2019;380(19):1843–52.

[27] Roncal-Jimenez CA, García-Trabanino R, Barregard L, Lanaspa MA, Wesseling C, Harra T, et al. Heat stress nephropathy from exercise-induced uric acid crystalluria: a perspective on Mesoamerican nephropathy. Am J Kidney Dis. 2016;67(1):20–30.

[28] Borg MA, Bi P, Nitschke M, Williams S, McDonald S. Ambient heat exposure and kidney function in patients with chronic kidney disease: a longitudinal cohort study. Lancet Planet Health. 2024;8(3):e156–66.

[29] Liu J, Varghese BM, Hansen A, Borg MA, Zhang Y, Driscoll T, et al. Association between heatwave exposure and rapid kidney function decline in older adults. Environ Health Perspect. 2024;132(4):047008.

[30] Kenny GP, Sigal RJ, McGinn R. Body temperature regulation in diabetes. Temperature (Austin). 2016;3(1):119–45.

[31] American Diabetes Association. Managing diabetes in the heat. Diabetes Care. 2024;47(Suppl 1):S1–S5.

[32] Isaka Y, Hayashi K, Aonuma K, Horio T, Terauchi Y, Yamada Y, et al. Association between heat exposure and hospitalization for diabetic ketoacidosis and hyperglycaemic crisis. Environ Int. 2022;167:107410.

[33] Westaway K, Frank O, Husband A, McClure A, Shute R, Edwards S, et al. Medicines can affect thermoregulation and accentuate the risk of dehydration and heat-related illness during hot weather. J Clin Pharm Ther. 2015;40(4):363–7.

[34] Goldenberg RM, Berard LD, Cheng AYY, Gilbert JD, Verma S, Woo VC, et al. SGLT2 inhibitor-associated diabetic ketoacidosis: clinical review and recommendations for prevention and diagnosis. Clin Ther. 2016;38(12):2654–64.

[35] Diaz-Ramos A, Eilbert W, Marquez D. Euglycemic diabetic ketoacidosis associated with sodium-glucose cotransporter-2 inhibitor use: a case report and review of the literature. Int J Emerg Med. 2019;12(1):27.

[36] Lalau JD, Kajbaf F, Protti A, Christensen MM, De Broe ME, Wiernsperger N. Metformin-associated lactic acidosis: current perspectives on causes and risk. Metabolism. 2017;77:26–34.

[37] Stollberger C, Lutz W, Finsterer J. Heat-related side-effects of neurological and non-neurological medication may increase heatwave fatalities. Eur J Neurol. 2009;16(7):879–82.

[38] Boeckmann M, Rohn I. Is planned adaptation to heat reducing heat-related mortality and illness? A systematic review. BMC Public Health. 2014;14:1112.