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Heatstroke at home: Prediction by thermoregulation modeling

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ABSTRACT

Background: As global warming continues, the incidence of heatstroke due to exposure to high temperature will become more prevalent. Although most heatstroke deaths occur at home, the effect of home environment on the development of heatstroke has not yet been addressed.

Objectives: To investigate the influences of home environmental parameters on the development of heatstroke using thermoregulation modeling.

Methods: A classical multi-segment multi-node human thermoregulatory model was extended to consider the role of dehydration in the sweating and cutaneous vasodilation during exposure to heat stress. The model was then used to systematically examine the effects of indoor critical environmental parameters on the time course of body core temperature (T_{core}) in the development of heatstroke, and to estimate the survival time (τ_s).

Results: Our extended thermoregulatory model predicted that T_{core} course during heat stress consists of three stages: an initial linear increase due to uncompensated heat load, an equilibrium plateau due to sweating, and a final rapid progression due to dehydration. Heatstroke occurred either early at the first stage due to the lack of thermoregulation in hot and humid environment or late at the third stage due to thermoregulatory breakdown caused by dehydration in warm and dry environment. Increasing air velocity delayed occurrence time of heatstroke in warm and dry environment but advanced the occurrence time in hot and humid environment. We obtained the survival time $\tau_s = \exp(50 - ISI)$ where $ISI = 44.2 + 0.02T_a + 0.209RH - 6.55RH/T_a$ is the indoor stress index, indicating that the time that heatstroke occurs was exponentially increased with the indoor air temperature (Ta) and relative humidity (RH).

Conclusions: We suggested a new thermoregulation model by considering the role of dehydration in the development of body core temperature during heat stress, which can be used to predict heatstroke at home. Easy-to-use indoor stress index and survival time table were obtained to early recognize and alert the risk of heatstroke.

1. Introduction

Global warming continues with no sign of slowing down [28], which results in more frequent, persistent, and intense heat waves [41]. Exposure to prolonged periods of high temperature during heat waves can cause heat-related illnesses [33] that cause excess mortality [62], such as 2003 European heat wave caused more than 7000 deaths [21].

Heatstroke, the most severe heat-related illness, is clinically defined as body core temperature (T_{core}) higher than 40.6 °C [6]. During heat waves, heatstroke occurred in an epidemic form, as witnessed by sharply increased admissions in both hospital emergency department (ED) visits and intensive care unit (ICU) [16]. Heatstroke is associated with high mortality rate, and also contributes to late deaths resulting from exacerbation of many pre-existing health conditions [30].

Heatstroke can rapidly become life threatening and people with severe heatstroke symptoms have little time to seek treatment in ED or hospitals [31]. It has been observed that most excess deaths were at home during heat waves [38,43], for example, during the 2003 European heat wave more than 50% of the fatalities in France occurred in homes [21]. However, so far studies of heat-related mortality have mainly focused on the increase in ambient temperature. The effect of indoor environmental parameters on heatstroke has never been investigated and thus warrants immediate attention.

Early recognition is an effective approach to prevent the risk of heatstroke. However, the time course of the development of heatstroke is unknown, because there is a lack of the clinical data. Our objective is to predict the time course of the development of heatstroke based on thermoregulation mechanism. Biologically speaking, heatstroke occurs

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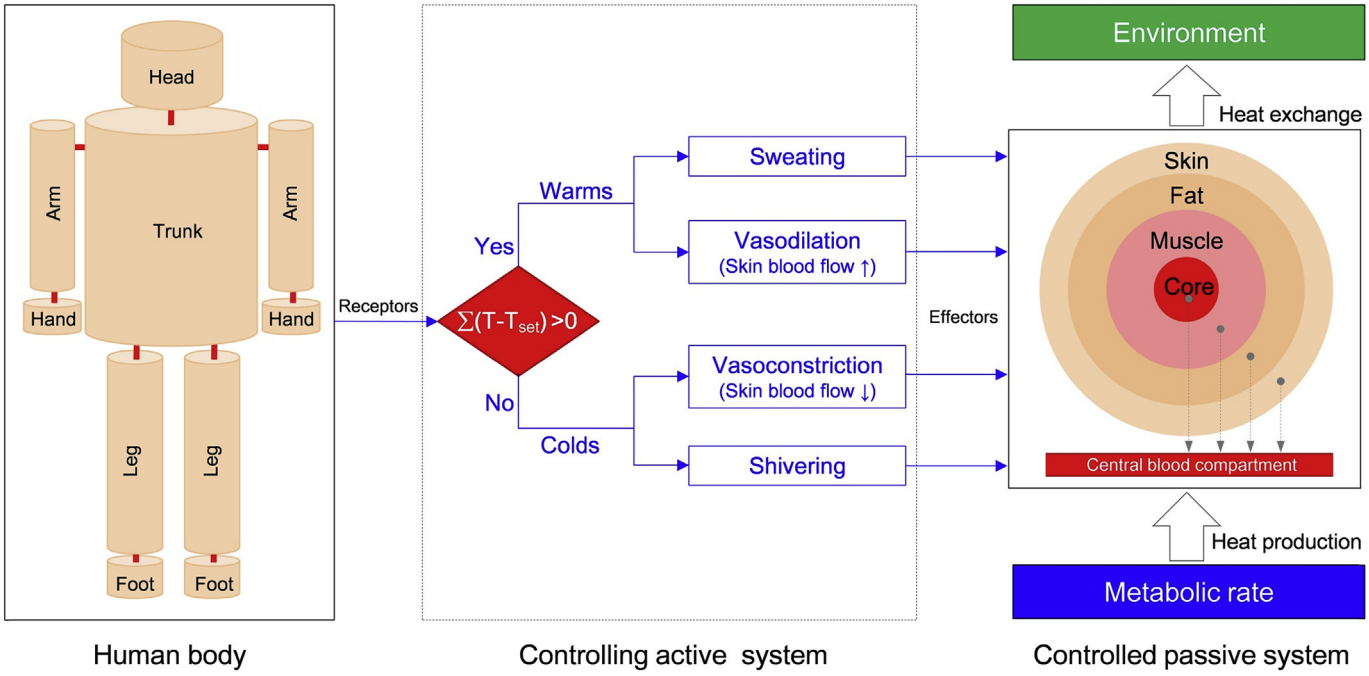

Fig. 1. Multi-segment multi-node human thermoregulatory model.

when the metabolic and environmental accumulated heat exceeds the body's ability to dissipate that leads to an excessive increase in T_{core} [13].

The objective of our work is to predict and hence early recognize heatstroke at home using human thermoregulatory model. We first extend the classical human thermoregulatory model so as to consider the role of high temperature in the development of heat-related illness and then analyze the effects of indoor environmental parameters on the time course of heatstroke development.

2. Methods

2.1. Human thermoregulatory modeling

A classical multi-segment multi-node human thermoregulation model [55,56] was implemented in our study. We considered a naked man with an average body weight of 74.1 kg and a surface area of 1.89 m^2. The model divided human body into 6 segments (head, trunk, arms, hands, legs and feet), each segment consisting of 4 concentric layers (core, muscle, fat and skin), and a central blood compartment (Fig. 1). The thermoregulation model is a negative feedback system that comprises controlling active and controlled passive sub-systems [49]. In the active system, the thermoafferent information is first transmitted by the skin thermoreceptors to central nervous system (CNS) where the information is integrated into thermoefferent signals (warms or colds). These signals are then forwarded by autonomic nervous system (ANS) to peripheral effectors that regulate cutaneous vasodilation and sweating (if warms) or cutaneous vasoconstriction and shivering (if colds). The effector responses together with metabolic rate finally alter the balance between heat gain and heat loss within the body. In the passive system, heat flow between adjacent layers is by conduction, all layers exchange heat by convection with a central blood compartment, and heat exchange between the skin and environment by convection, radiation, evaporation and respiration.

In order to predict the time course of the development of heatstroke during exposure to high temperature, we improved the classical thermoregulation model by considering the effect of dehydration (*de*), percentage of water loss due to sweating to the total body weight (74.1 kg) defined as follows

$$de = \frac{\int_0^{\tau} \text{Sweat} \times dt}{74.1} \times 100\,(\%) \tag{1}$$

Classical thermoregulatory model was based on set-point (fixed threshold) concept [8], i.e. the strength of the efferent signals depended on the comparison between the afferent signals and set-point temperatures (T_{set}). However, recent experimental studies have found that the threshold temperature is not fixed but varies within a zone [8,42,49]. Mounting evidence shows that *de* not only decreases the skin blood flow by increasing the threshold for cutaneous vasodilation but also degrades the sweat response by increasing the threshold temperature and decreasing the sensitivity [22,42,44]. Therefore, we considered the role of dehydration *de* in the thermoregulation model [55,56] as shown in Fig. 2:

$$\text{Sweat} = (0.2898 - \beta \times de) \times (T - (T_{set} + \alpha \times de)) + 0.0336 \times (\text{Warms} - \text{Colds}) \tag{2}$$

$$\text{Vasodilation} = 117 \times (T - (T_{set} + \gamma \times de)) + 7.5 \times (\text{Warms} - \text{Colds}) \tag{3}$$

where T_{set} is set-point temperature, Warms and Colds are integrated signals from the skin thermoreceptors, and the parameters α, β, γ are summarized in Table 1. It is clear that the effect of *de* was not considered in the classical thermoregulatory model. In our model, we set $\alpha = 0.06$ and $\beta = 0.068$ according to a recent experiment on the role of *de* in the thermoregulatory sweating [44], and set $\gamma = 0.06$ based on an experiment [20] and modeling [32]. Therefore, our work assumed that 1% dehydration (*de*) elevates thresholds for sweating and cutaneous vasodilation by 0.06 °C and decreases sensitivities for sweating by 0.068 kg/(hr·°C). The effect of the parameters and validation were in "4.1. Validation".

2.2. Survival time

Survival time (τ_s) is defined as the maximal tolerance time before heatstroke occurs (T_{core} = 40.6 °C) during heat exposure.