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Why not 35°C? Reasons for reductions in limits of human thermal tolerance and their implications

Comment on: Vecellio DJ, Kong Q, Kenney WL, Huber M. Greatly enhanced risk to humans as a consequence of empirically determined lower moist heat stress tolerance. Proc Natl Acad Sci U S A. 2023;120(42):e2305427120. doi: 10.1073/pnas.2305427120.

2023 was the hottest summer in recorded history globally, with heatwaves impacting nearly all regions of the world during the course of the year. It pushed the planet closer to the 1.5°C warming limit (for a single year) that most scientists agree needs to be the ceiling for climate change-induced warming before ecological degradation begins to rapidly increase. It also turned out that the most extreme conditions during the year put some regions close to the limits of what the human body can tolerate during extreme heat events.

In our recent study using future projections from state-of-the-art climate model simulations [1], we found large swaths of the world's population, primarily located in South and East Asia, the Middle East, and sub-Saharan Africa, will be exposed to environmental heat stress that exceed limits of human heat tolerance with as little as 1.5–2°C of warming above preindustrial levels, another indicator of the drastic impacts climate change will place on human health. In worlds with warming over 2°C, annual hours of exposure to uncompensable environmental heat stress would exponentially increase in the regions above and would begin to emerge in other regions such as North and South America at 3°C and Australia at 4°C. The novelty of the study lies with the empirically obtained limits to human heat tolerance it used by recruiting young, healthy, unacclimatized subjects as part of the Penn State University Human Environmental Age Thresholds (PSU HEAT) study [2]. Using data from those experiments, we found in an earlier study [3] that even in populations that should thermoregulate at near peak efficiency, wet-bulb temperature (T_{wb}) thresholds for heat stress compensability were closer to 30.6°C in warm-humid conditions (relative humidity values greater than 50% and temperatures 40°C or below) with a linear decrease across hotter and drier environments. These limits were significantly less than the widely cited 35°C T_{wb} limit theorized as an “upper adaptability limit” to extreme heat via loose biophysical principles by Sherwood and Huber in a 2010 study, hereby referred to as SH10 [4].

Here, we provide a brief explanation of the most evident reasons that physiological critical limits are lower than those based on biophysical theory (Figure 1). That is, why do the least vulnerable, healthiest humans not reach the theoretical SH10 upper limit for thermal balance that has been used to communicate extreme heat tolerance in the literature and mass media for over the past decade-plus? Additionally, we point out additional research that has now been completed, and those studies yet to be performed, to develop a fuller understanding of critical environmental thresholds for diverse populations across the world.

Many of SH10's assumptions were based on interactions between the skin surface and the overlying atmosphere and how that affects the human energy balance. The human skin plays a large role in allowing the body to maintain homeostasis via its interactions with both the ambient environment and the body's internal core [5]. As part of SH10's assumptions, a maximum, clamped skin temperature of 35°C was assumed, which would have maintained a core-to-skin temperature gradient to allow for efficient flow of heat away from the body given core temperatures between 37°C and 38°C. However, in empirical testing, skin temperatures ranged from 36°C to 38.5°C at the critical environmental limits (i.e. environments associated with excessive heat storage relative to heat loss and failure to maintain core temperature), increasing as a function of the dry-bulb temperature (T_{db}) [2]. With higher skin temperatures associated with cutaneous vasodilation, dry heat gain, and reduced evaporative heat transfer, the efficiency of moving heat from core to skin was diminished. In environments in which T_{db} exceeds skin temperature, the SH10 model does not

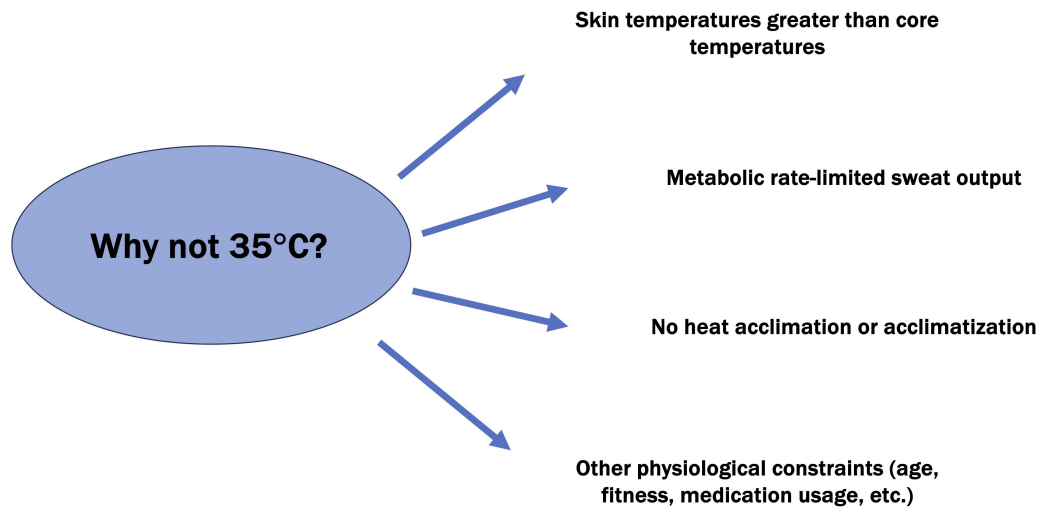


Figure 1. Schematic listing some of the reasons why the theorized 35°C wet-bulb temperature upper limit threshold for human adaptability to extreme heat falls short under realistic conditions.

account for dry heat gain through convection and radiation. Skin temperatures are highly influenced by sweating as the evaporation of sweat is the primary physiological means of body cooling for humans. However, sweating is a physiologically controlled process. For example, sweat rate, as tied to required evaporative loss, typically exhibits a non-linear relationship with metabolic workload [6]. In the PSU HEAT study, participants exerted themselves at low metabolic intensities, averaging 83.3 W/m² [2,3]. This led to relatively low sweat rates (~97–183 g m⁻² h⁻¹) which increased with skin temperature. The SH10 model assumes a constant sweat rate, full skin wettedness, and continuous evaporational cooling as the ambient environment permits. However, sweating in humans is not as straight-forward as implemented into SH10’s best-case theory. Sweat rate, as well as skin blood flow, are on the efferent arm of multiple non-thermoregulatory reflexes associated with blood pressure regulation, osmoregulation, etc. As core (or mean body) temperature increases linearly, sweat gland output – and sweating – increase after a lag period but then plateau. Multiple stimuli contribute to this, including increased sweat gland ductal resorption. Additionally, during prolonged sweating, especially in humid environments, sweat rate can decrease as eccrine gland pores swell, known as hidromeiosis, though this usually takes hours. Additionally, while not explicitly stated, SH10 assumed unimpeded evaporation from the body while PSU HEAT participants and humans in everyday scenarios wear clothing that imparts resistance to evaporative heat loss.

Other issues likely played a role in deviating from the theoretical threshold. Subjects in PSU HEAT studies were not acclimated (experiments were spaced out so subjects did not become used to the heat) or acclimatized (all subjects lived in the temperate central Pennsylvania climate which does not experience extreme heat found in the experimental protocols). More chronic exposure to extreme heat conditions can lead to the development of advanced physiological protections such as improved cardiovascular efficiency and sweating and lower core temperatures before and during heat stress. Epidemiological evidence shows that there is a latitudinal (and annual average temperature) dependence on minimum mortality temperatures across the globe, indicating prolonged exposure to higher temperatures and higher humidity provide protection to environmental conditions [7]. Human responses to environmental heat stress are not homogeneous (i.e. they do not behave like physical wet-bulb thermometers). As homeotherms who regulate core temperature using a variety of thermal and non-thermal mechanisms, a simple biophysical model cannot fully explain internal biological processes or variability across the human population. This variability can be captured in empirical studies involving large numbers of human subjects and used to determine confidence intervals, etc. It is important to note that the results from the set of PSU HEAT papers in young, healthy men and women represent a “best-case, worst-case scenario” Future research should be done in these “best-case” populations of

young, healthy participants from regions more appropriate for acclimation and acclimatization such as the desert southwest of the USA. (hot-dry conditions) or sub-tropical and equatorial regions of the globe (e.g. South Asia, sub-Saharan Africa) which are hot and humid with lesser temperature variability for longer periods of time.

To better understand the diversity in ranges of tolerances to extreme heat in humans, “worst-case,” more vulnerable populations that are associated with diminished thermoregulatory capacity should also be studied. For example, the PSU HEAT team followed up on the studies of young, healthy populations by examining critical environmental limits in adults aged 65 and above, finding that even relatively healthy older adults experienced uncompensable heat stress in conditions less warm and less humid than the younger population [8]. However, increased variability also existed within this cohort, hopefully providing insight into what comorbidities or lifestyle choices may better suit elderly individuals for dealing with extreme heat.

Climate change will remain a constant well into the future and will bring with it increases in extreme heat events which will in turn have large implications for human health in the coming decades. It will take a transdisciplinary effort to address the impacts climate change will have on different populations across the world and how thermal physiology will dictate the heterogeneous responses to a warmer world.

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