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The influence of pre-warming on the physiological responses to prolonged intermittent exercise

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Abstract

To examine the influence of pre-warming on the physiological responses to prolonged intermittent exercise in ambient temperatures of $21.5 \pm 0.6^\circ\text{C}$ and relative humidities of $35.7 \pm 5.4\%$ (mean $\pm$ s), six healthy men performed intermittent treadmill running (30-s bouts at 90% of maximal oxygen uptake separated by 30-s static recovery periods) to exhaustion after active pre-warming, passive pre-warming and pre-exercise rest (control). Exercise time to exhaustion was significantly different between all conditions (active, 51.8 ± 7.2 min; passive, 38.5 ± 11.1 min; control, 72.0 ± 17.2 min; $P < 0.05$). These changes in performance time were closely associated with a significant decline in both the rate of heat storage and heat storage capacity ($P < 0.05$). Rectal temperature, heart rate and ratings of perceived exertion were significantly higher during exercise in the two pre-warming conditions than in the control condition ($P < 0.05$). Ratings of perceived exertion were also significantly higher during exercise following passive pre-warming compared with active pre-warming ($P < 0.05$). During exercise there were no significant differences in serum prolactin, plasma norepinephrine and plasma free fatty acid concentrations between conditions. We conclude that both active and passive pre-warming promote a reduction in prolonged intermittent exercise capacity in environmental temperatures of 21°C compared with pre-exercise rest. These performance decrements were dependent upon the mode of pre-warming and closely reflected alterations in body heat content.

Keywords: Exhaustion, heat storage capacity, pre-warming

Introduction

The impact of pre-exercise warming in determining the capacity to perform prolonged sub-maximal exercise in ambient temperatures of 21°C has recently been demonstrated (Gregson, Drust, Batterham, & Cable, 2002). Results indicated that changes in the level of body temperature associated with pre-exercise active and passive pre-warming techniques reduce exercise time to exhaustion during prolonged sub-maximal exercise. Such alterations in performance were suggested to be mediated through mechanisms associated with the subsequent increase in thermal strain rather than the effects of elevated temperature on metabolism (Febbraio *et al.*, 1996; Gonzalez-Alonso, Teller, Andersen, Jensen, & Nielsen, 1999; Parkin, Carey, Zhao, & Febbraio, 1999).

Most studies that have increased core body temperature (using pre-warming strategies) to deter-

mine its influence on exercise performance have used continuous exercise patterns (Gonzalez-Alonso *et al.*, 1999; Gregson *et al.*, 2002). Consequently, there is little evidence regarding the effects of such thermal manipulations on the capacity to perform prolonged periods of intermittent exercise. Intermittent exercise patterns are characterized by repeated bouts of high-intensity exercise separated by periods of low-intensity exercise or static recovery. Such activity patterns form the basis of invasive field games, such as soccer, where repeated changes in exercise intensity occur over an extended period of time.

The physiological responses to intermittent exercise have been compared with those to continuous exercise at the same average exercise intensity, in an attempt to evaluate differences in the physiological demands associated with the two different exercise patterns (Cable & Bullock, 1996; Kranning & Gonzalez, 1991). Intermittent exercise has frequently been associated with an increased core

body temperature (Cable & Bullock, 1996; Kranning & Gonzalez, 1991) compared with continuous exercise. Such increases in thermal strain associated with intermittent exercise could reflect the decline in skin blood flow associated with high-intensity exercise ($> 80\%$ of maximal oxygen uptake) (Kranning & Gonzalez, 1991) and/or increased sweat rates associated with intermittent exercise (Cable & Bullock, 1996).

As a consequence of the increased thermoregulatory load induced by intermittent exercise (compared with continuous exercise), pre-exercise warming strategies may have implications for prolonged intermittent exercise performance, since changes in both cardiovascular (Gonzalez-Alonso, Mora-Rodriguez, Below, & Coyle, 1997) and metabolic (Febbraio, 1999) responses to exercise are related to increases in core temperature and the level of dehydration. Evidence from those investigations that have adopted continuous exercise protocols indicate that any potential changes in intermittent performance could be mediated by differences in the time to attain a high core body temperature (rectal temperature $> 39^{\circ}\text{C}$) rather than the effects of temperature of metabolism (Gonzalez-Alonso *et al.*, 1999; Gregson *et al.*, 2002). These observations are supported by reports which indicate that fatigue during prolonged intermittent exercise in the heat (30°C , 24–66% relative humidity) is associated with a high internal body temperature (Morris, Nevill, & Williams, 2000). Alternatively, any effects of pre-warming on intermittent capacity may more closely reflect alterations in body heat content rather than the attainment of a high internal body temperature (Webb, 1995). Webb (1995) has proposed that the body regulates heat content rather than core body temperature, with changes in body heat content driving deep body temperature. This theory of heat regulation is supported by the suggestion that it is not an absolute core temperature that the body tolerates before exhaustion occurs, but a given increase in body heat content (Cheung & McLellan, 1998a; Sawka *et al.*, 1992; McLellan, Gannon, Zamecnik, Gil, & Brown, 1999; McLellan, Smith, Gannon, & Zamecnik, 2000).

The aims of the present study were to determine the influence of active and passive pre-exercise warming on the ability to perform a prolonged period of intermittent exercise in laboratory temperatures of 21°C , and to evaluate the metabolic and thermoregulatory responses to exercise in such conditions. It was hypothesized that both active and passive pre-warming strategies would reduce intermittent exercise capacity to the same extent. In addition, it was proposed that decrements in performance would be mediated through mechanisms associated with the subsequent elevation in

body temperature rather than through the effects of pre-warming on metabolism.

Methods

Participants

Six healthy men (mean $\pm$ s: age 25 ± 6 years, height 1.81 ± 0.3 m, body mass 77.0 ± 5.6 kg, body fat $11.2 \pm 2.9\%$, sum of seven skinfold thicknesses 80.2 ± 16.6 mm, estimated muscle mass 43.6 ± 3.3 kg, $\dot{V}\text{O}_{2\text{max}}$ 4.46 ± 0.5 l $\cdot$ min $^{-1}$) were recruited. All participants were well-trained soccer players and were not acclimatized to heat. The participants were familiarized with the experimental procedures and associated risks, and gave their written informed consent to participate, in accordance with the university's ethical procedures.

Experimental design

Before the experimental trials, all participants completed pre-test assessments for determination of maximal oxygen uptake ($\dot{V}\text{O}_{2\text{max}}$) (used for work-rate prescription) and body composition. Maximal oxygen uptake was assessed during continuous incremental treadmill running to volitional exhaustion (Power Jog GX 200, Sport Engineering Ltd, UK). Body composition was determined by the measurement of skinfold thicknesses using Harpenden skinfold callipers (HSK BI, Batty International, UK) and limb girths corrected for skinfold thicknesses. Subsequent estimation of percentage body fat (Jackson & Pollock, 1978; Siri, 1956) and muscle mass (Martin, Spenst, Drinkwater, & Clarys, 1990) were made using the appropriate equations.

Each participant performed three experimental trials. The trials consisted of intermittent treadmill running to volitional fatigue following either active or passive pre-warming or no prior manipulation of body temperature (control). The intermittent protocol consisted of repeated 30-s bouts of high-intensity treadmill running ($90\% \dot{V}\text{O}_{2\text{max}}$) separated by 30-s static recovery periods performed until volitional fatigue. Both the pre-test assessments and experimental trials were undertaken without the use of fan-assisted air movement in laboratory ambient temperatures (dry bulb) of $21.5 \pm 0.6^{\circ}\text{C}$ (relative humidity of $35.7 \pm 5.4\%$).

On the morning of the exercise trials, participants arrived at the laboratory at 08.00 h after an overnight fast, having refrained from exercise, alcohol, tobacco and caffeine during the previous 24 h. The participants also recorded their nutritional and fluid intake in the 24 h preceding the first exercise trial. This record was photocopied and returned to the participants to permit them to replicate their nutritional and

fluid intake in the 24 h preceding the remaining trials. Only water intake was permitted before entering the laboratory on the morning of the exercise trials. The trials were conducted one week apart in a randomized order. All trials were conducted at the same time of day to avoid the circadian variation in internal body temperature (Reilly & Brooks, 1986). The participants were not allowed to consume fluid at any time during any of the trials.

On arrival at the laboratory, anthropometric measurements were obtained, which included height (m) and nude body mass (kg) (GEC Avery, BSI Testing, UK). The participants then rested in a seated position for 30 min in shorts, socks and training shoes [in laboratory ambient temperatures (dry bulb) of $21.5 \pm 0.6^\circ\text{C}$ and $35.7 \pm 5.4\%$ relative humidity], before undertaking one of the three trials. Active heating required the participants to undertake treadmill running at an exercise intensity of $70\% \dot{V}\text{O}_{2\text{max}}$ until a rectal temperature of 38°C was attained. During the passive pre-warming procedure, the participants were partially immersed in a heated water tank (44°C) to the level of the gluteal fold until a rectal temperature of 38°C was attained. It was acknowledged that measurement of rectal temperature during passive heating would partially reflect temperature of the blood leaving the legs rather than be representative of deep core temperature (unpublished observations); however, assessment of oesophageal temperature could not be undertaken at the time. Once a rectal temperature of 38°C was attained, the participants were moved from the treadmill/water tank and placed in a seated position for a maximum of 10 min. The inclusion of this period enabled collection of the necessary data following pre-warming and before the onset of exercise, while ensuring rectal temperature remained at 38°C before starting the intermittent protocol. At the cessation of the 30-min standardization period in the control trials, the participants undertook a further 10-min rest period to maintain consistency in experimental methodology in all trials. The participants then completed the intermittent exercise protocol until volitional exhaustion, as defined by their inability to maintain the required treadmill speed. After exercise, each participant's nude body mass was measured and body mass loss, corrected for respiratory (Mitchell, Nadel, & Stolwijk, 1972) and metabolic water loss (Kenney, 1998), was taken to represent sweat loss.

Rectal, mean skin and muscle temperatures

The participants inserted a rectal probe 10 cm beyond the external anal sphincter. The probe was then attached to a squirrel data logger (1250, Grant Instruments, UK) for the recording of rectal temperature. Skin thermistors were attached to the

upper chest, lower forearm, upper thigh and medial side of the calf for the calculation of weighted mean skin temperature (T_{skin}), using the equation of Ramanathan (1964). As a result of the partial immersion associated with the passive pre-warming protocol, skin thermistors were removed from the lower limb sites (thigh and calf) before the start of this trial. Consequently, skin temperature measurements during the course of the passive pre-warming trial were only recorded from the chest and forearm. The thigh and calf thermistors were applied following the completion of passive pre-warming for the calculation of T_{skin} during exercise.

Baseline values for rectal temperature and skin temperature were recorded following the 30-min standardization period. Rectal temperature and skin temperature were monitored continuously and recorded at 5-min intervals during both pre-warming and the subsequent intermittent exercise bout. Muscle temperature was assessed at rest and at the start and end of the intermittent protocol using a needle thermistor inserted into the vastus lateralis (13050, ELAB, Denmark). The depth of insertion was standardized at 3 cm. Muscle temperature was recorded using an ELAB electronic measuring system (CTF 9004, ELAB, Denmark).

Mean body temperature, thermal gradient and heat storage

Rectal temperature (T_{rec}) and T_{skin} were used to calculate mean body temperature (T_{b}) (Lee & Haymes, 1995) and the thermal gradient ($T_{\text{rec}} - T_{\text{skin}}$) during each experimental condition. The rate of heat storage ($\dot{S}$; $\text{W} \cdot \text{m}^{-2}$) was calculated as described previously (Lee and Haymes, 1995):

$$\dot{S} = 0.97 \cdot m_{\text{b}} \cdot (\Delta T_{\text{b}} / \Delta t) / A_{\text{D}}$$

where 0.97 is the specific heat capacity of body tissue ($\text{W} \cdot \text{kg}^{-1}$), m_{b} is body mass, $\Delta T_{\text{b}} / \Delta t$ is the change (Δ) in body temperature (T_{b}) divided by time (Δt) (h), and A_{D} is body surface area (m^2) calculated according to DuBois and DuBois (1916). Heat storage capacity (S ; $\text{kJ} \cdot \text{kg}^{-1}$) was calculated from $\dot{S}$ and time as follows:

$$S = \dot{S} A_{\text{D}} \cdot b_{\text{m}}^{-1} \cdot (60 \cdot \text{time}) \cdot 1000^{-1}$$

(McLellan *et al.*, 1999). Total heat storage capacity for each condition was calculated by summing S from the pre-exercise, transition and exercise periods.

Blood sampling and analyses

A venous cannula (Venflon 2, BOC Ohmeda, Sweden) was inserted in an ante-cubital vein and

a baseline sample for determination of all blood-borne substrates was taken following the 30-min rest period. The cannula was kept patent by immediate infusion of saline (2 ml) following insertion, and at the end of each subsequent blood sample. All venous blood samples were collected using a vacutainer system (Vacutainer Systems, Becton Dickinson, Europe). An initial 3-ml sample of blood was dispensed into a vacutainer tube containing ethylenediamine tetra-acetic acid (EDTA), and immediately used for the analysis of blood glucose (B-Glucose Photometer, Hemocue, Sweden) and blood lactate (GM7, Analox Instruments, Hammersmith, UK). A second sample (3 ml) was then taken and immediately centrifuged (10 min at $3000 \text{ rev} \cdot \text{min}^{-1}$; CR 312, Jouan, France), with the resulting supernatant being used for determination of plasma-free fatty acid (FFA) concentrations using a commercially available kit (NEFA-C, Wako, Neuss, Germany). A further sample (6 ml) was dispensed into a plain serum tube and was centrifuged within 3 h of sampling. The supernatant was frozen at -80°C for subsequent determination of serum prolactin, using a sandwich immunometric assay procedure (Elecsys, Roche, Germany). Serum prolactin was taken as a neuroendocrine marker of brain serotonergic [5-HT] activity (Cowen, Anderson, & Graham-Smith, 1990). A final sample (7 ml) was dispensed into a vacutainer tube containing EDTA and was immediately centrifuged, with the supernatant being frozen at -80°C for determination of plasma catecholamine concentrations (norepinephrine and epinephrine) using automated sample trace enrichment and dialysis with electrochemical detection (Asted, Anachem, UK). All analyses were performed in duplicate. Blood samples for determination of glucose, lactate, FFA and prolactin concentrations were taken after the end of pre-warming and before the start of the intermittent exercise bout. Glucose, lactate and FFA concentrations were also noted during the intermittent protocol at 15-min intervals and at the point of exhaustion. Plasma catecholamines and serum prolactin concentrations were determined at exhaustion.

Heart rate, gas analyses and subjective perception of effort

Heart rate was monitored continuously and recorded at 5-min intervals throughout pre-warming and the intermittent exercise bout (PE 4000, Polar, Finland). Expired air was collected at the start and finish of the pre-warming protocols using Douglas bags. The bags were immediately analysed to determine oxygen and carbon dioxide concentrations (Servomex Systech ZR89314, Systech Instruments, UK). The expired

volume was determined through a Harvard dry gas meter, and calculation of $\dot{V}\text{O}_2$ and the respiratory exchange ratio was undertaken on a laboratory-based PC (Opus Technology, UK). Samples were also taken at 15-min intervals (during the 30-s recovery period) during the intermittent exercise bout and at the point of exhaustion. At equivalent times, ratings of perceived exertion, using a 10-point category scale, were also taken (Borg, 1982).

Statistical analyses

All data are presented as the mean $\pm$ standard deviation (s). A two-way (condition $\times$ time) analysis of variance (ANOVA) with repeated measures was undertaken for the pre-warming and exercise periods to determine any treatment differences. Statistical analyses for the exercise period were only applied to the time points at which data were available for all participants ($n = 6$). Following observation of a main effect of condition, a repeated ANOVA was used to determine at which time points a significant effect was observed. *Post-hoc* analysis by paired Student's *t*-test (with Bonferroni correction of α) was undertaken to examine which trials were significantly different from each other. A one-way ANOVA was used to determine any difference in total sweat loss and performance time between conditions.

Simple effect size, estimated from the ratio of the mean difference to the pooled standard deviation, was also calculated. This was used to provide a measure of the magnitude of difference between conditions, where failure to observe statistical significance may have reflected the small sample used (Vincent, 1995). A correction factor for effect sizes derived from small samples was also applied (Becker, 1998). The α level for the evaluation of statistical significance was set at $P < 0.05$.

Results

The duration of pre-warming required to attain a T_{rec} of 38°C was similar for both the active (14.9 ± 4.4 min) and passive (18.0 ± 6.1 min) pre-warming protocols ($P > 0.05$). Exercise time to exhaustion was significantly reduced following both the active (51.8 ± 7.2 min; 95% confidence interval [CI] for the difference -31.79 to -8.55 min) and passive (38.5 ± 11.1 min; 95% CI for the difference -48.81 to -18.07 min) pre-warming conditions compared with the control condition (72.0 ± 17.2 min) ($P < 0.05$). Time to exhaustion following passive pre-warming was also significantly reduced compared with the active pre-warming condition (95% CI for the difference -25.31 to -1.23) ($P < 0.05$).

Thermoregulatory responses

At the onset of exercise, T_{rec} (active, $38.0 \pm 0.1^\circ\text{C}$; passive, $38.1 \pm 0.2^\circ\text{C}$; control, $37.2 \pm 0.4^\circ\text{C}$), T_b (active, $37.4 \pm 0.2^\circ\text{C}$; passive, $37.7 \pm 0.2^\circ\text{C}$; control, $36.5 \pm 0.3^\circ\text{C}$) and muscle temperature (active, $37.1 \pm 1.0^\circ\text{C}$; passive, $37.0 \pm 1.0^\circ\text{C}$; control, $34.6 \pm 0.6^\circ\text{C}$) were significantly elevated after active and passive pre-warming compared with the control condition ($P < 0.05$). Significant elevations in T_{rec} , T_b (Figures 1A and B) and muscle temperature were noted during exercise under all conditions ($P < 0.05$). During the initial 10 min of exercise, T_{rec} and T_b were significantly greater after active and passive pre-warming compared with the control condition ($P < 0.05$) (Figures 1A and B). Large effect sizes also indicated that T_{rec} and T_b remained higher with both forms of pre-warming than in the control condition after 15 min and 20 min of exercise. No differences in T_{rec} , muscle temperature or T_b were reported between the two pre-warming conditions at the onset of exercise or at any time during exercise.

At the point of exhaustion, T_{rec} (active, $39.6 \pm 0.4^\circ\text{C}$; passive, $39.3 \pm 0.4^\circ\text{C}$; control, $39.9 \pm 0.3^\circ\text{C}$) and T_b (active, $38.9 \pm 0.4^\circ\text{C}$; passive,

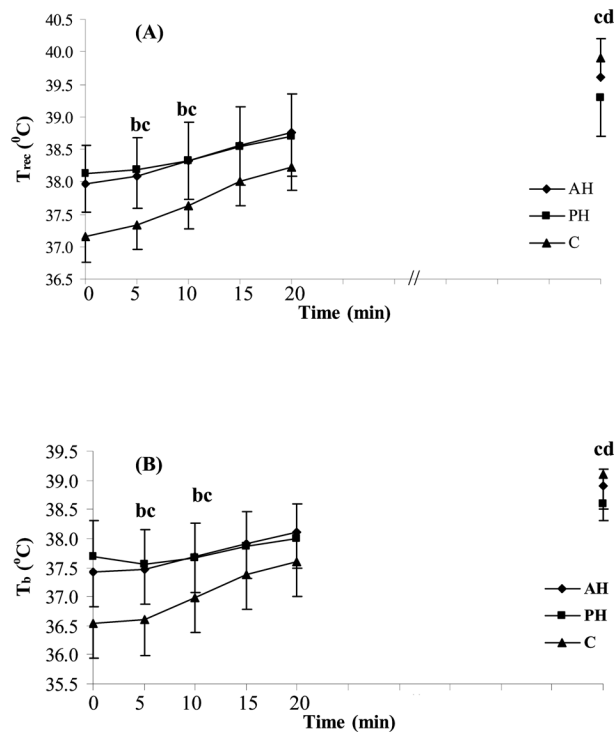


Figure 1. (A) Rectal temperature (T_{rec}) and (B) mean T_b (T_b) responses during prolonged high-intensity intermittent exercise in the active pre-warming ($\blacklozenge$), passive pre-warming ($\blacksquare$) and control ($\blacktriangle$) conditions ($n = 6$, mean $\pm$ s). Significant difference ($P < 0.05$): b, active pre-warming *vs* control; c, passive pre-warming *vs* control; d, active pre-warming *vs* passive pre-warming.

$38.6 \pm 0.4^\circ\text{C}$; control, $39.1 \pm 0.3^\circ\text{C}$) were significantly higher in the control and active pre-warming conditions compared with passive warming ($P < 0.05$). No significant differences in T_{rec} or T_b were observed between the control and active pre-warming conditions at the point of exhaustion. The differences observed in T_{rec} (effect size = 0.9) and T_b (effect size = 0.7) between the active pre-warming and control conditions were, however, associated with a large effect size. Muscle temperature responses were similar under all conditions at exhaustion (active, $39.3 \pm 0.5^\circ\text{C}$; passive, $39.1 \pm 0.5^\circ\text{C}$; control, $39.3 \pm 0.4^\circ\text{C}$). Mean skin temperature and the thermal ($T_{\text{rec}} - T_{\text{skin}}$) gradient were not significantly different between any of the conditions at the onset of exercise or during exercise. At the point of exhaustion, values remained similar under all conditions. Total sweat production, estimated from loss of body mass, was not significantly different between the active (1.51 ± 0.4 l), passive (1.27 ± 0.5 l) and control trials (1.45 ± 0.3 l).

Both the rate of heat storage and heat storage capacity (Figures 2A and B) were significantly different as a result of active and passive pre-warming and pre-exercise rest ($P < 0.05$). No differences in the rate of heat storage or heat storage capacity were observed during the transition period (Figures 2A and B). The rate of heat storage and heat storage capacity during exercise were significantly different between all conditions ($P < 0.05$) (Figures 2A and B). Total heat storage capacity (i.e. warm-up + transition + exercise) was not significantly different between conditions (active, 8.1 ± 0.7 kJ $\cdot$ kg $^{-1}$; passive, 7.2 ± 1.4 kJ $\cdot$ kg $^{-1}$; control, 8.7 ± 1.5 kJ $\cdot$ kg $^{-1}$) (Figure 2B). The observed effect sizes, however, suggested that total heat storage was reduced following passive pre-warming compared with both the active pre-warming (effect size = 0.9) and control (effect size = 1.0) trials.

Metabolic responses

At the onset of exercise, heart rate was significantly higher in both pre-warming conditions than in the control condition ($P < 0.05$) (Table I). Heart rate was significantly higher during the initial 20 min of exercise after both forms of pre-warming compared with the control trial ($P < 0.05$). Heart rate responses were, in contrast, similar at the onset of exercise and during exercise between the two pre-warming conditions. At exhaustion, heart rate was similar under all conditions (Table I).

At the onset of exercise, concentrations of blood glucose, blood lactate, plasma FFA and serum prolactin were similar under all conditions (Table I). Blood lactate concentration increased significantly during exercise under all conditions

($P < 0.05$) (Table I). No significant difference in blood lactate concentration was observed between any of the conditions during exercise (Figure 3A). Blood lactate concentration after 15 min of exercise

was, however, elevated in the passive pre-warming condition compared with the active pre-warming (effect size = 1.0) and control conditions (effect size = 0.8). No significant difference in blood lactate concentration was observed between conditions at exhaustion (Table I).

A significant elevation in blood glucose concentration was only observed during exercise ($P < 0.05$) (Table I). After 15 min of exercise, blood glucose concentration was significantly higher in the passive pre-warming condition than in the other two conditions ($P < 0.05$) (Figure 3B). No significant differences were observed between the active pre-warming and control conditions. At exhaustion, blood glucose concentration remained significantly higher in the passive pre-warming condition than in the control trial ($P < 0.05$). No differences were observed at exhaustion between the pre-warming conditions and the control condition (Table I).

Plasma FFA concentrations were not significantly altered under any condition (Table I). During exercise, concentrations of serum prolactin and plasma norepinephrine were significantly increased under all conditions ($P < 0.05$) (Table I). At exhaustion, serum prolactin and plasma norepinephrine concentrations were not significantly different between exercise conditions (Table I). Significant elevations in plasma epinephrine concentrations during exercise were observed only under pre-warming conditions ($P < 0.05$) (Table I). At exhaustion, epinephrine concentration was significantly higher under both pre-warming conditions than in the control condition ($P < 0.05$) (Table I). Concentrations of epinephrine were not significantly different between the two pre-warming conditions at exhaustion.

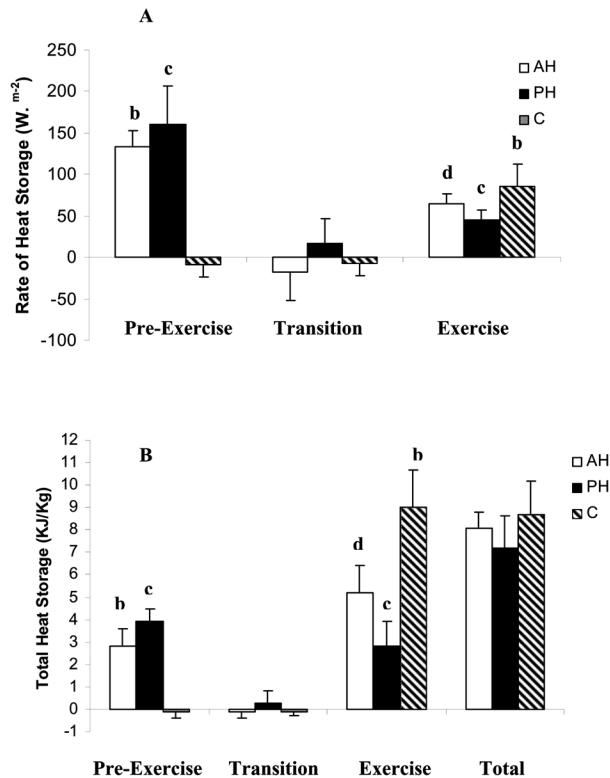


Figure 2. (A) Rate of heat storage and (B) heat storage capacity in the active pre-warming (□), passive pre-warming (■) and control (▨) conditions ($n = 6$, mean $\pm$ s). Significant difference ($P < 0.05$): b, active pre-warming vs control; c, passive pre-warming vs control; d, active pre-warming vs passive pre-warming.

Table I. Metabolic responses to exercise in the active pre-warming (AH), passive pre-warming (PH) and control (C) conditions ($n = 6$; mean $\pm$ s)

	Onset of exercise			Exhaustion		
	C	AH	PH	C	AH	PH
Heart rate (beats $\cdot$ min $^{-1}$)	70.8 $\pm$ 7.4	101.0 $\pm$ 13.3 ^b	84.0 $\pm$ 15.3 ^c	178.8 $\pm$ 15.9 ^a	177 $\pm$ 17.5 ^a	176.8 $\pm$ 18.1 ^a
$\dot{V}O_2$ (l $\cdot$ min $^{-1}$)	0.4 $\pm$ 0.1	0.7 $\pm$ 0.2	0.7 $\pm$ 0.2	1.8 $\pm$ 0.2 ^a	2.1 $\pm$ 0.5 ^a	1.9 $\pm$ 0.4 ^a
RER	0.9 $\pm$ 0.2	1.0 $\pm$ 0.2	0.8 $\pm$ 0.1	0.9 $\pm$ 0.1	1.0 $\pm$ 0.1	0.9 $\pm$ 0.1
Blood lactate (mmol $\cdot$ l $^{-1}$)	0.8 $\pm$ 0.3	1.6 $\pm$ 0.3	0.9 $\pm$ 0.3	2.5 $\pm$ 0.7 ^a	2.8 $\pm$ 0.7 ^a	3.3 $\pm$ 1.1 ^a
Blood glucose (mmol $\cdot$ l $^{-1}$)	4.3 $\pm$ 0.3	4.5 $\pm$ 0.6	4.4 $\pm$ 0.5	4.9 $\pm$ 1.0 ^c	5.4 $\pm$ 1.2	6.0 $\pm$ 1.4 ^a
FFA (mmol $\cdot$ l $^{-1}$)	0.6 $\pm$ 0.1	0.7 $\pm$ 0.3	0.7 $\pm$ 0.4	0.6 $\pm$ 0.2	0.4 $\pm$ 0.3	0.6 $\pm$ 0.3
Prolactin (ng $\cdot$ ml $^{-1}$)	13.9 $\pm$ 2.8	15.3 $\pm$ 3.2	19.0 $\pm$ 6.7	37.6 $\pm$ 23.3	38.2 $\pm$ 31.0	37.7 $\pm$ 20.7
Epinephrine (nmol $\cdot$ l $^{-1}$)	0.3 $\pm$ 0.1	0.4 $\pm$ 0.1	0.3 $\pm$ 0.1	0.3 $\pm$ 0.1 ^c	0.6 $\pm$ 0.2 ^{a, b}	0.7 $\pm$ 0.1 ^a
Norpinephrine (nmol $\cdot$ l $^{-1}$)	1.6 $\pm$ 0.9	2.2 $\pm$ 0.7	2.8 $\pm$ 1.7	7.1 $\pm$ 4.2 ^a	8.9 $\pm$ 3.6 ^a	10.2 $\pm$ 1.9 ^a

Significant difference ($P < 0.05$): ^a difference from the onset of exercise; ^b difference between active pre-warming and control; ^c difference between passive pre-warming and control. RER = respiratory exchange ratio

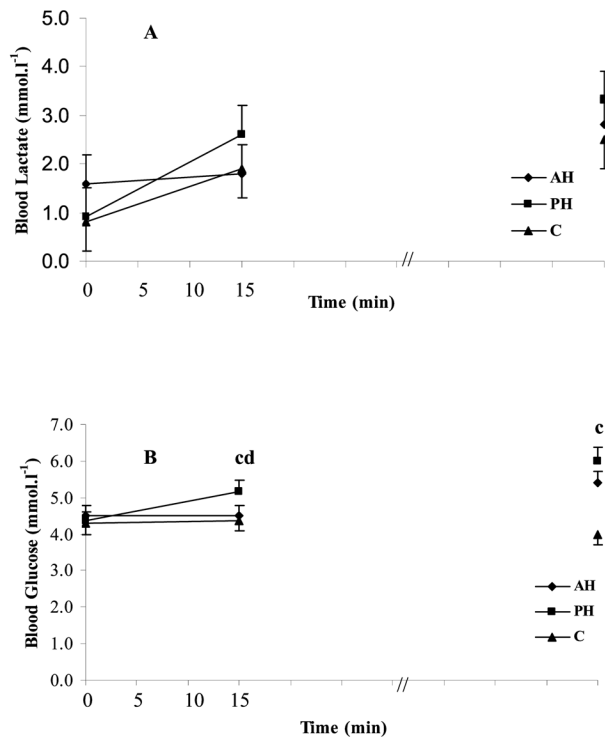


Figure 3. (A) Blood lactate and (B) blood glucose concentration during prolonged high-intensity intermittent exercise in the active pre-warming (◆), passive pre-warming (■) and control (▲) conditions ($n = 6$, mean $\pm$ s). Significant difference ($P < 0.05$): c, passive pre-warming *vs* control; d, active pre-warming *vs* passive pre-warming.

Subjective responses

Subjective perception of effort was not significantly different between any of the conditions at the onset of exercise. Ratings of perceived exertion increased significantly during exercise under all conditions ($P < 0.01$). During the initial 20 min of exercise, these ratings were significantly higher in both pre-warming conditions compared with the control trial ($P < 0.05$). A significantly higher rating was also observed in the passive compared with the active pre-warming trial at both 15 min (active, 4 ± 1 ; passive, 7 ± 3) and 20 min (active, 5 ± 1 ; passive, 7 ± 2) of exercise ($P < 0.05$). There was no significant difference in ratings of perceived exertion between conditions at exhaustion.

Discussion

The results of this study indicate that a pre-exercise elevation in core T_b , promoted by both active and passive pre-warming strategies, significantly reduces prolonged intermittent exercise capacity at ambient temperatures of 21°C compared with pre-exercise rest. Furthermore, the extent to which pre-warming reduces intermittent performance depends on the

method of pre-warming adopted. In the current study, passive pre-warming promoted a significantly greater decline in prolonged intermittent exercise performance than active pre-warming.

Muscle glycogen availability can affect the performance of prolonged high-intensity intermittent exercise (Balsom, Gaitanos, Soderlund, & Ekblom, 1999). Direct assessment of muscle metabolites was not undertaken in the present study; consequently, the role of substrate availability in the aetiology of fatigue is difficult to elucidate. The observed changes in blood-borne substrates during exercise, however, indicate that differences in performance between the three experimental conditions are unlikely to have stemmed from the effects of pre-warming on metabolism (Gonzalez-Alonso *et al.*, 1999; Gregson *et al.*, 2002). Blood glucose concentrations following both forms of pre-warming remained at or above corresponding values in the control condition (Figure 3B) and concentrations of FFA were similar under all conditions.

Blood glucose concentration in all conditions was also well maintained at exhaustion compared with resting values, indicating that carbohydrate availability did not appear to be the limiting factor at the point of fatigue (Febbraio *et al.*, 1996; Parkin *et al.*, 1999). These observations are supported by the results of studies that have looked at the effects of carbohydrate availability on time to exhaustion (Balsom *et al.*, 1999; Coggan & Coyle, 1987; Nassis, Williams, & Chisnal, 1998). Time to exhaustion at 70% $\dot{V}\text{O}_{2\text{max}}$ is associated with the depletion of muscle glycogen. This depletion is seen to occur at an exercise duration of between 100 and 170 min. Fatigue in both pre-warming conditions was associated with exercise durations of less than 60 min. This would seem to suggest that muscle glycogen concentration is not limiting under such conditions. In contrast with the two forms of pre-warming, exercise duration in the control condition approached 100 min in some participants. It is therefore possible that fatigue in the control condition may have been mediated by the availability of muscle glycogen (Febbraio *et al.*, 1996; Parkin *et al.*, 1999).

The significantly higher blood glucose concentration and increased blood lactate response after 15 min of exercise in the passive pre-warming condition, compared with the other two conditions (Figure 2A and B), has also previously been observed during prolonged continuous exercise to fatigue (Gregson *et al.*, 2002). These differences are surprising given the similarities in factors (e.g. rectal and muscle temperatures) in the two pre-warming conditions that are important in determining the metabolic response (Febbraio, 1999). It is possible that the increases in both plasma glucose and lactate

could simply reflect inherent changes in plasma volume, rather than an alteration in metabolism (Kargotich, Goodman, Keast, & Morton, 1998), as percent change in plasma volume during exercise could not be determined in the current investigation due to the postural differences associated with active and passive pre-warming (Harrison, 1985). Alternatively, passive pre-warming may have exacerbated body water loss (compared with exercise-induced active pre-warming and the control condition) due to an increased reliance upon evaporative heat loss mechanisms in response to the greater external heat load associated with this mode of heating (Nielsen, 1987). This could have led to differences in skin blood flow (Rowell, 1974) and/or elevations in sympathoadrenal activity (Hargreaves, Dillo, Angus, & Febbraio, 1996). Such alterations could impact upon the metabolic response to exercise.

In the present study, intermittent exercise performance after active and passive pre-warming was associated with a significantly higher T_{rec} and T_b (Figures 1A and B), heart rate and ratings of perceived exertion compared with the control condition. Such differences in thermoregulatory variables between experimental conditions suggests that alterations in thermal strain could have played a major role in the development of fatigue, as suggested by Galloway and Maughan, (1997), Gonzalez-Alonso *et al.* (1999), Morris *et al.* (2000) and Gregson *et al.* (2002).

Several studies have reported that exercise time to exhaustion under conditions of increased thermoregulatory strain frequently coincides with the attainment of a high internal T_b (e.g. $T_{\text{rec}} > 39^\circ\text{C}$) that is independent of the initial starting temperature (Gonzalez-Alonso *et al.*, 1999; Gregson *et al.*, 2002; Hales, Hubbard, & Gaffin, 1996). In contrast with such reports, the present results indicate that significant differences in T_{rec} were apparent at exhaustion between the two pre-warming conditions and between passive pre-warming and the control condition (active, $39.6 \pm 0.4^\circ\text{C}$; passive, $39.3 \pm 0.4^\circ\text{C}$; control, $39.9 \pm 0.3^\circ\text{C}$; $P < 0.05$), despite similar muscle temperatures (active, $39.3 \pm 0.5^\circ\text{C}$; passive, $39.1 \pm 0.5^\circ\text{C}$; control, $39.3 \pm 0.4^\circ\text{C}$). The large effect size (0.9) also suggests that final T_{rec} was different between the active pre-warming and control conditions. These findings lend support to previous studies that have shown that exhaustion from heat strain occurs over a broad range of core temperatures (Bridges, Weller, Rayson, & Jones, 2003; McLellan *et al.*, 2000; Sawka *et al.*, 1992), rather than a specific critical core temperature (Gonzalez-Alonso *et al.*, 1999; Gregson *et al.*, 2002).

Webb (1995) has suggested that the body regulates heat content, rather than core temperature

per se. McLellan *et al.* (2000), in line with this theory of heat regulation, have proposed that exhaustion in the heat may depend on the body's tolerance to a given increase in body heat content rather than the attainment of an absolute core temperature. These observations are consistent with the close association between changes in body heat content and exercise tolerance time reported under both compensable (Gonzalez-Alonso *et al.*, 1999; Gregson *et al.*, 2002) and uncompensable heat stress (Cheung & McLellan, 1998a; McLellan *et al.*, 2000).

The present results provide support for the theory that body heat regulation may be a key determinant of prolonged exhaustive exercise. Changes in exercise time to fatigue under both pre-warming conditions corresponded closely with a reduction in both the rate of heat storage and heat storage capacity (Figures 2A and B) compared with the control condition. Declines in exercise performance after passive versus active pre-warming were also consistent with a reduced rate of heat storage and heat storage capacity (Figures 2A and B). Interestingly, despite these differences in rate of heat storage and heat storage capacity, total heat storage capacity remained similar in the active pre-warming and control conditions when changes in heat storage capacity during the pre-exercise, transition and exercise periods were summed (Figure 2B). In contrast, total heat storage capacity was reduced after passive pre-warming compared with active pre-warming (effect size = 0.9) and the control condition (effect size = 1.0) ($P > 0.05$). This inconsistency in total heat storage capacity has also previously been noted under conditions of uncompensable heat stress, when pre-exercise elevations in core T_b are induced by mild hypohydration and elevated ambient temperatures (Cheung & McLellan, 1998a,b; McLellan *et al.*, 2000).

It is unclear based upon the current results why total heat storage during the passive pre-warming condition was reduced compared with the active pre-warming and control conditions. Due to its large specific heat capacity, body water content has been shown to be an important determinant of the body's heat storage capacity (Cheung & McLellan, 1998a; Kay & Marino, 2000). Greater body water loss during the passive pre-warming strategy (compared with exercise-induced active pre-warming and control conditions), as suggested earlier, may therefore explain the reduced total heat storage after passive pre-warming. Such changes in hydration status could, however, be questioned in light of the failure to observe any difference in total sweat loss between conditions or differences in T_{rec} and heart rate after active and passive pre-warming. Future work should focus upon the thermoregulatory responses to active

and passive pre-warming procedures in an attempt to resolve the current experimental findings.

Differences in performance and associated changes in the rate of heat storage, heat storage capacity and ratings of perceived exertion after active and passive pre-warming contrast with our previous research using continuous exercise (Gregson *et al.*, 2002). It is not clear based upon the available evidence why such discrepancies should exist between the two different modes of exercise. Intermittent exercise is frequently associated with an increased thermoregulatory and cardiovascular strain compared with continuous exercise (Cable & Bullock, 1996; Kranning & Gonzalez, 1991). It is possible, therefore, that potential differences in skin blood flow and/or sweat rate between active and passive pre-warming may have been magnified to some extent during the mode of exercise used in the present study, leading to changes in the rate of heat storage, heat storage capacity and associated performance levels.

The results of the present study show that active pre-warming induced by exercise and passive pre-warming by partial immersion in a heated water tank reduce the ability to perform prolonged intermittent exercise in ambient temperatures of 21°C. The results also indicate that profound physiological differences may be evident between the two pre-warming protocols, as the performance of prolonged intermittent exercise further declined after passive pre-warming. The changes in exercise capacity were closely associated with declines in the rate of heat storage and heat storage capacity and an increased perception of effort. Pre-exercise changes in thermal strain (T_{rec}), together with alterations in body fluid content (associated with different pre-warming procedures), would have contributed to the observed differences in body heat storage.

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