International Journal of Sports Physiology and Performance, 2008, 3, 331-346

Physiological Responses to Cold Water Immersion Following Cycling in the Heat

Shona L. Halson, Marc J. Quod, David T. Martin, Andrew S. Gardner, Tammie R. Ebert, and Paul B. Laursen

Cold water immersion (CWI) has become a popular means of enhancing recovery from various forms of exercise. However, there is minimal scientific information on the physiological effects of CWI following cycling in the heat. ***Purpose:*** To examine the safety and acute thermoregulatory, cardiovascular, metabolic, endocrine, and inflammatory responses to CWI following cycling in the heat. ***Methods:*** Eleven male endurance trained cyclists completed two simulated ~40-min time trials at 34.3 ± 1.1°C. All subjects completed both a CWI trial (11.5°C for 60 s repeated three times) and a control condition (CONT; passive recovery in 24.2 ± 1.8°C) in a randomized cross-over design. Capillary blood samples were assayed for lactate, glucose, pH, and blood gases. Venous blood samples were assayed for catecholamines, cortisol, testosterone, creatine kinase, C-reactive protein, IL-6, and IGF-1 on 7 of the 11 subjects. Heart rate (HR), rectal (T_{re}), and skin temperatures (T_{sk}) were measured throughout recovery. ***Results:*** CWI elicited a significantly lower HR (CWI: Δ116 ± 9 bpm vs. CONT: Δ106 ± 4 bpm; $P = .02$), T_{re} (CWI: Δ1.99 ± 0.50°C vs. CONT: Δ1.49 ± 0.50°C; $P = .01$) and T_{sk}. However, all other measures were not significantly different between conditions. All participants subjectively reported enhanced sensations of recovery following CWI. ***Conclusion:*** CWI did not result in hypothermia and can be considered safe following high intensity cycling in the heat, using the above protocol. CWI significantly reduced heart rate and core temperature; however, all other metabolic and endocrine markers were not affected by CWI.

Keywords: Recovery, cryotherapy, thermoregulation, core temperature

Cold water immersion (CWI) and other forms of hydrotherapy are becoming increasingly popular tools to enhance recovery from training and competition in elite athletes. While anecdotal support for the benefits of CWI is positive, little scientific evidence exists to substantiate its popularity. Indeed the physiological, thermoregulatory, and endocrine responses to cold water immersion protocols commonly used by athletes are unknown. Further, many elite athletes use CWI

Halson, Quod, Martin, Gardner, and Ebert are with the Department of Physiology, Australian Institute of Sport, Belconnen, ACT, Australia; and Laursen is with the School of Exercise, Biomedical, and Health Sciences, Edith Cowan University, Western Australia, and the School of Human Movement and Sport Sciences, University of Ballarat, Ballarat, Victoria, Australia.

after exercising in hot environments and the physiological and thermoregulatory consequences of this are unclear.

Exercising in hot environments can result in core temperatures above 40°C.[1] CWI is an aggressive form of cooling that can be used to reduce body temperature when an individual is hyperthermic or suffering from heat stress.[2] During whole-body CWI, the large surface area exposed to water results in a conductive heat transfer that is approximately 25 times greater than that of air.[3] Previous research in the area of cooling efficiency during hyperthermia has demonstrated that cooling athletes in various water temperatures (ranging from 2°C to 20°C) results in safe and efficient cooling of hyperthermic individuals.[4] However, the subjects used in many cooling studies are typically nonelite athletes, whose body composition may be vastly different from an athlete. Many athletes who use postexercise cooling strategies are very lean and have little insulation to serve as protection against the low water temperatures. For this reason, it is important to determine if the thermoregulatory effects are greater in lean individuals and whether CWI is safe in such a population.

CWI has been suggested to have a number of acute physiological, metabolic, hormonal, and thermoregulatory responses. The most investigated of these responses is changes in the cardiovascular system, with documented reductions in heart rate, cardiac output, and increases in arterial blood pressure and peripheral resistance.[5] Immersion in cold water also causes a drive to maintain core body temperature, resulting in increased oxygen consumption and local vasoconstriction.[5] Vasoconstriction of peripheral vessels may cause a reduction in fluid entering the interstitial space and may result in reduced inflammation as a consequence of muscle damage.[5] Finally, cold water immersion may be effective in reducing nerve transmission, and analgesia and a reduction in muscle spasm may occur.[6] Therefore, the stretch reflex response and spasticity of the muscle are decreased resulting in a reduction of the pain-spasm cycle.[6]

There is a scarcity of information on the acute metabolic and hormonal effects of CWI. An understanding of how the human body responds to CWI after exercise can aid in the determination of the validity of this method for enhancing recovery and its safety in individuals with elevated core temperatures.

The aims of the present investigation were twofold. Firstly, to determine the safety of CWI following exercise in the heat in lean individuals and secondly to examine the acute physiological, perceptual, metabolic, endocrine, and inflammatory responses to CWI following ~40 minutes of high-intensity cycling in the heat.

Methods

Subjects

Eleven endurance-trained cyclists (age: 23.8 ± 1.6 y; V_{O2peak}: 71.3 ± 1.2 $mL \cdot kg^{-1} \cdot min^{-1}$; sum of 7 skinfolds: 49.9 ± 2.8 mm; mean ± SD) previously unfamiliar with cold water immersion volunteered to complete the study, which was approved by both the Australian Institute of Sport and the University of Ballarat

Human Research Ethics Committees. All subjects provided written informed consent and were free from any known illness, cardiovascular concerns, and Raynaud's disease at the start of the study.

Approach to the Problem and Methodological Design

To examine the physiological and endocrine responses to cold water immersion following maximal exercise in the heat, subjects performed ~40 minutes of cycling in a hot environment, followed by either intermittent exposure to an ~11.5°C bath or passive recovery in a random and counterbalanced order (Figure 1).

Exercise Protocol

Subjects were instructed to abstain from intense exercise in the 24-h period before each experimental trial and were asked to record any training that was completed during the study. Subjects were provided with instructions for consuming appropriate amounts of fluid and carbohydrate during the study and were requested to complete a food diary on the day before each experimental trial (L. Burke, dietician, Australian Institute of Sport).

Each subject reported to the laboratory within the 14 days before commencement of the study to complete a progressive exercise test for the determination of maximal aerobic power (MAP). This test was conducted on a Velotron cycle ergometer (Racermate Inc., Seattle, Washington), which was fitted with the individual subject's cycling pedals and shoes. After a 10-minute warm-up at 100 W, the test protocol started at 100 W and increased by 50 W every 5 minutes until volitional exhaustion. If the subject finished part way through a 5-minute stage, MAP was prorated; eg, if a subject finished 2 minutes 30 s into the 300-W stage, their MAP would be $[(2.5 \text{ min} \div 5 \text{ min}) \times 50] + 300 = 325$ W.

Also at this time, descriptive measures of body mass, height and body composition were performed. Skinfold measurements were assessed across seven sites (triceps, subscapular, biceps, supraspinale, abdominal, mid-thigh and calf), using calibrated Harpenden skin fold calipers (British Indicators, England) by a Level 2 ISAK accredited anthropometrist.

Subjects completed ~40 minutes of cycling on a calibrated Lode Excalibur Sport cycle ergometer (Groningen, Netherlands) in an environmental chamber maintained at 34.3 ± 1.1°C and 41.2 ± 3.0% relative humidity (RH). The first 20 minutes of the exercise protocol was performed at a fixed workload that corresponded to 74.4 ± 4.1% of predetermined maximal aerobic power. The final ~20 minutes consisted of a time trial whereby subjects were required to complete the same amount of work (343.2 ± 32.4 kJ) as was completed in the fixed component of the time trial as quickly as possible. No warm-down was completed. This protocol was chosen to represent the typical duration of an Olympic cycling time trial event and aimed to induce a significant increase in core temperature. The fixed portion of the protocol was chosen to evaluate the physiological responses to a prior cooling intervention which will be presented elsewhere (M. Quod, unpublished data).

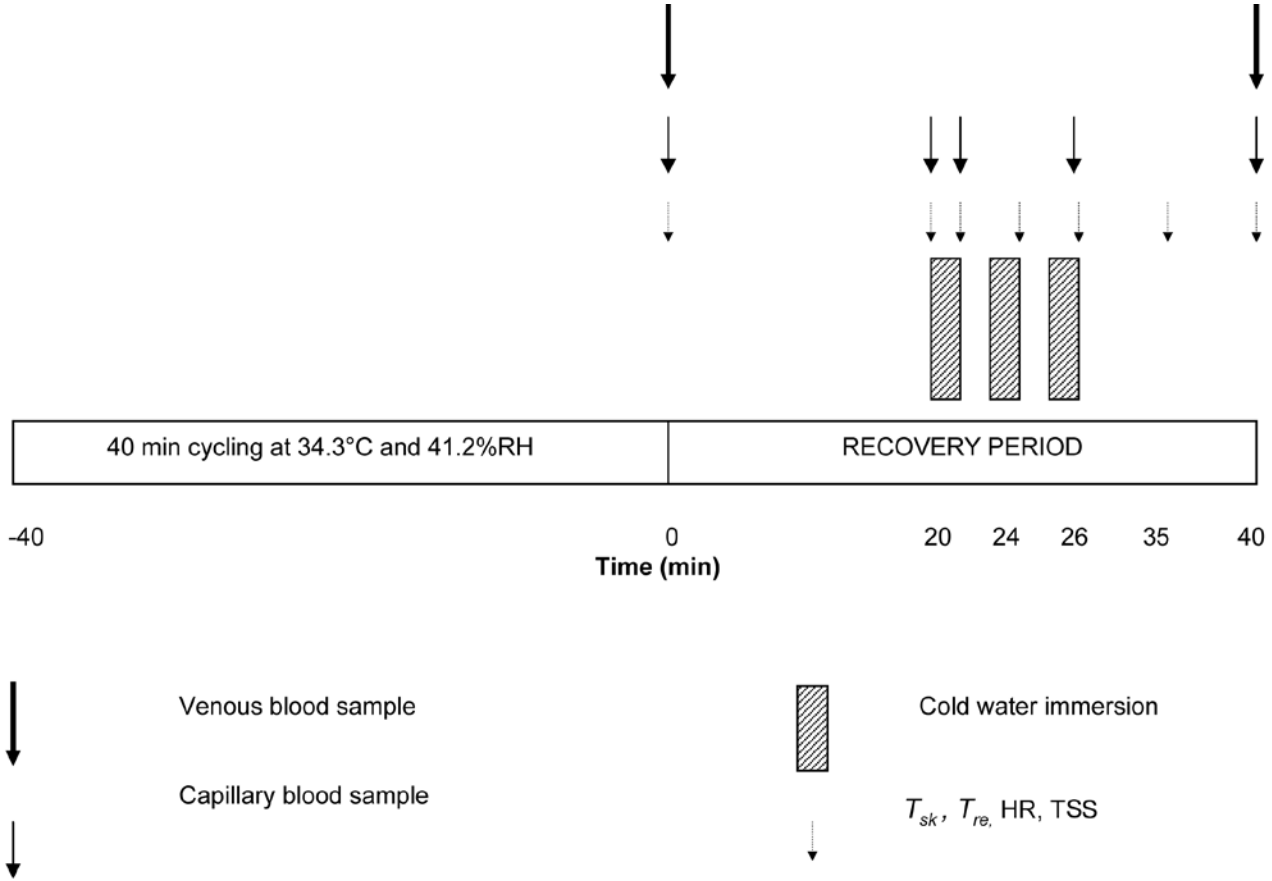

Figure 1 — Schematic representing study design (T_{sk} = mean skin temperature , T_{re} = rectal temperature, HR = Heart rate, TSS = Thermal Sensation Scale).

Cold Water Immersion (CWI) Protocol

The recovery period lasted for a total of 40 minutes. Twenty minutes following the completion of the exercise trial, subjects began the immersion protocol and were monitored for the subsequent 20 minutes. This time frame was chosen as it represents a practical duration for recovery following exercise. Subjects were immersed in 11.5 ± 0.3°C water to the level of the mesosternale in a 1.6 m long × 0.6 m wide × 0.8 m high inflatable pool (Portacovery, Australia) for 60 s. Temperature was maintained through the addition of ice and water. The water in the bath was not circulated. Each of the three immersions lasted 60 s and were separated by 120 s of seated rest. This protocol was chosen to ensure the subjects could tolerate whole-body immersion at 11.5°C. Further, in a practical setting, intermittent exposure allows a number of athletes to complete the protocol in a relatively short period. Finally, pilot work highlighted the need to submerge the armpits to ensure rapid cooling occurred. The duration of exposure may have been shorter than that of previous research[7]; however the current study employed whole-body immersion and not just that of single limbs. During the control (CONT) trial, subjects sat passively in the same room as the CWI trial which was maintained at 24.2 ± 1.8°C and 45.6 ± 6.5%RH, with no airflow. During both CWI and CONT subjects wore only cycling shorts. During the 120 s of seated rest in the CWI trial, subjects were allowed to sit with a towel wrapped around their upper body.

Hormonal, Metabolic, and Temperature Measurements

Heart rate, rectal and skin temperatures, lactate, glucose, pH, chloride, potassium, bicarbonate, sodium, PO_2, and PCO_2 were measured before initial exposure and after each CWI exposure during the recovery period. Testosterone, cortisol, growth hormone (GH), prolactin, adrenaline, noradrenaline, creatine kinase (CK), C-reactive protein (CRP), insulin-like growth factor-1 (IGF-1), and interleukin-6 (IL-6) were measured immediately after exercise and at the end of the recovery period (40 minutes post exercise) in 7 out of 11 subjects. Plasma measures were not made in 4 of the 11 subjects as these subjects were competing in an international event the following day and were unwilling to be cannulated.

Rectal temperature was measured using a disposable rectal probe (Monatherm, Mallinckrodt Medical, St. Louis, MO, USA), which was inserted to at least 12 cm beyond the anal sphincter. Skin temperature probes (YSI 409, Yellow Springs OH, USA) were fixed to the forearm, calf, thigh, chest, and forearm using adhesive tape (Transpore, 3M, St Paul MN, USA) according to previously published methods.[8] Rectal and skin temperatures were recorded from an 8-channel digital thermometer (Zentemp 5000, Zencor Pty Ltd, Australia). Rectal and skin temperatures were then used to calculate the mean skin temperature (T_{sk}) according to the equation established by Ramanathan[9] and the mean body temperature (T_b) as described by Schmidt and Bruck.[10]

$$T_{sk} = 0.3 \times (T_{Chest} + T_{Forearm}) + 0.2 \times (T_{Thigh} + T_{Calf})$$

Equation 1: Ramanathan equation[9] for calculation of the mean skin temperature (mean T_{sk}).

$$T_b = 0.87\ T_{re} + 0.13\ T_{sk}$$

Equation 2: Equation for the calculation of the mean body temperature (T_b)[10]

Cooling rates were calculated by determining the change in T_{re} immediately before CWI and immediately after the final immersion and dividing this by the time taken to complete the CWI protocol (7 minutes).

Blood Handling, Storage, and Analyses

Twenty minutes before each of the experimental trials, a 20 gauge Teflon cannula (Surflo, Terumo Corporation, Japan) was inserted into an antecubital vein and attached to a sterile three-way stopcock to allow for blood sampling in 7 of the 11 subjects. Two blood samples were drawn, one immediately upon cessation of exercise and the other at the end of the 40-minute recovery period. A 10-mL venous sample was drawn and divided into two 2-mL tubes prepared with K_3EDTA and one 6-mL serum separator tube prepared with a clot activator. The K_3EDTA tubes were immediately centrifuged (4000 rpm, 5 minutes, 4°C), and the serum separator tubes were allowed to clot before being centrifuged. The supernatant from each tube was then transferred to 1.5-mL Eppendorf tubes and frozen at −20°C before shipment and permanent storage at −80°C for later analysis. Fingertip capillary blood samples were collected in 100-μL heparinized capillary tubes (Clinitubes, Radiometer Medical, Copenhagen, Denmark) and were immediately analyzed for lactate, glucose, sodium, potassium, chloride, pH, PO_2, and PCO_2 using a blood-gas analyzer (ABL 700series, Radiometer Medical, Copenhagen, Denmark).

Plasma cortisol and testosterone were assayed by chemiluminescence–competitive immunoassay (Immulite, DPC, USA), which has an interassay coefficient of variation (CV) of 7.6% and 7.7%, respectively. GH, prolactin, IGF-1, IL-6, and CRP were measured by chemiluminescence-immunometric assay (Immulite, DPC, USA), which has an interassay CV of 6.0%, 9.5%, 3.5%, 4.6%, and 10%, respectively. Plasma creatine kinase (CK) activity was determined at 30°C using an enzymatic assay with an interassay coefficient of variation of 1.4% (Roche Diagnostics, Mannheim). Plasma adrenaline and noradrenaline were determined by an enzyme-linked immunosorbent assay (ELISA; IBL, Hamburg, Germany), which has an interassay CV of 13%.

Perceptual Responses

At regular intervals throughout the trials, subjects were asked to rate their perception of thermal comfort according to a 0-to-8 Thermal Sensation Scale as described by Young et al.[11] Following CWI, subjects were given a questionnaire that asked their perception of physical and mental recovery, leg soreness, and general fatigue (on a scale of 1 to 10). Subjects were given the same questionnaire at the same time point in the CONT condition.

Statistical Analysis

A 2 × 2 repeated measures analysis of variance was performed to determine differences between the treatment and control groups for metabolic and temperature

variables. Pairwise comparisons were then performed to determine where differences existed. Pairwise comparisons were performed on hormonal variables, markers of inflammation and perceptual responses. Significance was set at $P < .05$. Data are presented as mean ± standard deviations.

Results

All subjects completed the exercise protocol with no differences observed between CWI and CONT in time trial performance (CWI: 356 ± 32; CONT: 347 ± 32 kJ), lactate (CWI: 15.2 ± 3.6; CONT: 15.0 ± 3.8 mmol.L^{-1}), HR (CWI: 197 ± 9; CONT: 196± 9 bpm), T_{re} (CWI: 39.74 ± 0.51°C; CONT: 39.54 ± 0.42°C), or T_{sk} (CWI: 33.13 ± 1.92°C; CONT: 33.65 ± 1.42°C), indicating a similar physiological state before the interventions (CWI or CONT). As expected, there were significant reductions in T_{re}, T_{sk}, HR, and lactate as a consequence of time following exercise at all time points in both conditions ($P < .05$). However, CWI resulted in a significantly lower T_{re} than CONT at 40 minutes after exercise, but at no other time point (Figure 2a; $P < .05$). There was also a significant difference in HR following CWI when compared with CONT, from 21 minutes post exercise (after initial CWI) through to 40 minutes post exercise (Figure 2b; $P < .05$). There were no significant differences in lactate concentrations at any time point between treatments (Figure 2c).

With the exception of the unsubmerged forehead, all skin temperatures significantly declined over time irrespective of treatment; however, the decline was significantly greater with CWI ($P < .05$). The significant decrease in skin temperature with CWI occurred primarily during the initial immersion and persisted throughout the 40-minute postexercise period. For comparison, T_{sk} and forehead temperatures are shown in Figure 3a and b.

T_{sk} significantly decreased over time following both treatments from 33.65 ± 1.42°C to 32.45 ± 1.57°C (–3.7%; $P < .05$) in CONT and 33.13 ± 4.92°C to 27.57 ± 1.94°C (–20.2%; $P < .05$) in the CWI condition. There was a significant difference between trials following the first CWI exposure and this difference continued throughout the 40-minute postexercise period ($P < .05$). T_b also decreased significantly over time from 38.78 ± 0.48°C to 37.31 ± 0.39°C (–3.8%; $P < .05$) in CONT and 38.89 ± 0.79°C to 36.43 ± 0.49°C (–6.3%; $P < .05$) in the CWI condition. Cooling rates were significantly different between CWI and CONT, with a greater cooling rate in the CWI condition (CWI: 0.09 ± 0.03°C/min; CONT: 0.001 ± 0.001°C/min; $P < .05$).

No significant differences were found between trials in pH, chloride, glucose, bicarbonate, potassium, sodium, or PCO_2 at any time point (Table 1). However, PO_2 was significantly lower following CWI exposure at 40 minutes post exercise ($P < .05$; Table 1).

Following exercise, plasma CK, IGF-1, testosterone, and GH all significantly declined over time in both treatments ($P < .05$); however, there was no difference in these variables between treatments. CWI also had no significant effect on plasma CRP, IL-6, or cortisol concentrations. Plasma prolactin concentrations appeared to decrease during the recovery period; however, this was not statistically significant and there was no effect of treatment on prolactin concentration

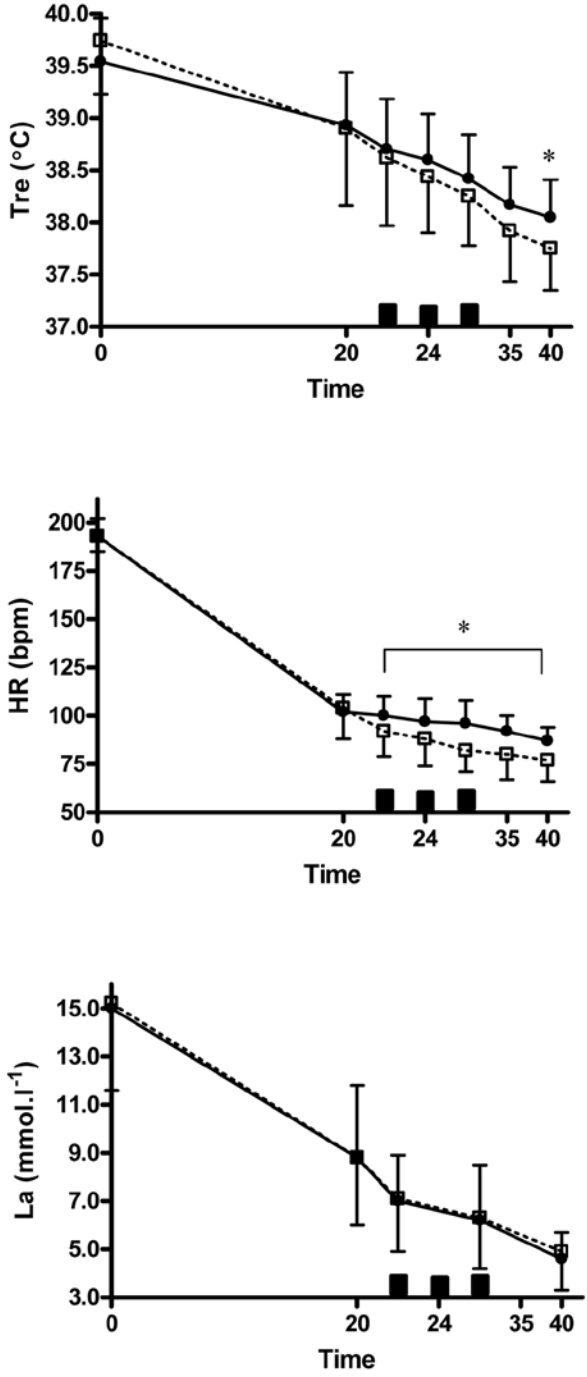

Figure 2 — Core temperature (Tre) (a), lactate (b), and heart rate (c) responses to cold water immersion (CWI) and passive recovery (CONT). * indicates significant difference between CONT and CWI.

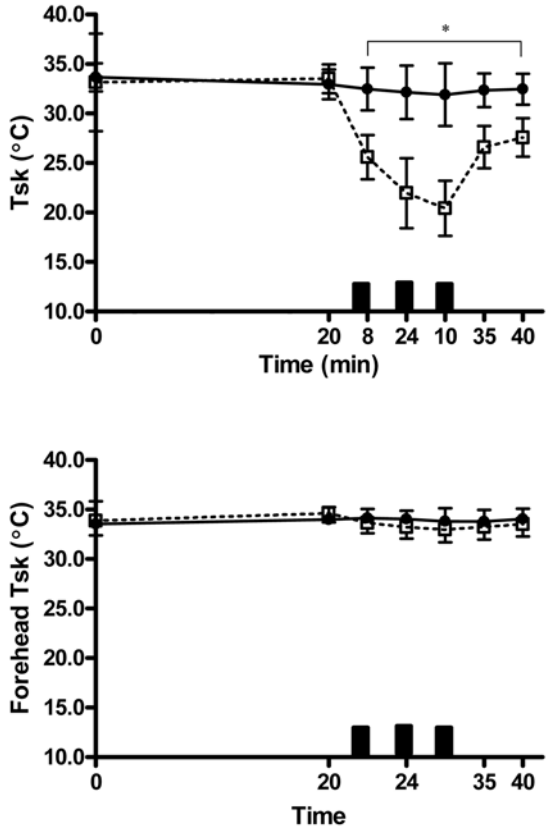

Figure 3 — Mean skin temperature (T_{sk}) (a) and forehead (b) skin temperature responses to cold water immersion (CWI) and passive recovery (CONT). * indicates significant difference between CONT and CWI.

(Table 2). Plasma adrenaline concentrations significantly declined in the 40-minute recovery period; however, there was no effect of CWI on this decline. Plasma noradrenaline remained unchanged after 40 minutes of recovery in both trials.

Thermal comfort for CWI was significantly lower compared with CONT between minutes 21 and 27, with no difference occurring at any other time points (Figure 4).

There were significant favorable effects of CWI compared with CONT (rating of physical recovery: CWI: 6.8 ± 1.5; CONT: 6.4 ± 1.7, rating of mental recovery: CWI: 6.7 ± 1.8; CONT: 6.1 ± 1.7, decreased muscle soreness: CWI: 3.8 ± 2.6; CONT: 5.0 ± 2.9 and decreased general fatigue: CWI: 5.3 ± 2.0; CONT: 6.3 ± 2.0).

Table 1 Metabolic Responses to Cold Water Immersion (CWI) and Passive Recovery (CONT); *Indicates Significant Difference Between CONT and CWI ($P < .05$)

		End of Exercise	+20 min	+40 min
pH	CONT	7.227 ± 0.064	7.330 ± 0.066	7.403 ± 0.052
	CWI	7.250 ± 0.062	7.359 ± 0.074	7.392 ± 0.049
	P value	0.157	0.150	0.530
Chloride ($mmol \cdot L^{-1}$)	CONT	110.50 ± 3.63	107.27 ± 2.61	107.09 ± 3.48
	CWI	110.55 ± 2.88	107.91 ± 2.43	105.82 ± 2.64
	P value	0.806	0.638	0.338
Glucose ($mmol \cdot L^{-1}$)	CONT	9.16 ± 2.39	8.41 ± 2.39	7.35 ± 2.20
	CWI	8.68 ± 2.14	8.05 ± 1.62	6.72 ± 0.54
	P value	0.394	0.092	0.394
Bicarbonate ($mmol \cdot L^{-1}$)	CONT	13.52 ± 2.37	17.83 ± 3.01	21.61 ± 2.67
	CWI	14.03 ± 2.03	18.64 ± 2.76	21.70 ± 1.68
	P value	0.421	0.138	0.893
Potassium ($mmol \cdot L^{-1}$)	CONT	6.05 ± 0.76	4.48 ± 0.36	4.73 ± 0.80
	CWI	6.01 ± 0.74	4.63 ± 0.67	4.25 ± 0.61
	P value	0.055	0.789	0.840
Sodium ($mmol \cdot L^{-1}$)	CONT	146.21 ± 3.25	142.6 ± 2.73	139.78 ± 6.54
	CWI	146.02 ± 3.05	143.23 ± 2.67	140.71 ± 2.33
	P value	0.467	0.082	0.609
PCO_2 (mmHg)	CONT	28.01 ± 2.74	30.62 ± 3.04	32.91 ± 3.37
	CWI	37.53 ± 1.81	29.77 ± 2.79	34.57 ± 3.06
	P value	0.531	0.596	0.208
PO_2 (mmHg)	CONT	78.36 ± 9.35	73.75 ± 13.72	67.71 ± 9.07
	CWI	77.95 ± 11.95	72.01 ± 11.56	59.46 ± 10.40*
	P value	0.559	0.058	0.015

Discussion

Cold water immersion has become a popular means of enhancing recovery from various forms of exercise. The rationale behind the usage of cold water immersion is based on empirical evidence supporting the use of cryotherapy for soft tissue injuries[12] as well as the benefits of cold water immersion for reducing core body temperature. Cold water immersion has also become popular among athletes involved in noncontact sports and sports with minimal muscle damage as a consequence of limited eccentric muscle contractions. In these particular sports, rationale has often been based on reducing core temperature, reducing lactate concentrations, and/or positively influencing a variety of hormone concentrations.

Table 2 Markers of Muscle Damage and Inflammation and Hormonal Responses to Cold Water Immersion (CWI) and Passive Recovery (CONT)

		End of Exercise (0 min)	End of Recovery (+40 min)
CK ($U \cdot L^{-1}$)	CONT	145.3 ± 56.6	127.1 ± 48.4
	CWI	150.3 ± 38.7	133.1 ± 33.7
	P value	0.870	0.814
CRP ($mg \cdot dL^{-1}$)	CONT	0.07 ± 0.07	0.05 ± 0.06
	CWI	0.06 ± 0.05	0.09 ± 0.11
	P value	0.312	0.354
IGF-1 ($ng \cdot mL^{-1}$)	CONT	304.4 ± 113.3	214.3 ± 51.6
	CWI	283.1 ± 127.2	195.8 ± 30.1
	P value	0.748	0.724
IL-6 ($pg \cdot mL^{-1}$)	CONT	4.9 ± 2.6	4.0 ± 1.6
	CWI	3.5 ± 1.0	4.0 ± 1.5
	P value	0.307	0.901
Prolactin ($ng \cdot mL^{-1}$)	CONT	36.8 ± 20.2	23.3 ± 9.4
	CWI	43.5 ± 29.0	24.3 ± 11.4
	P value	0.608	0.870
Growth Hormone ($mIU \cdot L^{-1}$)	CONT	40.2 ± 9.7	12.4 ± 8.7
	CWI	36.5 ± 10.8	9.2 ± 5.5
	P value	0.607	0.491
Cortisol (nM)	CONT	338.6 ± 125.1	479.7 ± 185.6
	CWI	353.1 ± 176.3	515.8 ± 229.5
	P value	0.882	0.737
Testosterone ($ng \cdot dL^{-1}$)	CONT	672.3 ± 185.3	499.0 ± 134.2
	CWI	615.6 ± 377.7	408.9 ± 236.3
	P value	0.791	0.596
Adrenaline ($pg \cdot mL^{-1}$)	CONT	323 ± 170	113 ± 19
	CWI	333 ± 119	115 ± 37
	P value	0.841	0.911
Noradrenaline ($ng \cdot mL^{-1}$)	CONT	1.864 ± 0.531	0.883 ± 0.338
	CWI	1.899 ± 0.586	0.771 ± 0.507
	P value	0.684	0.841

Subjects in the current study did not demonstrate physiological or thermoregulatory responses that would contraindicate the use of cold water immersion while possessing core temperatures above 39°C. Importantly, while CWI significantly decreased T_{re}, at no time did core temperature decline to a point where the subjects were at risk for hypothermia. Previous research in the area of cooling

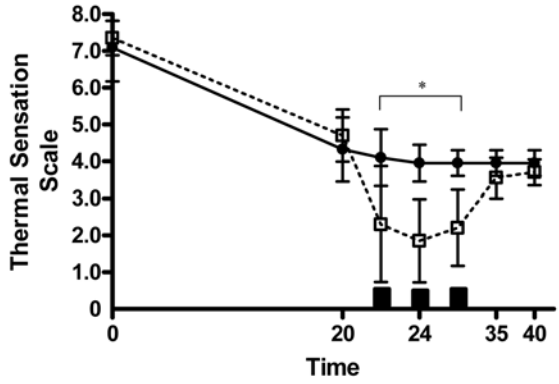

Figure 4 — Thermal Sensation Scale ratings in response to cold water immersion (CWI) and passive recovery (CONT). * indicates significant difference between CONT and CWI.

after hyperthermia examined cooling in circulated water at temperatures of 2°C, 8°C, 14°C, and 20°C for durations of 8.8 ± 4.5, 14.5 ± 5.3, 17.3 ± 4.5 and 18.2 ± 12.9 minutes, respectively.[13] Results of this study demonstrated that cooling individuals to a T_{re} of 38.6°C (for 2°C and 8°C water immersion) and a T_{re} of 37.8°C (for 14°C and 20°C water immersion) will result in the elimination of 100% of the heat gained by the exercise without causing hypothermia.[4] Proulx et al[4] exercised subjects for ~45 minutes and subjects reached an average T_{re} of 39.8°C; in the current study, subjects exercised for ~40 minutes and upon completion of cycling had reached a T_{re} of 39.54 ± 0.42°C (CONT) and 39.74 ± 0.51°C (CWI). The current study, however, had a lower cooling rate than any of those found with the various water temperatures used by Proulx et al[4] (0.35°C/min; 0.19°C/min; 0.15°C/min; and 0.19°C/min for 2°C, 8°C, 14°C, and 20°C, respectively). This difference may be explained by the lower duration of immersion in the current study (total immersion time of 3 min) and due to the fact that the cooling strategy was intermittent. However, the cooling rate of 0.09°C/min is similar to that of Wyndham et al,[14] who cooled hyperthermic subjects (T_{re} 40°C) in a noncirculated 14.4°C water bath. Water circulation displaces the boundary layer of water adjacent the subject's skin, which ensures the temperature gradient between the skin and the water is maintained, resulting in higher cooling rates. This effect may also explain some of the differences between the current study and that of Proulx et al.[4]

The objective of cooling in hyperthermic individuals is to reduce body temperature to a safe level as quickly as possible.[13] However, overcooling is also possible, bought about by an "after drop" in core temperature and can result in hypothermia. In an attempt to identify a safe level of cooling, research suggests that cooling individuals to a T_{re} of 38.6°C (for 2°C and 8°C water immersion) and a T_{re} of 37.8°C (for 14°C and 20°C water immersion) will not result in an after drop that may cause hypothermia.[4] Given that the T_{re} of subjects in the current study was 38.25 ± 0.47°C upon completion of the CWI protocol and their T_{re} was 37.75 ± 0.4°C at the end of the entire recovery period, it can be suggested that the CWI

protocol employed did not result in hypothermia and can be considered safe for these individuals.

The magnitude of the physiological response to cold water immersion depends on numerous factors including the duration of exposure, water temperature, water circulation, body movement, body size, body fat levels, whether the individual's face is submerged, and the presence or absence of clothing.[15] The results of the current study show an immediate decrease in heart rate following the initial cold water immersion. Heart rate has been shown to decline by 3 to 15%, resulting from an increase in central blood volume expansion and an increase in stroke volume.[5] It is commonly believed that CWI enhances lactate removal through a "flushing" effect. The results of this study do not support that belief, with no influence of CWI on lactate concentrations. It has been suggested that the vasoconstriction resulting from CWI may result in a redistribution of blood flow away from the skin toward the muscle and core. Even though this may or may not occur, there appears to be no effect on lactate concentrations.

Previous research has suggested that CWI may be an effective treatment for muscle damage,[7,16] through decreasing skin, muscle, and intra-articular temperatures; metabolism; inflammation; blood flow; pain; muscle spasm; and increasing tissue stiffness. Results from the present investigation did not identify changes in markers of muscle damage or inflammation as a consequence of cold water immersion. Creatine kinase, C-reactive protein, IL-6, and TNF-α were not affected by cold water immersion. However, as cycling involves predominantly concentric muscle actions, and the athletes in the present investigation were highly trained, the degree of muscle damage would not be expected to be high. In addition, the timing of sampling for these markers may have been unsuitable to observe changes in inflammation as this process may occur beyond a 40-minute postexercise period.

It is often anecdotally reported that cryotherapy may induce alterations in the hormonal milieu. Decreases in catabolic hormones (cortisol) and increases in anabolic hormones (testosterone, growth hormone, IGF-1) are thought to promote faster muscle recovery. The results of this study do not support this suggestion, with no changes observed in any of the endocrine parameters assessed. Previous research has also demonstrated no change in testosterone concentrations in response to cold water stimulation.[17] It was hypothesized that cold water immersion may have had an influence on the stress hormone prolactin, as prolactin concentrations have been associated with core temperature increases while exercising in the heat.[18] However, this was not observed in the acute recovery period in the present investigation, possibly due to the high intersubject variation of this measure. In addition, it should be noted that blood sampling was from the periphery and many of the measured variables may have important autocrine, paracrine, or intracellular functions that may be impacted by local cooling or altered perfusion that may not be reflected accurately from sampling at the periphery.

One of the postulated mechanisms behind physiological changes associated with CWI is related to hydrostatic pressure. While this was not assessed in the current study, hydrostatic pressure can enhance the return of fluid from the muscles into the blood.[5] This is suggested to enhance the removal of metabolic waste thereby enhancing recovery from exercise.[5] However, this was not indirectly

observed in the current study where no acute changes in any of the metabolic variables measured were noted.

Subjective reports of heightened recovery are commonly observed following cold water immersion, in particular after exercising in warm or hot conditions. Subjects in the present investigation reported reduced levels of fatigue and soreness and an increased perception of physical and mental recovery following CWI. While we did not incorporate a placebo control and thus a treatment effect cannot be dismissed, athletes commonly report enhanced feelings of alertness following cold water immersion. Cold water immersion and other forms of cryotherapy have strong evidence supporting their analgesic effects following muscle damage and soreness,[6] and this reduced pain sensation may have contributed to the reduced perception of soreness and fatigue. The mechanisms behind this increased perception of recovery require further research attention. The decreased thermal comfort reported during the CWI period is reflective of anecdotal reports of discomfort during CWI. These anecdotal reports are often heightened in lean subjects who possess less muscle and adipose tissue to serve as effective insulation. The subjects in the current study were very lean and this may have contributed to the low ratings of thermal comfort reported. However, at the end of the 40-minute recovery period, thermal comfort had returned to levels not different from CONT.

Practical Significance

The significant reductions in skin and core temperatures observed in the current study may be of particular benefit for athletes who are required to perform more than one bout of exercise in warm or hot environments within a short time frame, or in athletes who have a sufficient break in play/activity (ie, halftime), where rapid cooling may enhance subsequent performance. In this manner, cold water immersion may act as a form of precooling for further exercise/performance. However, this recovery aid may only benefit performance in warm-to-hot environments and during prolonged sporting events. Cold water immersion has been shown to reduce power output and heart rate in cyclists when conducted between two maximal 30-s sprints in thermoneutral conditions,[19] whereas the positive effects of precooling have been observed before longer duration, submaximal exercise in warm-to-hot conditions (for review, see Marino[20]).

CWI results in safe and rapid cooling in very lean individuals. The strategy incorporated in the present investigation did not result in an excessive post-CWI decrease in core temperature that could lead to hypothermia and thus can be safely prescribed to healthy individuals. The current findings relate only to predominantly concentric activity over an acute period of time. CWI may result in significant changes following other forms of activity (weight training, running, contact sports, etc) where there is increased eccentric activity or muscle damage through contact or impact.

Conclusions

In summary, cold water immersion resulted in a significant decrease in heart rate and core and skin temperatures in the 40 minutes following cycling in the heat. While cold water immersion has been suggested to enhance recovery by reducing

lactate concentrations and altering hormone concentrations, this does not appear to be the cause of the perceived enhanced recovery in this investigation. The cold water immersion protocol used in the present investigation did not result in hypothermia. Finally, cold water immersion may be an effective and safe tool when a reduction in core temperature may be of benefit to subsequent performance or perceptions of fatigue.

Acknowledgments

The authors would like to thank the subjects for their participation and enthusiasm. This research was supported by an Australian Sports Commission Grant and the University of Ballarat.

References

1. Coris EE, Ramirez AM, Van Durme DJ. Heat illness in athletes: the dangerous combination of heat, humidity and exercise. *Sports Med*. 2004;34:9–16.
2. Hadad E, Rav-Acha M, Heled Y, Epstein Y, Moran DS. Heat stroke :a review of cooling methods. *Sports Med*. 2004;34:501–511.
3. Lee DT, Toner MM, McArdle WD, Vrabas IS, Pandolf KB. Thermal and metabolic responses to cold-water immersion at knee, hip, and shoulder levels. *J Appl Physiol*. 1997;82:1523–1530.
4. Proulx CI, Ducharme MB, Kenny GP. Safe cooling limits from exercise-induced hyperthermia. *Eur J Appl Physiol*. 2006;96:434–445.
5. Wilcock IM, Cronin JB, Hing WA. Physiological response to water immersion: a method for sport recovery? *Sports Med*. 2006;36:747–765.
6. Meeusen R, Lievens P. The use of cryotherapy in sports injuries. *Sports Med*. 1986;3:398–414.
7. Eston R, Peters D. Effects of cold water immersion on the symptoms of exercise-induced muscle damage. *J Sports Sci*. 1999;17:231–238.
8. Jirak Z, Jokl M, Stverak J, Pechlat R, Coufalov H. Correction factors in skin temperature measurement. *J Appl Physiol*. 1975;38:752–756.
9. Ramanathan NL. A New Weighting System for Mean Surface Temperature of the Human Body. *J Appl Physiol*. 1964;19:531–533.
10. Schmidt V, Bruck K. Effect of a precooling maneuver on body temperature and exercise performance. *J Appl Physiol*. 1981;50:772–778.
11. Young AJ, Sawka MN, Epstein Y, Decristofano B, Pandolf KB. Cooling different body surfaces during upper and lower body exercise. *J Appl Physiol*. 1987;63:1218–1223.
12. Bleakley C, McDonough S, MacAuley D. The use of ice in the treatment of acute soft-tissue injury: a systematic review of randomized controlled trials. *Am J Sports Med*. 2004;32:251–261.
13. Proulx CI, Ducharme MB, Kenny GP. Effect of water temperature on cooling efficiency during hyperthermia in humans. *J Appl Physiol*. 2003;94:1317–1323.
14. Wyndham CH, Strydom NB, Cooke HM, et al. Methods of cooling subjects with hyperpyrexia. *J Appl Physiol*. 1959;14:771–776.
15. Wittmers LE, Savage MV. Cold Water Immersion. In: Burr RE, ed. *Medical Aspects of Harsh Environments*. Bethesda, Maryland: United States Government Printing; 2001:531–552.
16. Merrick MA, Rankin JM, Andres FA, Hinman CL. A preliminary examination of cryotherapy and secondary injury in skeletal muscle. *Med Sci Sports Exerc*. 1999;31:1516–1521.

17. Sakamoto K, Wakabayashi I, Yoshimoto S, Masui H, Katsuno S. Effects of physical exercise and cold stimulation on serum testosterone level in men. *Nippon Eiseigaku Zasshi*. 1991;46:635–638.
18. Bridge MW, Weller AS, Rayson M, Jones DA. Responses to exercise in the heat related to measures of hypothalamic serotonergic and dopaminergic function. *Eur J Appl Physiol*. 2003;89:451–459.
19. Schniepp J, Campbell TS, Powell KL, Pincivero DM. The effects of cold-water immersion on power output and heart rate in elite cyclists. *J Strength Cond Res*. 2002;16:561–566.
20. Marino FE. Methods, advantages, and limitations of body cooling for exercise performance. *Br J Sports Med*. 2002;36:89–94.