Protecting vulnerable populations in extreme heat – a growing and pervasive health challenge

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Background: defining extreme heat vulnerability

Extreme heat is a growing human health challenge, largely driven by two factors—the magnitude of heat stress experienced and a person's ability to thermoregulate.[1] Together, these factors dictate whether core (internal) body temperature will be maintained close to normal levels (~37 °C) or will rise (aka hyperthermia). As such, an increase in the magnitude of heat stress and/or an impaired ability to thermoregulate will exacerbate the development of hyperthermia. This, in turn, increases the risk of developing heat illness, including heat exhaustion, heat stroke (the only potentially fatal heat illness), and heat injury that is mainly characterized by heart and kidney strain. Thus, extreme heat is associated with population-level risks of increased morbidity and mortality.[2]

Heat stress is defined as the total heat load experienced by the body and is largely dictated by environmental heat exposure and internal heat production, a byproduct of metabolism.[1] Due to climate change, the severity, frequency and duration of extreme heat exposure is forecasted to increase in the coming years.[3] That said, heat is not simply a problem of the future. Indeed, extreme heat is already the deadliest weather-related event in the U.S.[3] Notably, not all people are equally vulnerable to the effects of this increasingly extreme heat.[3] For example, some people in low socioeconomic situations can be at heightened risk of heat illness, at least partially due to insufficient access to air conditioning and an inability to escape the heat.[2] Moreover, even for a given level of heat stress the magnitude of hyperthermia varies between individuals. This variability is driven by the capacity to thermoregulate, which can be modified by age, health status, medication use, hydration status, and acclimatization.[1] This pathway by which heat stress increases the risk of developing hyperthermia and heat illness, and the modifying factors, is depicted in Fig. 1.

During heat stress, elevations in body temperature are initially sensed by central and peripheral thermoreceptors. Integration of these thermal afferent signals in the central nervous system ultimately triggers, via sympathetic nerves, activation of thermoregulation-mediated heat loss responses to offload excess body heat to the environment and limit the development of hyperthermia.[1] These responses primarily include increases in skin blood flow to promote the movement of body heat from the core to the periphery and sweating, which promotes evaporative heat loss from the skin.[1] Thus, any factors that modify cardiovascular control or limit evaporative heat loss increase the risk of developing hyperthermia. For example, high humidity (i.e., the amount of water vapor in the air) hinders sweat evaporation, making air temperature alone an incomplete picture of environmental heat exposure.[4] Moreover, dehydration, certain medical conditions (e.g., cardiovascular disease) and certain medications (e.g., diuretics) can impair the cardiovascular response to heat stress, while older age and anticholinergic medications, for example, can impair the sweating response.[1] Notably, heat acclimatization, an adaptation to repeated heat stress exposures, improves heat loss, and can mitigate the development of hyperthermia.[1] That said, while the hyperthermia mitigating effect of heat acclimatization in young, healthy people is clear, the capacity for heat acclimatization to lessen the risk of heat illness in other groups of people (e.g., older adults, people with cardiovascular disease, etc.) is not well known.

Case study: the unique vulnerability of outdoor workers

Due to a combination of frequent extreme heat exposure and intense manual labor, which elevates heat production, outdoor workers are uniquely vulnerable to extreme heat.[5] Thus, occupation should be considered an important, though inadequately studied, social determinant of health. Strikingly, it is estimated that 2.4 billion people worldwide are exposed to heat stress in the workplace.[5] It is interesting to note that ninety percent of the time this heat exposure occurs outside of heatwaves,[5] likely highlighting the importance of heat production in occupational heat stress exposure. Thus, it is perhaps not surprising that hyperthermia is common in workers in the summer months,[6] with construction and agriculture workers being considered at the highest risk. This heightened risk of hyperthermia in the

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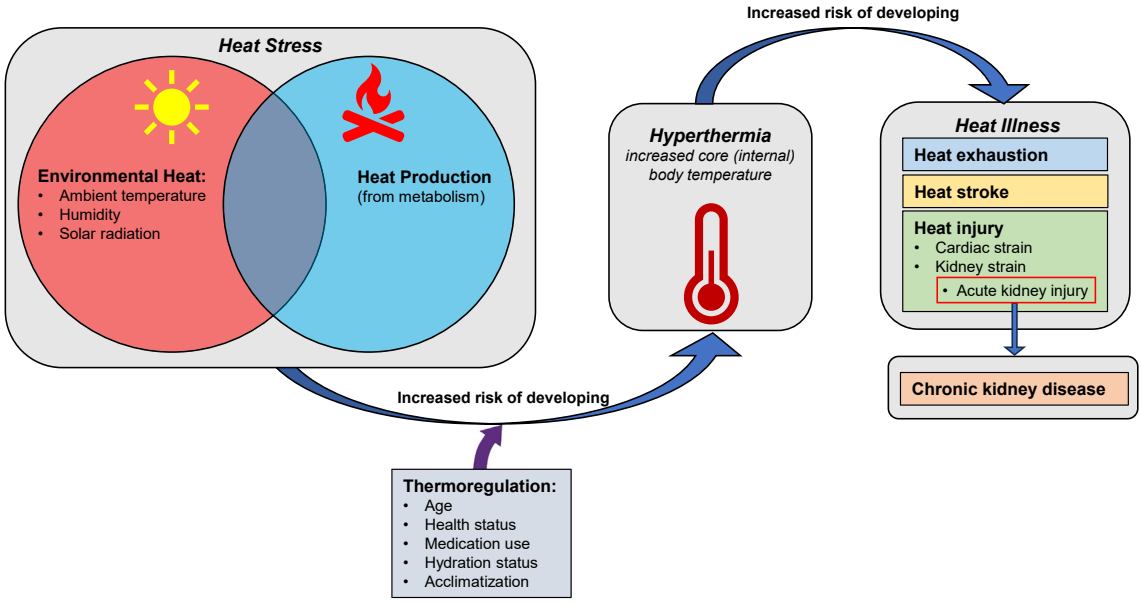

Fig. 1: The way heat stress, a function of environmental and metabolic heat, increases the risk of developing hyperthermia and heat illness, and examples of the responses that modify thermoregulation. The role of heat injury provoked acute kidney injury and the link to chronic kidney disease is used as an example by which heat stress and hyperthermia increases the risk of acute illness and chronic disease.

workplace elevates the risk of heat illness, occupational injuries, and death. For example, a recent analysis indicates that heat stress causes ~23 million non-fatal injuries and almost 19,000 deaths in workplace worldwide every year.[5] Importantly, the rate of heat-related occupational injuries and deaths[5] has increased in recent years, despite no change in the rates of occupational injuries and fatalities overall.

Not included in these statistics is the fact that approximately 26 million workers are living with heat attributable chronic kidney disease (CKD).[5] While CKD, defined as a sustained reduction in kidney function, is traditionally associated with hypertension and diabetes, this heat-attributed CKD is characterized by occupational origins and a nontraditional etiology (termed CKD of non-traditional causes or CKDnt).[6] CKDnt is arguably the first chronic disease attributed to heat stress. As such, while concomitant exposure to environmental toxins (e.g., contaminated drinking water) and infectious disease may play a role, a leading hypothesis is that CKDnt is primarily caused by a singular episode of hyperthermia-induced acute kidney injury (AKI) or repeated bouts of subclinical AKI.[6] Indeed, approximately 15% of outdoor workers frequently exposed to heat stress develop AKI.[5] The risk of developing hyperthermia-induced AKI during work is modified by several intra-personal factors.[6] For example, dehydration, defined as the net loss of body water, worsens the risk of hyperthermia-mediated AKI. Notably, dehydration is common during work-related heat stress as fluid replacement via drinking does not always keep pace with fluid lost due to sweating. Moreover, older age, hypertension and diabetes, traditional CKD risk factors, heighten the risk of hyperthermia-mediated AKI.[7] Importantly, the U.S. workforce is aging and has a relatively high prevalence of both hypertension and diabetes,[6] highlighting the pervasive nature of heat stress vulnerability.

Protecting vulnerable populations: new horizons

Great strides have been made to identify risk factors underlying vulnerability and to identify mitigation strategies to protect vulnerable populations. For example, low-cost cooling strategies, such as self-dousing with water and electric fan use, have been identified to overcome some of the socioeconomic barriers underlying vulnerability.[4] Moreover, observations that hot nighttime temperatures heighten the risk of occupational heat illness the following day raises important considerations regarding housing conditions.[8] Nevertheless, protecting heat vulnerable populations requires efforts that extend beyond the identification of risk factors. To this end, several important knowledge gaps exist that would safeguard the health of some of the most vulnerable groups in the face of climate change. Below two of these knowledge gaps are introduced.

Work-related heat stress interventions

Current worker protections against extreme heat include rest, hydration, and shade (to limit exposure to solar

radiation) to mitigate the development of hyperthermia in the face of heat stress. That said, in the U.S. there is not yet a federal heat standard providing universal protections, and only a handful of U.S. states have rules mandating rest and hydration under heat stress conditions. Nevertheless, the effectiveness of rest, hydration and shade interventions appears promising. For example, the primary role of heat stress in the AKI-to-CKDnt etiology is perhaps best exemplified in observations that rest, shade, and hydration interventions in agriculture workers at risk of CKDnt reduces the occurrence of AKI during a work shift and mitigates declines in kidney function across the heat season.[9] That said, given the complexities associated with conducting randomized controlled trials (the standard for demonstrating causality in clinical studies) in occupational settings, these studies can be compromised by aspects of study design and implementation. Thus, novel study designs capable of better inferring causality are required to invoke changes needed to universally implement rest, hydration, and shade interventions in workplaces where heat stress is common.

Non-cooling focused treatments

First line treatment for heat illness is removal from heat exposure and initiating cooling to lessen the magnitude of hyperthermia. For example, immediate treatment of heat stroke by immersion in ice water results in a rapid reduction in core temperature and is nearly universally regarded as effective.[10] While other cooling strategies (e.g., ice sheet cooling, forearm immersion, etc.) are also effective, their efficacy at reducing core temperature is nowhere near that of ice water immersion. However, ice water immersion may not be feasible in many instances such as remote locations, on poorly resourced job sites, etc., where heat stress and hyperthermia are common.[1] Thus, there is a need to develop therapies that do not require aggressive cooling. Such therapies could target some of the molecular pathways involved in hyperthermia-induced cellular and organ injury and could be used alongside more feasible yet less aggressive cooling modalities. Accomplishing this objective, however, requires an improved understanding of how hyperthermia activates the immune system and how the subsequent hyper-inflammatory state contributes to the development of heat stroke and cell and organ injury, particularly in the heart and kidneys. Such information is not only vital towards the development of novel therapies but would also inform new heat illness prevention strategies and risk identification pathways that have important implications for policy and policy implementation.

To put it simply, it's hot now and the future will be hotter, but actions taken today will help protect even the most vulnerable tomorrow.

Declaration of interests

The authors have no conflicts of interest to disclose as it relates to the preparation of this manuscript.

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