Influence of hypohydration on intermittent sprint performance in the heat

Neil S. Maxwell[1]
Richard W.A. Mackenzie[1,2]
David Bishop[3]

[1] Chelsea School Research Centre, University of Brighton
[2] Now based in the School of Life Sciences, University of Westminster
[3] Facoltà di Scienze Motorie, Università di Verona

This is a copy of an article published in the International Journal of Sports Physiology and Performance, 4 (1). pp. 54-67, March 2009. It is available on the publisher's website at:

http://journals.humankinetics.com/ijspp-back-issues/IJSPPVolume4Issue1March

International Journal of Sports Physiology and Performance, 2009, 4, 54-67

Influence of Hypohydration on Intermittent Sprint Performance in the Heat

Neil S. Maxwell, Richard W.A. Mackenzie, and David Bishop

Purpose: To examine the effect of hypohydration on physiological strain and intermittent sprint exercise performance in the heat (35.5 ± 0.6°C, 48.7 ± 3.4% relative humidity). ***Methods:*** Eight unacclimatized males (age 23.4 ± 6.2 y, height 1.78 ± 0.04 m, mass 76.8 ± 7.7 kg) undertook three trials, each over two days. On day 1, subjects performed 90 min of exercise/heat-induced dehydration on a cycle ergometer, before following one of three rehydration strategies. On day 2, subjects completed a 36-min cycling intermittent sprint test (IST) with a −0.62 ± 0.74% (euhydrated, EUH), −1.81 (0.99)% (hypohydrated$_1$, HYPO$_1$), or −3.88 ± 0.89% (hypohydrated$_2$, HYPO$_2$) body mass deficit. ***Results:*** No difference was observed in average total work (EUH, 3790 ± 556 kJ; HYPO$_1$, 3785 ± 628 kJ; HYPO$_2$, 3647 ± 339 kJ, $P = 0.418$), or average peak power (EUH, 1315 ± 129 W; HYPO$_1$, 1304 ± 175 W; HYPO$_2$, 1282 ± 128 W, $P = 0.356$) between conditions on day 2. Total work and peak power output in the sprint immediately following an intense repeated sprint bout during the IST were lower in the HYPO$_2$ condition. Physiological strain index was greater in the HYPO$_2$ vs. the EUH condition, but without changes in metabolic markers. ***Conclusion:*** A greater physiological strain was observed with the greatest degree of hypohydration; however, sprint performance only diminished in the most hypohydrated state near the end of the IST, following an intense bout of repeating sprinting.

Keywords: physiological strain index, hyperthermia, cycling, hydration status

Intermittent sprint exercise, resembling the work:rest ratios of many team sports, is associated with a high level of metabolic heat production.[1] When this exercise is performed in hot conditions, this heat load is exacerbated. A higher thermal strain has been associated with a reduced sprint performance when intermittent sprint exercise is performed in hot compared with temperate conditions.[1–3] Some evidence exists to suggest that intermittent sprint exercise performance is not affected[4] or improved[5] in the heat, but differences in the duration and number of sprints and recovery periods between sprints may account for these discrepancies. It is unlikely that the subjects in those studies would have experienced the level of heat strain commonly observed in team games, due to the brevity of the sprint protocols, although core temperatures were not reported to confirm this.

Maxwell and Mackenzie are with Chelsea School Research Centre, University of Brighton, Eastbourne, U.K., and Bishop is with Facoltà di Scienze Motorie, Università di Verona, Verona, Italy.

Limited evidence exists of the physiological mechanisms that underpin the decline in intermittent sprint performance in the heat.

Hypohydration has been commonly cited as one mechanism underlying the heat-induced decrements in endurance performance.[6] Gonzalez-Alonso et al[7] observed an additive effect from hypohydration and heat on increasing cardiovascular strain when performing submaximal exercise. Unfortunately, inconsistent results that can be explained from the choice and subsequent interpretation of methodological designs has led to a poor understanding of the relationship between hypohydration and anaerobic performance.[8] Watson et al[9] found 50-m, 200-m, and 400-m sprint times to be maintained after diuretic-induced dehydration (2.5%). Magal et al[10] found moderate hypohydration (<3%) under field-based heat stress conditions to decrease tennis related sprint performance. Greater impairments with increasing fluid deficits were also observed in repeated resistance exercise performance.[11] It remains unclear if repeated sprint exercise performance that is common to many team games differs in a hypohydrated compared with a euhydrated state in hot conditions.

Although trying to replicate levels of hypohydration that athletes might encounter, the decline in intermittent sprint running observed by Maxwell et al[3] only examined the effect of a 2% body mass deficit in temperate conditions. Sweat losses among elite team sports confirm that postmatch water deficits following exercise in the heat can be >2% due to variability in sweat response.[12] From examining sweat responses of elite professional soccer players training for 90 min in the heat, Shirreffs et al[13] observed average body mass changes of 1.6%, but ranging from 0.7 to 3.2%. If sufficient rehydration does not exist before a second training session in the day, or before a match on a subsequent day, games players may begin a match in an acutely hypohydrated state. Therefore, the purpose of this study was to investigate whether intermittent sprint exercise performance in the heat declines with hypohydration.

Methods

Subjects

Eight unacclimatized, male games players (mean ± SD: age 23.4 ± 6.2 years, height 1.78 ± 0.04 m, mass 76.8 ± 7.7 kg, peak aerobic power [$\dot{V}O_{2peak}$] 59.9 ± 8.0 $mL \cdot kg^{-1} \cdot min^{-1}$, sum of skinfolds 27.9 ± 7.2 mm) were recruited to participate in this investigation. Subjects were informed of the purpose, procedures, and possible risks of this study, which had the approval from the university's research ethics committee, before giving their written informed consent. Subjects were asked to maintain their normal diet and training, but replicate their dietary habits before subsequent trials and abstain from alcohol, caffeine, or vigorous exercise in the 24 hours before testing.

Experimental Design

This study involved three preliminary visits followed by three experimental trials, with each trial conducted over two days, in a crossover, randomized order one week apart. Each experimental trial required subjects to exercise on day 1 in a hot

environment (35.5 ± 0.6°C, 48.7 ± 3.4% relative humidity) to achieve a hypohydrated state (~2% below baseline body mass) before they were assigned one of three rehydration strategies. On day 2, subjects performed the intermittent sprint test (IST) in the hot conditions described earlier in a different hydration state. All cycle tests were conducted on an air-braked cycle ergometer (Evolution Pty. Ltd., Adelaide, Australia) that monitored power output continuously and was modified to reduce the convective cooling effect from the ergometer flywheel. To control for diurnal variations in body temperature, day 1 and day 2 experimental trials were conducted at the same time of day, respectively.

Experimental Procedures

Preliminary Visits. Anthropometric data were determined on the first visit with skinfolds assessed from four sites (biceps, triceps, subscapular, and supra-iliac crest) using calipers (Harpenden Instruments, West Sussex, UK), as described by Durnin & Wormersley,[14] before subjects were familiarized to the graded exercise test (GXT). The GXT was performed on the second visit to establish lactate threshold (LT) and peak aerobic power (Vo_{2peak}). The GXT commenced at 70 W and power increased by 30 W every 3 minutes while cycling at 80 rpm until volitional exhaustion. The LT was calculated using the modified Dmax method.[15] On the third visit, subjects performed three 4-s sprints to determine a criterion sprint profile in a nonfatigued state before performing a familiarization of the IST (Figure 1).

Intermittent Sprint Test. The IST was designed to mimic the physiological demands of one half of a typical team-sport game[16] and involved 36 minutes of repeated-sprint exercise divided into 2-min periods of a 4-s sprint, 100 seconds of active recovery at 35% of Vo_{2peak}, and 16 seconds of passive rest. The active recovery intensity was calculated from regression analysis of power output against

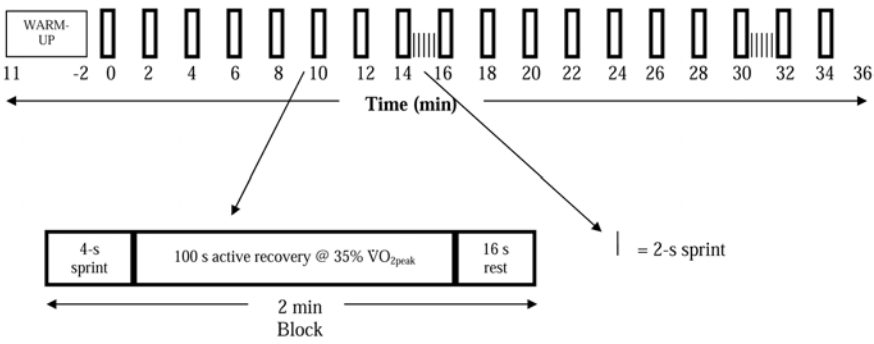

Figure 1 — Schematic representation of the intermittent sprint test. Each 2-min block comprised a 4-s maximal cycle sprint, 100- s cycling at 35% Vo_{2peak} and 16- s passive rest. There were also two repeated-sprint bouts which occurred during the 100-s active recovery following sprint 8 and 16 that comprised five, 2-s sprints separated by ~18- s cycling at 35% Vo_{2peak}.

oxygen uptake during the GXT. After the 8th and 16th 4-s sprint during the IST, a repeated sprint bout (RSB) was performed comprising five 2-s sprints with 18 seconds of active recovery between sprints. The RSBs were designed to mimic an intense period of sprinting, commonly experienced within team games.[16] Peak power output (PPO) was recorded as the highest value during each 4-s sprint. No fluid was permitted by subjects throughout the IST.

Experimental Trial—Day 1. On day 1 (1300–1500) of each experimental trial, following the recording of baseline measures, subjects performed exercise/heat-induced dehydration (EH-ID) to hypohydrate by ~2% of body mass. The EH-ID comprised the IST, 15 minutes of passive rest, and 30 minutes of exercise at 50% of $\dot{V}O_{2peak}$ in the heat.

Subjects were randomly assigned to one of three rehydration conditions designed to begin the IST either 0%, 2%, or 4% hypohydrated. Subjects in the 0% (EUH) and 2% ($HYPO_1$) hypohydrated conditions ingested a volume of water that equaled 150% and 100% of the body weight lost during day 1, respectively. The EUH trial also involved the subjects drinking 0.5 L of water 30 minutes before going to bed and within 30 minutes of arising the next morning. Subjects in the 4% hypohydrated condition ($HYPO_2$) received no fluids.

Experimental Trial—Day 2. On day 2 (0800–1000) each subject performed the IST following a standard warm-up that involved 5 minutes of cycling at 50% $\dot{V}O_{2peak}$, two repeats of 30 seconds of cycling at 70% of $\dot{V}O_{2peak}$ alternated with 30 seconds of passive rest and two 4-s all-out sprints separated by 2 minutes of cycling at 35% $\dot{V}O_{2peak}$.

Measures

Nude body mass was measured before exercise to the nearest 0.01 kg using electronic balance scales (ED3300, Sauter, Germany) and on completion of the IST. The change from baseline to postexercise body mass was used to calculate the precise fluid deficits. Fluid loss was then estimated from the difference between before and after nude body mass, and sweat rate was calculated.

During the GXT, expired air was continuously analyzed for O_2 and CO_2 concentrations using Ametek gas analyzers (SOV S-3A11 and COV CD-3A, Applied Electrochemistry, Pittsburgh, PA). Ventilation was recorded every 15 seconds using a turbine ventilometer (225A, Morgan, Kent, England). The gas analyzers were calibrated before and verified after each test (BOC Gases, Chatswood, Australia). The ventilometer was calibrated preexercise and verified postexercise using a 1-L syringe.

Heart rate (HR) was monitored continuously throughout all testing using short-distance telemetry (Sports Tester PE-4000, Bodycare Products Ltd., Warwickshire, UK). Readings of core temperature (T_{re}) were taken continuously on a 1000-series, 8-bit Squirrel meter logger (Grant Instruments Ltd., Cambridge, UK) using a rectal probe inserted ~10 cm beyond the anal sphincter. The physiological strain index (PSI) was calculated from exercising HR and T_{re} and categorized from 0 (no strain) to 10 (very high strain) using the following equation:[17]

$$PSI = 5(T_{ret} - T_{re0}) \times (39.5 - T_{re0})^{-1} + 5(HR_t - HR_0) \times (180 - HR_0)^{-1}$$

where T_{re0} and HR_0 are the initial T_{re} and HR, respectively, and T_{ret} and HR_t are simultaneous measurements taken at any time.

A hyperemic ointment (Finalgon, Boehringer Ingelheim, Germany) was applied to the earlobe of each subject 5 minutes before initial blood sampling. Glass capillary tubes were used to collect 35 μL of blood during the GXT (D957G-70 to 35, Clinitubes, Radiometer Copenhagen) and 2 × 125 μL of blood during the IST (D957G-70 to 125, Clinitubes). Capillary blood samples were taken at rest following a 20-min stabilization period and immediately following each 3-min stage of the GXT. Capillary blood samples were also taken at rest, after warm-up, following the RSBs during the IST, and on completion of the IST; all in a seated position.

Whole blood pH, lactate ($[La^-]$), hemoglobin ([Hb]), and hematocrit (Hct) concentration were determined using a blood gas analyzer (ABL 625, Radiometer Copenhagen) calibrated using precision standards and assessed by external quality controls. Percentage change in plasma volume was estimated from whole blood Hct and [Hb] values.[18]

Urine samples were collected into 20-mL glass containers on arrival to the laboratory. Urine samples were immediately analyzed for urine color (U_{col}) and specific gravity (U_{sg}), before 5 mL was drawn off and frozen at −70°C in inert microcentrifuge tubes for later analysis of urine osmolality (U_{osm}). Urine color was determined independently by two investigators using an 8-point color chart; specific gravity was measured by pipetting 1 mL of urine onto the plate of a handheld refractometer (URICON-N_E 2722, Atago Co., Ltd.). Before measurement of U_{osm}, the sample was thawed, incubated in a 37°C water bath for 15 minutes and mixed with a vortex shaker. Triplicate measurements of U_{osm} were taken on each sample using a freeze-point depression osmometer (210, Fisk Associates, Burlington, MA).

Subjects' perception of effort during exercise was determined from a 15-point rating of perceived exertion (RPE) scale,[19] whereas thermal sensation (TS) and perceived thirst (PT) were determined using an 8-category[20] and 9-category[21] scale, respectively. Whole-body ratings of TS and PT were recorded at rest, after warm-up, and 1 minute into every fifth 2-min block during the IST. Measurements of RPE were recorded after warm-up and at the same time points as TS and PT during the IST.

Statistical Analyses

All data were checked for normality and where there was a significant violation of sphericity the Huynh–Feldt correction factor was used. Two-way repeated-measures ANOVA was used to test for interaction within dependent variables (condition × time). When significance was obtained, Tukey's honestly significant difference post hoc test was performed to identify pairwise differences between means. Friedman's two-way ANOVA was used to test for differences in RPE, TS, and PT. The software SPSS (version 12.0) was used. Pearson correlation coefficients were calculated to examine the relationship between change in hypohydration and change in sprint performance indices as calculated from the criterion sprint data of the preliminary visits. Statistical significance was set at $P < .05$ and all values are reported as means (SD).

Results

Hydration Indices

Hydration markers on day 1 and 2 are shown in Table 1. EH-ID reduced body mass for all conditions from baseline to postexercise values. Both Hct and [Hb] were greater post EH-ID for EUH, $HYPO_1$ and $HYPO_2$, whereas U_{osm} and U_{sg} demonstrated no differences from the EH-ID used during day 1 (Table 1). Following EH-ID, U_{col} was greater in the $HYPO_1$ condition only ($P = .02$).

The EUH trial was not different for any hydration marker between baseline values day 1 and 2, although body mass did approach a significant reduction between days (–0.62 ± 0.74%, $P = .053$, Table 1). EH-ID combined with the respective rehydration strategy caused body mass deficits of –1.79% and –3.89% in the $HYPO_1$ ($P < .002$) and $HYPO_2$ ($P < .001$) trials, respectively. Values for U_{osm}, U_{sg}, and U_{col} significantly increased between day 1 and 2 for both hypohydration conditions (Table 1).

Performance Indices

Despite a trend for a decreased total work with increasing hypohydration (Figure 2), this was not significant between conditions (EUH, 3790 ± 556 kJ; $HYPO_1$, 3785 ± 628 kJ; $HYPO_2$, 3647 ± 339 kJ [$P = 0.418$]). Power analysis, indicated that 55 subjects would have been required to detect a significant difference in total work throughout the IST at the 5% level with 91% power. Similarly, PPO decreased with increasing hypohydration (Figure 3), but this was not significant (EUH, 1315 ± 129 W; $HYPO_1$, 1304 ± 175 W; $HYPO_2$, 1282 ± 128 W [$P = .356$]). Peak power output relative to body mass ($PP{\cdot}kg^{-1}$) was not different between the EUH (17.3 ± 1.7 W), $HYPO_1$ (17.2 ± 1.8 W), and $HYPO_2$ (17.3 ± 1.9 W [$P = 0.932$]) conditions.

No difference was found between hydration conditions after RSB_1 for total work (EUH, 3396 ± 558 kJ; $HYPO_1$, 3551 ± 556 kJ; $HYPO_2$, 3431 ± 389 kJ), PPO (EUH, 1178 ± 257 W; $HYPO_1$, 1211 ± 167 W; $HYPO_2$, 1194 ± 121 W), or $PP{\cdot}kg^{-1}$ (EUH, 16.4 ± 1.5 W; $HYPO_1$, 16.1 ± 1.7 W; $HYPO_2$, 16.1 ± 1.4 W). There were also no differences in RSB_2 for total work between EUH (3463 ± 273 kJ) and $HYPO_1$ (3205 ± 622 kJ), or $HYPO_1$ and $HYPO_2$ (3189 ± 261 kJ). Peak power output showed no differences between EUH (1227 ± 108 W) and $HYPO_1$ (1167 ± 209 W) or between $HYPO_1$ and $HYPO_2$ (1125 ± 134 W). Differences were observed between EUH and $HYPO_2$ for both total work ($P = .004$) and PPO ($P = .01$). Peak power output relative to body mass showed no difference between all three hydration conditions in RSB_2 (EUH, 16.2 ± 0.7 W; $HYPO_1$, 15.2 ± 1.9 W; $HYPO_2$, 15.1 ± 1.4 W).

Physiological and Perceptual Indices

No differences were observed for resting HR in both hypohydrated ($HYPO_1$, 62 ± 15 $beats{\cdot}min^{-1}$; $HYPO_2$, 65 ± 10 $beats{\cdot}min^{-1}$) compared with the EUH (58 ± 10 $beats{\cdot}min^{-1}$, $P = .58$) conditions. Heart rate increased similarly in all conditions throughout the IST, but it was higher in $HYPO_2$ compared with EUH condition

Table 1 Mean (SD) Hydration Markers at Rest and Postexercise on Day 1 and Day 2 in the EUH, $HYPO_1$ and $HYPO_2$ Conditions

	EUH				$HYPO_1$				$HYPO_2$			
	Rest (day 1)	Post-Exercise (day 1)	Rest (day 2)	Post-Exercise (day 2)	Rest (day 1)	Post-Exercise (day 1)	Rest (day 2)	Post-Exercise (day 2)	Rest (day 1)	Post-Exercise (day 1)	Rest (day 2)	Post-Exercise (day 2)
BM (kg)	77.5 (7.3)	75.6** (7.2)	77.1 (7.2)	75.8** (7.2)	77.3 (7.8)	75.5** (7.6)	75.9** (7.9)	75.0** (8.1)	77.4 (7.8)	75.5** (7.6)	74.4** (7.9)	73.5** (7.8)
Hct (%)	48.1 (2.4)	50.1* (2.1)	48.0 (2.1)	50.3* (2.3)	48.0 (3.0)	51.5** (2.7)	48.9 (2.9)	51.5** (3.3)	48.6 (2.8)	52.5** (2.5)	51.0** (2.8)	52.6** (2.6)
Hb (g/dL)	15.7 (0.8)	16.4* (0.7)	15.7 (0.7)	16.4** (0.8)	15.7 (1.0)	16.8* (0.9)	16.0 (1.0)	16.9** (1.1)	15.9 (0.9)	17.2** (0.8)	16.6** (0.9)	17.2** (0.9)
PV (%)	/	−8.2 (6.0)	0.2 (10.5)	−7.5 (6.4)	—	−12.8 (10.1)	−3.2 (11.4)	−10.2 (2.6)	—	−11.6 (6.4)	−9.4 (3.8)	−10.4 (7.5)
U_{OSM} (mOsm/L)	372 (269)	341 (263)	241 (168)	218 (152)	353 (187)	379 (165)	803** (129)	794 (96)	252 (135)	391 (270)	902** (93)	843** (81)
U_{SG} (g/mL)	1.011 (0.008)	1.009 (0.007)	1.007 (0.005)	1.007 (0.004)	1.012 (0.004)	1.012 (0.006)	1.025** (0.005)	1.024 (0.005)	1.008 (0.006)	1.009 (0.004)	1.027** (0.003)	1.027 (0.002)
U_{COL}	2.4 (1.4)	2.6 (1.1)	1.8 (1.0)	2.5 (0.9)	3.0 (1.3)	3.8 * (1.4)	5.4** (1.1)	5.8 (1.2)	2.0 (1.1)	2.5 (0.9)	6** (0.0)	6.5* (0.5)

Note. BM body mass; Hct, hematocrit; Hb, hemoglobin; PV, plasma volume; U_{OSM}, urine osmolality; U_{SG}, urine specific gravity; U_{COL}, urine color; TS, thermal sensation; comparisons made between rest (day 1) against post exercise (day 1), rest (day 1) against rest (day 2) and between rest (day 2) against post exercise (day 2).

*Denotes alpha value of $P < .05$ and **denotes alpha value of $P < .01$.

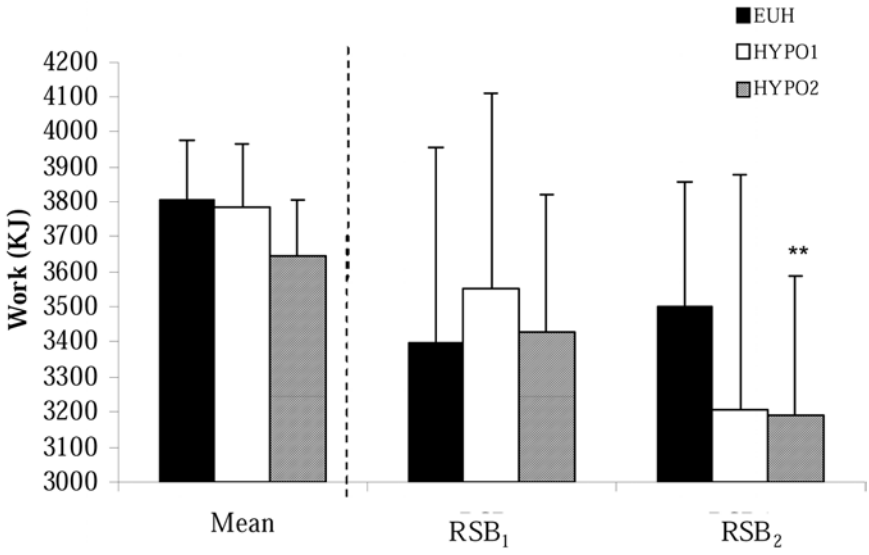

Figure 2 — Average total work during the Intermittent Sprint Test in the EUH, $HYPO_1$ and $HYPO_2$ trials. Left of the dotted line denotes average total work of sprints 1 to 18, while right of the dotted line denotes average total work for the sprint completed immediately following repeated sprint bout 1 (RSB_1) and 2 (RSB_2). ** denotes significant main effect between $HYPO_2$ and EUH for RSB_2 average total work ($P = .004$). Values are mean (*SD*).

($P = .01$). Peak HR was higher ($P = .01$) in the $HYPO_2$ (165 ± 16 beats·min^{-1}) vs. EUH (155 ± 17 beats·min^{-1}) and $HYPO_1$ (159 ± 19 beats·min^{-1}) conditions

Resting T_{re} was higher with increasing hypohydration (EUH, 36.7 ± 0.2°C; $HYPO_1$, 36.8 ± 0.3°C and $HYPO_2$, 36.9 ± 0.3°C), although this was only significant between $HYPO_2$ and EUH ($P = .03$). During the IST, mean T_{re} (EUH, 37.7 ± 0.3°C; $HYPO_1$, 37.8 ± 0.3°C and $HYPO_2$, 38.1 ± 0.3°C) and peak T_{re} (EUH, 38.1 ± 0.9°C; $HYPO_1$, 38.6 ± 0.3°C and $HYPO_2$, 38.9 ± 0.4°C) were higher ($P = .03$) in the $HYPO_2$ compared with the EUH condition. On average, T_{re} rose 0.4°C for every percent body mass lost. Physiological strain index throughout the IST was higher ($P = .001$) in $HYPO_2$ (6.0 ± 0.8) compared with both the $HYPO_1$ (5.4 ± 1.0) and EUH (5.1 ± 1.0) conditions. Peak PSI values showed a similar trend with $HYPO_2$ (8.1 (1.1)), being higher than both EUH (7.2 ± 1.3) and $HYPO_1$ (7.6 ± 1.1) conditions ($P = .01$).

Fluid loss was greater within the EUH (1.23 ± 0.24 L) compared with $HYPO_1$ (0.84 ± 0.42 L) and $HYPO_2$ (0.99 ± 0.19 L) conditions, but no difference was found between hypohydrated conditions. Consequently, sweat rates were different in the hypohydrated ($HYPO_1$, 1.36 ± 0.35 L·h^{-1}; $HYPO_2$, 1.32 ± 0.26 L·h^{-1}) compared with the EUH (1.67 ± 0.31 L·h^{-1}) conditions, but not different between hypohydrated conditions. Thermal sensation, RPE, and PT all increased significantly ($P < .001$) with increasing hypohydration.

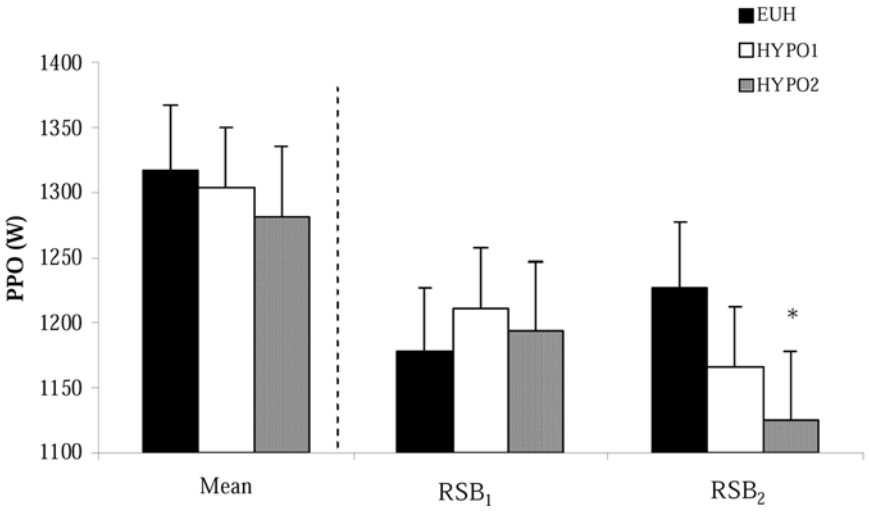

Figure 3 — Peak Power Output (PPO) during the Intermittent Sprint Test in the EUH, $HYPO_1$ and $HYPO_2$ trials. Left of the dotted line denotes PPO of sprints 1 to 18, while right of the dotted line denotes PPO for the sprint completed immediately following repeated sprint bout 1 (RSB_1) and 2 (RSB_2). * denotes significant main effect between $HYPO_2$ and EUH for RSB_2 PPO ($P = .01$). Values are mean (SD).

Blood Indices

No differences were observed for either pH or [La^-] as a result of performing the IST in the three hydration conditions or in plasma volume changes (Table 2).

Correlation Analyses

A weak relationship ($r = 0.25$, $P = .25$) was observed between the percent decrease in body mass and the percent change in total work taken from criterion sprint data. A similar pattern was seen in PPO. Relationships between PSI and total work, PPO, and change in body mass were $r = .30$ ($P = .16$), $r = 0.36$ ($P = .09$), and $r = 0.44$ ($P = .03$), respectively.

Discussion

This study determined whether male games players, who were hypohydrated by as much as 4% body mass from prior exercise/heat-induced dehydration, could sustain intermittent sprint exercise performance that mimicked team sport activity[16] under heat stress conditions. The principal finding of this study was that despite a greater physiological strain with the greatest level of hypohydration, reductions in work done and PPO were only observed after a second, intense repeated sprint bout during the latter stages of the IST. No relationship was observed between changes in these performance indices and percent change in hypohydration.

Table 2 Mean (SD) pH and Blood Lactate [La] at Rest, Post Warm-Up, Post Repeated Sprint Bout 1 (RSB_1), Post Repeated Sprint Bout 2 (RSB_2) and Post Intermittent Sprint Test (IST) in the EUH, $HYPO_1$, and $HYPO_2$ Conditions on Day 2

		EUH	$HYPO_1$	$HYPO_2$
pH	**Rest**	7.41 (0.02)	7.41 (0.02)	7.41 (0.02)
	Post Warm-up	7.39 (0.02)	7.40 (0.03)	7.40 (0.02)
	RSB_1	7.37 (0.03)	7.38 (0.05)	7.39 (0.03)
	RSB_2	7.39 (0.04)	7.40 (0.03)	7.39 (0.03)
	Post IST	7.37 (0.02)	7.37 (0.03)	7.39 (0.02)
[La] ($mmol \cdot L^{-1}$)	**Rest**	0.68 (0.19)	0.73 (0.21)	0.74 (0.13)
	Post Warm-up	3.4 (1.7)	3.44 (1.76)	3.31 (1.05)
	RSB_1	7.48 (2.68)	6.80 (3.49)	7.54 (2.37)
	RSB_2	7.48 (2.99)	6.57 (2.10)	7.53 (2.46)
	Post IST	5.23 (2.09)	5.40 (2.49)	5.05 (2.29)

Previous investigations have observed a reduced intermittent sprint exercise performance in the heat compared with temperate conditions and this has been associated with a greater physiological strain.[1–3] The PSI was no different between $HYPO_1$ and EUH conditions, suggesting that the PSI may not be sensitive to detect small changes in hydration status. However, Moran et al[22] did find physiological strain to be ranked correctly over a range of hydration states (1.1 to 4.2%) using the PSI. Using the same index, physiological strain was also found to be different after prior exercise that resulted in 3% and 5% body mass deficits while under heat stress conditions.[22] The PSI may not be suited to intermittent exercise, however, as it takes into account only initial and final values of heart rate and core temperature.

For every percent body mass lost, T_{re} rose on average 0.4°C, which is higher than the 0.1 to 0.25°C reported for endurance exercise.[7,23] Although body mass was significantly lower in the $HYPO_1$ vs. EUH condition on day 2, this rise in T_{re} either had little physiological significance, or there had been sufficient time since the EH-ID for the redistribution of fluid from intra- and extracellular spaces of the muscle and skin to maintain blood volume.[24] That the blood hydration indices were not different from rest day 1 in the $HYPO_1$ trial, but were in the $HYPO_2$ trial support this. Alternatively, with such modest levels of hypohydration in the $HYPO_1$ trial, significant cardiovascular and thermoregulatory adjustments were not needed to meet the demands of performing the IST in the heat. In contrast, during the $HYPO_2$ trial a decreased blood volume at rest and during exercise will have increased blood viscosity, reduced venous return and cardiac filling, and decreased stroke volume and cardiac output—all resulting in a decreased physiological tolerance. Core temperature (T_{re}) and HR adjustments will have compensated for the increased level of hypohydration and may have prevented a significant reduction of cardiac output during the IST. However, following RSB_2 stroke volume may have been reduced where cardiac output could not be maintained,

even with elevations in heart rate.[25] The combination of intermittent sprint exercise and heat stress may have masked the consequences of mild, but not severe levels of hypohydration.

Several factors could explain the reduced sprint performance following RSB_2 of the $HYPO_2$ trial. An altered muscle metabolism and/or muscle blood flow and reduced central drive to exercise have been linked to declines in exercise performance in the heat. As skeletal muscle glycogen resynthesis is unaffected by hypohydration,[26] EH-ID is unlikely to have altered glycogen storage between conditions when subjects commenced the IST on day 2. Neither [La^-] nor pH were different between conditions, which is consistent with research that has observed the debilitating effects of heat stress during intermittent sprint exercise without alterations in muscle metabolism,[3] or blood metabolites.[1,3,5]

Indirect measurements of blood flow taken during moderate exercise-heat stress suggest that an increase in muscle blood flow is not warranted. During severe exercise-heat stress, skeletal muscle blood flow is decreased.[27] The redistribution of blood flow to the cutaneous vasculature may have reduced the cardiac output that perfused the contracting muscle in the RSB_2 of the $HYPO_2$ trial and/or a decreased effective central blood volume causing a decreased venous return and cardiac filling. Both the vasodilatory and hypovolemic consequences of the heat stress and hypohydration, respectively, could have independently or simultaneously limited cardiac output in the $HYPO_2$ trial.

Behavioral changes (ie, confusion, loss of coordination, syncope) are often associated with hyperthermia-induced fatigue and indirect evidence that central nervous system function is altered in the heat. Increased central fatigue has been explained through a reduction in voluntary muscle activation (VMA) and associated with increases in RPE.[28] Ratings of perceived exertion and TS were both significantly higher in the $HYPO_2$ trial. If a reduced central drive existed following the RSB_2 of the IST in the $HYPO_2$ trial, an inadequate input to the motorneuron pool and/or the inability to respond to adequate efferent input could explain this. Either could have adjusted muscle recruitment and power output after the RSB_2. Under hyperthermic conditions, Todd et al[29] observed that a decline in VMA was associated with T_{re} of ~38.5°C. This T_{re}, although achieved by different heating methods, is similar to the peak T_{re} values in $HYPO_1$ and $HYPO_2$, indicating that moderate hyperthermia could affect the central nervous system without a "critical" T_{re} of 40°C.

An anticipatory thermogenesis regulation may react to the expected rate of heat storage and respond through feedback to the brain.[29,30] The lower total work and PPO in RSB_2 of the $HYPO_2$ trial may have related to an anticipated rate of heat production during the IST. In contrast, the EUH trial may have attenuated any subconscious down-regulation of motor units. The last sprint of the IST across conditions may add support for this anticipatory response. Despite a lower total work and PPO in the penultimate sprint (ie, after RSB_2) in $HYPO_2$ vs. EUH and $HYPO_1$, there was no difference in either sprint performance index in the last sprint between conditions. An anticipatory pacing strategy during intermittent sprint exercise in the heat has previously been observed[31] and may support a central governor causing an anticipatory thermogenic response.

Practical Applications

EH-ID used in this study may mimic a situation that games players might encounter on arrival at a second training session of the day, or on a subsequent day in a hypohydrated state. Games players may be able to tolerate hypohydration up to 4% of body mass without a significant decline in sprint performance while exercising in the heat, but when a critical intense period in the game ensues a body mass deficit of 4% could cause sprint performance to diminish.

Limitations

The IST was based on motion analysis of international men's field hockey[16] and designed to mimic the physiological demands of one half of a typical team-sport game. However, cycling that only consisted of one half will have reduced the external validity of this study. If a running model had been used while being hypohydrated, it is conceivable that less weight would have transferred into less energetic cost, negating any negative effect from the hypohydration and consequently, sprint performance may have been better maintained throughout the entire IST. It is also possible that extending the protocol to a second half would have accentuated any hypohydration effect. The ecological validity of this study could have been enhanced with a rest period before a second half to mimic a game scenario.

Conclusions

In conclusion, while the exercise-heat stress appeared to mask the consequences of hypohydration during most of the IST, it was only when a second intense, repeated bout of sprinting took place toward the end of the IST in the most hypohydrated state that sprint performance declined. Peripheral markers of fatigue could not explain this reduction in repeated sprint performance. A central governor, possibly that mediates an anticipatory thermogenic response, may explain why sprint performance was reduced after an intense repeated sprint bout, although this can only be indirectly inferred from these findings.

Acknowledgments

The authors are grateful for the technical assistance of Mr. Dave Gould of the Western Australian Institute of Sport and Mr. Tony Robey of the University of Western Australia.

References

1. Falk B, Radom-Isaac S, Hoffman JR, et al. The effect of heat exposure on performance of and recovery from high-intensity, intermittent exercise. *Int J Sports Med*. 1998;19:1–6.
2. Maxwell NS, Gardner F, Nimmo MA. Intermittent running: muscle metabolism in the heat and effect of hypohydration. *Med Sci Sports Exerc*. 1999;31:675–683.

3. Morris JG, Nevill ME, Lakomy HKA, Nicholas C, Williams C. Effect of a hot environment on performance of prolonged, intermittent, high intensity shuttle running. *J Sports Sci*. 1998;16:677–686.
4. Backx KM, McNaughton L, Crickmore L, Palmer G, Carlisle A. Effects of differing heat and humidity on the performance and recovery from multiple high intensity, intermittent exercise bouts. *Int J Sports Med*. 2000;21:400–405.
5. Ball D, Burrows C, Sargeant AJ. Human power output during repeated sprint cycle exercise: the influence of thermal stress. *Eur J Appl Physiol*. 1999;79:360–366.
6. Sawka MN, Young AJ, Francesconi RP, Muza SR, Pandolf KB. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J Appl Physiol*. 1985;59:1394–1401.
7. Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, Coyle EF. Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. *J Appl Physiol*. 1997;82:1229–1236.
8. Judelson DA, Maresh CM, Anderson JM, et al. Hydration and muscular performance. *Sports Med*. 2007;37(10):907–921.
9. Watson G, Judelson DA, Armstrong LE, Yeargin SW, Casa DJ, Maresh CM. Influence of diuretic induced dehydration on competitive sprint and power performance. *Med Sci Sports Exerc*. 2005;37(7):1168–1174.
10. Magal M, Webster MJ, Sistrunk LE, Whitehead MT, Evans RK, Boyd JC. Comparison of glycerol and water hydration regimens on tennis-related performance. *Med Sci Sports Exerc*. 2003;35:150–156.
11. Judelson DA, Maresh CM, Farrell MJ, et al. Effect of hydration state on strength, power and resistance exercise performance. *Med Sci Sports Exerc*. 2007;39(10):1817–1824.
12. Burke L. Fluid balance during team sports. *J Sports Sci*. 1997;15:287–295.
13. Shirreffs SM, Aragon-Vargan LF, Chamorro M, Maughan RJ, Serratosa L, Zachwieja JJ. The sweating response of elite professional soccer players to training in the heat. *Int J Sports Med*. 2005;26:90–95.
14. Durnin J, Womersley J. Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br J Nutr*. 1974;32:77–97.
15. Bishop D, Jenkins DG, Mackinnon LT. The relationship between plasma lactate parameters, Wpeak and 1-h cycling performance in women. *Med Sci Sports Exerc*. 1998;30(8):1270–1275.
16. Spencer M, Lawrence S, Rechichi C, Bishop D, Dawson B, Goodman C. Time motion analysis of elite field hockey during several games in succession: a tournament scenario. *J Sci Med Sport*. 2005;8(4):382–391.
17. Moran DS, Shitzer A, Pandolf KB. A physiological strain index to evaluate heat stress. *Am J Physiol Regul Integr Comp Physiol*. 1998a;275:R129–R134.
18. Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol*. 1974;37:247–248.
19. Borg GA. Psychological basis of physical exertion. *Med Sci Sports Exerc*. 1982;14:377.
20. Toner MM, Drolet LL, Pandolf KB. Perceptual and physiological responses during exercise in cool and cold water. *Percept Mot Skills*. 1986;62:211–220.
21. Engell DB, Maller O, Sawka MN, Francesconi RN, Drolet L, Young AJ. Thirst and fluid intake following graded hypohydration levels in humans. *Physiol Behav*. 1987;40:229–236.
22. Moran DS, Montain SJ, Pandolf KB. Evaluation of different levels of hydration using a new physiological strain index. *Am J Physiol*. 1998b;273(3):R854–R860.
23. Montain SJ, Coyle EF. Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. *J Appl Physiol*. 1992;73:903–910.

24. Nose H, Mack GW, Shi XR, Nadel ER. Shift in body fluid compartments after dehydration in humans. *J Appl Physiol*. 1988;65:318–324.
25. Rowell LB. Human cardiovascular adjustments to exercise and thermal stress. *Physiol Rev*. 1974;54:75–159.
26. Neufer PD, Sawka MN, Young AJ, Quigley MD, Latzka WA, Levine L. Hypohydration does not impair skeletal muscle glycogen resynthesis after exercise. *J Appl Physiol*. 1991;70:1490–1494.
27. Gonzalez-Alonso J, Calbet JAL, Nielsen B. Metabolic and thermodynamic responses to dehydration-induced reductions in muscle blood flow in exercising humans. *J Physiol*. 1999;520:577–589.
28. Nybo L, Nielsen B. Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. *J Appl Physiol*. 2001;91:2017–2023.
29. Todd G, Butler JE, Taylor JL, Gandevia SC. Hyperthermia: A failure of the motor cortex and the muscle. *J Physiol*. 2005;563:621–631.
30. Marino FE, Lambert MI, Noakes TD. Superior performance of African runners in warm humid but not in cool environmental conditions. *J Appl Physiol*. 2004;96:124–130.
31. Castle PC, Macdonald AL, Philp A, Webborn A, Watt PW, Maxwell NS. Pre-cooling leg muscle improves intermittent sprint exercise performance in hot, humid conditions. *J Appl Physiol*. 2005;100:1377–1384.