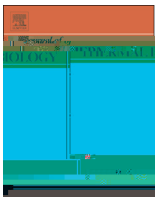


Isothermic and fixed intensity heat acclimation methods induce similar heat adaptation following short and long-term timescales

Oliver R. Gibson [a,*], Jessica A. Mee [a], James A. Tuttle [b], Lee Taylor [b], Peter W. Watt [a], Neil S. Maxwell [a]

[a] Centre for Sport and Exercise Science and Medicine (SESAME), Exercise in Extreme Environments Laboratory, University of Brighton, Welkin Human Performance Laboratories, Denton Road, Eastbourne, UK
[b] Muscle Cellular and Molecular Physiology (MCMP) and Applied Sport and Exercise Science (ASEP) Research Groups, Department of Sport Science and Physical Activity, Institute of Sport and Physical Activity Research (ISPAR), University of Bedfordshire, Bedford Campus, Polhill Avenue, Bedfordshire, UK

ve 90 min sessions) utilising either fixed intensity (50%

$\dot{V}_{O_2\ ea}$), continuous isothermic (target rectal temperature 38.5 °C for STHA and LTHA), or progressive isothermic heat acclimation (target rectal temperature 38.5 °C for STHA, and 39.0 °C for LTHA). Identical heat stress tests followed STHA and LTHA to determine the magnitude of adaptation.

All methods induced equal adaptation from baseline however isothermic methods induced adaptation and reduced exercise durations (STHA¼ ▫ 66% and LTHA¼ ▫ 72%) and mean session intensity (STHA¼ ▫ 13% $\dot{V}_{O_2\ ea}$ and LTHA¼ ▫ 9% V

1.

Repeated exposure to stressful hot environments initiates the heat-adapted phenotype. The heat-adapted phenotype is acquired most effectively when hot and humid environmental conditions and physical work (intensity, duration and frequency) interact to stress thermoregulatory and cardiovascular systems (Sawka et al., 2011); this process is known as heat acclimation (Garrett et al., 2011). Primary adaptations induced by heat acclimation include decreased core temperature (Armstrong and Maresh, 1991; Buono et al., 1998; Garrett et al., 2011) and reduced heat storage (Aoyagi et al., 1997

(W kg^{-1}) was calculated to describe the initial requirements of each heat acclimation method in accordance with the guidelines of Cramer and Jay (2014).

$\dot{V}_{O2\ ea}$ (L min^{-1}) was determined from an incremental test on a cycle ergometer (Monark e724, Vansbro, Sweden) in temperate laboratory conditions (20 °

mounted a cycle ergometer (Monark, e724, Vansbro, Sweden) located inside the environmental chamber where conditions were consistent for all groups (40.2 ± 0.4 °C, 39.0 ± 7.8% RH). The FIXED participants performed 10 sessions of 90 min of continuous cycling exercise at a workload corresponding to 50% $\dot{V}_{O2\ ea}$

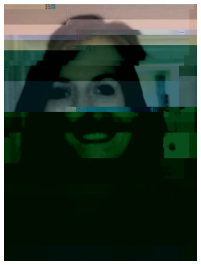
all demonstrated improvements overall as the number of heat acclimation sessions increased. Post-hoc analysis revealed resting T_{rec} was reduced ($p < 0.05$) from session one before session eight,

matching of training stimulus is required. Exercising durations were lower in isothermic methods (66–79% of session duration) compared to fixed intensity heat acclimation (> 99% of session

phenomenon, although the molecular role of prostaglandin E2 (PGE2), cyclooxygenase (COX)-2 and orexin cannot be excluded (Shin et al., 2013). Change in T_{rec} did not attenuate from HST1 following STHA or LTHA, therefore adaptations did not offset rate of heat gain (Schlader et al., 2011a). Reduced HR following STHA, combined with lower T_{rec}, indicated lower overall physiological strain during HST2, but a further five days of any heat acclimation method did not elicit further adaptations at HST3 (Kampmann et al., 2008

isothermic heat acclimation under conditions of greater exogenous heat stress than forthcoming competition, to facilitate maximal thermal adaptation for reduced exercise training requirement. The reduction in training volume being an essential component of the taper (Spilsbury et al., 2014), establishing cardiorespiratory, vascular, haematological and neuromuscular changes which ultimately contribute towards optimal performance (Mujika et al., 2004). Additionally due to the greater absolute $\dot{V}_{O_2}$, and consequently metabolic heat production, for the same relative workload athletes typically exhibit, the rate of rectal temperature increase is

heat acclimation in moderately and highly trained athletes. Sports Med. 41, 757–771. http://dx.doi.org/10.2165/11587320-000000000-00000.
Gibson, O.R., Dennis, A., Parfitt, T., Taylor, L., Watt, P.W., Maxwell, N.S., 2014. Extracellular Hsp72 concentration relates to a minimum endogenous criteria during acute exercise-heat exposure. Cell Stress Chaperones 19, 389–400. http:



received her undergraduate degree in Sport Science from the University of Brighton in 2010. Jessica began her Ph.D. at the University of Brighton in 2011, which is examining the thermo tolerance and adaptation to the heat in females. Jessica is a technical instructor for the undergraduate and postgraduate