Current evidence does not support an anticipatory regulation of exercise intensity mediated by rate of body heat storage

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A growing body of literature has offered purported evidence of a “central governor” mechanism providing an “anticipatory” control of exercise performance to prevent catastrophic failures of homeostasis in any physiological system (13, 14, 16). The notion that sensations such as “thirst” and “perceived exertion” cause a preemptive reduction in motor unit recruitment and therefore exercise performance to avoid otherwise damaging levels of dehydration and skeletal muscle energy, respectively, (17, 24) have initiated considerable debate (24, 28). However, despite being heavily cited as a progressive exercise model to evaluate performance in the heat (1–3, 5, 10, 13, 15), the claim that uncontrollable hyperthermia is prevented via a feedforward calculation of the rate of body heat storage (26) remains relatively uncontested.

It is clear that hyperthermia causes fatigue and that performance degradation and subsequent exhaustion during exercise in a hot environment coincides with the attainment of a high core temperature (8); the critical values of which appear to be influenced by multiple factors including dehydration and skin temperature (25) as well as central fatigue (18). However, when considering the known time courses of thermogenesis and thermolysis during exercise, the evidence of an anticipatory feedforward mechanism regarding the rate of body heat storage (26) appears fundamentally flawed.

The principal evidence is the report (26) that subjects self-regulating cycling power output at a fixed rating of perceived exertion under three separate air temperatures (T_a = 15°C, 25°C, and 35°C) only showed significantly different rates of body heat storage during the initial 4-min of exercise despite an air temperature difference of 20°C between the hottest and coolest conditions. These supposed early heat storage differences were thought to be “fed forward” with power output subsequently regulated to elicit similar near-zero rates of body heat storage, irrespective of environmental conditions.

By definition, rate of body heat storage is the instantaneous difference between the rates of metabolic heat production and net heat loss by combined evaporative and dry heat exchange. At the onset of exercise, heat production is instantly elevated due to the liberation of energy supplying the demands of working muscle groups. In contrast, rate of net heat loss responds much slower with a slight delay followed by an exponential increase with a time constant of ~10 min (12, 27). This inertia gives an increase in body heat content and a concurrent rise in core temperature eliciting reflex heat loss mechanisms of sweating and cutaneous vasodilatation to eventually compensate for elevated heat production. A heat storage rate of zero only occurs after ~20 to 40 min of constant exercise (12, 27), and this is only possible if heat production is within the thermolytic capacity of the person, primarily determined by acclimation and hydration status as well as environmental biophysics (i.e., ambient temperature, evaporative capacity, clothing).

The crux of the feedforward mechanism was the early difference in rate of body heat storage between 15 and 35°C [Fig. 3 (26)]. The source of these differences was the negative values estimated at 15°C. After 1 min, heat storage was reportedly ~85 kJ/min (~1,420 W) and external work was 245 W [Fig. 2 (26)]. Cycling work efficiency is ~29% (29). Therefore, heat production after 1 min would be ~625 W. For body heat storage to be ~1,420 W, a net heat loss of 2,045 W is required. Since minimal changes in sweating and vasodilation occur after 1 min, heat loss must primarily be via dry heat exchange. Ambient air temperature was 15.1°C with 10 km/h (2.8 m/s) air movement. According to heat balance calculations (7) that employ a conservative estimation (0.2 clo) of dry clothing insulation (no clothing details were provided), rate of dry heat exchange after 1 min was ~360 W. In fact, even if the participants were nude this value would only be ~570 W. The reported values are therefore clearly inconceivable. Equally unlikely is despite power output only declining by ~25 W after 4 min at 15°C [Fig. 2 (26)], rate of net heat loss reduced from 2,045 to 540 W (rate of heat storage was reportedly ~0 W). Environmental conditions remained unchanged and sudomotor/vasomotor activity if anything would have increased slightly [core temperature increased; Fig. 5 (26)].

Rates of body heat storage were estimated by calculating minute-by-minute changes in body heat content; which itself was estimated using the product of body mass; an estimated average specific heat (C_p) of 3.47 kJ·kg^{-1}·°C^{-1}; and volume-weighted (mean) body temperature (T_{body}) derived from the two-compartment thermometry model of “core” using rectal temperature (T_{rec}) and “shell” using mean skin temperature (T_{sk}) (4). Relative contributions of each compartment were determined by fixed sum-to-one weighting coefficients (6). Since mass and C_p remained constant throughout, changes in the rate of body heat storage were solely dependent on minute-by-minute changes in T_{body}, or more specifically T_{rec}, and/or T_{sk}. Only minor early changes in T_{rec} occurred, so the single difference between the 15°C and 35°C conditions to be fed-forward within the first 4 min of exercise were changes in T_{sk} [~3°C at 15°C; +2°C at 35°C; Fig. 6 (26)]. Forty percent of T_{sk} was derived from the legs (21); therefore initial changes in T_{sk} were likely due to altered dry heat exchange of the legs when they began moving.
Absolute power output (and therefore heat production) during exercise declined fastest at 35°C [Fig. 4B (26)]. Therefore relative to 15°C, the rate of net heat loss (dry + evaporative heat exchange) required for heat balance (i.e., zero rate of heat storage) at 35°C was lower. However, nearly all net heat loss at 35°C would have occurred by evaporation since $T_{sk}$ (storage) at 35°C was lower. However, nearly all net heat loss exchange) required for heat balance (i.e., zero rate of heat exchange) by-minute changes in $T_{re}$ particularly when the primary source of body heat storage would not be adequately reflected by minute-wise changes in $T_{sk}$ were negative, which by their model would imply early changes in brain and/or muscle temperature, but no such anticipatory reduction in exercise intensity may occur relative to “downregulate” RPE eliciting an increase in exercise intensity. An alternative thermal evidence lies with the early changes in $T_{sk}$. While the authors do cite the primary role of afferent information from thermoreceptors (26), early changes in $T_{sk}$ were minor; and at 15°C changes in $T_{sk}$ were negative, which by their model would “downregulate” RPE eliciting an increase in exercise intensity. An anticipatory reduction in exercise intensity may occur relative to early changes in brain and/or muscle temperature, but no such reaction currently exists.

REFERENCES