

Research Article

Health effects of heat exposure and air pollution in general medical practice

Emmanuel Santos ^{1*}, Maria Clarissa Recto ²¹ Adventist University of the Philippines College of Medicine, Philippines² Isabela State University College of Medicine, Philippines* Correspondence: santos_e09872@gmail.com<https://doi.org/10.59652/fjapkt79>

Abstract: Heat exposure and air pollution are no longer peripheral public health concerns; they are routine clinical exposures that affect patients seen in general medical practice. Adults with cardiovascular disease, chronic respiratory disease, chronic kidney disease, diabetes, frailty, severe mental illness, pregnancy, outdoor work exposure, poor housing, and limited access to cooling are at particular risk. Heat can precipitate heat exhaustion, heatstroke, dehydration, electrolyte disorders, acute kidney injury, syncope, falls, myocardial infarction, arrhythmia, heart failure decompensation, stroke, respiratory deterioration, and medication-related harm. Air pollution, especially fine particulate matter, ozone, and nitrogen dioxide, contributes to cardiovascular events, respiratory exacerbations, daily mortality, asthma attacks, chronic obstructive pulmonary disease exacerbations, and wider cardiometabolic risk. The combined exposure of hot weather and polluted air is clinically important because heat can increase ozone formation, wildfire smoke can raise particulate exposure, and physiological stress from heat may lower the reserve needed to tolerate cardiorespiratory insults. This review examines the health effects of heat exposure and air pollution in general medical practice, with emphasis on climate-sensitive illness, cardiovascular events, respiratory exacerbations, kidney injury, and medication safety during heatwaves. PubMed-indexed evidence from systematic reviews, meta-analyses, cohort studies, clinical reviews, and pharmacoepidemiological studies was reviewed. A practical clinical approach should include seasonal risk identification, anticipatory counselling, medication review, kidney and electrolyte risk assessment, respiratory action planning, cardiovascular safety advice, and clear thresholds for urgent assessment. Heat and air pollution should be treated as modifiable exposure risks in routine care. General practitioners are well placed to identify vulnerable patients before extreme weather occurs, rather than only managing the clinical consequences after exposure.

Keywords: Heat exposure, air pollution, heatwaves, climate-sensitive illness, cardiovascular events, respiratory exacerbations, acute kidney injury, medication safety, PM2.5, ozone, general practice

Introduction

General medical practice is increasingly being asked to manage illness shaped by the physical environment. A patient with heart failure becomes dizzy during a heatwave after continuing high-dose diuretics. A patient with chronic obstructive pulmonary disease develops breathlessness during a wildfire smoke episode. An older adult living alone presents with confusion and acute kidney injury after several hot nights. A patient with asthma has repeated reliever use during high ozone days. These cases do not arrive labelled as climate-sensitive illness. They arrive as ordinary consultations, emergency calls, and hospital admissions.

The clinical problem is that heat exposure and air pollution are usually treated as background conditions rather than active risk factors. The weather is noted, but not entered into the diagnostic reasoning. The air quality warning is seen as public advice, not as part of chronic disease care. Medication lists are reviewed for renal function, falls, and interactions, but not always for hot weather.

Received: 15 February 2026

Accepted: 15 June 2026

Published: 28 June 2026

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This needs to change. The 2024 Lancet Countdown reported that heat-related mortality in people older than 65 years increased by 167% compared with the 1990s, and that several climate-related health indicators had reached record levels (1). Large multi-country analyses have also shown that non-optimal temperatures contribute substantially to mortality, even though the distribution of risk varies by region and by heat versus cold exposure (3,4). Ambient air pollution remains one of the major environmental causes of morbidity and mortality worldwide, with particulate matter and gaseous pollutants linked to cardiovascular and respiratory harm (18-23).

In general practice, the question is not whether climate change exists. The question is how to care for patients whose health risks are already modified by heat, humidity, smoke, ozone, particulate matter, housing, occupation, medication exposure, and chronic disease. This review focuses on that clinical question.

Review frame

This article is a narrative literature review using PubMed-indexed sources. The literature base included systematic reviews, meta-analyses, cohort studies, large time-series analyses, clinical reviews, and pharmacoepidemiological studies relevant to heat exposure, air pollution, cardiovascular outcomes, respiratory exacerbations, kidney injury, and medication safety.

Search concepts included heat exposure, heatwaves, high ambient temperature, heat-stroke, cardiovascular events, respiratory admissions, chronic obstructive pulmonary disease exacerbation, asthma exacerbation, acute kidney injury, kidney disease, particulate matter, PM2.5, ozone, nitrogen dioxide, medication safety, diuretics, anticholinergic drugs, psychotropics, antihypertensives, and general practice. Only PubMed-indexed references were retained in the final reference list.

The article is written for general medical practice. It therefore gives priority to clinical recognition, vulnerable groups, medication review, prevention, and referral thresholds rather than atmospheric science or climate modelling.

Heat exposure as a clinical stress test

Heat harms health through direct thermoregulatory failure and through decompensation of chronic disease. Heat illness exists on a spectrum from heat cramps and heat exhaustion to heatstroke. Heatstroke is the most severe presentation and is characterised by marked hyperthermia with central nervous system dysfunction; rapid recognition and cooling are central to survival (8,9). Yet most heat-related harm in general medicine is not classic heatstroke. It is dehydration, syncope, electrolyte imbalance, renal impairment, delirium, cardiac decompensation, and worsening respiratory disease.

The body responds to heat by increasing skin blood flow and sweating. These mechanisms protect core temperature, but they require cardiovascular reserve, fluid availability, renal adaptation, intact sweating, and adequate behavioural response. Older adults have reduced thermoregulatory reserve. Patients with heart failure may not tolerate peripheral vasodilation or fluid shifts. Patients with chronic kidney disease may not compensate safely for dehydration. Patients with dementia, psychosis, alcohol dependence, disability, or social isolation may not recognise danger or act early.

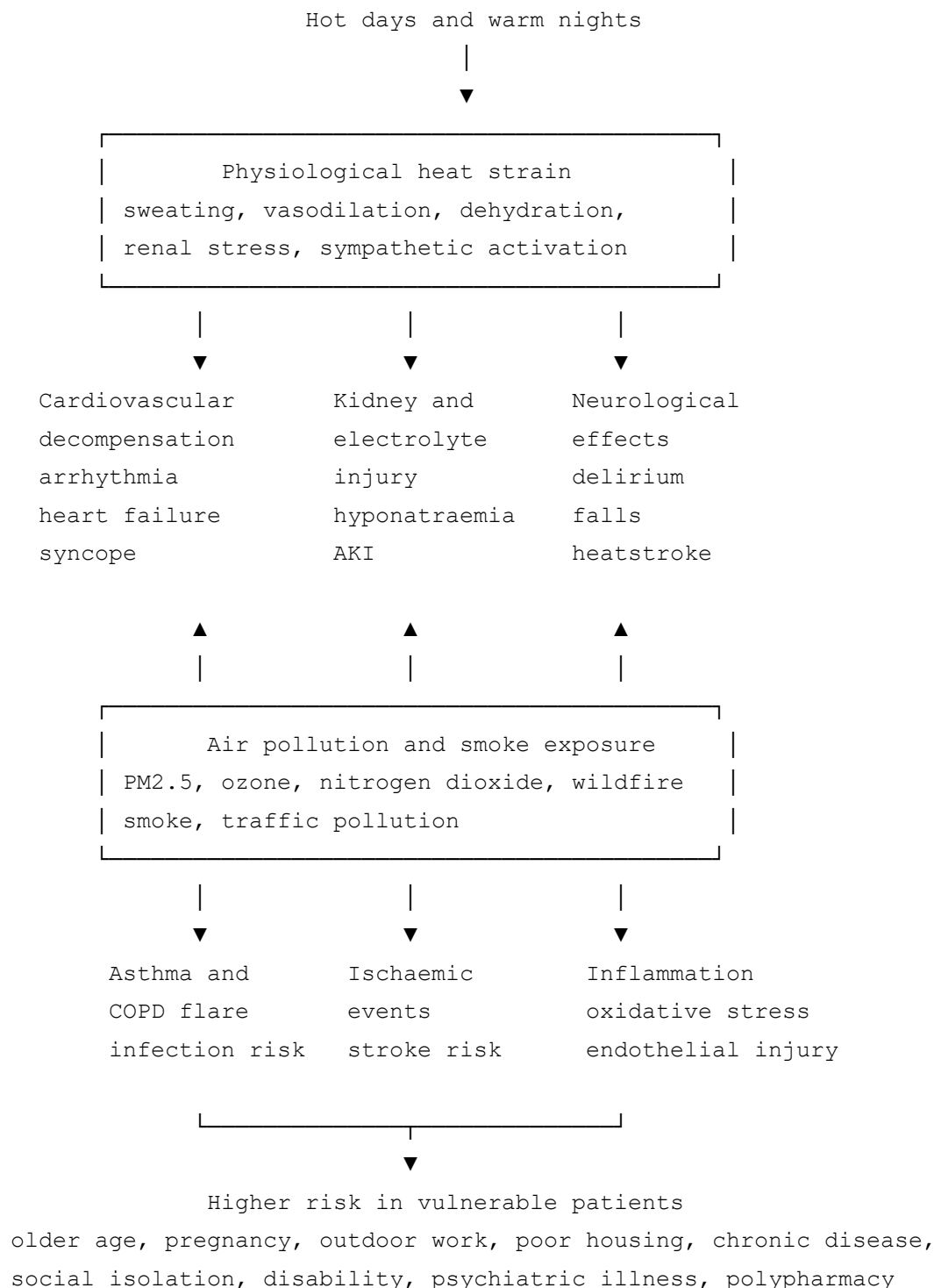
Extreme heat also changes how chronic disease is experienced. A patient with stable angina may become symptomatic because heat increases cardiovascular strain. A patient with hypertension may become hypotensive if dehydrated or if antihypertensive treatment is too intensive for the exposure conditions. A patient with diabetes may be more vulnerable to dehydration, infection, renal injury, and medication storage problems. A patient with chronic respiratory disease may breathe faster in hot conditions and may be simultaneously exposed to ozone or smoke.

A large study of older adults in the United States found that periods of extreme heat were associated with increased risk of hospitalisation for fluid and electrolyte disorders, renal failure, urinary tract infection, septicaemia, and heat stroke (5). This pattern is familiar in

practice. Heat rarely attacks only one organ system. It exposes the weak points in the patient's physiology and living conditions.

Figure 1

The environmental exposure web in general practice



Air pollution as a daily clinical exposure

Air pollution is a mixture of particulate and gaseous exposures. Fine particulate matter with aerodynamic diameter of 2.5 µm or less is clinically important because it can penetrate deep into the lungs and is associated with systemic effects. Ozone is a secondary pollutant

formed through photochemical reactions and can rise during hot, sunny conditions. Nitrogen dioxide is strongly related to combustion sources, especially traffic. Wildfire smoke can produce acute peaks in particulate matter and irritant gases.

The cardiovascular evidence for particulate pollution is mature. The American Heart Association scientific statement concluded that the evidence supports a causal relationship between PM_{2.5} exposure and cardiovascular morbidity and mortality (20). A later JACC review described PM_{2.5} as a major environmental risk factor for global cardiovascular mortality and disability, with short-term increases linked to acute cardiovascular events and longer-term exposure linked to larger cumulative risk (21).

Large epidemiological studies support these conclusions. In a study of 652 cities, short-term exposure to PM₁₀ and PM_{2.5} was independently associated with daily all-cause, cardiovascular, and respiratory mortality (19). In the United States Medicare population, long-term exposure to PM_{2.5} and ozone was associated with increased all-cause mortality, including at levels below some regulatory standards at the time (18). Nitrogen dioxide has also been linked to mortality in multi-city analyses; a 398-city study found that a 10 µg/m³ increase in NO₂ on the previous day was associated with increases in total, cardiovascular, and respiratory mortality (23).

Respiratory effects are clinically visible. Air pollution can aggravate asthma, chronic obstructive pulmonary disease, rhinitis, respiratory infection risk, and exercise limitation. A systematic review and meta-analysis found that selected outdoor pollutants were associated with asthma exacerbations in children and adults, including significant associations for PM_{2.5}, nitrogen dioxide, carbon monoxide, and ozone (24). Another meta-analysis reported that short-term exposure to major air pollutants increased the burden of acute COPD exacerbations (25).

The general practitioner does not need to measure individual inhaled dose. The practical task is to identify patients for whom a high pollution day is not just uncomfortable but clinically dangerous.

Chart 1

Two-exposure risk field for hot and polluted days

		Air pollution level	
		lower	higher
lower heat		Baseline zone	Irritant zone
		Chronic disease care as usual	Asthma, COPD, angina, outdoor exertion risk
higher heat		Heat strain zone	Compound exposure zone
		dehydration, falls, AKI, syncope	heat illness plus respiratory and cardiac exacerbation risk

This chart is intended for clinical risk framing. Many dangerous days fall in the compound exposure zone, especially during heatwaves with ozone elevation, wildfire smoke, stagnant air, or poor indoor cooling.

Cardiovascular events during heat and pollution exposure

Cardiovascular disease is one of the main pathways through which both heat and air pollution increase morbidity and mortality. Heat exposure increases skin blood flow, heart rate, plasma volume stress, and myocardial workload. Dehydration can increase blood viscosity and reduce renal perfusion. In susceptible patients, these changes may contribute to myocardial infarction, arrhythmia, heart failure, stroke, hypotension, and syncope.

A systematic review and meta-analysis of heat exposure and cardiovascular outcomes found elevated risk of cardiovascular morbidity and mortality, with greater vulnerability in older adults, women, and people living in tropical or subtropical climate zones (6). Studies of cause-specific cardiovascular mortality also show that extreme temperature associations vary by disease subtype and population vulnerability (7).

Air pollution acts through overlapping but distinct mechanisms: autonomic imbalance, endothelial dysfunction, oxidative stress, vascular inflammation, thrombosis, atherosclerotic progression, and plaque instability (20,21). Short-term increases in PM_{2.5} can trigger acute cardiovascular events in susceptible individuals, while long-term exposure contributes to the development and progression of cardiovascular disease.

The combined exposure of heat and air pollution is especially relevant to cardiovascular practice. Hot sunny days can increase ozone formation, while stagnant air can trap pollutants. A study of ozone and heatwave exposure found that combined exposure contributed to cardiovascular mortality, with older adults and women more vulnerable (28). The clinical implication is simple: a high-temperature day with poor air quality should be treated as higher risk than a hot day alone for patients with coronary disease, heart failure, arrhythmia, previous stroke, or resistant hypertension.

General practice can intervene before the emergency. Patients with heart failure need advice on weighing, fluid balance, warning signs, and when to seek review. Patients on diuretics, renin-angiotensin system blockers, sodium-glucose cotransporter-2 inhibitors, lithium, or nephrotoxic medicines may need specific sick-day and hot-day planning. Patients with angina should be advised to avoid outdoor exertion during peak heat and pollution. Patients with atrial fibrillation or anticoagulation should understand dehydration and fall risk.

Respiratory exacerbations and polluted heat

Respiratory patients are often the first to notice polluted air. They report chest tightness, cough, wheeze, sputum change, reduced walking distance, nocturnal symptoms, or increased reliever use. During hot weather, respiratory burden may rise through several routes: ozone exposure, particulate matter, wildfire smoke, increased ventilation rate, dehydration of airways, indoor mould after humid periods, and avoidance of usual physical activity.

Asthma exacerbation data are consistent enough to support practical advice. Outdoor air pollution has been associated with moderate and severe asthma exacerbations in systematic review evidence (24). Short-term exposure to ozone, nitrogen dioxide, and sulphur dioxide has also been associated with asthma-related emergency visits and hospital admissions (29). For COPD, short-term exposure to air pollutants increases exacerbation risk, and PM_{2.5} exposure has been associated with COPD hospitalisations and emergency events (25,26).

Heat may worsen respiratory risk directly and indirectly. Hot air can increase ventilatory demand. Ozone is more likely to rise on hot, sunny days. Wildfire events often bring both heat and smoke. Power failure during heatwaves can stop home nebulisers, oxygen concentrators, fans, and air conditioning. Patients may keep windows open for cooling and thereby increase smoke or pollution exposure.

Respiratory planning should therefore be seasonal. Patients with asthma and COPD should have an action plan before high-risk months. Inhaler technique, controller adherence, rescue medication availability, spacer use, and vaccination status should be reviewed. Patients using home oxygen or nebuliser equipment need a power-outage plan. During smoke or high

ozone days, advice may include staying indoors in a cooler clean-air room, reducing outdoor exertion, using air filtration where available, and seeking early review if reliever need increases.

Kidney injury and electrolyte disorders

The kidney is a sensitive indicator of heat vulnerability. Heat exposure can reduce effective circulating volume, increase sweating losses, concentrate urine, worsen nephrolithiasis risk, and precipitate acute kidney injury. In patients with chronic kidney disease, diabetes, heart failure, older age, diuretic use, non-steroidal anti-inflammatory drug use, or renin-angiotensin system blockade, the margin of safety can be narrow.

High temperature has been associated with kidney disease outcomes. A systematic review found that high temperatures and heatwaves were associated with elevated risk of kidney disease outcomes, particularly urolithiasis (12). An analysis of emergency department visits found dose-dependent associations between extreme heat exposure and kidney disease visits (13). Occupational heat exposure has also been linked to kidney injury and disease, especially in workers performing physically demanding labour in hot environments (11). Mechanistic reviews describe heat stress, muscle injury, reduced renal blood flow, dehydration, and fructose metabolism as possible contributors to kidney inflammation and injury (14).

General medical practice should treat heat as a kidney risk modifier. A patient with estimated glomerular filtration rate of 45 mL/min/1.73 m² may be stable in winter and unstable during a heatwave if fluid intake falls, sweating rises, and diuretic or NSAID exposure continues. A patient with recurrent stones may have higher risk during hot weather because of lower urine volume. Outdoor workers may repeatedly experience subclinical dehydration and kidney stress.

The practical response is targeted. Patients with chronic kidney disease, heart failure, diabetes, older age, or previous AKI should receive hot-weather advice. This includes maintaining safe fluid intake according to their condition, avoiding unnecessary NSAIDs, recognising dizziness or reduced urine output, and seeking timely testing if unwell. For some patients, a heatwave medication plan should be documented before the season starts.

Table 1 Clinical manifestations by exposure route and time course

Exposure route	Hours to days	Days to weeks	Clinical notes for general practice
Direct heat exposure	Heat exhaustion, syncope, dehydration, heatstroke, delirium	Functional decline, poor sleep, falls, decompensation of chronic disease	Ask about indoor temperature, cooling access, fluid intake, and living alone
Hot nights	Poor sleep, fatigue, worsening cognition	Higher cumulative stress in older adults and chronic disease	Night-time heat may be missed if only daytime temperature is considered
PM2.5 exposure	Wheeze, dyspnoea, chest tightness, angina, arrhythmia symptoms	Increased cardiorespiratory events and healthcare use	Wildfire smoke can create rapid spikes even outside usual pollution seasons
Ozone exposure	Asthma flare, cough, reduced exercise tolerance	Recurrent symptoms during hot sunny periods	Patients may need outdoor activity timing advice
Nitrogen dioxide exposure	Airway irritation, asthma/COPD worsening	Higher respiratory and cardiovascular mortality associations in population studies	Traffic exposure matters for commuters and outdoor workers

Exposure route	Hours to days	Days to weeks	Clinical notes for general practice
Heat plus pollution	Mixed heat strain and cardiorespiratory symptoms	Compound risk in frail and multimorbid patients	Treat as a higher-risk exposure state, not two separate mild risks
Heat plus medication vulnerability	Hypotension, AKI, hyponatraemia, falls, confusion	Recurrent summer admissions if not recognised	Review diuretics, anticholinergics, psychotropics, NSAIDs, antihypertensives

Medication safety during heatwaves

Medication safety is one of the most practical areas where general practice can prevent harm. Medicines can increase heat-related risk through several mechanisms: impaired sweating, reduced thirst, sedation, cognitive impairment, volume depletion, electrolyte imbalance, hypotension, reduced cardiac output, impaired renal perfusion, and altered drug storage.

A clinical pharmacy review described how medicines may accentuate dehydration and heat-related illness, especially in older people taking multiple medicines, and highlighted diuretics, anticholinergics, psychotropics, and combinations such as diuretics with ACE inhibitors or angiotensin receptor blockers as important concerns (15). In older Medicare beneficiaries with chronic conditions, heatwaves were associated with a 21% to 33% increase in heat-related hospitalisations across medication classes; the study also found that potentially heat-sensitising medicines were common and that risk existed throughout the warm season, not only during formal heatwaves (16).

Experimental evidence is more selective. A 2024 systematic review and meta-analysis of medications and core temperature during heat stress found moderate-quality evidence that drugs with high anticholinergic properties increased core temperature at air temperatures of 30°C or higher and reduced sweating. Non-selective beta-blockers, adrenaline, and anti-Parkinson agents also increased core temperature in the analysed studies, while evidence for several other drug classes was limited and often based on healthy young men rather than older multimorbid patients (17). This nuance matters. Clinicians should not make broad, automatic medication changes during hot weather. They should identify plausible risk, individualise advice, and avoid abrupt discontinuation of essential medicines.

Anticholinergic burden is a recurrent concern. During heatwaves, drugs with anticholinergic effects may reduce sweating and impair cognition. A study of older hospitalised adults found that exposure to drugs with anticholinergic effects during heatwave periods was associated with differences in length of stay, although not mortality (18). Psychotropic drugs also need attention because they may impair alertness, behavioural response, sweating, or central thermoregulation, and because severe mental illness itself is associated with higher heat vulnerability.

Medication storage is another neglected issue. Insulin, some injectables, inhalers, and other medicines may be affected by high temperatures. Patients who travel, live without reliable cooling, or experience power outages may need specific storage advice.

Table 2 Medication review during hot weather

Medicine or group	Heat-related concern	Suggested clinical response
Loop and thiazide diuretics	Volume depletion, hypotension, AKI, electrolyte disturbance	Review indication, dose, recent renal function, weight plan, and fluid advice

Medicine or group	Heat-related concern	Suggested clinical response
ACE inhibitors and ARBs	AKI risk during dehydration, hypotension when volume depleted	Provide sick-day and hot-day advice rather than automatic stopping
SGLT2 inhibitors	Volume depletion risk, genital infection risk, ketoacidosis concern during illness	Reinforce hydration, sick-day rules, and early review if unwell
NSAIDs	Reduced renal perfusion, AKI risk during dehydration	Avoid unnecessary use in heat-vulnerable patients
Anticholinergic medicines	Reduced sweating, urinary retention, confusion, heat intolerance	Reduce avoidable anticholinergic burden where clinically possible
Antipsychotics and sedatives	Impaired thermoregulation, sedation, reduced behavioural response	Review necessity, dose, support, hydration, and monitoring
Beta-blockers	Reduced cardiac output and possible thermoregulatory effects, especially non-selective agents	Avoid abrupt cessation; review symptoms, blood pressure, and heart rate
Lithium	Toxicity risk with dehydration and renal impairment	Provide clear fluid, illness, and testing advice
Insulin and temperature-sensitive medicines	Reduced potency if overheated, storage failure during power cuts	Provide practical storage plan and backup cooling arrangements
Laxatives	Dehydration and electrolyte disturbance if overused	Review dose in frail adults and those with kidney disease

Who is most vulnerable

Vulnerability is not only biological. It is also social and environmental. Older adults, infants, pregnant women, outdoor workers, people with chronic disease, people with disability, people living alone, people with severe mental illness, people with substance use disorders, and people without access to cooling are repeatedly identified as higher-risk groups (2,30). Poor housing, top-floor rooms, urban heat islands, low tree cover, homelessness, poverty, and inability to leave unsafe indoor temperatures can all convert hot weather into clinical danger.

The 2021 western North America heat dome showed the importance of indoor temperature, social vulnerability, age, and neighbourhood conditions. A study of community deaths in greater Vancouver found that heat dome mortality was associated with deprivation, lower neighbourhood greenness, older age, and sex; high indoor temperatures likely played an important role (30). This type of evidence is highly relevant to general practice. Patients rarely volunteer that their home becomes dangerously hot. Clinicians may need to ask.

A heat-risk history can be brief:

Can the patient keep one room cool?
Do they live alone?
Can they drink safely, or are fluids restricted?
Do they have heart failure, kidney disease, dementia, severe mental illness, COPD, asthma, diabetes, or mobility limitation?
Do they work outdoors?

Do they use diuretics, anticholinergics, psychotropics, NSAIDs, lithium, insulin, or multiple antihypertensives?
Do they understand when to seek help?

This is not social work replacing medicine. It is medicine recognising exposure.

Figure 2 Seasonal preparation model for general practice

BEFORE HOT SEASON

Identify high-risk patients
Review chronic disease control
Check medication risk
Update asthma/COPD and heart failure plans
Discuss cooling and social support



WHEN HEAT OR POLLUTION ALERT IS ISSUED

Send targeted advice where possible
Reduce outdoor exertion
Use clean/cool indoor space
Check vulnerable patients living alone
Clarify fluid and medication instructions



DURING CLINICAL CONTACT

Ask about exposure, indoor heat, air quality, hydration
Look for AKI, hypotension, electrolyte disturbance, hypoxia
Assess respiratory and cardiovascular decompensation
Treat heat illness urgently when suspected



AFTER THE EVENT

Review admissions, near misses, medication harms
Update patient-specific plans
Record vulnerability in the medical file
Improve recall list for next alert

General practice assessment during hot or polluted periods

Clinical assessment should integrate exposure history with ordinary medical reasoning.
In hot weather, a patient with dizziness should be asked about fluid intake, indoor

temperature, sweating, urine output, diuretics, antihypertensives, alcohol, diarrhoea, and time spent outdoors. A patient with confusion should be assessed for heat illness, dehydration, infection, medication toxicity, hyponatraemia, and renal impairment. A patient with breathlessness should be asked about air quality, smoke exposure, inhaler use, oxygen equipment, chest pain, and heart failure symptoms.

Vital signs can mislead if not interpreted in context. Fever may be absent in heat exhaustion. Skin may not always be dry. Older adults may present with weakness or delirium rather than classic symptoms. A normal clinic oxygen saturation does not exclude an impending asthma or COPD exacerbation. Blood pressure may be lower than the patient's usual level even if it appears acceptable.

Basic investigations often include renal function, electrolytes, glucose, full blood count, urinalysis where appropriate, ECG for cardiovascular symptoms, and oxygen saturation for respiratory symptoms. In suspected heatstroke, urgent transfer and immediate cooling should not wait for laboratory confirmation. In patients with COPD or asthma, peak flow or spirometry may be useful outside acute severe distress, but treatment should not be delayed when clinical severity is clear.

Table 3 Clinical red flags during heat or pollution events

Presentation	Red flags	Likely action
Heat exposure	Confusion, collapse, seizure, core temperature concern, anhidrosis or severe hyperthermia	Emergency assessment and active cooling
Cardiovascular symptoms	Chest pain, syncope, new arrhythmia, severe hypotension, acute heart failure signs	Urgent ECG, acute care referral, fluid status assessment
Respiratory symptoms	Severe breathlessness, cyanosis, silent chest, inability to speak, low oxygen saturation	Emergency asthma/COPD pathway and referral
Kidney risk	Oliguria, rising creatinine, severe dehydration, hyperkalaemia, hyponatraemia	Same-day blood tests or acute care depending severity
Neurological symptoms	New delirium, severe weakness, falls, reduced consciousness	Assess for heat illness, infection, electrolyte disorder, medication toxicity
Medication-related concern	Lithium toxicity symptoms, excessive diuresis, NSAID use with dehydration, sedative accumulation	Hold or adjust only with clinical plan; test renal function and electrolytes
Social risk	Living alone in overheated home, no access to fluids or cooling, severe frailty	Activate family, community, social, or emergency support

Prevention and patient communication

Advice must be practical enough to use. Telling patients to “avoid heat” is weak if they live in one hot room, work outdoors, or cannot afford cooling. Advice should be matched to the patient's life.

For heat, patients can be advised to drink safely according to their condition, avoid heavy exertion during peak heat, use cool showers or wet cloths, spend time in cooler public places where available, check on vulnerable relatives, and seek help early for confusion, fainting, chest pain, severe breathlessness, reduced urine, or persistent vomiting. Patients with heart

failure or kidney disease need individualised fluid advice rather than generic instructions to drink large volumes.

For air pollution, patients with asthma, COPD, cardiovascular disease, and pregnancy may be advised to monitor air quality alerts, reduce outdoor exertion when air quality is poor, keep rescue medication available, use masks appropriate for particulate exposure where feasible, create a cleaner indoor room, and seek review if symptoms increase. During wildfire smoke events, ordinary cloth masks are not sufficient for fine particulate protection; however, mask feasibility depends on age, respiratory disease, fit, and local guidance.

For medications, the message should be careful: do not stop medicines abruptly. Instead, patients should know which medicines need review if they become dehydrated, dizzy, febrile, unable to drink, or acutely unwell. Written hot-day or sick-day instructions are safer than improvised changes during a crisis.

Discussion

Heat exposure and air pollution have become clinically relevant exposures in adult general medical practice. The evidence does not support treating them as rare events. Heatwaves, hot nights, wildfire smoke, ozone episodes, traffic-related pollution, and poor indoor environments now interact with the chronic disease burden that general practitioners already manage.

The strongest clinical message is that these exposures amplify existing vulnerability. Heat does not only cause heatstroke. It destabilises heart failure, worsens kidney function, increases fall risk, contributes to delirium, complicates diabetes care, and changes medication safety. Air pollution does not only irritate lungs. It contributes to asthma and COPD exacerbations, cardiovascular events, mortality, inflammation, endothelial dysfunction, and systemic risk.

The second message is that risk is compound. Heat and air pollution may occur together, and the combined exposure can be worse than either alone. Ozone formation is favoured by sunlight and heat. Wildfires can occur during hot dry periods and produce high particulate exposure. Patients may open windows for cooling and increase smoke exposure. Power cuts can remove both cooling and respiratory equipment. This compound-risk framing is closer to clinical reality than a single-exposure model.

The third message is that general practice can act before exposure. Many interventions are simple: identify high-risk patients, update chronic disease plans, check medication lists, provide written hot-weather instructions, ask about housing and cooling, advise respiratory patients before smoke events, and arrange early testing for kidney or electrolyte risk. These actions are not dramatic, but they are exactly the sort of anticipatory care that prevents admissions.

Medication management needs particular caution. Some medicines plausibly and demonstrably increase heat vulnerability, but the evidence is uneven across drug classes. Automatic stopping of cardiovascular, psychiatric, or renal medicines during heatwaves may cause harm. A better model is patient-specific planning: which medicines matter for this patient, under which circumstances should they seek advice, and what monitoring is needed if they become unwell?

There are also evidence gaps. Many heat-medication studies are observational or involve younger healthy volunteers rather than older multimorbid patients. Individual exposure is difficult to measure because outdoor temperature does not equal indoor temperature, and ambient air pollution does not equal personal inhaled dose. Social vulnerability is often poorly captured in electronic health records. Trials of clinical interventions during heatwaves and pollution episodes remain limited. General practice needs more implementation research on recall systems, pharmacist-supported heat medication reviews, indoor-temperature screening, and targeted alert messaging.

The larger public health implications are clear, but this article's focus is clinical. A general practitioner cannot reduce regional PM_{2.5} alone or redesign urban heat islands from the consultation room. However, clinicians can identify patients at high risk, translate warnings into actionable medical advice, and document environmental vulnerability as part of care. This is increasingly part of safe chronic disease management.

Conclusion

Heat exposure and air pollution are important contributors to illness in general medical practice. They worsen cardiovascular disease, trigger respiratory exacerbations, increase kidney and electrolyte risk, contribute to delirium and falls, and alter medication safety during hot weather. Patients with older age, multimorbidity, frailty, chronic kidney disease, heart failure, COPD, asthma, diabetes, severe mental illness, pregnancy, outdoor work exposure, poor housing, and social isolation need particular attention.

Clinical management should be anticipatory. Before hot seasons and pollution-prone periods, general practices should identify vulnerable patients, review medication risk, update asthma and COPD plans, assess heart failure and kidney disease safety, and discuss realistic cooling and exposure-reduction strategies. During heatwaves or poor air quality events, clinicians should actively ask about exposure, indoor temperature, hydration, air quality, medication use, urine output, breathing symptoms, and social support.

Medication safety should be individualised. Diuretics, renin-angiotensin system blockers, NSAIDs, lithium, anticholinergics, psychotropics, beta-blockers, SGLT2 inhibitors, and temperature-sensitive medicines require careful review in context, but abrupt discontinuation without a plan should be avoided.

The central change is conceptual. Heat and air pollution should not be treated as background weather conditions. They are clinically meaningful exposures that interact with chronic disease, medication use, housing, occupation, and social vulnerability. General practice is one of the best places to recognise this risk early and reduce avoidable harm.

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