Impact of Two Different Body Mass Management Strategies on Repeat Rowing Performance

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[1]Department of Physiology, Australian Institute of Sport, Belconnen, Australian Capital Territory, AUSTRALIA; [2]School of Human Movement Studies, The University of Queensland, Brisbane, Queensland, AUSTRALIA; and [3]Department of Mathematics and Statistics, The University of Melbourne, Melbourne, Victoria, AUSTRALIA

ABSTRACT

SLATER, G. J., A. J. RICE, R. TANNER, K. SHARPE, D. JENKINS, and A. G. HAHN. Impact of Two Different Body Mass Management Strategies on Repeat Rowing Performance. *Med. Sci. Sports Exerc.*, Vol. 38, No. 1, pp. 138–146, 2006. **Purpose:** The present study was conducted to examine the impact of acute weight loss on repeat 2000-m rowing ergometer performance during a simulated multiday regatta, and to compare two different body mass management strategies between races. **Methods:** Competitive rowers (N = 16) were assigned to either a control (CON), partial recovery ($REC_{partial}$), or complete recovery ($REC_{complete}$) group. Volunteers completed four trials, each separated by 48 h. No weight restrictions were imposed for the first trial. Thereafter, athletes in $REC_{partial}$ and $REC_{complete}$ were required to reduce their body mass by 4% in the 24 h before trial 2, again reaching this body mass before the final two trials. No weight restrictions were imposed on CON. Aggressive nutritional recovery strategies were used in the 2 h following weigh-in for all athletes. These strategies were maintained for the 12–16 h following racing for $REC_{complete}$ with the aim of restoring at least three quarters of the original 4% body mass loss. Postrace recovery strategies were less aggressive in $REC_{partial}$; volunteers were encouraged to restore no more than half of their initial 4% body mass loss. **Results:** Acute weight loss increased time to complete the first "at-weight" performance trial by a small margin (mean 3.0, 95% CI −0.3 to 6.3 s, P = 0.07) when compared with the CON response. This effect decreased when sustained for several days. Aggressive postrace recovery strategies tended to eliminate the effect of acute weight loss on subsequent performance. **Conclusion:** Acute weight loss resulted in a small performance compromise that was reduced or eliminated when repeated over several days. Athletes should be encouraged to maximize recovery in the 12–16 h following racing when attempting to optimize subsequent performance. **Key Words:** MAKING WEIGHT, RECOVERY, REHYDRATION

The international governing body of rowing (FISA) discourages lightweight rowers from using acute weight loss to achieve specified body mass limits. This can be justified by both the potential health (1) and performance (5) implications of making weight. Despite this, a proportion of lightweight rowers continue to make use of acute body mass management techniques before competition, typically reducing body mass by 3–4% in the days before competition (10,27). The implications of acute weight loss on rowing performance remain an issue of conjecture. After inducing a 5.2% decrease in body mass over 24 h, Burge et al. (5) observed a 22-s increase in simulated 2000-m ergometer time among a group of internationally competitive oarsmen. More recent work by our group, however, suggests the performance implications of moderate acute weight loss (2.1-s increase in 2000-m ergometer time trial performance following 4% body mass loss in 24 h) is much smaller, possibly because our use of aggressive nutritional recovery strategies following weigh-in (28).

The implications of maintaining lower than normal body mass on repeat performance throughout the course of a multiday regatta remains to be investigated. Because lightweight rowers weigh in not less than 1 h and no more than 2 h before the start of each race (33), competing in a multiday regatta demands consideration of body mass management and recovery strategies between races. We have previously shown that a proportion of athletes are conservative with nutrient intake, both during recovery following weigh-in and also between days of racing (27). This mini-

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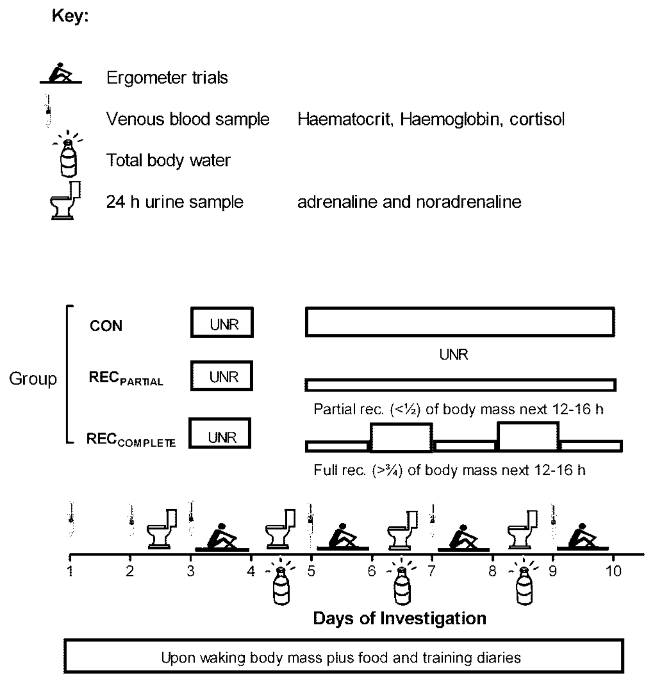

FIGURE 1—An overview of the investigation, incorporating assessment of acute weight loss (4% over 24 h) and different body mass management or recovery strategies (control (CON) vs partial ($REC_{partial}$) and complete ($REC_{complete}$) recovery) between races on performance throughout a simulated regatta. UNR, unrestricted body mass.

mizes weight gain and, hence, reduces the need for repetition of acute weight loss in preparation for subsequent races during the following 24 to 48 h. This approach, however, could potentially impair recovery between days of racing. The influence of nutritional recovery strategies implemented following each race on subsequent performance during a regatta is yet to be assessed.

The present study, therefore, was conducted to examine whether acute weight loss techniques repeatedly used to achieve specified body mass limits over several days of racing would affect rowing ergometer performance. We hypothesized that the continued reliance on acute weight loss techniques before each race throughout a simulated regatta would result in progressively greater performance decrements. We also hypothesized that the use of aggressive nutritional recovery following each race would minimize fatigue and, hence, tend to ameliorate performance decrements for the later, and most important, races of the regatta when compared with conservative postrace recovery strategies.

METHODS

A total of 16 male lightweight rowers who had previously competed in the Australian Rowing Championships volunteered for this investigation. An overview of the study is shown in Figure 1, replicating wherever possible the demands of a major international regatta such as the World Championships or Olympic Games. Volunteers were fully informed of the nature and possible risks of the investigation before giving their written informed consent to participate. The investigation was approved by the human research ethics committee of the Australian Institute of Sport.

All volunteers were required to adhere to a standardized training program for 3 wk before the study to prepare them for racing, including two 2000-m ergometer time trials, which acted as familiarization trials. Athletes maintained a daily log of duration, mode, intensity, and frequency of training beginning 3 wk before and continuing throughout the experimental period. The diary was used to assess compliance with the training program and to monitor body mass management strategies during the simulated regatta.

On their first visit to the laboratory, athletes performed a progressive maximal test on a rowing ergometer (Concept 2C, Morrisville, VT). The test protocol was modified from that previously described (12) and consisted of three submaximal workloads and one maximal workload, each of 4-min duration and separated by 1-min recovery intervals. Submaximal steady-state workloads equated to 50, 65, and 80% of average power output from the maximal 2000-m ergometer time trials conducted in the 3-wk lead-in period. The ergometer was secured firmly to the ground, placed no closer than 1 m from a wall, and the drag factor was set at 120. 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 (24). $\dot{V}O_{2peak}$ 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), or within-subject standard deviation, of 1.8%.

On the same day, measurement of hemoglobin (Hb) mass (Hb_{mass}) was made using a CO-rebreathing technique modified by Burge and Skinner (6). The alterations used two doses of 99.5% CO, which were rebreathed for 10 min each (20-mL initial dose and a second dose of 1.25 mL of CO per kilogram of body mass), and percent HbCO was measured on capillary instead of venous blood (2). An average of percent HbCO of four capillary blood samples determined on an ABL700 Series blood-gas analyzer (Radiometer Medical, Copenhagen, Denmark) for both CO doses was obtained, and the change in percept HbCO was used to calculate Hb_{mass} (6). This was repeated again at the end of the investigation, approximately 24 h following the final ergometer time trial and after full restoration of body mass. Typical error of measurement for Hb_{mass} in our laboratory is 2.0%. Following CO-rebreathing, a full anthropometric profile was undertaken by an International Society of Kinanthropometry accredited level III anthropometrist using techniques previously described (22). The mean of duplicate or median of triplicate measurements was used to create a four-way fractionation of body mass, partitioning total body mass into fat mass, muscle mass, bone, and residual masses using the phantom model (9).

Treatments. During informal consultation, four athletes acknowledged they had never used acute weight loss before competition (i.e., "natural lightweights"). Consequently, they were assigned to the control group (CON). The

12 remaining athletes were all experienced in the use of acute weight loss techniques and were ranked according to previous 2000-m ergometer time trial performances. The ranking was used to assign athletes to two fitness matched groups: a partial recovery group ($REC_{partial}$) and a complete recovery group ($REC_{complete}$). Groups differed only in recovery goals implemented in the 24 h following the first two "at-weight" ergometer time trial. Volunteers assigned to $REC_{partial}$ were to restore no more than half of the initial 4% body mass loss. For athletes assigned to $REC_{complete}$, aggressive recovery strategies were used in the 12–16 h following at-weight ergometer trials with the aim of restoring at least three quarters of the 4% body mass loss experienced in the previous 24 h. To assist athletes in managing their body mass, bladder voided, nude mass was monitored on waking, following lunch, and again after the evening meal.

Athletes performed four 2000-m maximal rowing ergometer time trials (TR#1–TR#4); each test was separated by 48 h and undertaken at the same time of day. For all groups, no weight restrictions were enforced for the first ergometer trial (TR#1). Thereafter, however, athletes in the $REC_{partial}$ and $REC_{complete}$ groups were required to reduce their body mass by 4% over a 24-h period before their second ergometer trial (TR#2), and to again reach this body mass before the remaining two ergometer trials (TR#3 and TR#4). No limits were imposed on techniques used to induce the specified weight loss. Athletes were asked, however, to replicate weight loss techniques used before TR#2 for subsequent trials, and this was verified using food and training diaries.

Training load was prescribed before each of the four ergometer trials, simulating training habitually undertaken in the 24 h before racing at a regatta. Additional training, however, was allowed to assist athletes in achieving body mass goals. For those athletes assigned to CON, dietary intake was standardized in the 24 h before all ergometer tests; whatever was consumed in the 24 h before TR#1 was also consumed for all remaining trials. Similarly, dietary intake was standardized before the final three time trials among athletes allocated to $REC_{complete}$ and last two trials in $REC_{partial}$.

The 24-h urinary collections were taken the day before each ergometer trial. Urine volume was quantified using a calibrated digital scale with a resolution of ±2 g (Tanita, Tokyo, Japan) adjusted for polyethylene bottle weight. A small aliquot of urine was stored at −20°C for later analysis of adrenaline (ADR) and noradrenaline (NAD) using high-performance liquid chromatography (Waters Australia, Rydalmere, Australia).

Hydration status was monitored throughout the investigation via the measurement of urinary osmolality (OSM) using an Osmomat 030-D cryogenic osmometer (Gonotec, Berlin, Germany). Additionally, urinalysis of fresh samples was done for the presence of ketones using reagent strips (Ketostix, Bayer Diagnostics Manufacturing Ltd., New South Wales). Dietary intake was also monitored each day with the aid of food diaries that were analyzed by a qualified dietitian using the Foodworks dietary analysis program (version 3.02, Xyris Software, Brisbane, Australia).

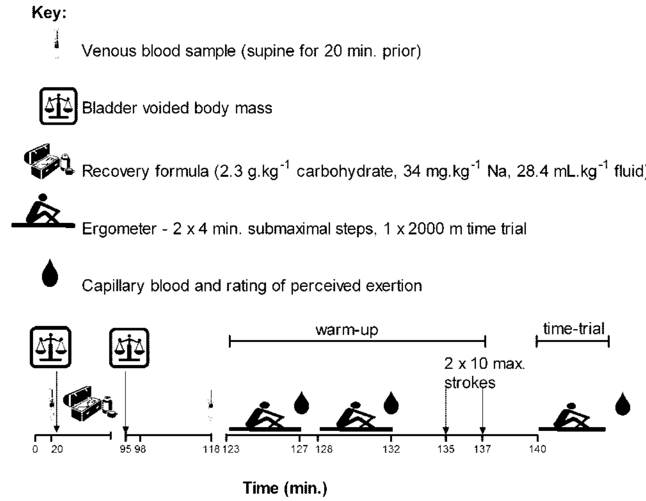

FIGURE 2—An overview of testing commitments undertaken during each 2000-m rowing ergometer time trial.

Experimental protocol. An overview of the testing schedule before each ergometer test is presented in Figure 2. Subjects arrived at the laboratory 140 min before the start of each 2000-m time trial. After lying supine for 20 min, 8 mL of blood was sampled via venipuncture without stasis from a superficial forearm vein using standard phlebotomy procedures. A total of 4 mL of blood was allocated to a serum separation tube and centrifuged at 4000 rpm for 5 min. The resultant serum was analyzed for cortisol (solid phase, competitive chemiluminescent enzyme immunoassay), β-hydroxy butyrate (β-HB) (kinetic enzymatic method), and total protein (TP) (colormetric assay); the former on an Immulite 1000 analyzer (DPC, Los Angeles, CA) and the latter two on a Hitachi 911 clinical chemistry analyzer (Roche Diagnostics, Mannheim, Germany). A further 4-mL aliquot of blood was mixed in a tube containing ethylene diaminetetraacetic acid. Hematocrit (Hct) and Hb concentration (Hb) were determined in triplicate using an automated flow cytometry hematology analyzer (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 (8). Changes in plasma volume were calculated and expressed relative to Hct and Hb concentrations averaged from the first 4 d of the investigation while volunteers were in a euhydrated state.

Following the first blood sample, bladder voided body mass was measured on a calibrated digital scale with a resolution of ±0.02 kg (A and D Co., Tokyo, Japan). Thereafter, subjects consumed a standard meal (toasted bread, Vegemite, Power Bar, Carboshotz, Gastrolyte, Gatorade, water). Food and fluid intake was prescribed following weigh-in for TR#2–TR#4 (2.3 $g \cdot kg^{-1}$ carbohydrate, 34 $mg \cdot kg^{-1}$ Na, and 28.4 $mL \cdot kg^{-1}$ fluid); fluid intake was *ad libitum* in TR#1, with equivalent choices made available. Fluid intake was prescribed to maximize plasma volume and fluid balance restoration (18). Fluid intake was quantified by weighing drink bottles before and after the recovery period. During this time, subjects remained within the controlled environment of the laboratory (21.1 ± 0.7°C, 29.0 ± 4.5% relative humidity).

Bladder voided body mass was again recorded before the warm-up. Thereafter, 2 mL of venous blood was again collected using previously described procedures and analyzed for Hct and Hb concentration. All urine produced during the recovery period was collected into 2-L polyethylene bottles and quantified using a calibrated digital scale with a resolution 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.

Mood was assessed toward the end of the recovery period using the Profile of Mood States-A (POMS-A) questionnaire (32). Additionally, immediately before each ergometer trial, athletes were asked to rate their "performance expectations" and "motivation" using a five-point Likert scale (1 = very poor, 5 = excellent).

Athletes initiated a standardized warm-up before each maximal 2000-m time trial, consisting of two submaximal workloads (50 and 65% of previously reported average 2000-m ergometer time trial power output), each of 4-min duration and separated by a 1-min passive recovery interval. After a further 3-min passive recovery, subjects were required to undertake 10 maximal race pace strokes, simulating the start of a race. This procedure was repeated 2 min later. Initiation of the maximal 2000-m time trial followed 3 min thereafter. Arterialized capillary blood was sampled immediately following the submaximal workloads and the 2000-m time trial, and analyzed without delay for pH in addition to glucose, bicarbonate (HCO^{3-}), and lactate (La^-) concentrations (ABL 725, Radiometer, Copenhagen, Denmark). The analyzer was calibrated daily in accordance with the manufacturer's specifications.

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

Total body water (TBW) was measured by the deuterium dilution technique on three separate occasions, approximately 24 h after each of the first three 2000-m ergometer trials, using procedures previously described (3). Briefly, each subject provided a small urine sample and then drank a 10% solution of deuterium (in the form of water (^2H_2O)) based on their body mass (0.5 g·kg^{-1}). The dose consumed was recorded to two decimal places of a gram. A further urine sample was collected 4 h later. The enrichment of the predose urine sample, the postdose urine sample, local tap water, and the dose given were measured using an isotope ratio mass spectrometer (Hydra, Europa Scientific, Crewe, UK) that was calibrated against Vienna Standard mean Ocean Water and International Atomic Energy Agency enriched standards. TBW was calculated in accordance with the recommendations of Schoeller et al. (26), who advocate a 3% correction factor for the exchange of ^2H_2O with labile hydrogen of protein and other body constituents. TBW measurements in the laboratory used for analysis have a TE of 0.88 L.

TABLE 1. Physiologic and anthropometric characteristics of volunteers (N = 16).

Variable	Control (N = 4)	$REC_{partial}$ (N = 6)	$REC_{complete}$ (N = 6)
Age (yr)	17.9 ± 0.1	21.0 ± 1.9	22.3 ± 3.8
Stretch stature (cm)	180.1 ± 3.4	183.4 ± 2.3	181.4 ± 3.2
Mass (kg)	72.3 ± 4.6	76.7 ± 1.2	75.0 ± 3.2
Fat (%)	8.5 ± 0.9	8.6 ± 1.2	8.7 ± 0.8
$\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	61.3 ± 4.7	60.5 ± 3.0	63.4 ± 3.2

Values are means ± SD. $\dot{V}O_{2peak}$, peak O_2 uptake. $REC_{partial}$, restoring less than half of the body mass lost in the subsequent 12–16 h; $REC_{complete}$, restoring at least three fourths of the body mass lost in the subsequent 12–16 h.

Statistical analysis. Performance, biochemical, and other parameters from the four ergometer trials were compared using general linear mixed modeling with group (CON, $REC_{partial}$, $REC_{complete}$) and trial order (TR#1–TR#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. Weight gain in the 12–16 h following racing was included as a covariate in the model to assess the influence of body mass management on subsequent performance. Comparisons between differences in performance for specific pairs of trials were made using *t*-tests for independent samples, without the assumption of equal variances. Standardized residuals were calculated for each observation. A value >3.0 was considered to be extreme or an "outlier" and all such observations were removed from analysis one at a time. The general linear modeling analysis was conducted using Statistica software for Windows (version 7.0, Statsoft, Tulsa, OK). Significance was accepted at $P < 0.05$. All data are reported as means ± SD, unless otherwise specified.

RESULTS

Characteristics of all 16 athletes who completed the investigation are presented in Table 1. Groups did not differ significantly in physiological or anthropometric traits.

Body mass. Percent body mass loss in the 24 h before TR#2 was greater for $REC_{partial}$ and $REC_{complete}$ compared with CON (CON, 0.6 ± 1.2%; $REC_{complete}$, 4.7 ± 0.2%; $REC_{partial}$, 5.1 ± 0.7%; $P < 0.001$) but was not different between interventions ($P = 0.700$). In contrast, body mass loss in the 24 h before TR#3 (CON, 0.4 ± 0.8%; $REC_{complete}$, 2.9 ± 0.4%; $REC_{partial}$, 1.8 ± 0.4%) was greater in $REC_{complete}$ when compared with both $REC_{partial}$ ($P = 0.007$) and CON ($P < 0.001$). A similar trend was evident before TR#4 (CON, 0.1 ± 0.5%; $REC_{complete}$, 2.7 ± 0.6%; $REC_{partial}$, 2.0 ± 0.7%; $P < 0.001$). Body mass gain in the 12–16 h following TR#2 (CON, 0.3 ± 0.4%; $REC_{complete}$, 2.9 ± 0.6%; $REC_{partial}$, 1.8 ± 0.8%; $P < 0.05$) was greater in $REC_{complete}$ than both $REC_{partial}$ ($P = 0.033$) and CON ($P <$ 0.001). A similar trend occurred following TR#3 (CON, 0.6 ±

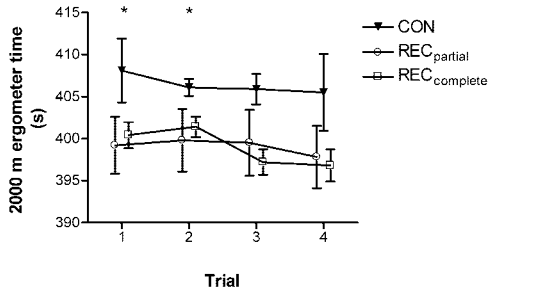

FIGURE 3—The impact of acute weight loss (4% over 24 h) and different body mass management strategies (partial ($REC_{partial}$) vs complete ($REC_{complete}$) recovery) on 2000-m ergometer time trial performance throughout the simulated regatta. Values are means ± SD for 16 volunteers. Main effect of trial (P = 0.027) * Significantly different from Trial 4 (P = 0.03).

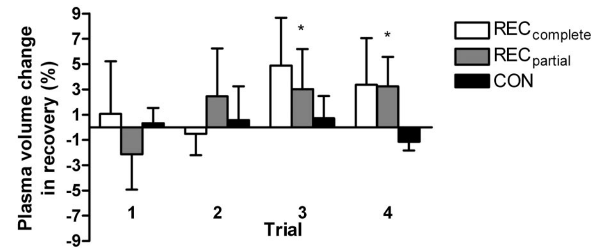

FIGURE 5—Acute plasma volume response to nutrient intake between weigh-in and racing throughout the simulated regatta. Values are means ± SD for 16 subjects. Main effect of trial (P = 0.011). Group-by-trial interaction (P = 0.036). * Significantly different from Trial 1 (P < 0.05).

1.1%; $REC_{complete}$, 2.8 ± 0.7%; $REC_{partial}$, 1.9 ± 0.3%; $P <$ 0.001).

Ergometer performance. Results of the four 2000-m ergometer time trials are summarized in Figure 3. A main effect of trial was evident (P = 0.027), with TR#4 faster than both TR#1 (P = 0.032) and TR#2 (P = 0.034). No main effect of group was observed (P = 0.139), nor an interaction between group and trial (P = 0.347). When two outliers (standardized residuals > 3.0) were omitted (Fig. 4), however, the group-by-trial interaction approached significance (P = 0.069). Aside from the performance outcome, outlying trials were unremarkable in their physiological response.

When data from $REC_{partial}$ and $REC_{complete}$ were combined for TR#1 and TR#2, respectively, acute weight loss increased 2000-m ergometer time by 2.89 s (95% CI −0.79 to 6.57, P = 0.110) when compared with the CON response. If the same two outliers are removed, the effect of acute weight loss for the first time increases to 3.00 s (95% CI −0.40 to 6.40, P = 0.070). The impact of acute weight loss was reduced or eliminated for the second at-weight trial (TR#3) for both $REC_{partial}$ (mean 2.50, 95% CI −4.28 to 9.29 s, P = 0.412) and $REC_{complete}$ (mean −1.02, 95% CI −7.28 to 5.25 s, P = 0.676); a positive value indicates a slower time and a negative value a faster time. This effect continued during TR#4 for both $REC_{partial}$ (mean 1.28, 95% CI −5.22 to 7.77 s, P = 0.635) and $REC_{complete}$ (mean −0.94, 95% CI −4.40 to 2.52 s, P = 0.530).

The influence of weight gain in the 12–16 h following racing on subsequent performance was small but favorable. For every kilogram gain in body mass, 2000-m ergometer time decreased by −0.84 s (95% CI −1.72 to 0.05 s, P = 0.064).

Plasma volume. Whereas plasma volume at weigh-in did not differ between groups (P = 0.864), a main effect of trial was evident (TR#1, 0.39 ± 2.47%; TR#2, 2.82 ± 3.64%; TR#3, 0.75 ± 3.67%; TR#4, 1.08 ± 21.1%; P = 0.020); plasma volume being compromised before TR#2 (P = 0.041) but not before TR#3 (P = 0.993) or TR#4 (P = 0.930) when compared with TR#1.

Acute plasma volume restoration in response to nutrient intake following weigh-in did not differ between groups (P = 0.394). A main effect of trial was evident (P = 0.011), however, with plasma volume restoration greater in TR#3 (P = 0.003) and TR#4 (P = 0.020) compared with TR#1. The interaction between main effects (P = 0.036) confirmed that this effect was most pronounced for $REC_{partial}$ (Fig. 5).

Hydration status. Urinary OSM increased in response to acute weight loss undertaken in the 24 h before TR#2, exceeding levels indicative of hypohydration (21) among both $REC_{partial}$ and $REC_{complete}$ (Fig. 6). This state of hypohydration was maintained throughout the remainder of

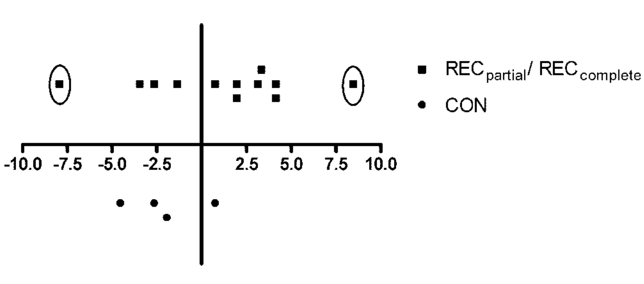

FIGURE 4—The impact of acute weight loss for the first time (TR#2) on 2000-m time trial performance for $REC_{partial}$ and $REC_{complete}$. Performance variance between TR#1 and TR#2 for CON are included for comparison. Negative values indicate a faster time, positive values a slower time. Circled values highlight individual performances considered outliers, as identified by standardized residuals > 3.0.

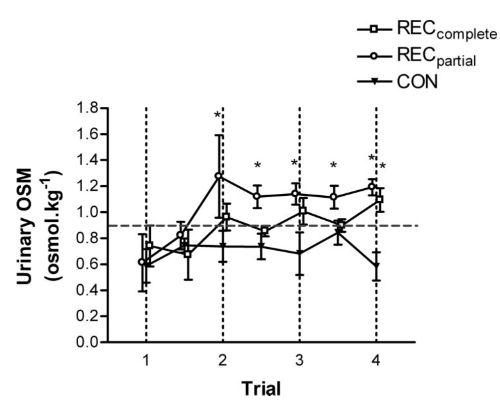

FIGURE 6—Hydration status throughout the simulated regatta as inferred from on-waking urine sample osmolality (OSM). A urinary OSM ≥0.90 osmol·kg^{-1} was considered indicative of hypohydration. Values are means ± SD for 16 subjects across 7 d of the investigation. * Significantly different from Trial 1 (P < 0.05). Main effect of group (P < 0.001). Main effect of time (P < 0.001). Group-by-trial interaction (P < 0.001).

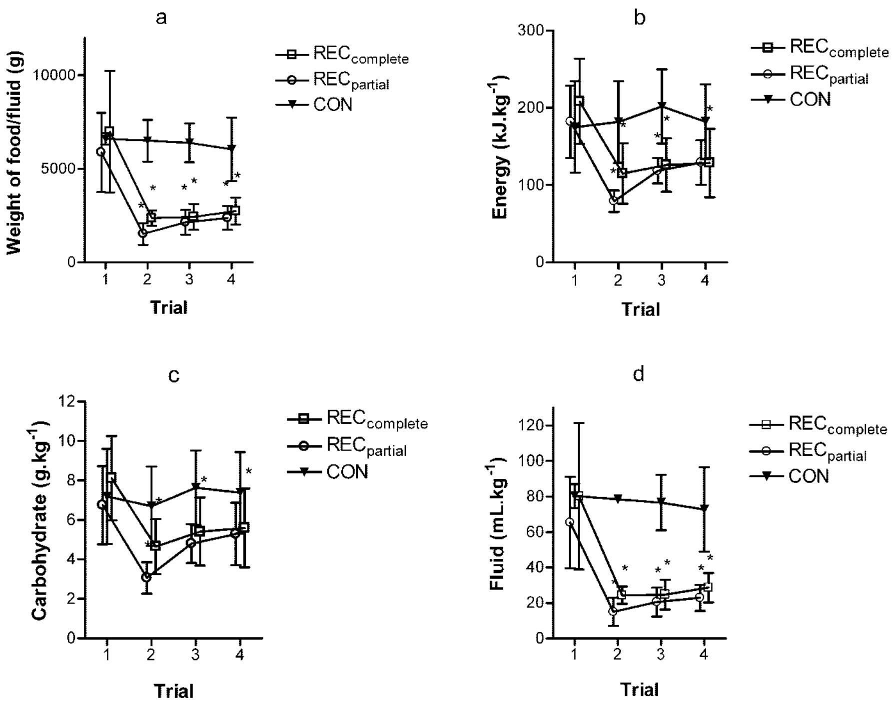

FIGURE 7—Nutrient intake, including the weight (a), energy (b), carbohydrate (c), and fluid (d) in the 24 h before ergometer trials. Values are means ± SD for 16 subjects. *Significantly different from Trial 1 ($P < 0.05$).

the simulated regatta for $REC_{partial}$ but not for $REC_{complete}$. At no stage did urinary OSM indicate a state of hypohydration among athletes allocated to CON.

Hormonal response. Although nicotinic acid dehydrogenase (NAD) levels were lower before TR#4 when compared with the other trials (TR#1, 385.8 ± 98.0 nmol·24 h^{-1}; TR#2, 371.3 ± 96.6 nmol·24 h^{-1}; TR#3, 367.5 ± 90.8 nmol·24 h^{-1}; TR#2, 313.8 ± 83.1 nmol·24 h^{-1}; $P = 0.002$), urinary concentrations did not differ between groups ($P = 0.720$). No interaction was evident between group and trial ($P = 0.11$). A similar response occurred for ADR. On-waking cortisol values did not change throughout the investigation ($P = 0.723$), nor did they differ between groups ($P = 0.125$). No interaction was evident between group and trial for cortisol ($P = 0.980$).

Total body water. Total body water levels were relatively stable throughout the simulated regatta with no main effects of group (CON, 48.0 ± 2.6 L; $REC_{complete}$, 49.1 ± 2.1 L; $REC_{partial}$, 49.9 ± 1.5 L; $P = 0.378$) or trial (TR#1, 49.7 ± 2.1 L; TR#2, 48.6 ± 1.8 L; TR#3, 49.6 ± 1.7 L; $P = 0.182$). Some evidence, however, indicated an interaction between group and trial ($P = 0.057$) with TBW following TR#2 significantly lower than TR#1 for $REC_{partial}$ (TR#1, 50.7 ± 1.2 L; TR#2, 48.7 ± 1.6 L; TR#3, 50.3 ± 1.0 L; $P = 0.014$).

Dietary intake. Acute weight loss was partially achieved via a reduction in the total volume of food and fluid intake in the 24 h before TR#2 when compared with TR#1 (Fig. 7). Consequently, 24-h energy, carbohydrate, and fluid intakes were also reduced. The reduction in nutrient intake for $REC_{complete}$ and $REC_{partial}$ in the day before TR#3 and TR#4 was similar to that initially achieved in the 24 h before TR#2. Nutrient intake did not differ between $REC_{complete}$ and $REC_{partial}$ in the 24 h before ergometer trials ($P > 0.05$).

Although nutrient intake in the 12–16 h following TR#1 was greater than that consumed following TR#2 and TR#3, few differences existed between groups (Fig. 8). Fluid intake ($P = 0.050$) and the weight of food and fluid consumed ($P = 0.050$) were lower in $REC_{partial}$ than CON. Nutrient intake did not differ between $REC_{complete}$ and $REC_{partial}$ ($P > 0.05$). Retention of ingested fluid between weigh-in and racing was greater when volunteers were at weight (TR#1, 61.6 ± 16.6%; TR#2, 82.9 ± 21.7%; TR#3, 84.2 ± 19.8%; TR#4, 83.0 ± 21.1%; $P < 0.001$), exceeding 92% for $REC_{partial}$ and $REC_{complete}$ during TR#2–TR#4.

Training. The total volume of training undertaken in the 24 h before time trials was greater before TR#2 compared with TR#1 ($P = 0.009$), but not when compared with the other trials (TR#1, 62.6 ± 7.2 min; TR#2, 85.1 ± 42.1 min; TR#3, 69.6 ± 14.0 min; TR#4, 69.6 ± 12.9 min; $P > 0.05$). Training load, however, did not differ between groups ($P = 0.141$).

Other variables.

Serum total protein concentration at weigh-in did not differ between groups ($P = 0.699$). A main effect of trial was apparent, however, (TR#1, 68.2 ± 2.9 g·L^{-1}; TR#2, 70.9 ± 3.4 g·L^{-1}; TR#3, 70.0 ± 2.3 g·L^{-1}; TR#4, 70.1 ± 2.3 g·L^{-1}; $P = 0.001$), as was an interaction between main effects ($P = 0.027$). Total protein concentration was higher for $REC_{complete}$ before TR#2 ($P = 0.001$) and TR#3 ($P = 0.043$), but not TR#4 ($P = 0.212$) when compared with TR#1. A similar response was evident for $REC_{partial}$.

No main effects or interactions between group and trial were evident for performance expectation, motivation, mood state, RPE, HR, blood metabolites, or indices of acid-base status ($P > 0.05$); data not presented here. Urinary

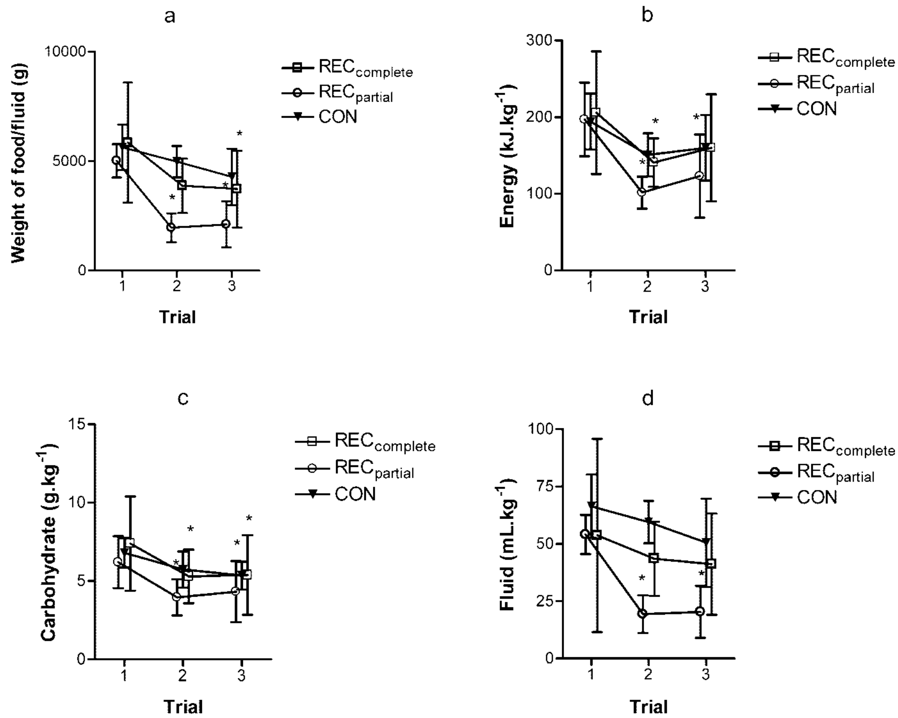

FIGURE 8—Nutrient intake, including the weight (a), energy (b), carbohydrate (c), and fluid (d) over the remainder of the day (12–16 h) following ergometer trials. Values are means ± SD for 16 subjects. * Significantly different from Trial 1 ($P < 0.05$).

ketones were absent throughout the simulated regatta. Hb mass did not vary throughout the investigation ($P = 0.136$).

DISCUSSION

The primary finding of this investigation is that the performance implications of acute weight loss within the range of 4–5% on rowing ergometer performance are small (3.0 s), but could still influence competition outcomes. These effects are further reduced or eliminated when repeated over several days; at-weight trials did not differ statistically from unrestricted trials. This is consistent with our previous findings suggesting that, when aggressive nutritional recovery strategies are used in the 2-h recovery period between weigh-in and racing, the implications of acute weight loss on a single performance effort are small (28). We have also shown that aggressive nutrient intake in the 12–16 h following racing (as inferred by body mass gain) further reduces the influence of acute weight loss on subsequent performance when athletes race every 48 h.

To our knowledge, this is the first investigation that has assessed the impact of acute weight loss on repeat 2000-m rowing ergometer time trial performance when weight loss strategies are maintained for several days during a simulated regatta. We hypothesized that the continued use of acute weight loss strategies throughout the simulated regatta would increase stress on the body and result in progressively greater performance decrements. In contrast, the influence of acute weight loss on performance appeared to reduce with subsequent trials, as did the stress hormone response, despite the athletes reaching similar degrees of hypohydration, as inferred from urinary OSM, before at-weight trials. This response was evident in addition to the order or learning effect observed. Although contrary to our initial hypothesis, a similar response has been observed in athletes participating in other weight category sports (29).

Among boxers, repeat simulated punching ergometer performance tended to be compromised for bouts separated by 48 h that followed a 3% body mass reduction induced via food and fluid restriction when compared with unrestricted bouts (29). Performance for the second body mass restricted trial improved when compared with the first, however, despite continued body mass loss, perhaps in part because of the reported order effect.

The present findings suggest that athletes may experience some degree of adaptation to the physiological state that results from acute weight loss. Indeed, in the present investigation, plasma volume was conserved before TR#3, despite athletes in the intervention groups decreasing their body mass by 2–3% in the previous 24 h and presenting with urinary OSM suggesting a state of hypohydration. An increase in total circulating protein has been proposed as a possible adaptation to hypohydration, increasing oncotic pressure and aiding redistribution of body water into the intravascular space (25). Whereas acute weight loss increased TP concentrations among athletes in $REC_{partial}$ and $REC_{complete}$, this response was acute and likely caused by the hemoconcentration accompanying hypohydration rather than a chronic adaptation.

Alternatively, the observed plasma volume response may merely reflect the method of monitoring plasma volume shifts in the present investigation. For the Hct–Hb method to be valid, the three following preconditions need to be fulfilled: (a) the number of circulating erythrocytes must be unchanged, (b) the sampling site must remain at the same level relative to the heart, and (c) the *F* cell ratio must remain unchanged (17). Although Hb mass remained stable throughout the investigation and sampling site was standardized, we were unable to monitor the F cell ratio. Acute hypohydration, however, does not appear to influence the F cell ratio (7), suggesting preconditions were met for the monitoring of plasma volume shifts via the Hct–Hb method.

If the response observed in the present investigation can be confirmed, it may alter guidelines for acute body mass management. Current guidelines encourage athletes to limit acute weight loss strategies to the final hours before competition to minimize the impact on training quality (33). In contrast, the present data suggest that optimal performances occurred at least 72 h following initiation of acute weight loss strategies. More research is needed to identify the optimal time frame in which to undertake acute weight loss before competition.

Most variables considered in the present investigation were not influenced by the subtle differences in weight loss or gain and nutrient intake between $REC_{complete}$ and $REC_{partial}$. Individual performances, however, tended to be better for $REC_{complete}$ in the later races of the simulated regatta, despite greater weight loss in the 24 h before time trials. We are unable, however, to offer conclusive reasoning why this response was evident, but propose that it may relate to subtle differences in training load or nutrient intake between $REC_{complete}$ and $REC_{partial}$, both before and following ergometer trials.

We initially hypothesized that, despite the need to repeatedly make weight, sustained use of aggressive nutritional recovery strategies would result in less physiological stress on the body and result in more favorable performance responses. Although indices of physiological stress were not different between interventions, the more aggressive recovery strategy resulted in more favorable performance responses. This alone provides sufficient justification for an athlete who has to "make weight" to achieve specified body mass limits and to be aggressive with nutritional recovery strategies, both before and after racing, especially if competing in a multiday regatta involving races every 48 h.

Our data contrast with the marked decrease in performance reported by Burge and associates (5) when only water was provided in recovery following acute weight loss (5.2%). Because the degree of hypohydration and time frame of body mass reduction were similar between the present study and the investigation by Burge et al. (5), maintenance of time trial performance in the present study may reflect differences in nutrient intake practices (e.g., carbohydrate and sodium) following weigh-in. Until a direct comparison between nutritional recovery formulas is undertaken, this hypothesis remains purely speculative.

Acute strategies used to make weight can reduce muscle glycogen stores by 30–50% (5,31). Although a 2-h period of recovery is insufficient for full restoration of muscle glycogen reserves (16,31), it is unlikely that carbohydrate reserves limit performance during a 5- to 7-min event (11), even when increased muscle glycogen utilization associated with hypohydration is considered (14). Although muscle glycogen stores are unlikely to limit rowing performance in a single 2000-m race, even after a 24-h period of dietary restriction to assist with acute weight loss (5), the impact of racing for 5–7 d, as occurs during a rowing regatta, on energy reserves remains unclear. Serum β-HB, an index of carbohydrate availability (30), did remain low however, and urinary ketones were absent throughout the investigation, suggesting carbohydrate reserves may not have been challenged.

The ergogenic potential of preexercise carbohydrate intake on endurance performance has been well researched (13,15). The influence of carbohydrate ingestion in the few hours before brief high intensity exercise has been less widely investigated, however, especially among athletes in weight category sports who routinely restrict energy intake before competition. In wrestlers who had an 8% loss of body mass in 4 d, Houston et al. (16) reported no benefit of nutrient intake (unspecified) during a 3-h recovery period in restoring indices of both aerobic and anaerobic performance. In contrast, refeeding wrestlers a high (75%) carbohydrate beverage after 3 d of energy restriction (75 $kJ \cdot kg^{-1}$) that resulted in a 3.3% body mass loss restored anaerobic performance after 5 h (23). An energy-matched, moderate (47%) carbohydrate intake did not restore performance in the test designed to replicate the demands of wrestling competition (8 $\times$ 15 s of maximal arm cranking with 20 s of active recovery between efforts), suggesting that a high carbohydrate intake following weigh-in may be important in maintaining performance. Carbohydrate intake following weigh-in in the present investigation may explain, at least in part, the enhanced performance response compared with that found by Burge et al. (5).

Whereas the volume of fluid ingested following weigh-in in the present study was similar to that provided by Burge et al. (5), dietary sodium intake differed between investigations. Unless the sodium content of a beverage is sufficiently high, much of the ingested fluid will merely contribute to urinary output and delay restoration of fluid balance (19). A sodium intake within the range of 50–60 $mmol \cdot L^{-1}$ is recommended for optimal rehydration (20), substantially greater than that provided by Burge et al. (5). We believe that the sodium contained in our recovery formula most likely assisted in restoration of fluid balance and subsequently minimized the impact of acute weight loss on performance.

We acknowledge the lack of randomization of "natural lightweights" between intervention and control groups. Given the amount of body mass loss required and the time frame allocated to this, however, it was deemed inappropriate to enforce acute weight loss strategies on athletes with no experience in the use of such techniques. Consequently, all comparisons between intervention and control groups should be considered observational.

In summary, the present investigation has shown that acute weight loss within the range of 4–5%, when combined with aggressive nutrient intake following weigh-in, has only a small impact on rowing ergometer performance and that this diminishes when acute weight loss is repeated over several days. Furthermore, more aggressive nutritional recovery strategies following racing appear to minimize or eliminate the impact of acute weight loss on subsequent performance. The impact of variation in specific nutrient intake in the recovery period, both before and after racing, warrants further investigation, as does the optimal time frame for acute weight loss before competition.

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