



Heat and Health 1

Hot weather and heat extremes: health risks

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This is the first in a *Series* of two papers on heat and health

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Hot ambient conditions and associated heat stress can increase mortality and morbidity, as well as increase adverse pregnancy outcomes and negatively affect mental health. High heat stress can also reduce physical work capacity and motor-cognitive performances, with consequences for productivity, and increase the risk of occupational health problems. Almost half of the global population and more than 1 billion workers are exposed to high heat episodes and about a third of all exposed workers have negative health effects. However, excess deaths and many heat-related health risks are preventable, with appropriate heat action plans involving behavioural strategies and biophysical solutions. Extreme heat events are becoming permanent features of summer seasons worldwide, causing many excess deaths. Heat-related morbidity and mortality are projected to increase further as climate change progresses, with greater risk associated with higher degrees of global warming. Particularly in tropical regions, increased warming might mean that physiological limits related to heat tolerance (survival) will be reached regularly and more often in coming decades. Climate change is interacting with other trends, such as population growth and ageing, urbanisation, and socioeconomic development, that can either exacerbate or ameliorate heat-related hazards. Urban temperatures are further enhanced by anthropogenic heat from vehicular transport and heat waste from buildings. Although there is some evidence of adaptation to increasing temperatures in high-income countries, projections of a hotter future suggest that without investment in research and risk management actions, heat-related morbidity and mortality are likely to increase.

Introduction

Exposure to high ambient temperatures causes needless suffering and death. Temperatures above long-term averages during summer months and discrete heat extremes (eg, heatwaves) are associated with excess mortality.¹ In high-income countries, heat is one of the largest weather-related causes of death.² In addition to mortality, hot weather and heat extremes are associated with increased emergency room visits and hospital admittance,^{3–5} increased deaths from cardiorespiratory and other diseases,⁶ mental health issues,⁷ adverse pregnancy and birth outcomes,⁸ and increased health-care costs.⁹ Most heat-related morbidity and mortality should be preventable with improved preparedness and avoidance of exposure. Studies consistently show that adults older than 65 years, people with cardiopulmonary and other chronic diseases,

and very young children are particularly vulnerable to the effects of heat, irrespective of income level or geographical region.^{10–14} There is greater knowledge about the burden of heat-related mortality in high-income countries, although excess mortality also occurs in low-income and middle-income countries (LMICs).¹⁵

Historical burdens of disease from the first two decades of the 21st century will be poor predictors of risks over coming decades. Climate change is increasing the frequency, intensity, and duration of heat extremes, putting more individuals, communities, and health systems at risk.¹⁶ European heat extremes in 2015, 2017, 2018, and 2019 were made more likely and more intense because of anthropogenic climate change.¹⁷ A prolonged Siberian heat extreme, in 2020, was almost impossible without climate change.¹⁸ On average between May and July, 2018, about 22% of the populated and agricultural areas north of 30° latitude had concurrent temperatures above the 90th percentile because of climate change.^{19,20} Projections show that the average high-exposure area expected to have warm and hot spells in the northern hemisphere could increase by about 16% per additional 1°C of global warming.¹⁹

Morbidity, mortality, and reductions in worker productivity are projected to increase with climate change, in the absence of strong adaptation and mitigation efforts, particularly in LMICs.^{21,22} Projections of heat-related morbidity and mortality are affected by assumptions about heat adaptation strategies (eg, technological and infrastructural); adaptation assumptions should be incorporated to provide robust projections of heat-related mortality.²³ Higher degrees of temperature change might

Key messages

- People are unnecessarily suffering and dying in the heat, with population health and community vulnerability often unrecognised
- Other heat-related effects include reduced occupational health and productivity, and increased sports illnesses and injuries
- The physiological limits of heat tolerance are finite and numerous cofactors substantially reduce these limits
- Without urgent investments in research and risk management actions, climate change will continue to increase heat-related hazards, and associated morbidity and mortality

decrease the extent to which morbidity and mortality are preventable because physiological limits could be reached even with adaptation measures. Heat action plans that incorporate early warning and response systems are essential for reducing vulnerability.²⁴

Preventing further heat-related risks in a warmer and more extreme climate in the future requires upscaling interventions, as reviewed in the second paper in this Series.²⁵ In this Series paper, we build a more holistic understanding of factors heightening or attenuating risks that need to be incorporated into heat action plans, also known as heat-health action plans, to enhance their effectiveness in a changing climate. We start by reviewing physiological responses and other factors affecting heat stress, illustrate possible future challenges to the general population from research on occupational and sports exposures, discuss the role of the built environment, and highlight megatrends that can alter risks.

Physiological factors affecting heat strain, illnesses, and death

The human body responds to heat stress in two primary ways: redistributing blood flow towards the skin (vasodilation) to improve heat transfer from muscles to skin and subsequently to the environment, and secreting sweat onto the skin, which subsequently evaporates and removes body heat.²⁶ The brain regulates these physiological heat loss responses, with additional thermal input from temperature-sensitive nerve cells in the skin and throughout the body.²⁷ This regulation can also be affected by non-thermal signals, such as from dehydration, metaboreceptors (a type of chemoreceptor that responds to metabolic products generated by exercising muscles), and cytokines.²⁷ These physiological heat stress responses are necessary to limit elevations in core temperature, and can affect people differently based on, for example, pre-existing medical conditions, with the possibility for negative effects on the body.

The redistribution and increased blood flow to the skin, due to cutaneous vasodilation, increases cardiac demand while decreasing the heart's filling pressure.^{28,29} These responses require the heart to pump harder and faster, increasing the local (coronary tissue) oxygen demand (figure). For people with pre-existing heart conditions, this response can lead to a mismatch between a high cardiac oxygen demand and a compromised cardiac oxygen delivery.³⁰ A sustained mismatch can lead to cardiac ischaemia, infarction, and ultimately, cardiovascular collapse.³⁰ The elevated cardiovascular strain from heat stress is a primary health concern during heat extremes as more older adults die from cardiovascular events than nearly all other heat-related causes of death combined.³¹ However, this observation might be illness-specific because some evidence³² suggests that particular illnesses, such as congestive heart failure, might benefit from high temperatures, whereas other evidence contradicts these

findings,³³ and further evidence has shown impaired heat loss responses in patients with congestive heart failure.^{34,35} Regardless, meta-analyses have clearly shown that all-cause cardiovascular illness is the primary cause of death during heatwaves,^{6,31} and as almost half a billion people are estimated to have cardiovascular disease globally,³⁶ any densely populated area affected by a heat extreme will be at risk for increased cardiovascular-related mortality.

Additionally, sweat production can lead to dehydration if the resultant body water deficits are not adequately replenished. Dehydration decreases blood volume that can eventually exacerbate cardiovascular strain³⁷ and also lead to acute kidney injury and failure (figure).^{38,39} Chronic dehydration can lead to kidney fibrosis and chronic kidney disease, which is of concern for outdoor workers in Mesoamerica, India, and other regions.⁴⁰ This condition worsens cardiovascular disease⁴⁰ and is more regularly reported during or following hot weather events.⁴¹

Under conditions of extreme heat stress, the thermoregulatory capacity of the body can be exceeded, resulting in illness due to overheating that can progress to heat stroke, which if untreated, can be fatal.^{26,42} High internal temperatures (39–40°C), combined with ischaemia and increased oxidative stress after blood redistribution, can cause cell, tissue, or organ damage, with the brain, heart, kidneys, intestines, liver, and lungs at the greatest risk (figure). Heat-derived lung damage, in the form of pulmonary oedema and acute respiratory distress syndrome, combined with the high rates of people with pre-existing respiratory conditions,³³ and increased pulmonary stress because of heat-related hyperventilation⁴³ and elevated air pollution during heatwaves,⁴⁴ is responsible for the second greatest source of mortality and morbidity during heatwaves, after cardiovascular disease.³¹ Due to the multifaceted source of damage, heat-derived injuries can remain hazardous even after cooling the body to normothermic temperatures.⁴⁵ Similarly, although most heat-related hospital admissions occur within 24 h of the onset of these events, cognitive⁴⁶ and organ dysfunction can persist for years following injury and render the injured individual at two to three-times greater risk of death for decades after injury.⁴⁷

Other factors affecting heat strain

The use of alcohol, medications, and illegal narcotics is associated with increased mortality during heat extremes.^{46,48–50} Many commonly prescribed medications, such as general anticholinergics, antidepressants, and opioids,⁵¹ and illegal narcotics such as cocaine,⁴⁸ might compromise physiological heat loss responses (panel). Although sweating reductions with some medications have been reported,⁴⁸ most medications have not been systematically studied in a thermoregulatory context and no studies have used ecologically valid doses in

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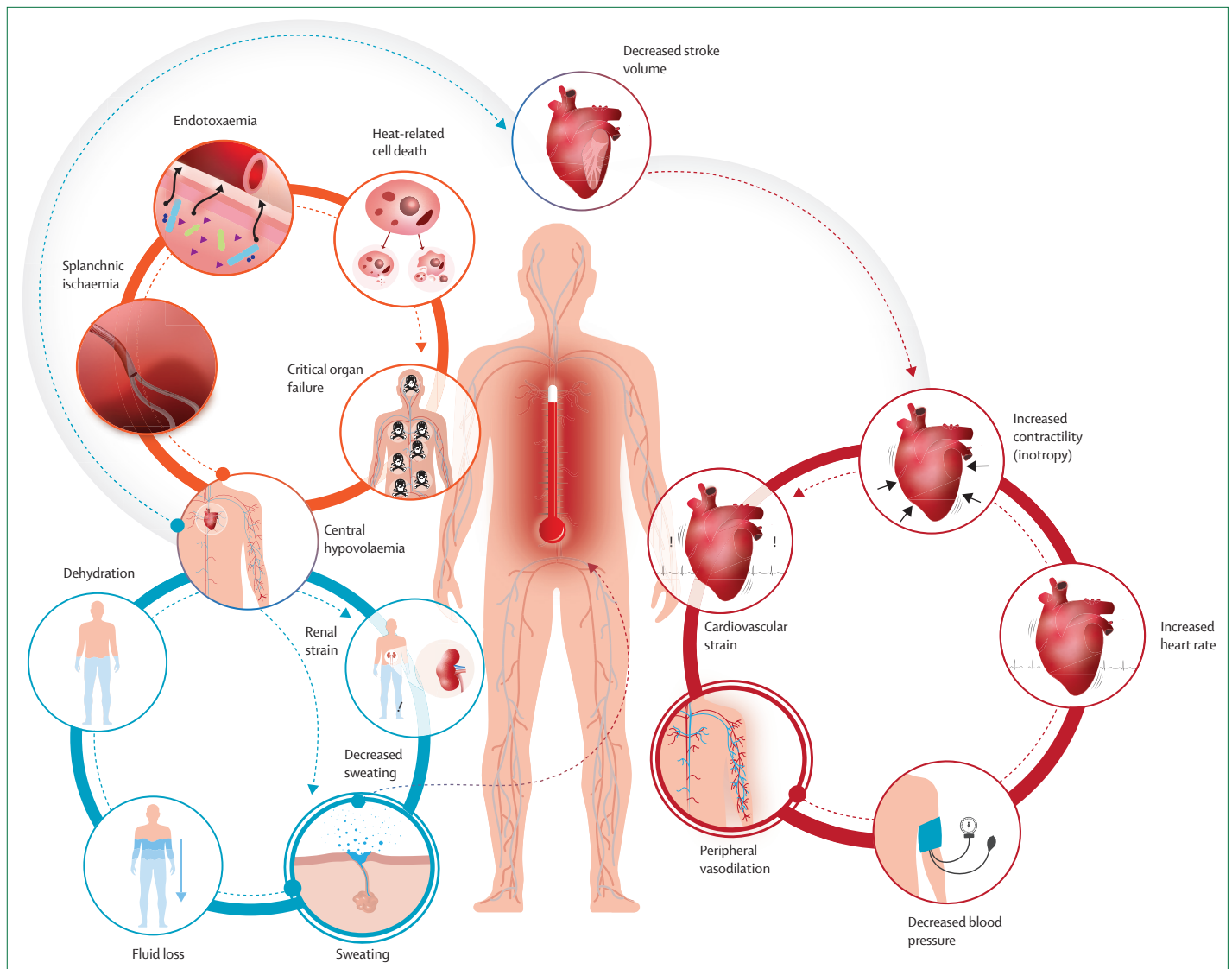


Figure: Illustration of the physiological pathways of human heat strain

realistic conditions of heat extremes. Empirical evidence of the exact effect of these medications is urgently needed.⁵⁰ A primary area of concern is a direct inhibition of acetylcholine, the neurotransmitter responsible for inducing sweating and, potentially, skin vasodilation.⁷⁷ Some medications⁵¹ and narcotics⁶⁸ might also directly influence the thermoregulatory control centre in the brain.

A reduced behavioural capacity to respond and adapt to extreme heat also elevates the risk of heat-related illness or injury. In particular, increased mortality during heat extremes⁴⁹ is associated with being confined to bed, living alone, being unable to care for oneself, not leaving the residence to cool down their body temperature, and having a pre-existing mental health condition. For individuals with pre-existing mental health conditions,

the elevated mortality rates are probably due to combinations of an inability to take necessary personal precautions to cool down, impairments in the thermoregulatory control system, and the effects of medications.⁷⁸

Individuals with paraplegia and tetraplegia have an inability to adequately control body temperatures in hot environments while resting and during exercise,⁷⁹ even with excellent fitness levels. However, the relative contributions of these factors are unclear and further studies are required to elucidate the relative effect.⁷⁸

Heat can also indirectly contribute to an increased risk of communicable diseases. In many tropical countries, hot temperatures are the leading barrier to overnight mosquito-net use; therefore, heat stress is a major factor aggravating risk of diseases such as dengue and malaria.^{80,81}

Panel: Epidemiological observations with evidence-based explanations

Physiological factors associated with increased risk of death

Cardiovascular disease⁴⁹

- Primary cause of death during heatwaves³¹
- As a pre-existing condition, predisposes individuals to coronary oxygen use versus delivery mismatches³⁰

Respiratory diseases (particularly chronic obstructive pulmonary disease)³¹

- As a pre-existing condition, predisposes individuals to death by respiratory disease³³
- Secondary cause of death during heatwaves³¹
- Acute respiratory distress syndrome brought on by immune response to heat-related cell death,⁵ and by increased air pollution with heatwaves⁴⁴

Cerebrovascular disease³¹

- Tertiary cause of death during heatwaves³¹
- Heat-related reductions in cerebral blood flow and damage to blood–brain barrier can lead to increased intracranial pressure, cerebral ischaemia, and possibly intracranial haemorrhage^{52,53}

Genitourinary disease (particularly chronic kidney disease)⁵⁴

- Potential cause of death³¹
- Daily dehydration (especially in the presence of other environmental factors) can lead to kidney fibrosis⁵⁵ and potentially failure⁵⁶
- Chronic kidney disease, in turn, predisposes individuals to cardiovascular events⁵⁷

Diabetes⁵⁸

- Potential cause of death during heatwaves³¹
- Reductions in skin blood flow and sweating for individuals with type 1 and type 2 diabetes related to extent of disease-associated complications (peripheral neuropathy), how well the disease is controlled, and how long the individual has lived with the disease⁵⁹

Dehydration⁵⁸

- Depending on the health-care system, identified as a cause of death during heatwaves⁵⁸
- Reduced sweat loss, skin blood flow, and increased core temperature of heat stress above approximately 2% total body-mass loss dehydration⁶⁰

Heat-related illness⁵⁸

- Advanced stages of heat illness or stroke can lead to lethal damage to the brain, kidneys, and liver⁶¹

Mental health illness or disorder⁴⁹

- Potential physiological thermoregulatory inhibitions (uncertain)
- Potential behavioural thermoregulation impairments (uncertain)
- Probable interactions with anticholinergic medications (eg, antidepressants)⁵¹

Hypertension⁴⁶

- Increases cardiovascular strain, increasing coronary oxygen demand⁶²

Younger than 1 year¹³ or 4 years (inconsistent)⁶³

- Infants have a lower heat loss capacity relative to adults⁶⁴
- Relatively high surface area allows them to absorb heat more quickly when air temperature is higher than skin temperature⁶⁴
- Inability to behaviourally thermoregulate (particularly to remove themselves from hot cars; probable but no definitive studies)

Older than 65 years⁶⁵

- Diminished sweating ability⁶⁶

Selected prescription medications⁴⁹

- Possibly alter the thermoregulatory centre (anticholinergics, antidepressants, antiepileptics, antihypertensives, muscle relaxants, and opioids)⁵¹
- Possibly alter sweat gland and cutaneous vasculature stimulation peripherally (anticholinergics, antihistamines, antipsychotics, antivertigo medications, bladder antispasmodics, gastric antisecretories, and muscle relaxants)⁵¹
- Note, no studies have verified these effects using regularly prescribed doses in humans in heatwave conditions

Cocaine⁴⁶

- Increases metabolism (possible, but uncertain)⁶⁷
- Impairs both sweating and vasodilatory heat loss responses⁶⁸
- Reduces perceptual heat sensitivity (ie, users feel cooler at equivalent temperatures compared with non-users)⁶⁸

Prescribed diuretics⁴⁶

- Increased water excretion might lead to dehydration and subsequent heat intolerance⁶⁹

Alcohol abuse⁴⁶

- No impairment (and possible enhancement) of sweating, vasodilation, and cool-seeking behaviour^{70–72}
- Increased diuresis leading to dehydration (effect probably small)⁷³
- Impaired decision making⁷⁴

Behavioural factors associated with increased risk of death

Visited by nurses (within the home)⁴⁹

- Indication of immunocompromised state and therefore impaired thermoregulatory control²⁷ or inability to deal with heat-related sepsis and coagulopathies;⁷⁵ or presence of other health conditions that could limit mobility and behavioural thermoregulation (uncertain)

Unable to care for themselves, did not leave home, or confined to bed⁴⁹

- Reduced thermoregulatory adaptive capacity (uncertain)
- Warmer indoor temperatures with low air flow

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- Isolated, unable to receive help from others (uncertain)
- Unable to behaviourally thermoregulate (uncertain)

*Lived alone*⁴⁹

- Unable to access help if needed (uncertain)

*Lack of air conditioning*⁶

- Higher indoor environment temperatures, leading to greater heat strain²⁶

Behavioural factors associated with decreased risk of death

*Visited air conditioned places*⁴⁹

- Provide physiological respite from heat by visiting cool environments⁷⁶

*Working air conditioner in house*⁴⁹

- Able to choose to lower environmental temperatures to reduce physiological strain⁷⁶

*Access to transportation*⁴⁹

- Able to visit others for help or travel to cooler areas⁷⁶

Heat extremes in countries with limited clean drinking water increase the threat of water-borne diseases as the volume needed to replace sweat losses and prevent dehydration might exceed supply.

Heat extremes are associated with higher mortality rates in infants, particularly neonates.¹³ The development of the thermoregulatory system is age dependent.⁶⁴ Infants could be at a higher risk due to morphological factors, such as a body surface area-to-mass ratio that can be 64% greater than adults,⁶⁴ allowing for more dry heat (convective) gain from the environment when air temperature exceeds skin temperature (approximately 35°C). This gain can be offset by the relatively greater capacity for evaporative cooling when there is no impairment to sweating, but dehydration (relative to mass) would subsequently occur at a faster rate.⁴² Irrespective of any morphological or physiological disadvantages, the primary cause of heat-related death in infants and toddlers is due to being forgotten or gaining unsupervised access to, or being left alone, in hot vehicles.⁸²

Without accounting for the many crucial interindividual factors of human thermoregulation mentioned previously and in the panel, as well as adaptation, current heat risk projections might create unreliable and unrealistic estimates of future adverse health outcomes.⁸³

Sport, exertional heat stress, and heat stroke

Although heat stress risk is often assumed lowest in elite athletes, cases of fatal exertional heat stroke have occurred in elite sports such as American football, rugby league, wrestling, and long distance running. Its incidence rate in sport is currently low, but fatal and life-threatening cases of exertional heat stroke are ten-times more common than serious cardiac events in warm-weather endurance events.⁸⁴ It is typically observed in otherwise healthy people and workers, after very high amounts of heat production associated with intense physical exertion, often coupled with clothing or equipment that impairs heat loss in temperate to hot conditions.^{85–87}

In 2019, examples of heat-related disruptions to major sporting events include the Women's Fédération Internationale de Football Association (FIFA) World Cup in France, the Australian Open tennis tournament in Melbourne, VIC, Australia, the Olympic test triathlon event in Tokyo, Japan, the World Track and Field Championships in Doha, Qatar, and the New York City Triathlon, NY, USA; each had either interrupted or postponed competition due to the anticipated high risk of exertional heat illness in competitors. Projections suggest that by 2085, very few major cities will be able to host the summer Olympic games due to heat-related risks for athletes.⁸⁸ Heat-related concerns could be even greater for the Paralympic games because they will involve more vulnerable populations.^{89–92}

Most community and professional sports health and safety policies to reduce heat-related risks (eg, International Olympic Committee and FIFA) are based on the index thresholds of shaded air temperature or the wet-bulb globe temperature (WBGT). Fixed air temperature thresholds for the suspension of play are inadvisable because the amount of heat strain induced by a particular temperature is affected by humidity, wind speed, sun exposure, clothing, and exercise intensity.⁹³ Although the WBGT index accounts, to a certain extent, for these other parameters, relatively expensive equipment is required for its accurate and direct measurement. An indoor WBGT formula that incorporates only temperature and natural wet bulb temperature is often estimated from ambient humidity values and, thus, will underestimate the risk of heat stress for people directly exposed to the sun and low wind speeds. Current estimated WBGT values issued by some weather forecast services (eg, Bureau of Meteorology in Australia) assume fixed cloud cover and wind, and similarly underestimate environmental risk of heat stress on clear and still days.⁹⁴ Furthermore, critical wet bulb globe temperature thresholds for modifying or suspending play to protect health should be specific for each sport and competition level.⁹⁵ Alternative heat stress indices should also be considered.⁹⁶

Community sport competitors might be at a greater risk of heat illness than elite and professional athletes due to a lower fitness and acclimatisation status;⁹⁷ and although they are probably protected by lower exercise intensity levels, less economical movement elicits a higher heat production for a given activity.⁹⁸ Summer sports and sports played in tropical and subtropical environments are projected to be the most affected by future warming. Physical activity decreases at average daily air temperatures exceeding 29–30°C, with older adults being most affected.^{99,100} Projections (in Perth, WA, Australia) indicate an eight to 50-fold increase in the number of days that will be unsafe for moderate physical activity by 2070.¹⁰¹ Sports requiring intense physical activity are at high risk, including those with prolonged environmental exposures (marathon, triathlon, and road cycling), high intensity sports, and those where specialised clothing or protective equipment impairs heat loss (eg, American football and cricket). Risks for youth athletes are poorly understood.

In summary, where and when amateur and sporting events can be held safely, and when individuals can engage in jogging, hiking, gardening, and other activities and hobbies safely, will shift in a warmer climate.

Heat exposure of outdoor workers

A warmer world is also projected to negatively affect outdoor employment, with solutions urgently needed for outdoor workers worldwide. The International Labour Organization estimates that more than 1 billion workers are exposed to high heat episodes, not all of which occur during summer months.¹⁰² Depending on assumptions of future socioeconomic development, these numbers are probably conservative.

For outdoor workers, high metabolic heat production associated with occupational tasks combined with high ambient and radiant heat, low air flow, and sometimes high humidity, add to human heat strain. Metabolic heat production can increase by more than 15-times from resting values of around 100 watts; occupational heat production of 200–500 watts is typical.¹⁰³ Internal heat production for a given task is very intensity-dependent; thus, pacing (ie, lowering of the intensity) should be, and often becomes, an integral part of behavioural thermoregulation for safety. Accordingly, workers exposed to elevated environmental heat will typically reduce their work output, taking more unplanned breaks or working at a slower pace than normal¹⁰⁴ to adjust the overall occupational heat stress. Workers following a fixed or externally dictated pace (eg, buckets per h) or piecemeal will face higher heat strain than those workers who are free to self-pace. For manual labourers (eg, agriculture and construction), productivity begins to decline at around 20°C and progressively decreases as temperatures rise.¹⁰⁵ Rest breaks that lower metabolic heat production are an effective heat mitigation method that might not appeal to some employers due to concerns

about labour output. However, without extra breaks, lower work efficiency¹⁰⁴ and higher rates of accidents and sick leave^{106,107} contribute to a reduction in overall productivity in the heat. Other relevant factors include personal protective equipment (eg, clothing and garments) with high evaporative resistance, and the cumulative effect of daily exposures to elevated temperatures.^{41,85}

Occupational field studies^{104,108,109} and controlled mechanistic laboratory studies¹¹⁰ have shown that hyperthermia provoked by heat stress directly impairs physical work capacity^{104,105,108,109} and tasks relying on complex cognitive functions or skilled motor performances.^{111,112} A meta-analysis of more than 447 million workers from over 40 occupations in 30 countries¹¹³ found that approximately a third of all workers exposed to occupational heat stress had negative effects, including an increased risk of hyperthermia and cardiovascular failure or collapse, and increased risk of acute kidney disease.

Many workers are repetitively exposed to daily occupational heat stress over extended periods, thereby making them more susceptible to both acute and chronic effects of heat strain.⁴¹ In occupational settings, and especially when workers are exposed to heat extremes during work and leisure time (including overnight), adequate rehydration appears to be challenging, as indicated by the high prevalence of low hydration status at the onset of work in occupations with high heat stress¹¹⁴ and accumulative negative effects on productivity following consecutive heat extremes.¹¹⁵

Built environment

The term built environment refers to the physical components of where we live and work, including buildings, streets, open urban spaces, and infrastructure. Urbanisation transforms the surface microclimate by modifying the radiative, thermal, moisture, and aerodynamic processes, and thereby affecting the urban surface where people live and work.¹¹⁶ One consequence of urban development is the urban heat island that results in cities being, on average, warmer than adjacent rural landscapes. The urban heat island is amplified at night when stored daytime heat is emitted and intensified with increasing city size and population density.¹¹⁶ The true intensity of the urban heat island should adequately account for rural characteristics in addition to urban predictors, such as population.¹¹⁷ In practice, intra-urban heat intensities are more predictive of true human exposures;^{117,118} hence, the aim should be to reduce the various negative effects of high concentrations of urban heat, rather than only focusing on mitigating the urban heat island intensity.¹¹⁹

Urban temperatures are enhanced by anthropogenic heat from vehicular transport, heat emitted from building energy waste, and minimally by human metabolic heat. Estimates of the anthropogenic heat fluxes are usually based on intensity of energy use within a city, specifically the average energy uses per capita multiplied by urban

population density. Increasing usage of air conditioning intensifies emissions of anthropogenic heat into the urban climate in a positive feedback spiral. In the summer of 2011, peak summer energy use in Beijing, China, was approximately 19 100 megawatts with more than 40% of that attributed to air conditioning load.¹²⁰ When such city-scale additional heat loads are included in global climate projections, rising household incomes result in a particularly rapid increase in demand for air conditioning¹²¹ in middle-income economies of the tropics.

The rapid growth and densification of cities simultaneously increases the concentrations of ambient air pollution produced by vehicles and transportation systems, buildings, and industry. Within urban canyons, the airflow and vertical exchanges of street pollutants can lead to long residence times of particulate and gaseous pollutants,^{122,123} thus increasing human exposure to pollution and causing potentially worse adverse health outcomes.¹²⁴

Megatrends affecting heat-related morbidity and mortality

The magnitude and pattern of future heat-related morbidity and mortality will depend on climate change and other important factors such as population growth and ageing,¹²⁵ urbanisation trends,¹²⁶ adaptation efforts,¹²⁷ and development choices.¹²⁸

Increases in the mean and extremes of global and regional temperatures have been observed since 1950, with confidence in the extent of change varying by region.¹²⁹ Projections indicate substantial warming in temperature extremes by the end of the century over most land areas; hot days, hot nights, and the duration and intensity of heat extremes are projected to increase in most world regions.¹³⁰ For example, in some regions, a 1-in-20-year annual hottest day could become a 1-in-2-year event by the end of this century.

There is a large range of possible regional climates for a given increase in global mean surface temperature above preindustrial levels. For example, the mean temperature anomaly in the Arctic at peak warming could range from 5·04°C (for a 66th percentile outcome)

to 6·29°C (for a worst case, 90th percentile). For the contiguous USA, the range is projected to be 2·57°C (for a 66th percentile) to 3·09°C (for a 90th percentile). These ranges imply a greater increase in the intensity of heat extremes than projections suggest. Most global climate models underestimate the extremes, indicating that future heat-related mortality risks could be even larger than projected in earlier studies.¹³¹

Without considering demographic change, achieving the Paris Agreement goal of limiting additional warming to below 2·0°C above preindustrial levels is projected to avoid substantial heat-related mortality.^{132–134} For example, in 15 cities in the USA, increasing mitigation ambition to achieve the 2·0°C goal could avoid 70–1980 annual heat-related deaths per city during heat extremes, and achieving the 1·5°C goal could avoid 110–2720 annual heat-related deaths.¹³² Such wide ranges in heat mortality are often due to high uncertainties across predictors of heat-related mortality (eg, human behaviour and adaptation);¹²⁷ whereas slower future population growth and more adaptation result in lower estimates of negative health outcomes.

Byers and colleagues¹³⁵ projected the global number of people exposed and vulnerable (living on <US\$10 per day) to heat extremes under assumptions of a socioeconomic development pathway characterised by moderate challenges to adaptation and mitigation, by degrees of future temperature change (table). Overall, the numbers of people exposed, and those both exposed and vulnerable, substantially increase with each additional unit of warming. The risks also vary substantially by location; missing information from Africa and parts of Asia means the risks there are probably underestimates.

Climate change is also affecting other hazards that can exacerbate adverse heat-related outcomes, including ground-level ozone concentrations¹³⁶ and wildfires (eg, increasing human exposures to particulate matter).¹³⁷

Other trends of importance include the rapid increase in global urban population from 751 million in 1950, to 4·2 billion in 2018. By 2030, the population living in urban areas is expected to reach 60% of the world total and the number of megacities exceeding 10 million inhabitants is expected to reach 43 megacities, up from 31 megacities in 2016.¹³⁸ Global population by the end of the century could be 6·9–12·6 billion, depending on assumptions about fertility, mortality, migration, and education across different development pathways; in 2050, the range is projected to be 8·5–10·0 billion.¹²⁵ A larger and older population would mean more people at greater risk of heat-related effects.

Discussion

People are unnecessarily suffering and dying during hot weather and heat extremes. Heat-related morbidity and mortality are expected to increase as climate change continues, with each additional unit of warming projected to further increase the risks. Robust evidence of the relationships between hot weather and morbidity

	Exposed	Exposed and vulnerable
1·5°C	3960 (3546–4508)	1187 (410–2372)
2·0°C	5986 (5417–6710)	1581 (506–3218)
3·0°C	7909 (7286–8640)	1707 (537–3575)

Data are millions of people (range), based on an estimated total world population of 9·2 billion in 2050. *Vulnerable people are those living on less than US\$10 per day. †Heat event exposure was calculated as the sum of days from heat events lasting 3 or more consecutive days above the historical 99th percentile daily mean wet bulb air temperature.¹³⁵

Table: Number of people globally projected to be exposed and vulnerable* to heat extremes of a socioeconomic development pathway with moderate challenges to adaptation and mitigation, by degrees of future temperature change†

and mortality is being augmented with growing evidence of other effects, including on occupational workers and professional and recreational athletes. Particularly in tropical regions, increased warming means that physiological limits could be reached regularly and more often in coming decades.

Without investment in research and implementation of needed risk management actions, a very different future awaits many people and communities. Higher means and extremes of temperature will lead to future summers characterised by higher morbidity and mortality, and important limitations and changes to what are today considered normal activities. In the second paper of this Series, Jay and colleagues²⁵ focus on heat action plans and other options that health authorities and partners can implement to increase resilience to higher ambient temperatures and more heat extremes. These actions are developed in the context of other trends, such as urbanisation and socioeconomic development, that could ameliorate or exacerbate heat-related hazards.

Contributors

KLE, AC, and OJ conceptualised the Series and contributed to outlining the approach and conclusions. KLE reviewed and synthesised elements of the review and was the overall editor. PB, CB, RdD, GH, YH, RSK, WM, AM, NBM, LN, SIS, JV, and OJ reviewed and synthesised elements of the review and contributed to editing.

Declaration of interests

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