

Postexercise whole-body sweating increases during muscle metaboreceptor activation in young men

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Abstract: We assessed the effect of metaboreceptor activation on whole-body evaporative heat loss (WB-EHL) in 12 men (aged 24 ± 4 years) in the early-to-late stages of a 60-min exercise recovery in the heat. Metaboreceptor activation induced by 1-min isometric-handgrip (IHG) exercise followed by 5-min forearm ischemia to trap metabolites increased WB-EHL by 25%–31% and 26%–34% during the ischemic period relative to IHG-only and control (natural recovery only), respectively, throughout recovery. We show that metaboreceptor activation enhances WB-EHL in recovery.

Key words: exercise, heat loss, calorimetry, nonthermal, isometric, sweat rate.

Résumé : On évalue chez 12 hommes (âgés de 24 ± 4 ans) l'effet de l'activation des métaborécepteurs sur la perte de chaleur de tout le corps par évaporation (« WB-EHL ») du début à la fin de la récupération de 60 min d'exercice dans la chaleur. L'activation des métaborécepteurs induite par 1 min de préhension manuelle isométrique (« IHG ») suivie de 5 min d'ischémie de l'avant-bras afin de coincer les métabolites engendre une augmentation de 25–31 % et 26–34 % de WB-EHL respectivement durant la période ischémique par rapport à IHG seul et au contrôle (récupération naturelle seulement) tout au long de la récupération. Nous démontrons que l'activation des métaborécepteurs accroît la WB-EHL durant la récupération. [Traduit par la Rédaction]

Mots-clés : exercice physique, perte de chaleur, calorimétrie, non-thermal, isométrique, taux de sudation.

Introduction

An increasing number of studies demonstrate that nonthermal factors associated with the activation of sensory end organs of baroreceptors, osmoreceptors, and metaboreceptors play an important role in the regulation of heat loss responses during heat stress (Kenny and Journey 2010; Kondo et al. 2010). For instance, studies show that independent of thermal control, nonthermal factors can simultaneously act on the thermoregulatory system to modulate heat dissipation during the postexercise recovery period (Kenny and Journey 2010; Kenny and McGinn 2017). Upon cessation of exercise, the rapid decrease in metabolic heat liberation is paralleled by a decline in local and whole-body heat loss to pre-exercise resting levels despite sustained elevations in body temperatures (i.e., core and muscle) and therefore body heat storage (Kenny et al. 2008; Kenny and Journey 2010). This response, which has been shown to last for up to 120 min, has been primarily attributed to the overriding influences of nonthermal factors on postexercise thermoregulatory function (Kenny et al. 2008; Kenny and Journey 2010).

Until recently, the nonthermal-mediated postexercise disturbance in thermal homeostasis was largely ascribed to a baroreflex-mediated response associated with postexercise hypotension (Kenny and Journey 2010). However, recent findings reveal that other nonthermal factors associated with metaboreceptor activation may be involved (McGinn et al. 2014; Paull et al. 2015; Kenny and McGinn 2017). The accumulation of metabolites within the active

skeletal muscle has been shown to stimulate group III and IV afferent neurons, which in turn evoke a reflex increase in muscle sympathetic nerve activity, known as the muscle metaboreflex (Rowell and O'Leary 1990). The activation of metaboreceptors have been shown to modulate heat loss responses during both passive heat stress and exercise (Crandall et al. 1998; Shibasaki et al. 2001; Boushel 2010) with recent studies extending their influence to the postexercise period (McGinn et al. 2014; Paull et al. 2015). These latter studies demonstrate that the influence of baroreceptors may be limited to the control of local skin blood flow whereas metaboreceptors, and not baroreceptors, may largely influence the regulation of local sweating (McGinn et al. 2014; Paull et al. 2015), which is an important avenue of heat dissipation during heat stress. Specifically, metaboreceptor activation, induced by a brief isometric handgrip (IHG) exercise followed by forearm ischemia to trap metabolites, was shown to delay the rapid reduction in local sweating occurring in the early stages of recovery (≤ 20 min) (McGinn et al. 2014; Paull et al. 2015) while increasing local sweating during the mid-to-late stages of recovery (30- to 60-min of recovery) relative to a natural recovery (no IHG or ischemia) (Paull et al. 2015). Despite regional variations in absolute local sweat rate, a consistent response was observed across different body regions (i.e., chest, forearm, and upper back) during the activation of metaboreceptors (Paull et al. 2015). Taken together, these findings indicate that the metaboreceptor-mediated modulation of local sweating may have an important effect on whole-body sweating. In this context, it is possible that changes in

Received 10 October 2017. Accepted 2 January 2018.

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*Glen P. Kenny currently serves as an Associate Editor; peer review and editorial decisions regarding this manuscript were handled by A. William Sheel and Terry Graham.

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metaboreceptor activity in the postexercise period may play an important role in the observed disruption of thermal homeostasis.

Thus, in the present study, we examined the effect of metaboreceptor activation on whole-body sweating by assessing whole-body evaporative heat loss (WB-EHL) during the early (15 min), mid (30 min), and late (45 min) stages of recovery following a 45-min moderate-intensity exercise bout in the heat (35 °C, 20% relative humidity). To evaluate this response, we used direct calorimetry to precisely measure WB-EHL (Kenny et al. 2017). We evaluated the hypothesis that following dynamic exercise, WB-EHL would increase in response to the activation of metaboreceptors induced by a period of post-IHG ischemia throughout the early-to-late stages of the 60-min recovery period relative to both IHG exercise only and a natural recovery.

Materials and methods

Ethical approval

The experimental protocol was approved by the University of Ottawa Health Sciences and Science Research Ethics Board and agrees with the *Declaration of Helsinki*. Written and informed consent was obtained from all participants prior to their participation in the study.

Participants

Twelve healthy habitually active young males (age: 24 ± 4 years; height: 1.7 ± 0.7 m; body mass: 76.0 ± 8.1 kg; body surface area: 1.90 ± 0.11 m²; peak oxygen consumption ($\dot{V}O_{2\text{peak}}$): 49.4 ± 7.7 mL O₂·kg⁻¹·min⁻¹; body fat percentage: $16.9\% \pm 5.4\%$) participated in this study.

Experimental design

Participants completed 1 preliminary and 3 experimental sessions each separated by at least 48 h. For all sessions, participants refrained from exercise, alcohol, or caffeine 24 h prior to experimentation. During the preliminary session, the participant's physical characteristics (height, weight, and body composition) were assessed. Additionally, aerobic fitness (defined by $\dot{V}O_{2\text{peak}}$) was determined through an incremental cycling exercise protocol. Participants performed two 5-s maximal voluntary contractions (MVC) to establish the relative intensity (determined using the higher of the 2 values) for the IHG exercise. The detailed procedures are reported elsewhere (McGinn et al. 2014; Paull et al. 2015). For each experimental session, following instrumentation, participants rested inside the direct air calorimeter regulated at 35 °C (20% relative humidity) for a 20-min resting period (pre-exercise resting) followed by a 45-min bout of cycling at a fixed rate of metabolic heat liberation of 400 W (to maintain a constant thermal drive for sweating between conditions) and a 60-min recovery. During recovery, they performed either (i) a 1-min IHG exercise at 60% of their predetermined MVC (IHG-only), (ii) a 1-min IHG at 60% of MVC followed by a 5-min forearm ischemia (IHG+OCC), or (iii) a natural recovery with no IHG or OCC (control). Forearm occlusion was achieved by inflating a blood pressure cuff to supra-systolic levels that was then deflated rapidly for a 5-min recovery period. The protocols were repeated in the early (15 min), mid (30 min), and late (45 min) stages of the 60-min recovery data collection period. During the period of muscle ischemia, it is thought that the accumulation of metabolites within the muscle triggers chemosensitive afferents (group III and IV afferents) and reflexively raises arterial blood pressure, which is used to confirm the activation of metaboreceptors (Rowell and O'Leary 1990).

Instrumentation

The modified Snellen direct air calorimeter was used to perform continuous measurements of WB-EHL and whole-body dry heat loss (WB-DHL), and metabolic heat liberation was calculated by indirect calorimetry (Kenny et al. 2017). Esophageal temperature

was measured using a thermocouple probe (Mallinckrodt Medical Inc., St-Louis, Mo., USA) estimated to be in the region bounded by the left ventricle and aorta. Skin temperature was measured using 0.3-mm diameter T-type (copper/constantan) thermocouples integrated into heat flow sensors (Concept Engineering, Old Saybrook, Conn., USA) affixed to the chest, biceps, thigh, and calf (Concept Engineering). Mean skin temperature was calculated using the following weightings: chest, 30%; bicep, 30%; thigh, 20%; and calf, 20% (Ramanathan 1964). Calorimetry and temperature data were recorded continuously on a personal computer with LabVIEW software (version 7.0; National Instruments, Austin, Texas, USA). Mean arterial blood pressure (MAP) (Finapres Medical Systems, Amsterdam, the Netherlands) was measured continuously. Isometric handgrip exercise was performed using a Smedley Hand Dynamometer (Model 19117, Stoelting Co, Wood Dale, Ill., USA).

Data analysis

Pre-exercise resting and end-exercise values were averaged over the last 5 min. For the postexercise recovery, values for each of the 3 recovery periods were assessed based on the 4 time stages for the metaboreceptor activation protocol as follows: (i) 1-min period preceding the IHG (Pre-IHG), (ii) last 15 s of the IHG exercise (IHG), (iii) the final 15 s of the postexercise forearm occlusion (OCC), and (iv) final 15 s of the post-OCC recovery period (REC). Body heat storage was calculated as the temporal summation of metabolic heat liberation and heat loss within the 5-min occlusion phase during each recovery stage.

Statistical analysis

A 1-way ANOVA with the repeated factor of condition (control, IHG-only, IHG+OCC) was conducted for all dependent variables for pre-exercise resting and end-exercise as well as for the change in body heat storage across conditions. When a main effect of condition was measured, post hoc analysis was conducted using paired-sample *t*-tests corrected for multiple comparisons using the Bonferroni procedure. During recovery, a 2-way repeated-measures ANOVA was performed to compare the dependent variables at each stage (pre-IHG, IHG, OCC, REC) as well as between the 3 experimental conditions (control, IHG-only, IHG+OCC) for each recovery period. When a significant main effect or interaction was observed, post hoc comparisons were carried out using paired-sample *t*-tests corrected for multiple comparisons using the Bonferroni procedure. Results are presented at means \pm SD. All statistical analyses performed using IBM SPSS Statistics (version 24.0; IBM Corp., Armonk, N.Y., USA).

Results

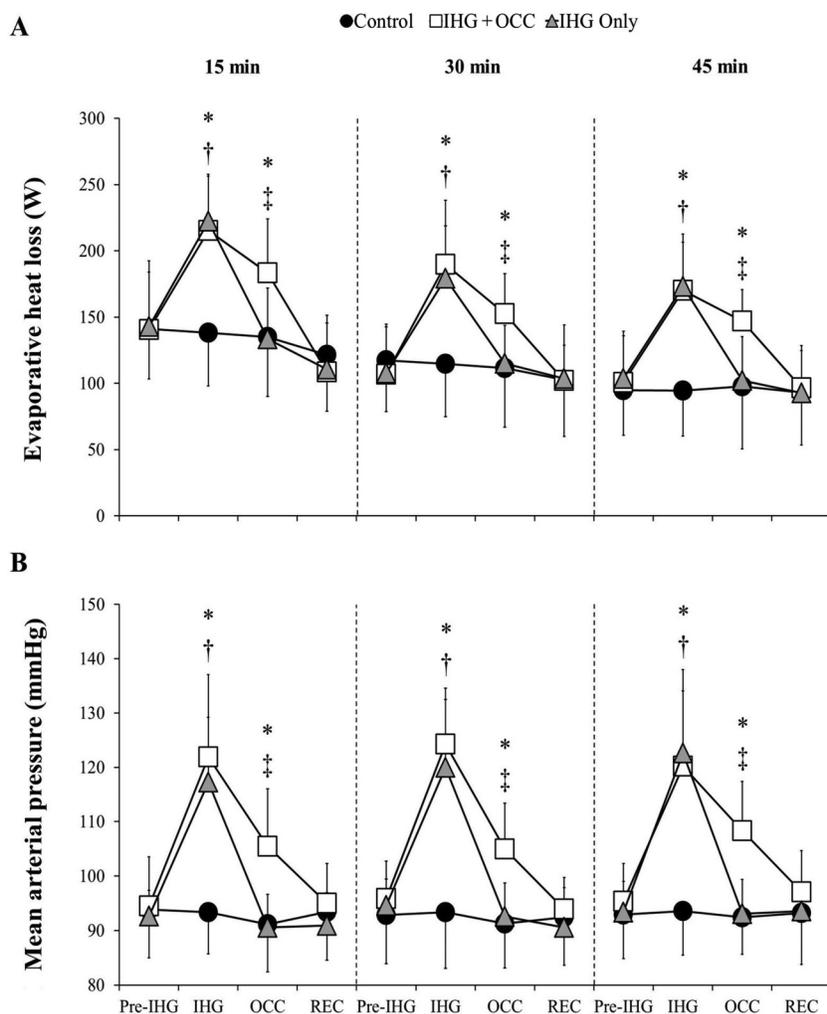
Pre-exercise resting and end-exercise

Pre-exercise resting and end-exercise responses for body temperatures (esophageal: 36.9 ± 0.3 °C, 37.3 ± 0.4 °C; mean skin: 34.4 ± 0.5 °C, 35.0 ± 0.5 °C), MAP (92 ± 5 mm Hg, 94 ± 7 mm Hg), metabolic heat liberation (108 ± 18 W, 390 ± 31 W), and heat loss (WB-EHL: 86 ± 38 W, 373 ± 51 W; WB-DHL: -10 ± 18 W, -25 ± 19 W), respectively, were similar between conditions (all $P > 0.05$).

Postexercise recovery

Metabolic heat liberation did not differ between conditions during pre-IHG, OCC, or REC phases in all recovery stages (all $P > 0.05$), averaging 113 ± 17 , 110 ± 19 , and 108 ± 15 W (early); 106 ± 13 , 108 ± 18 , and 104 ± 12 W (mid); and 110 ± 16 , 114 ± 17 , and 107 ± 14 W (late) across conditions, respectively. Metabolic heat liberation was also similar between conditions during IHG exercise during the early stage ($P = 0.11$; mean across conditions: 123 ± 18 W), yet was elevated in the mid and late stages relative to control (107 ± 15 and 101 ± 11 W, respectively) in the IHG+OCC condition (124 ± 21 and 124 ± 18 W, respectively; $P = 0.02$ and $P = 0.01$) and in the IHG-only condition in the mid-stage (122 ± 12 W; $P = 0.04$), but not in the late stage (114 ± 18 W; $P = 0.26$). WB-DHL did not differ between conditions or

Fig. 1. Whole-body evaporative heat loss (W) (A) and mean arterial pressure (mm Hg) (B) during pre-isometric hand grip exercise (pre-IHG, 1 min prior to IHG), last 15 s of isometric handgrip exercise (IHG), last 15 s of forearm occlusion (OCC), and last 15 s of recovery from forearm occlusion (REC, 5 min after the end of OCC) during control, IHG-only, and IHG+OCC conditions. Measurements were performed in the early (15 min), mid (30 min), and late (45 min) stages of postexercise recovery. *, Significantly different between IHG+OCC and control. †, Significantly different between IHG-only and control. ‡, Significantly different between IHG+OCC and IHG-only. Significance level accepted at $P \leq 0.05$. Values are presented as means \pm SD.



phases in each recovery stage (all $P > 0.05$), averaging -5 ± 17 , -2 ± 19 , and 1 ± 18 W across phases during the early, mid, and late stages, respectively. For all recovery stages, WB-EHL increased during the IHG exercise for the IHG-only and IHG+OCC conditions relative to control (all $P < 0.05$), and remained elevated during the OCC phase for the IHG+OCC condition relative to the control and IHG-only conditions (all $P < 0.05$; Fig. 1). Heat stored during OCC was reduced during the early, mid, and late stages in the IHG+OCC condition (-20 ± 10 , -15 ± 2 , and -10 ± 2 kJ, respectively) relative to control (-9 ± 3 , -2 ± 3 , and 2 ± 10 kJ, respectively; all $P = 0.03$) as well as between IHG-only (-6 ± 6 kJ) and control during the mid-stage ($P = 0.04$).

In all recovery stages, MAP did not differ between conditions during pre-IHG and REC phases (all $P > 0.05$), but increased during IHG exercise for IHG-only and IHG+OCC conditions relative to the control condition (all $P < 0.05$) and remained elevated during OCC in the IHG+OCC condition relative to the IHG-only and control conditions (all $P < 0.05$; Fig. 1). Esophageal and mean skin temperatures remained elevated above baseline but similar between conditions during the early (esophageal: 37.1 ± 0.3 °C, mean skin: 34.7 ± 0.5 °C), mid (esophageal: 37.1 ± 0.3 °C, mean skin: $34.6 \pm$

0.5 °C), and late stages (esophageal: 37.1 ± 0.3 °C, mean skin: 34.6 ± 0.4 °C; all $P > 0.05$).

Discussion

We show for the first time that the activation of muscle metaboreceptors augments WB-EHL (and therefore whole-body sweating) and that the level of influence (as defined by the relative increase in WB-EHL of ~ 45 W) remains constant throughout the early to late stages of recovery. Specifically, we showed that metaboreceptor activation induced by 1-min IHG exercise followed by 5-min forearm ischemia to trap metabolites increased WB-EHL by 25%–31% and 26%–34% during the ischemic period relative to IHG-only and control (no IHG or ischemia, natural recovery), respectively, throughout recovery. Moreover, despite the relatively short duration of the period of stimulation (i.e., 5-min ischemia), the metaboreceptor-mediated increase in evaporative heat loss was associated with a concomitant reduction in body heat storage. Taken together, our findings demonstrate that the metaboreceptor modulation of whole-body sweating is an important factor mediating the postexercise disturbance in thermal homeostasis.

Previous studies have demonstrated that the activation of metaboreceptors play an important role in the regulation of local forearm sweating (McGinn et al. 2014; Paull et al. 2015) as well as back and upper chest sweating (Paull et al. 2015) during the postexercise period. This response was shown to be independent of any changes in baroreceptor loading status that occurs during the occlusion period (McGinn et al. 2014). While previous studies showed that the activation of metaboreceptors preserved but did not augment local sweating in the early stages of recovery (i.e., ≤ 20 min) (McGinn et al. 2014; Paull et al. 2015), we observed a consistent increase in whole-body sweating throughout the early to late stages of recovery. This disparity may in part be due to differences in the level of hyperthermia achieved during the exercise. In previous studies, the level of hyperthermia prior to the start of the metaboreceptor activation protocol was greater (as defined by a greater rise in core temperature of ~ 0.3 – 0.6 °C; equivalent to ≥ 37.4 °C) relative to that recorded in the present study (~ 37.0 °C). In keeping with this observation, previous work showed that thermal control predominates over nonthermal factors at core temperatures above ~ 37.4 °C (Gagnon et al. 2008). Differences in the duration of the period of ischemia may also contribute to this response. While prior studies employed a 2-min period of ischemia, we used an extended period of ischemia (5 min) to ensure that a metaboreceptor-mediated response, if any, would be captured in our calorimetric measurement of whole-body sweating. It remains to be determined if the increase in whole-body sweating would have been observed with a shorter period of ischemia.

In summary, we show that metaboreceptor activation enhances WB-EHL and therefore whole-body sweating, throughout the early-to-late stages of the postexercise recovery period in the heat. This was associated with a 56%, 88%, and 115% greater amount of heat stored during the natural recovery (i.e., control) relative to recovery with the activation of metaboreceptors (i.e., IHG+OCC) performed in the early, mid, and late stages, respectively. Our findings demonstrate that independent of thermal control (i.e., changes in skin and/or core temperatures), changes in nonthermal metaboreceptor activity during the postexercise period play an important role in the observed disruption in whole-body thermal homeostasis. In the context of exercise or work performed in the heat, our findings demonstrate that nonthermal factors can influence the rate of heat dissipation and therefore the restoration of thermoregulation after exercise in the heat.

Conflict of interest statement

No conflict of interest, financial or otherwise, are declared by the author(s). The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

Author contributions

G.P.K. and B.J.F. conceptualized and designed the research; B.J.F., M.P.P., D.T.L., and A.W.D. performed experiments; B.J.F. and M.P.P. analyzed the data; all authors interpreted the results of experiments; G.P.K. drafted the manuscript; all authors edited

and revised the manuscript and approved final version of manuscript.

Acknowledgements

We thank all the participants who volunteered for the present study. This research was supported by the Natural Sciences and Engineering Research Council (RGPIN-298159-2009, RGPIN-2014-06313, held by Dr. Glen P. Kenny). Disclaimer: The findings and conclusions in this article are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health.

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