Metabolic Habituation Following Repeated Resting Cold-Water Immersion Is Not Apparent During Low-Intensity Cold-Water Exercise

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Abstract This project examined the effects of repeated, resting cold-water immersion on metabolic heat production and core temperature defence during subsequent rest and exercising immersions. Seven males undertook 15 days of cold-water adaptation, immersed to the fourth intercostal space, with cold-water stress tests (CWST) on days 1, 8 and 15 ($18.1 \pm 0.1^\circ\text{C}$: 60 min seated, followed by 30 min cycling ($1 \text{ W}\cdot\text{kg}^{-1}$)), and 90-min resting immersions ($18.4 \pm 0.4^\circ\text{C}$) on each of the intervening days. Adaptation elicited an habituated thermogenic response during the rest phase of CWST3 beyond 20 min, compared to CWST1 ($P < 0.05$), with oxygen consumption averaging $11.15 (\pm 0.25) \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ and $8.61 (\pm 0.90) \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ by 50 min, for CWST1 and CWST3, respectively. During exercise, this metabolic blunting was only apparent over the first 10-min period (60–70 min). No significant differences were observed during either the rest or exercise phases of the CWSTs for oesophageal temperature (T_{es}). While repeated cold-water exposures produced an habituated-thermogenic response, for an equivalent drop in T_{es} during rest, neither this response, nor an elevated thermogenesis, were apparent during subsequent cold-water exercise. *J Physiol Anthropol* 20 (5): 263–267, 2001 <http://www.jstage.jst.go.jp/en/>

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Introduction

Water offers negligible thermal insulation at the skin surface, and during cold immersion, skin temperature

(T_{sk}) rapidly falls towards water temperature (Nadel, 1977). Notwithstanding this lower T_{sk} , more rapid convectional heat losses occur, with T_{c} falling two-five times more quickly compared with that observed in air at the same temperature (Hong, 1984). When immersion is accompanied by exercise, the rate of T_{c} decline is often (Cannon and Keatinge, 1960; Keatinge, 1961), though not always accelerated (Craig and Dvorak, 1968; McArdle et al., 1984). This disparity stems not only from differences in water temperature and subject adiposity, but from variations in both exercise intensity and mode. Certainly, low-intensity exercise may accelerate T_{c} decline, relative to the resting state, as it disturbs the trapped boundary layer, reduces total insulation due to an elevated muscle blood flow, and increases the exposed skin surface area. The current investigation focussed upon such low-intensity exercise, with particular attention to the effects of repeated resting cold-water immersion upon metabolic heat production (thermogenic response) and T_{c} defence during subsequent cold-water exercise.

Thermal adaptations which enhance survival take morphological, physiological, biochemical, and behavioural forms (Werner, 1994). For instance, during shorter-term heat adaptation, thermoeffector function is enhanced, as typified by an enhanced sweating response (Nadel et al., 1974; Libert et al., 1983); a positive phenotypic adaptation (Mekjavić and Bligh, 1987). However, repeated cold exposures can elicit either positive (acclimation) or negative (habituation) adaptation (Young, 1996). Humans may display three distinct, but not necessarily exclusive, adaptation patterns, including: metabolic, insulative and hypothermic adaptation (Hammel, 1963). The metabolic adaptation is typified by an increased metabolic heat

production (Keatinge, 1961; Scholander et al., 1958), and is observed in many species (Feist and White, 1989), but is infrequently seen in humans. The insulative adaptation response is characterised by a lower skin temperature during cold stress, while T_c and metabolic rate remain near pre-exposure levels (Hong, 1973; Park et al., 1983). Hypothermic adaptation is associated with a greater fall in T_c and an habituated metabolic heat production (Brück et al., 1976; Young et al., 1986), and was classically described in Australian Aborigines (Hicks and Matters, 1933). This response is most common following intermittently-applied cold exposure, such as that employed by the current study. In this report, we address the interaction of such a metabolic habituation upon T_c defence during post-adaptation, cold-water exercise.

Only one group has previously addressed the possible carry-over effects from resting cold adaptation into a subsequent exercising cold exposure (Golden and Tipton, 1988). Using a short, rigorous regimen, Golden and Tipton (1988) found no significant adaptation differences during exercise immersion when compared prior to, and following, six resting cold-water immersions (15°C, 40 min). Since an habituated-metabolic response, accompanied by a greater fall in T_c , was apparent on the final adaptation day, it was postulated that exercise obscured the possible benefits of the resting adaptation, accelerating heat loss in excess of heat conservation (Golden and Tipton, 1988). However, it is possible that six immersions were too few to allow this habituation response to manifest itself during exercise. Since the authors did not report T_c across adaptation days, the thermal strain remained unquantified. In addition, it is not known whether the cold-water exercise response was protocol specific, or was generalised across adaptation regimens eliciting different thermal strain levels. To evaluate these issues in the current investigation, we increased the duration of each cold exposure, eliciting an average rectal temperature (T_{re}) reduction of 1.87°C, we doubled the number of consecutive cold exposures, and we studied both rest and exercise within a single experimental session, to better observe the possible carry over of a resting metabolic habituation into the exercising state.

Methods

Seven healthy, active males (24.7 SD 8.7 yr; mass 74.4 SD 6.4 kg; height 176.8 SD 7.8 cm; sum of eight skinfolds 107.4 SD 20.4 mm), participated in this study during the Autumn months, which was approved by the University of Wollongong's Human Experimentation Ethics Committee. Subjects were exposed to cold-water stress tests (CWST), immersed to the fourth intercostal space (seated), in well-stirred water, on days 1, 8 and 15 of a 15-

d cold-water adaptation protocol (18.1°C SD 0.1°C, air temperature 21.3°C SD 0.9°C). Each CWST consisted of two phases: seated, slightly reclined rest (60 min); followed by cycling in the same posture (30 min at 1 W·kg⁻¹; Collins Pedalmate, Warren E. Collins Inc., MA). The workload range corresponding with this relative intensity was 62–82 W, and is indicative of the routine work intensity of military divers. Cold-water adaptation began the day following CWST1, and continued on days 2–7 and 9–14. Acclimation involved 90-min, cold-water immersions (18.4°C SD 0.4°C, air temperature 21.3°C SD 1.0°C), with the subjects seated at rest at the same immersion depth. Subjects wore only swimming costumes for all exposures.

On CWST days, fasting, euhydrated subjects were fed (38 kJ·kg⁻¹; 7 ml·kg⁻¹ fluid) and rested 4.5 h, in an air temperature of approximately 22°C, before baseline data were collected. Body core (oesophagus and rectum) and skin temperatures were measured (1 Hz). The oesophageal temperature (T_{es}) thermistor (Edale Instruments, Cambridge, U.K.) was inserted ~40 cm from the nares (Mekjavić and Rempel, 1990). Rectal temperature (T_{re}) was monitored 12 cm beyond the anal sphincter (Yellow Springs Instruments Co. Inc., Yellow Springs, OH), and was also measured during acclimation days. Skin temperatures were measured using thermistors at eight sites: forehead, scapula, upper chest, upper arm, forearm, hand, thigh and calf (EU type, Yellow Springs Instruments Co. Inc., Yellow Springs, OH). Thermistors were calibrated against a certified reference thermometer (Dobbie Instruments, Sydney, Australia), and temperatures were recorded using a portable data logger (Grant Instruments Ltd., U.K.). Mean skin temperature ($\bar{T}_{sk}$) was determined as an area-weighted average (International Standards Organisation, 1992), while mean body temperature ($\bar{T}_b$) was derived: $\bar{T}_b = (0.67 \cdot \bar{T}_{es}) + (0.33 \cdot \bar{T}_{sk})$. Oxygen consumption ($\dot{V}_{O_2}$) was measured every 10 min during immersion, over a 3-min period (Quinton Instrument Company, Q-Plex I, Seattle, WA). Cardiac frequency (f_c) was recorded at 0.2 Hz (Polar Electro SportTester, model PE3000, Finland).

Multifactorial, repeated-measures analysis of variance was used to determine differences between acclimation trials (CWST1, 2 and 3) and across exposure time. Tukey's WSD test was used to isolate significant differences, with *alpha* set at 0.05. Unless otherwise stated, data are reported as means with standard errors of the means.

Results

All subjects completed every exposure, without early termination, experiencing a mean T_{re} change, across adaptation days, of -1.87°C (± 0.20). On the CWST days, the respective pre-exposure (baseline) for T_{es} and T_{re}

remained unchanged, with progressive declines observed during the course of each CWST ($P < 0.05$). Due to the separate rest and exercise phases, two distinct T_c responses were evident within each CWST. The 60-min resting phase elicited an initial T_{es} rise (Fig. 1), which is typically observed upon suddenly moving to a cooler temperature. Cold-induced vasoconstriction is maximal under such conditions, instantaneously reducing heat loss by elevating tissue insulation, decreasing conductive heat transfer, and minimising exposure of blood to the cold. These events precede shivering, positively disturbing heat balance (Hardy, 1954). This transient T_{es} rise was followed by an approximately linear decline, producing a mean drop of -0.52°C (± 0.07) across the three CWSTs (Figure 1). During the CWSTs, T_{re} showed a significantly larger decrease, averaging -1.16°C (± 0.14). Despite absolute differences between T_c measurement sites, adaptation did not produce significant differences in either the T_{es} or T_{re} responses between CWST days ($P > 0.05$, Fig. 1).

During the 30-min exercise period, further biphasic decreases in T_{es} occurred, with changes of -0.55°C (± 0.15), -0.62°C (± 0.15), and -0.65°C (± 0.16), following CWST1, 2, and 3, respectively (Figure 1; $P > 0.05$). Average decreases in T_{re} were: -0.58°C (± 0.14), -0.84°C (± 0.11), and -0.68°C (± 0.12) following CWST1, 2, and 3, respectively. Final T_{es} were: 35.78°C (± 0.18 , CWST1), 35.80°C (± 0.23 , CWST2) and 35.74°C (± 0.19 , CWST3; $P > 0.05$; Figure 1), demonstrating that the 15-d resting acclimation regimen did not provide a thermal benefit, in terms of T_c defence, during subsequent cold-water exercise.

Following the rest phase, $\bar{T}_{sk}$ remained unchanged during exercise, and was not significantly different between CWSTs ($P > 0.05$). Furthermore, no differences were observed in $\bar{T}_{br}$, or the temperature gradient between T_{es} and the immersed $\bar{T}_{sk}$ ($P > 0.05$). Evidently, tissue heat conductance, beyond the first 5–10 min, remained relatively constant during the exercise phase of each CWST, despite repeated cold-water exposure. Beyond the initial, cold-induced tachycardia, cardiac frequency decreased during immersion, and was significantly lower during the resting phase of CWST3 compared to CWST1, at 30 and 40 min of immersion ($P < 0.05$). Exercise produced a return of f_c to pre-immersion resting levels, mirroring the increased metabolic demand of the active muscles. However, no acclimation-induced response differences were observed for f_c during this phase of the CWSTs ($P > 0.05$).

Although $\dot{V}_{O_2}$ increased within the resting phase of each CWST, shivering thermogenesis was blunted (habituated) during the resting phase of CWST3, compared to CWST1 beyond 20 min ($P < 0.05$; Figure 2). Exercise induced further $\dot{V}_{O_2}$ increases in all CWSTs. However, a significant metabolic blunting during exercise was only apparent at

Fig. 1 Oesophageal temperature (T_{es}) during 90-min, cold-water stress tests (CWST: 18.1°C) on days one, eight and 15 of cold-water acclimation (90-min rest; 18.5°C). Insert shows T_{es} averaged over the rest phase of each CWST.

Fig. 2 Oxygen consumption ($\dot{V}_{O_2}$) during 90-min cold-water stress tests (CWST: 18.1°C) on days one, eight and 15 of cold-water acclimation (90-min rest; 18.5°C). Inserts show cumulative $\dot{V}_{O_2}$ change ($\text{ml.kg}^{-1}.\text{min}^{-1}$) from pre-immersion and pre-exercise baselines. Significant differences: * = between CWST1 and 3.

the first sampling point, reflecting the residual affects of metabolic habituation during the previous 60 min of resting immersion. Beyond this time, $\dot{V}_{O_2}$ was equivalent between trials ($P > 0.05$; Fig. 2).

Discussion

This is only the second investigation to evaluate the effects of cold-water habituation upon thermal homeostasis during subsequent cold-water exercise (Golden and Tipton, 1988). The current investigation utilised a longer adaptation regimen, and increased the

thermal impulse, demonstrating that during the 60-min rest phase of CWST3, there was a pronounced blunting of cold-induced thermogenesis, which occurred concomitantly with an equivalent T_{c} defence. While such metabolic habituation is not a novel observation (Golden and Tipton, 1988; Janský et al., 1996), it is usually associated with greater falls in T_{c} . In addition, it was found that this habituation was apparently overridden during post-adaptation, low intensity cold-water exercise.

In the resting state, under the current conditions, it is possible that T_{c} maintenance was facilitated primarily by increasing tissue insulation, which occurred concomitantly with an habituated metabolic response. Muscle accounts for about 80% of total body insulation for resting subjects in cool water (Veicsteinas et al., 1982). Shivering not only disturbs the boundary layer, but facilitates heat loss by increasing muscle perfusion. Thus, if the water temperature is low enough, then a slight elevation in shivering may actually increase heat loss more than it does heat production (Tipton; personal communication). It is possible that, under these circumstances, a dampened thermogenic response may itself serve a protective function, by minimising the T_{c} reduction that would normally accompany cold stress. During exercise, convective cooling is dramatically elevated as the cycle cadence rhythmically displaces water close to the skin, and further increases muscle blood flow, thus eliciting the biphasic T_{es} reduction (Fig. 1). In this circumstance, a blunted metabolic response would reduce net heat production, compromising thermal balance. In the current study, shivering and exercise-induced thermogenesis were insufficient to offset heat loss, and T_{c} declined rapidly, and at a rate equal to that observed prior to adaptation. Thus, under circumstances where enhanced thermogenesis could have proven beneficial, it was still not evident. Furthermore, while it is possible that the resting metabolic adaptation was obscured during exercise (Golden and Tipton, 1988), there was no evidence of a blunted thermogenic response while subjects exercised in cold water, with both T_{es} and $\dot{V}_{\text{O}_2}$ being equivalent to the values observed during CWST1. Therefore, no indication was apparent for any form of metabolic adaptation during the exercise phase.

Using the above data in conjunction with the heat balance equation, and assuming a constant tissue insulation, it was predicted that the T_{c} decline could have been countered, had the work rate been increased to approximately 1.3 watts·kg⁻¹. However, in the absence of either a work rate elevation, or a greater adaptation-induced thermogenesis, a progressive T_{c} decline for the current subjects would have occurred under all thermal states where water temperature was less than about 21–22°C.

It is concluded that, while a longer cold-water

adaptation regimen acts to attenuate the metabolic response for an equivalent drop in T_{c} at rest in the cold, it has minimal impact upon shivering thermogenesis during cold-water exercise of low intensity. Thus, across a range of levels and durations of imposed thermal strain during resting cold-water adaptation, it appears that the metabolic responses observed during cold-water exercise appear to be similar before and after adaptation.

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