Impact of Acute Weight Loss and/or Thermal Stress on Rowing Ergometer Performance

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ABSTRACT
SLATER, G. J., A. J. RICE, K. SHARPE, R. TANNER, D. JENKINS, C. J. GORE, and A. G. HAHN. Impact of Acute Weight Loss and/or Thermal Stress on Rowing Ergometer Performance. Med. Sci. Sports Exerc., Vol. 37, No. 8, pp. 1387–1394, 2005. Purpose: The impact of acute weight loss on rowing performance was assessed when generous nutrient intake was provided in 2 h of recovery after making weight. Methods: Competitive rowers (N = 17) completed four ergometer trials, each separated by 48 h. Two trials were performed after a 4% body mass loss in the previous 24 h (WT) and two were performed after no weight restrictions, that is, unrestricted (UNR). In addition, two trials (1 × WT, 1 × UNR) were in a thermoneutral environment (NEUTRAL, mean 21.1 ± SD 0.7°C, 29.0 ± 4.5% RH) and two were in the heat (HOT 32.4, ± 0.4°C, 60.4 ± 2.7% RH). Trials were performed in a counterbalanced fashion according to a Latin square design. Aggressive nutritional recovery strategies (WT 2.3 g·kg−1 carbohydrate, 34 mg·kg−1 Na, 28.4 mL·kg−1 fluid; UNR ad libitum) were employed in the 2 h after weigh-in. Results: Both WT (mean 2.1, 95% CI 0.7–3.4 s; P = 0.003) and HOT (4.1, 2.7 – 5.4 s; P < 0.001) compromised 2000-m time-trial performance. Whereas WT resulted in hypohydration, the associated reduction in plasma volume explained only part of the performance compromise observed (0.2 s for every 1% decrement). Moreover, WT did not influence core temperature or indices of cardiovascular function. Conclusions: Acute weight loss compromised performance, despite generous nutrient intake in recovery, although the effect was small. Performance decrements were further exacerbated when exercise was performed in the heat. Key Words: MAKING WEIGHT, REHYDRATION, RECOVERY, HEAT

Lightweight rowing is unique among weight category sports. Whereas the development of strength, anaerobic power and anaerobic capacity are dominant characteristics of successful athletes in combat sports such as wrestling (16), aerobic capacity is arguably more critical to competitive success among rowers (18). Debate continues as to the influence of hypohydration on anaerobic performance (27), although there is little doubt hypohydration induced during exercise compromises aerobic performance (for recent review see Barr (2)). Likewise, tolerance to aerobic exercise is compromised by hypohydration induced before exercise (29), at least when no attempt is made to restore fluid balance before exercise. However, the performance implications of acute weight loss induced before exercise when an attempt is made to restore fluid balance before competition, as occurs in many weight category sports, has not been thoroughly investigated.

The impact of acute weight loss on rowing performance has received little attention despite the overwhelming majority of lightweight rowers acknowledging the use of acute weight loss techniques (12). After inducing a 5.2% decrease in body mass over a 24-h period, Burge et al. (7) observed a 22-s increase in time to complete a 2000-m ergometer time trial. Although similar decrements in performance associated with dehydration have been observed in other high-intensity aerobic activities (32), the recovery strategy utilized by Burge and associates (7) was not ideal; volunteers consumed only 1.5 L of tap water after weigh-in, and no food was provided despite a restriction in dietary intake during the previous 24 h. In practice, lightweight rowers eat and drink in the 2 h between weigh-in and racing, and although little is known of specific food/fluid choices, weight category athletes tend to ingest a large amount of energy in an attempt to recover before competition (12).

The primary aim of the present study was to examine the effect of acute weight loss on ergometer performance among lightweight rowers when aggressive nutritional recovery strategies are employed in the 2 h between weigh-in and competition. A secondary aim was to investigate the strength of association between rowing performance, changes in plasma volume and core temperature under different environmental conditions. It was hypothesized that despite the utilization of aggressive recovery strategies, acute weight loss would compromise rowing performance, particularly in the heat.

METHODS

Experimental approach. The performance implications of moderate (4%), acute weight loss on rowing per-
formance were assessed via 2000-m ergometer time trials, each separated by 48 h. Considering that regattas are typically undertaken during summer, performances were assessed under both thermoneutral and thermally stressful conditions. In an attempt to maximize subsequent performance, aggressive nutritional recovery strategies were enforced before each time trial, ensuring athletes had restored at least a portion of the body mass loss incurred over the previous 24 h.

**Subjects.** Seventeen nationally competitive male ($N = 8$) and female ($N = 9$) lightweight rowers participated in this investigation. Volunteers were fully informed of the nature and possible risks of the investigation before giving their written informed consent, which was consistent with the human subject policy of the American College of Sports Medicine. The investigation was approved by the human research ethics committee of the Australian Institute of Sport.

As the investigation was undertaken during midwinter, it was assumed that none of the athletes were heat acclimated. All volunteers were required to adhere to a standardized training program for the 4 wk before the study in an attempt to prepare them for racing. Athletes maintained a daily log of duration, mode, intensity, and frequency of training beginning 4 wk before and continuing throughout the experimental period. The diary was used to assess compliance to the training program.

To ensure maintenance of estrogen and progesterone levels, female volunteers were prescribed a monophasic oral contraceptive, beginning 4 wk before and continuing throughout the experimental period. An aliquot of serum (1 mL) collected at each performance test was frozen at $-80^\circ$C for later analysis of progesterone and estrogen using chemiluminescence technology (Vitros Eci, Ortho-Clinical Diagnostics, Raritan, NJ).

On their first visit to the laboratory, athletes performed a progressive maximal test on a rowing ergometer (Concept 2, Morrisville, VT). The test protocol was modified from that previously described (13) and consisted of three submaximal workloads and one maximal workload, each of 4 min duration and separated by 1-min recovery intervals. Throughout the testing period, mixed expired air passed through a fully automated, first principles, indirect calorimetry system (Australian Institute of Sport, Belconnen, ACT, Australia). The operation and calibration of this system has been described elsewhere (25). $V\text{O}_2\text{peak}$ was defined as the highest $O_2$ uptake athletes attained during two consecutive 30-s sampling periods. In our laboratory this technique has a typical error (TE) of 1.8%.

**Treatments.** Athletes were ranked according to gender and previous 2000-m ergometer time-trial performances. The ranking was used to assign athletes to four fitness and gender-matched groups that were counterbalanced for test order according to a Latin-square design. The remaining subject was randomly allocated to one of the four groups.

Athletes performed four maximal 2000-m ergometer time trials, each test separated by 48 h and undertaken at the same time of day. Two ergometer trials were undertaken follow-

![FIGURE 1—An overview of testing commitments undertaken during each 2000-m ergometer time trial.](image)

ing a 4% loss of body mass (i.e., “at-weight,” WT) in the preceding 24 h; the body mass specified for the first WT trial was specified as the required body mass for the second WT trial. No weight limit was specified in the remaining tests, that is, unrestricted (UNR). No restrictions were imposed on techniques utilized to induce weight loss. However, athletes were required to replicate weight loss techniques employed before their first WT trial for the subsequent WT trial.

Two ergometer trials (1 × WT, 1 × UNR) were performed in a thermoneutral environment (NEUTRAL; mean 21.1 ± SD 0.7°C, 29.0 ± 4.5% relative humidity) whereas the remaining two (1 × WT, 1 × UNR) were completed in a climate chamber at 32.4 ± 0.4°C and 60.4 ± 2.7% relative humidity (HOT). This study design created four different test conditions; unrestricted body mass in a thermoneutral environment (UNRNEUTRAL), unrestricted body mass in a thermally challenging environment (UNR_HOT), acute weight loss in a thermoneutral environment (WTNEUTRAL), and acute weight loss in a thermally challenging environment (WT_HOT). In our laboratory, the 2000-m time trial has a TE of 1.6%.

**Experimental protocol.** An overview of the testing schedule before each ergometer test is presented in Figure 1. Subjects presented to the laboratory 140 min before they were scheduled to start each 2000-m time trial. After assuming a rowing ergometer specific position on a plinth (back support at 115°), legs extended, arms by the side) for 20 min, 12 mL of blood was sampled via venipuncture without stasis from a superficial forearm vein using standard phlebotomy procedures. A total of 6 mL of blood was placed in a serum separation tube and centrifuged at 4000 rpm for 5 min. Half of the resultant serum was stored at $-80^\circ$C and later analyzed for progesterone, estrogen, and osmolality (OSM), the later undertaken in duplicate via the freezing point depression method using an Osmomat 030-D cryogenic osmometer (Gonotech, Berlin, Germany). A further 2-mL aliquot of blood was mixed in a tube containing ethylene diaminetetraacetic acid (potassium salt). Hemato-

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**Venous blood sample (Hct, Hb, OSM, progesterone, oestrogen)**

**Bladder voided body mass**

**Recovery formula (2.3 g kg$^{-1}$ carbohydrate, 34 mg kg$^{-1}$ Na, 28.4 ml kg$^{-1}$ fluid)**

**Ergometer - 2 x 4 min. submax, warm-up, 1 x 2000 m time trial**

**Capillary blood, rectal temp., ratings of perceived exertion and thermal stress**

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**Time (min)**

0 30 60 90 120 150 180 210 240 270 300 330 360 390 420 450 480 510 540 570 600 630 660 690 720 750 780 810 840 870 900 930 960 990 1020 1050 1080 1110 1140
crit and hemoglobin concentration were measured in triplicate using an automated flow cytometry hematology analyser (ADVIA 120, Bayer Diagnostics, Tarrytown, NY) with the mean result used in analysis. Relative changes in plasma volume were calculated using the method employed by Dill and Costill (10). Changes in plasma volume were calculated and expressed relative to hematocrit and hemoglobin concentrations measured in the euhydrated state.

After the first blood sample, bladder voided body mass was measured on a calibrated digital scale with a precision of ± 0.02 kg (A and D Co., Tokyo, Japan). Thereafter subjects consumed a standard meal (toasted bread, Veg-emite™, Power Bar™, Carboshotz™, Gastrolyte™, Gatorade™, water). Food and fluid intake was prescribed in the WT trials (2.3 g·kg⁻¹ carbohydrate, 34 mg·kg⁻¹ Na, and 28.4 mL·kg⁻¹ fluid); fluid intake was ad libitum in UNR trials although equivalent choices were made available. Fluid intake was prescribed in the WT trials to maximize plasma volume and fluid balance restoration (17). Fluid intake was quantified by weighing drink bottles before and after the recovery period.

Bladder-voided body mass was again recorded 103 min after presenting to the laboratory. All urine produced during the recovery period was collected into 2-L polyethylene bottles and quantified using a calibrated digital scale with a precision of ± 2 g (Tanita, Tokyo, Japan). Percent fluid retention was calculated from weighted inventories of fluid intake (inclusive of food water content) and urinary volumes in the recovery period. Insensible water loss was assumed to be similar between experimental trials.

Before sitting on the ergometer, a disposable thermistor (Mallinckrodt Medical, St. Louis, MO) was self-inserted 15 min prior to warm-up before each maximal 2000-m time trial. Arterialized capillary blood was sampled immediately after the submaximal workloads and the 2000 m time trial, and immediately analyzed for pH plus glucose, bicarbonate [HCO₃⁻] and lactate [La⁻] concentrations (ABL 725, Radiometer, Copenhagen, Denmark). The analyser was calibrated daily in accordance with the manufacturer’s specifications.

Average power (W), heart rate (HR), thermal sensation (TSE), rating of perceived exertion (RPE), and T_re were recorded upon completion of each of the workloads. HR during each ergometer test was monitored using short-range telemetry (Vantage, Polar Electro OY, Kempele, Finland) whereas RPE and TSE were ascertained using the 15-point Borg scale (4) and 17-point thermal sensation scale (33), respectively.

**Hydration status.** Overnight fasted, bladder voided, nude body mass was measured soon after waking each day of the investigation on a calibrated digital scale with a resolution of ± 0.02 kg (A and D Co., Tokyo, Japan). Before weigh-in each morning, a mid stream urine sample (approximately 20 mL) was collected during bladder evacuation and analyzed in duplicate for OSM.

**Dietary intake.** Athletes maintained food diaries throughout the investigation. Acknowledging the limitations of self-reported dietary survey methodology (8), all athletes were provided with household measures to assist in quantifying food/fluid intake. Furthermore, athletes were provided with advice on maintaining dietary logs and met with a qualified dietitian periodically to ensure compliance with recommendations. Errors associated with the analysis of diaries is also recognized. Given this, the recommendations of Braakhuis et al. (5) were implemented before analysis of diaries, which were evaluated and analyzed using the Foodworks dietary analysis program (version 3.02, Xyris Software, Brisbane, Australia).

Food and training diaries were used to standardize dietary intake and training in the 24 h before testing. Whatever was consumed in the 24 h before the first UNR and WT trials was replicated in the respective second trials. Similarly, training load was prescribed before each of the time trials, simulating training habitually undertaken in the 24 h before racing. However, athletes were permitted to undertake additional exercise before the first WT trial to assist in achieving specified body mass requirements. This was then replicated for the second WT trial.

**Statistical analyses.** Changes in the mean of variables from the four ergometer trials were compared using a linear mixed-effects model with gender, body mass (UNR, WT), environmental conditions (NEUTRAL, HOT), and trial number (1,2,3,4) as fixed effects, and subject as a random effect. The same procedure provided 95% confidence intervals (CI), the likely range of true values, for all estimates. Plasma volume (relative to the euhydrated state) at the end of the recovery period was included as a covariate in the mixed-effects model to assess the influence of plasma volume on ergometer performance. Likewise, temperature gain throughout the ergometer trial was included as a covariate to determine the influence of thermal load on performance. Biochemical parameters, including plasma volume, were compared using the mixed model analysis with gender and body mass as fixed effects and subject as a random effect. All subsequent results are reported as mean of the difference between WT and UNR values (plus HOT and NEUTRAL), together with 95% CI for the difference and a P value for testing mean difference = 0. The mixed-method analysis was conducted using S-Plus (Insightful Corporation, Seattle, WA) software. All other statistical analyses were undertaken by using Statistica software for Windows (version 6.0, StatSoft, Tulsa, OK). All data are reported as means ± SD, unless otherwise specified.
TABLE 1. Physiological and anthropometric characteristics of participants.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Male (N = 8)</th>
<th>Female (N = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>22.3 ± 3.9</td>
<td>22.6 ± 4.1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>183.2 ± 1.8</td>
<td>171.7 ± 5.0</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>74.2 ± 1.3</td>
<td>63.2 ± 2.6</td>
</tr>
<tr>
<td>VO₂peak·L⁻¹·min⁻¹</td>
<td>4.7 ± 0.2</td>
<td>3.5 ± 0.2</td>
</tr>
<tr>
<td>VO₂peak·mL⁻¹·kg⁻¹·min⁻¹</td>
<td>64.5 ± 2.5</td>
<td>55.0 ± 3.1</td>
</tr>
</tbody>
</table>

Values are means ± SD. VO₂peak: peak O₂ uptake.

Correction for posture. Hematocrit and hemoglobin values measured for comparison of posture were analyzed using a paired t-test. Mean differences for hematocrit and hemoglobin between sampling postures were used as correction factors for the raw data when assessing plasma volume shifts throughout the period of testing. A correction factor for both hematocrit (0.7%) and hemoglobin (0.26 g·dL⁻¹) was applied to the first blood sample of each ergometer test after a pilot study (N = 15) revealed posture-induced hemodilution when sampled on the plinth compared to the ergometer.

RESULTS

General descriptive data. Characteristics of all 17 subjects who completed the investigation are presented in Table 1. No effect of time was evident for either estrogen (P = 0.60) or progesterone (P = 0.50) levels. Data for males and females were therefore combined for all subsequent statistical analyses.

Ergometer performance. Results of the four 2000-m ergometer time trials are summarized in Table 2. Acute weight loss compromised performance, at least during the first WT trial (P = 0.01, Fig. 2). However, when acute weight loss was undertaken for the second time, the performance decrement was no longer statistically different either 48 h (P = 0.54) or 96 h (P = 0.12) after the first WT trial. When data were collapsed across WT trials, the performance compromise averaged 2.1 s (95% CI 0.7–3.4; P = 0.003) or a mean of 0.7% (95% CI 0.2–1.2).

Initial exposure to HOT compromised performance (P < 0.01) and subsequent performance remained slower in HOT, whether 48 h (P = 0.05) or 96 h (P = 0.04) after initial exposure. When data were collapsed across HOT trials, performance compromise averaged 4.1 s (95% CI 2.7–5.4; P < 0.001) or a mean of 1.1% (95% CI 0.7–1.5). No interaction was evident between WT and HOT (P = 0.38).

The effect of gender was significant (mean 56.4, 95% CI 47.5–65.2 s; P < 0.01), with little evidence of interaction between gender and other fixed effects (P > 0.05).

Heart rate. HOT resulted in an elevation in HR (1.8, 95% CI 0.6–3.1 beats·min⁻¹; P < 0.01). WT did not influence HR response (0.2, 95% CI -1.1 to 1.4 beats·min⁻¹; P = 0.78) during the 2000-m time trials, nor was there an interaction between HOT and WT (P = 0.96).

Perception of effort. Trial 4 was perceived to be harder than trial 1 (P = 0.003). After accounting for this order effect, neither HOT (−0.1, 95% CI −0.3 to 0.2; P = 0.47) nor WT (0.2, 95% CI −0.1 to 0.4; P = 0.18) influenced RPE during time trials.

Core temperature and thermal sensation. HOT resulted in an elevation in Tₚₑ during time trials when compared to NEUTRAL (0.2, 95% CI 0.0–4.0°C; P = 0.05). However, WT did not influence Tₑ response (0.1, 95% CI −0.1 to 0.2°C; P = 0.53), nor was there an interaction between these factors (P = 0.80). The influence of core temperature gain on performance during ergometer tests approached significance (P = 0.09). On average, core temperature gain throughout a time trial increased as er-
compromised before WT time trials (0.7–4.8%; during the recovery period for WT than UNR (2.7, 95% CI 0.8–1.1; P < 0.001). No effect was evident for WT (0.1, 95% CI 0.0–0.3; P = 0.25).

All athletes experienced hyperthermia (≥1°C gain) during WT, but 25–31% of subjects did not become hyperthermic during UNR, and WT trials. T$_{re}$ reached or exceeded 39°C for only one athlete each during UNR, WT, and WT and three during UNR (Fig. 3); this was achieved by different athletes under each condition.

**Plasma volume.** WT induced a compromise in plasma volume (−5.7, 95% CI −7.5 to −3.9%; P < 0.001) compared with UNR. Change in plasma volume was greater during the recovery period for WT than UNR (2.7, 95% CI 0.7–4.8%; P = 0.01). Despite this, plasma volume remained compromised before WT time trials (−3.1, 95% CI −4.9 to −1.3%; P = 0.04) (Fig. 4). The influence of plasma volume decrement on ergometer performance at the end of the recovery period was small but approached significance (P = 0.08). For every 1% decrease in plasma volume there was, on average, an associated increase in 2000-m ergometer time of 0.2 s (95% CI 0.0–0.4).

**Hydration status.** WT resulted in an elevation in serum OSM upon weigh-in (0.010, 95% CI 0.005–0.015 osmol·kg$^{-1}$; P < 0.01), exceeding levels indicative of hypohydration (Fig. 5). However, HOT did not influence serum OSM (−0.004, 95% CI −0.008 to 0.001 osmol·kg$^{-1}$; P = 0.13), nor was there an interaction between these factors (P = 0.87).

**Body mass.** WT was associated with a 4.3% body mass reduction (95% CI 4.0–4.6%). When body mass loss was assessed according to the sequence of WT trials, an order effect was evident with acute weight loss the first time (4.5%, 95% CI 3.9–5.1%) greater than the second (3.9%, 95% CI 3.4–4.4%; P = 0.05). However, when the time frame between WT trials was considered, no significant differences were observed in weight loss in the previous 24 h (P = 0.17). Ingestion of the recovery formula restored approximately one-third of the body mass loss (1.6%, 95% CI 1.4–1.8%; P < 0.001) (Table 3). The amount of body mass loss did not strongly influence ergometer performance (P = 0.39).

**Blood metabolites and acid–base status.** HOT resulted in an elevation in La$^{-1}$ (1.1 mmol·L$^{-1}$, 95% CI 0.5–1.7; P = 0.001). However, WT did not influence blood La$^{-1}$ (P = 0.29), nor was there an interaction between these factors (P = 0.12). Other indices of acid–base status followed a similar trend. Blood glucose levels were elevated after HOT (0.5, 95% CI 0.0–1.0 mmol·L$^{-1}$; P = 0.04). WT did not influence the blood glucose response (−0.2, 95% CI −0.7 to 0.3 mmol·L$^{-1}$; P = 0.45).

**Training volume and dietary intake.** Training volume increased in the 24 h before WT when compared to UNR (33.7, 95% CI 20.0–47.3 min; P < 0.001). Over the same time period, total volume of food and fluid intake decreased (−2520, 95% CI −2939 to −2102 g; P < 0.001). Accordingly, fluid (−32.3, 95% CI −38.4 to −26.1 mL·kg$^{-1}$; P < 0.001), total energy (−97, 95% CI −112 to −83 kJ·kg$^{-1}$; P < 0.001), carbohydrate (−3.4, 95% CI −4.1 to −2.7 g·kg$^{-1}$; P < 0.001), fat (−0.6, 95% CI −0.7 to −0.4 g·kg$^{-1}$; P < 0.001), and protein (−1.1, 95% CI −1.3 to −0.9 g·kg$^{-1}$; P < 0.001) intakes decreased for WT compared with UNR (Table 4). No main effects of trial number were evident for training volume or nutrient intake in the 24 h before ergometer trials (P > 0.05). Nutrient intake following weigh-in was greater for WT compared with UNR (Table 4).

![FIGURE 3—Effect of acute weight loss (4% over 24 h) and a thermal challenge (32.4 ± 0.4°C, 60.4 ± 2.7% humidity) on core temperature during submaximal workloads and 2000-m ergometer time trials. SUB1 submaximal workload one, SUB2 submaximal workload two.](Image)

![FIGURE 4—Effect of acute weight loss (4% over 24 h) and a thermal challenge (32.4 ± 0.4°C, 60.4 ± 2.7% humidity) on plasma volume. For each condition, the left bar represents plasma volume change at weigh-in, the right bar represents plasma volume change (relative to the euhydrated state) before warm-up. Values are means ± SD for 15 subjects (blood samples were not obtained from two subjects).](Image)

![FIGURE 5—Effect of acute weight loss (4% over 24 h) on serum OSM at weigh-in. A serum OSM ≥ 0.30 osmol·kg$^{-1}$ is indicative of hypohydration. Values are means ± SD for 17 subjects.](Image)
TABLE 3. Body mass changes throughout the simulated regatta (values are means ± SD).

<table>
<thead>
<tr>
<th>Body Mass</th>
<th>Temp.</th>
<th>24 h pre–Weigh-in</th>
<th>Weigh-in</th>
<th>90 min post–Weigh-in</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unrestricted</td>
<td>Neutral</td>
<td>74.0 ± 1.3</td>
<td>73.7 ± 1.2</td>
<td>74.6 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>Hot</td>
<td>74.0 ± 1.2</td>
<td>74.1 ± 1.5</td>
<td>74.8 ± 1.3</td>
</tr>
<tr>
<td>Weight restricted</td>
<td>Neutral</td>
<td>74.1 ± 1.1</td>
<td>70.8 ± 1.2</td>
<td>72.6 ± 1.3</td>
</tr>
<tr>
<td></td>
<td>Hot</td>
<td>74.2 ± 1.7</td>
<td>70.9 ± 1.3</td>
<td>72.7 ± 1.3</td>
</tr>
<tr>
<td></td>
<td>Female (kg)</td>
<td>63.2 ± 2.6</td>
<td>63.6 ± 2.4</td>
<td>64.3 ± 2.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>63.1 ± 2.1</td>
<td>63.5 ± 2.4</td>
<td>64.2 ± 2.3</td>
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<tr>
<td></td>
<td></td>
<td>63.1 ± 2.3</td>
<td>60.5 ± 2.2</td>
<td>62.3 ± 2.1</td>
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<tr>
<td></td>
<td></td>
<td>63.1 ± 2.1</td>
<td>60.8 ± 2.2</td>
<td>62.4 ± 2.0</td>
</tr>
</tbody>
</table>

95% CI 1.6–2.6 kJ·kg⁻¹; P < 0.001), carbohydrate (0.1, 95% CI 0.1–0.2 g·kg⁻¹; P < 0.001), fluid (8.6, 95% CI 7.1–10.1 mL·kg⁻¹; P < 0.001), and sodium (7.4, 95% CI 5.9–8.9 mg·kg⁻¹; P < 0.001) intakes were higher. Retention of ingested fluid during the recovery period between weigh-in and racing was also greater during WT (48.0, 95% CI 41.1–55.0%; P < 0.001) intakes were higher. Retention of ingested fluid during the recovery period between weigh-in and racing was also greater during WT (48.0, 95% CI 41.1–55.0%; P < 0.001). The nutritional recovery formula was well tolerated, with no reports of intestinal discomfort or nausea.

DISCUSSION

The primary finding of this investigation is that both acute weight loss (4.3%) and exposure to a heat challenge compromise rowing ergometer performance. Whereas a reduction in exercise performance is consistent with findings from the only other investigation to assess the implications of acute weight loss on rowing ergometer time trial performance, the absolute decrement in the present investigation was approximately one tenth of that previously reported among athletes of similar caliber (7). Our attenuated response likely reflects the use of aggressive nutritional recovery strategies between weigh-in and racing.

The influence of acute weight loss on performance was similar in magnitude to the normal random variation in 2000-m ergometer time trials observed in our laboratory (0.5%) and elsewhere (0.6%) (30). Hopkins and associates (15) suggest an effect greater than 0.4–0.7 of the typical within-athlete random variation in performance is likely to influence competitive outcomes. Among the present group of athletes this equates to an effect greater than 0.8–1.4 s and 0.9–1.6 s for male and females, respectively. As such, both acute weight loss and heat exposure are likely to compromise performance in a 2000-m ergometer time trial.

The present data suggest the performance implications of acute weight loss may be reduced when undertaken a second time. That is, the athlete may somewhat adapt to acute weight loss. Such adaptations have been proposed by other investigators, possibly via adjustment in fluid regulatory hormones (11) or an increase in total circulating protein (29). Although plausible, the present data can neither support nor refute these mechanisms. Furthermore, results may merely reflect the reduced sample size and thus statistical power of the second body mass restricted trial (two groups undertaking acute weight loss 48 h later (N = 8) or 96 h (N = 9) after the first body mass restricted trial) compared with the first (N = 17).

**Hypohydration and performance.** The performance decrement associated with acute weight loss among lightweight rowers has been attributed almost exclusively to a reduction in plasma volume (7), suggesting maintenance of cardiovascular function is essential if performance is to be maintained during high intensity exercise lasting 7–8 min. Indeed, hypohydration and the associated plasma volume decrement negatively influence an array of parameters, including heart rate, stroke volume, cardiac output, and core temperature (3,21). Hence, our attempt to optimize intake of nutrients, and particularly fluid, in the recovery period between weigh-in and racing.

When nutrient intake during recovery after weigh-in was administered in accordance with current guidelines (31), the present data show improvements in plasma volume. Although not fully restored, the deficit in plasma volume was small (i.e., 1–3% of euhydrated levels) and approached levels (1.6–2.6%) below which performance is unlikely to be impaired (1,7). Nonetheless, the decrement accounted for part of the compromise in ergometer performance.

Vasoconstriction of peripheral vascular beds and/or redistribution of blood from inactive skeletal muscle (19) in response to elevations in angiotensin and vasopressin may have compensated for the slight reduction in blood volume and thus assisted in maintaining cardiac output (26). Whereas the present data suggest minimal performance implications with small reductions in plasma volume, the

TABLE 4. Nutrient intake 24 h prior to each ergometer test and during the recovery period following weigh-in.

<table>
<thead>
<tr>
<th>Ingestion Period</th>
<th>Nutrient Intake</th>
<th>UNRNEUTRAL</th>
<th>UNRHOT</th>
<th>WTNNEUTRAL</th>
<th>WTNHOT</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 h pretesting</td>
<td>Total weight (kg)</td>
<td>4.23 ± 1.76</td>
<td>4.42 ± 1.66</td>
<td>1.61 ± 1.22</td>
<td>1.81 ± 1.48</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Energy (kJ·kg⁻¹)</td>
<td>164 ± 44.6</td>
<td>169 ± 56.6</td>
<td>66 ± 34.3</td>
<td>71.9 ± 37.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Carbohydrate (g·kg⁻¹)</td>
<td>6.1 ± 1.7</td>
<td>6.5 ± 2.7</td>
<td>2.7 ± 1.4</td>
<td>2.9 ± 1.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Protein (g·kg⁻¹)</td>
<td>1.6 ± 0.4</td>
<td>1.7 ± 0.5</td>
<td>0.6 ± 0.4</td>
<td>0.6 ± 0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Fat (g·kg⁻¹)</td>
<td>0.8 ± 0.6</td>
<td>0.9 ± 0.4</td>
<td>0.3 ± 0.2</td>
<td>0.3 ± 0.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Fluid (mL·kg⁻¹)</td>
<td>516 ± 244.4</td>
<td>566 ± 273</td>
<td>194 ± 158</td>
<td>224 ± 201</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Energy (kJ·kg⁻¹)</td>
<td>435 ± 3.2</td>
<td>436 ± 3.2</td>
<td>457 ± 2.6</td>
<td>457 ± 2.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Carbohydrate (g·kg⁻¹)</td>
<td>2.2 ± 0.2</td>
<td>2.2 ± 0.2</td>
<td>2.3 ± 0.1</td>
<td>2.3 ± 0.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Sodium (mg·kg⁻¹)</td>
<td>28.2 ± 3.5</td>
<td>28.1 ± 5.1</td>
<td>34.0 ± 1.8</td>
<td>34.0 ± 1.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Fluid (mL·kg⁻¹)</td>
<td>19.6 ± 4.2</td>
<td>19.7 ± 4.2</td>
<td>28.4 ± 5.5</td>
<td>28.4 ± 5.5</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Level of significance specified is for a main effect of acute weight loss. Values are means ± SD.
threshold below which performance is compromised may depend on environmental stressors coupled with the exercise task (27).

A dissociation between plasma volume decrement and performance has been observed in other investigations using performance trials of similar duration to that used in the present study (24). This suggests that factors other than changes in plasma volume may be responsible, at least in part, for poorer performance following acute weight loss. Indeed, in addition to fluid restriction, athletes also increased their training volume and decreased total energy (and carbohydrate) intake in the 24 h before body mass restricted trials. The influence of these factors on performance, either independently or in combination, cannot be discounted.

Energy depletion and performance. The present data do not allow speculation as to whether the present protocol influenced muscle glycogen stores. It is reasonable to assume that given dietary carbohydrate intake was reduced by more than 50% and activity levels were increased in the 24 h before ergometer trials requiring acute weight loss, a reduction in muscle glycogen concentration may have occurred. However, it is unlikely that carbohydrate availability limits performance during a 5- to 7-min event (7), even when increased muscle glycogen utilization associated with hypohydration is considered (14).

Training load and performance. The independent performance implications of additional long duration training in the 24 h before body mass restricted ergometer trials can not be addressed in the present investigation. However, such strategies do not comply with current tapering guidelines (22) and thus cannot be discounted as an independent factor influencing performance. The independent performance implications of energy/carbohydrate restriction and increased training before competition warrants further investigation among lightweight rowers.

Body mass and performance. Considering the association between fat free mass and rowing ergometer performance (9), it could be argued that the performance decrement observed in response to acute weight loss may be ascribed, at least in part, to a reduction in body mass alone, independent of metabolic factors. However, unlike fat free mass, which includes total body water, active skeletal muscle mass is unlikely to have been reduced to any significant degree during a 24-h period of energy restriction, suggesting potential force generating capacity was unlikely to have been influenced by the acute period of body mass reduction. Thus, any performance compromise observed was unlikely a consequence of reductions in body mass alone.

Core temperature and performance. The attainment of a critically high body temperature (39–40°C) has been proposed as the main factor limiting endurance performance in hot environments (23). Whereas hyperthermia developed in response to the exercise demands of the present study, irrespective of environmental conditions and hydration status, few athletes achieved a core temperature commonly associated with fatigue. However, rather than there being a critical temperature at which fatigue is induced, a continuum of performance decrement with thermal strain is likely. In addition, the impact of hyperthermia on exercise performance is influenced by factors such as duration/intensity of exercise plus hydration and nutritional status, fitness and motivation of the athlete (23).

Hypohydration did not influence thermoregulatory response to approximately 7 min of rowing in a thermoneutral environment in our study. This contrasts with previous research that has assessed the influence of hypohydration on both cardiovascular and thermoregulatory responses to sustained exercise of 1 h. A recent study has shown that 5% hypohydration (without rehydration) compromises cardiovascular function and thermoregulation during 1 h of moderate-intensity exercise in both temperate and hot environments, albeit to a smaller degree in the temperate conditions (6). The discrepancy in response under temperate conditions may reflect the aggressive recovery strategies and shorter exercise duration used in the present investigation.

As rowing competitions are typically undertaken in the summer months, strategies for minimizing the impact of hyperthermia on performance should be considered by oarsmen, especially those individuals in which hydration status is compromised due to acute weight loss techniques. Whereas heat acclimation is one such strategy, the ensuing favorable thermoregulatory response is ameliorated by hypohydration (28). With this in mind, other strategies such as precooling (20) during the recovery period between weigh-in and racing may be beneficial to further attenuate the performance compromise associated with a thermal challenge.

In summary, the present investigation has shown that modest acute weight loss (4% of body mass in 24 h) compromises rowing ergometer time trial performance (0.7%), even when aggressive nutritional recovery strategies are utilized following weigh-in. Whereas a small reduction in plasma volume persisted until the end of the recovery period, this only partially accounted for the performance decrement. Exposure to the thermally stressful environment caused a greater decrease in ergometer performance (1.1%). Consequently, when competing in thermally stressful conditions, lightweight oarsmen should not only optimize nutrient intake following weigh-in, they should also give special consideration to strategies such as precooling that can assist in minimizing the thermal load.

We are particularly grateful for the support of the Australian Institute of Sport Rowing Program, especially head coach Reinhold Batschi and scholarship coach Connie Vanderwerp.

This research was supported via a grant obtained from the Australian Institute of Sport.
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