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Title: The Effect of Water Loading on Acute Weight Loss Following Fluid Restriction in Combat Sports Athletes

Submission Type: Original research

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Abstract

Novel methods of acute weight loss practiced by combat sport athletes include 'water loading'; the consumption of large fluid volumes for several days prior to restriction. We examined claims this technique increases total body water losses, while also assessing the risk of hyponatremia. Male athletes were separated into control (CON, n=10) and water loading (WL, n=11) groups and fed a standardised energy-matched diet for 6 days. Day 1-3 fluid intake was 40 $mL \cdot kg^{-1}$ and 100 $mL \cdot kg^{-1}$ for CON and WL, respectively with both groups consuming 15 $mL \cdot kg^{-1}$on Day 4 and following the same rehydration protocol on Days 5-6. We tracked body mass (BM), urine sodium, specific gravity (USG) and volume, training-related sweat losses and blood concentrations of renal hormones and urea and electrolytes (U+Es) throughout. Physical performance was assessed pre-and post-intervention. Following fluid restriction, there were substantial differences between groups in the ratio of fluid input/output (39%, $p < 0.01$, ES=1.2) and BM loss (0.6%BM, p=0.02, ES=0.82). Changes in USG, U+Es and renal hormones occurred over time ($p < 0.05$), with an interaction of time and intervention on blood sodium, potassium, chloride, urea, creatinine, USG and vasopressin ($p < 0.05$). Measurements of U+E remained within reference ranges and no differences in physical performance were detected over time or between groups. Water loading appears to be a safe and effective method of acute BM loss under the conditions of this study. Vasopressin regulated changes in aquaporin channels may potentially partially explain the mechanism of increased body water loss with water loading.

Introduction

Combat sport athletes commonly manipulate body mass (BM) prior to competition, attempting to gain real or perceived advantages by competing in weight divisions lighter than their day-to-day BM (Franchini, Brito et al. 2012). Aside from chronic fat mass reductions, athletes acutely reduce BM pre-weigh-in. Common and effective methods include active and passive sweating, diuretics, fluid and sodium restriction (reducing body water) and reduction of gut contents via laxative use, fasting, reducing food volume and reduced carbohydrate and/or fibre intake (Franchini, Brito et al. 2012, Reale, Slater et al. 2016).

'Water loading' is a recent addition to these methods; purportedly decreasing BM via increased urine production (Reale, Slater et al. 2016). This technique involves consuming large fluid volumes (i.e. 7-10+ L/d) for several days followed by fluid restriction; allegedly manipulating renal hormones and urine output, thus increasing fluid losses relative to fluid restriction following ad-libitum fluid intake (Reale, Slater et al. 2016). Anecdotes exist among body builders and power lifters as well as in combat sports. Two recent investigations have confirmed the use of water loading in UK combat sport athletes (Crighton, Close et al. 2015, Matthews and Nicholas 2016) and data from this group indicates >40% of Australian Olympic combat sport athletes have used this method at some stage (Reale et al. 2017). However, these athletes commonly manipulate sodium and other nutrients alongside fluid intake while 'making weight', thus confounding the ability of anecdotal 'evidence' to provide insights into its efficacy. Given the prevalence of use, the lack of scientific investigation and the potential risk of hyponatremia associated with consuming large volumes of fluid, further research is warranted. Accordingly, the aim of this study was to examine water loading in a controlled setting, investigating the efficacy, safety and potential underlying mechanisms.

Methods

Overview

This study was conducted at the Australian Institute of Sport as a parallel intervention. Subjects were separated into a control (CON) or intervention group (water loading (WL)). The Human Research Ethics Committee of the University of the Sunshine Coast approved the study. Subjects provided written informed consent prior to participation. The project took place over eight days: two 'pre' testing days prior to intervention (Day -1 and 0), six intervention days (Day 1-6) and post' testing (Day 6). See Figure 1 for study overview. Figure 2 summarises timelines and details of key data collection points.

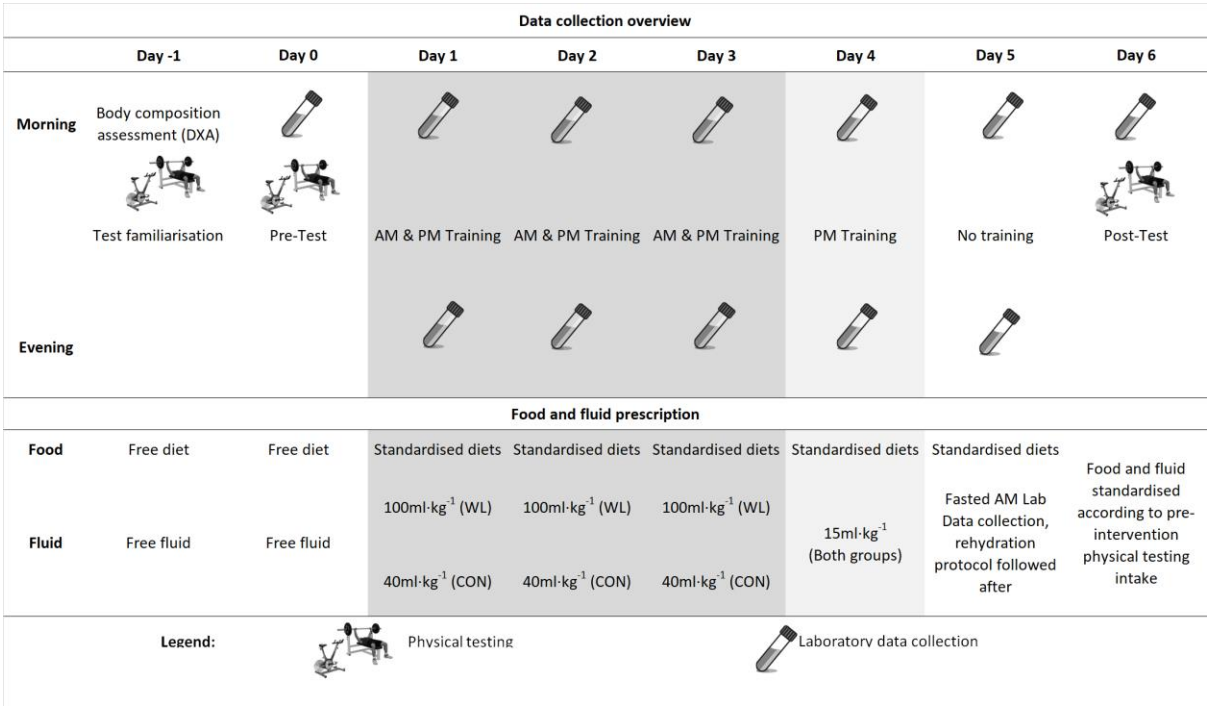

Figure 1. Study outline: DXA- dual energy x-ray absorptiometry, WL – water loading group, CON – control group

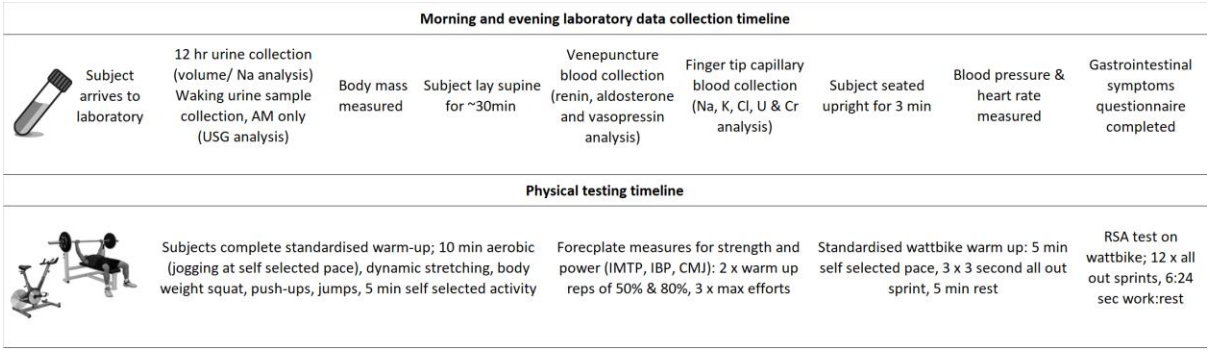

Figure 2. Laboratory data collection and Physical testing undertaken on days -1 and 0 (pre-intervention) and day 6 (post-intervention): USG – urine specific gravity, Na – sodium, K – potassium, Cl – chloride, U – urea, Cr – creatinine, IMTP – isometric mid thig pull, IBP – isometric bench press, CMJ – counter movement jump, RSA – repeated sprint ability

Subjects

Subjects were 22 male grapplers (jiu-jitsu, judo and wrestling athletes) with at least 4 years competition experience, currently training ≥8 hours per week. One subject withdrew from the study prior to completion for reasons unrelated to the intervention, thus 21 were included in the final analysis. Subjects were stratified into blocks, matching for BM and then simple randomisation was used to place subject into CON (n = 10; 77.2±8.7kg, 178.9±5.7cm, 15.1±4.2% body fat, 24.9±4.0years) and WL groups (n = 11; 77.8±8.0kg, 176.2±6.4cm, 15.5±2.9% body fat, 28.3±3.5years). All subjects reported having lost weight in order to make weight in the past with the four indicating previous water loading experience allocated evenly between the WL and CON groups.

Body composition assessment

On Day -1, body composition was assessed by a trained technician, using dual energy x-ray absorptiometry (iDEXA GE Healthcare, Madison, WI) according to the standardised protocol developed at the Australian Institute of Sport (Nana, Slater et al. 2016).

Physical performance testing

Physical performance measures included maximal isometric strength, lower body power and repeated sprint ability (RSA) tests (Figure 2). Subjects performed familiarisation sessions on Day -1, with pre-intervention testing undertaken on Day 0 and replicated on Day 6. Testing occurred at the same time daily, following morning blood collection and a standardised breakfast. It was conducted by the same scientists in a noise sensitive laboratory. Instructions to give maximal effort were provided prior to, but not during testing.

Testing consisted of a standardised warm-up followed by 3 maximal efforts of; countermovement jump (CMJ), isometric mid-thigh pull and isometric bench press conducted on a force plate. Testing was completed according to the methodology used by Halperin et al (Halperin, Williams et al. 2016). Subjects then performed the RSA test after a cycle ergometer warm-up (Wattbike Ltd, Nottingham, UK). Handlebar/saddle position were self-selected and replicated between trials.

Diets

Standardised diets during the intervention provided an energy content of 125 kJ·kg FFM^{-1} to meet resting requirements, plus additional energy accounting for exercise induced thermogenesis (estimated based on BM and training duration (Montoye 2000)). This represents a mild energy restriction of ˜14-18 kJ·kg FFM^{-1}, maintaining moderate energy availability (Loucks 2004): protein: 2.2-2.5g·kg FFM^{-1}, carbohydrate: 5-6g·kg BM^{-1} and fat: 1-2 g·kg BM^{-1}. Sodium prescription was ~300mg·Mj^{-1} and fibre 10-13 g, representing a reduced residue diet recommended to athletes “making weight” as a means to reduce the weight of gut content/ overall BM. Main meals were consumed in the presence of researchers and subjects verified all snacks were consumed as prescribed. No differences existed in dietary intake between the groups.

Fluid prescription

During Days 1-3 of the intervention, fluid intake (tap water) was clamped at 100 mL·kg^{-1} BM for WL and 40 mL·kg^{-1} BM for CON. On Day 4, both groups restricted intake to 15 ml·kg^{-1} BM. No fluid was consumed on Day 5 until after the morning laboratory data collection. Both groups followed the same re-hydration protocol after this point; fluid intake of 30 mL·kg^{-1} BM + 150% of the BM loss incurred during the fluid restriction period (morning of Day 4 until post Day 5 data collection). Daily fluid targets were divided into an hourly volume to be consumed during waking hours.

Training

The training schedule aimed to replicate combat sport athletes' competition preparation, consisting of two training sessions daily during Days 1-3, one session on Day 4 and no training on Day 5. All subject completing the same training sessions throughout the study.

Laboratory data collection

The standardised protocol for laboratory data collection (Figure 2) involved morning testing following an overnight fast (no food or fluid) at 7 am (Day -1 to Day 6) and evening testing at 6 pm (Day 1-5). No food was consumed for ˜3 h and no fluid for ˜1 h prior to the 6pm testing. Each time point involved the collection of urine, venous and capillary blood, BM measurements, blood pressure, heart rate, and completion of a gastro intestinal (GI) symptoms questionnaire.

Body mass

BM measurements were conducted after bladder voiding using the BWB800S digital BM scales (Tanita, Tokyo, Japan). In addition to laboratory data collection time points, naked BM was measured before and after training sessions and used alongside urine output and fluid intake to estimate sweat losses (i.e. sweat loss = BM change + fluid intake – urine output).

Urine collection and analysis

Waking urine samples were analysed for specific gravity (USG) using the UG-1 digital refractometer (ATAGO, Tokyo, Japan). Twenty-four-hour urine collection was undertaken Days 1-6 in 2 collection periods daily. Sodium concentration was determined using the B-722 Laqua twin (Horiba, Kyoto, Japan).

Blood collection and hormone analysis

Phlebotomists collected venous blood (3 x EDTA tubes, 1 x serum separated tube (SST), totalling 26.5 ml per collection point) following ~30 min supine rest for renal hormones measurement (vasopressin, renin and aldosterone). Samples were mixed and allowed to clot (when appropriate) before being centrifuged and frozen at -80^{o} until analysis. Vasopressin concentrations were determination using the Buhlmann Vasopressin double-antibody radioimmunoassay method (Buhlmann Laboratories, Schönenbuch, Switzerland) based on the method of Glick and Kagan (Glick and Kagan 1979). Inter-assay CV and intra-assay CV for vasopressin determination are between 1.8–3.5% and 5.6–9.5% respectively.2.3-9.5% 6.8-13.0%. Aldosterone and renin concentrations were determined by a LIAISON Analyzer (DiaSorin Inc, Via Crescentino, Italy), using a competitive assay (sheep monoclonal antibody) and a sandwich chemiluminescence immunoassay (specific mouse monoclonal antibody)

respectively (Derkx, De Bruin et al. 1996, Cartledge and Lawson 2000). Inter-assay and intra-assay coefficients of variability (CV) for renin determination are between 2.1–2.4% and 6.8–7.3%, respectively. Inter-assay CV and intra-assay CV for aldosterone determination are between 1.8–3.5% and 5.6–9.5% respectively.

Fingertip capillary blood (95uL) was then collected to analyse concentrations of sodium, potassium, chloride, urea and creatinine using the i-STAT Point of Care device and chem8+ cartridges (Abbott Laboratories, Abbott Pak, IL, USA). Inter-assay CV and intra-assay CV for all blood chemistry measures are ≤3.5% except for urea which at low concentration (1.7 mmol/L) is ≤11.2%.

Heart rate and blood pressure

Following blood collection, resting blood pressure and heart rate measurements were taken using the HEM-7325 automatic blood pressure monitor (Omron Healthcare, Kyoto, Japan).

Gastrointestinal symptoms

Three questions relating to GI symptoms associated with fluid intake from a validated questionnaire (Bovenschen, Janssen et al. 2006) were administered. Subjects rated nausea, bloating, and loss of appetite using a 1-7 Likert scale.

Data analysis

Conventional statistical analysis was used to calculate mean ± SD for each variable. Where appropriate, results were analysed and reported as absolute and/or delta scores. D'Agostino-Pearson and Levene's tests were used to assess normality and homogeneity respectively, where data were not

distributed normally, square root transformations were performed in order to achieve normality. Repeated measures two-way-ANOVAs with Bonferroni post-hoc tests were used to compare between groups and across time using the PRISM v 6.0 statistical analysis package (GraphPad Software, San Diego, California, USA). When data were transformed to achieve normality, statistical analyses were completed on the transformed data, with back transformed data being displayed in tables and figures for ease of visualisation. Significance was set at $p<0.05$. Additionally, 95% confidence intervals (CI95%) and Cohen d effect sizes (ES) were reported when appropriate. Magnitudes of ES were classified as trivial (0–0.19), small (0.20–0.49), medium (0.50–0.79) and large (≥0.80) (Cohen 1992).

Results

Body mass

BM changes are displayed in Figure 3. Time had a significant effect on cumulative and day-to-day BM changes and an interaction between time and fluid intake existed. Within both groups, a significant cumulative change in BM change across each successive day existed, with the exception of Day 3 to Day 4 ($P < 0.05$). Rehydration returned BM at Day 6 to levels equivalent to Days 3 and 4.

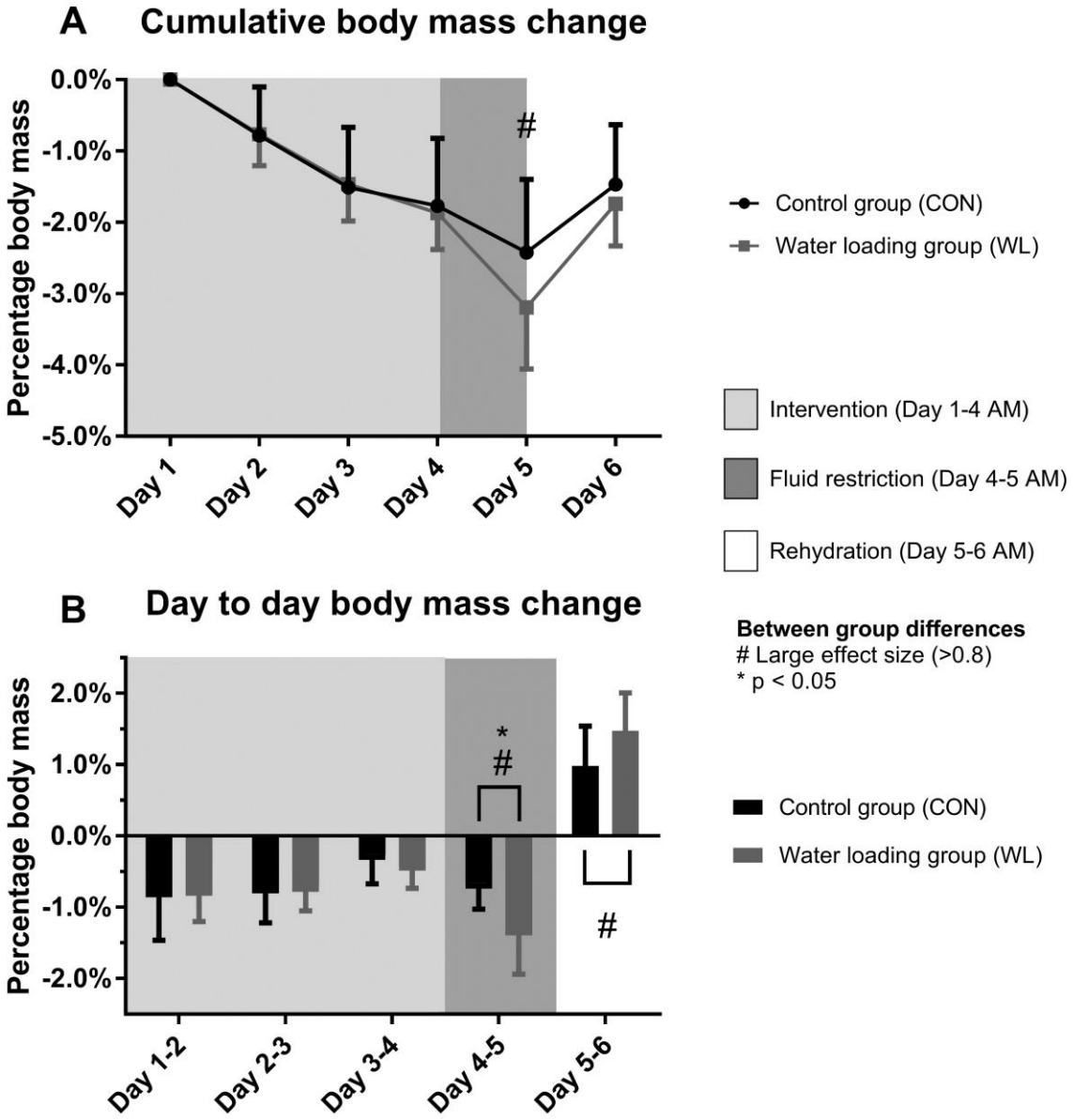

Figure 3. Changes in body mass across the 6-day intervention. Cumulative body mass (BM) change between groups, expressed as percentage of BM ±SD normalised to day 1 (A). Day to day BM change, expressed as percentage of BM ±SD (B). Main effect of time on cumulative BM change and day to day BM change ($p<0.0001$). Interaction between treatment and time on cumulative BM change ($p=0.027$) and day to day BM change ($p=0.02$). Within both groups; significant differences were found for cumulative BM change between successive days except for Day 3 to Day 4. Rehydration returned BM at Day 6 to levels equivalent to Day 3/4

Fluid balance and urine analysis

Fluid balance and urine analyses are displayed in Figure 4. There was a main effect of time for all measures, a main effect of fluid intake on USG, and an interaction between fluid intake and time for USG and fluid output.

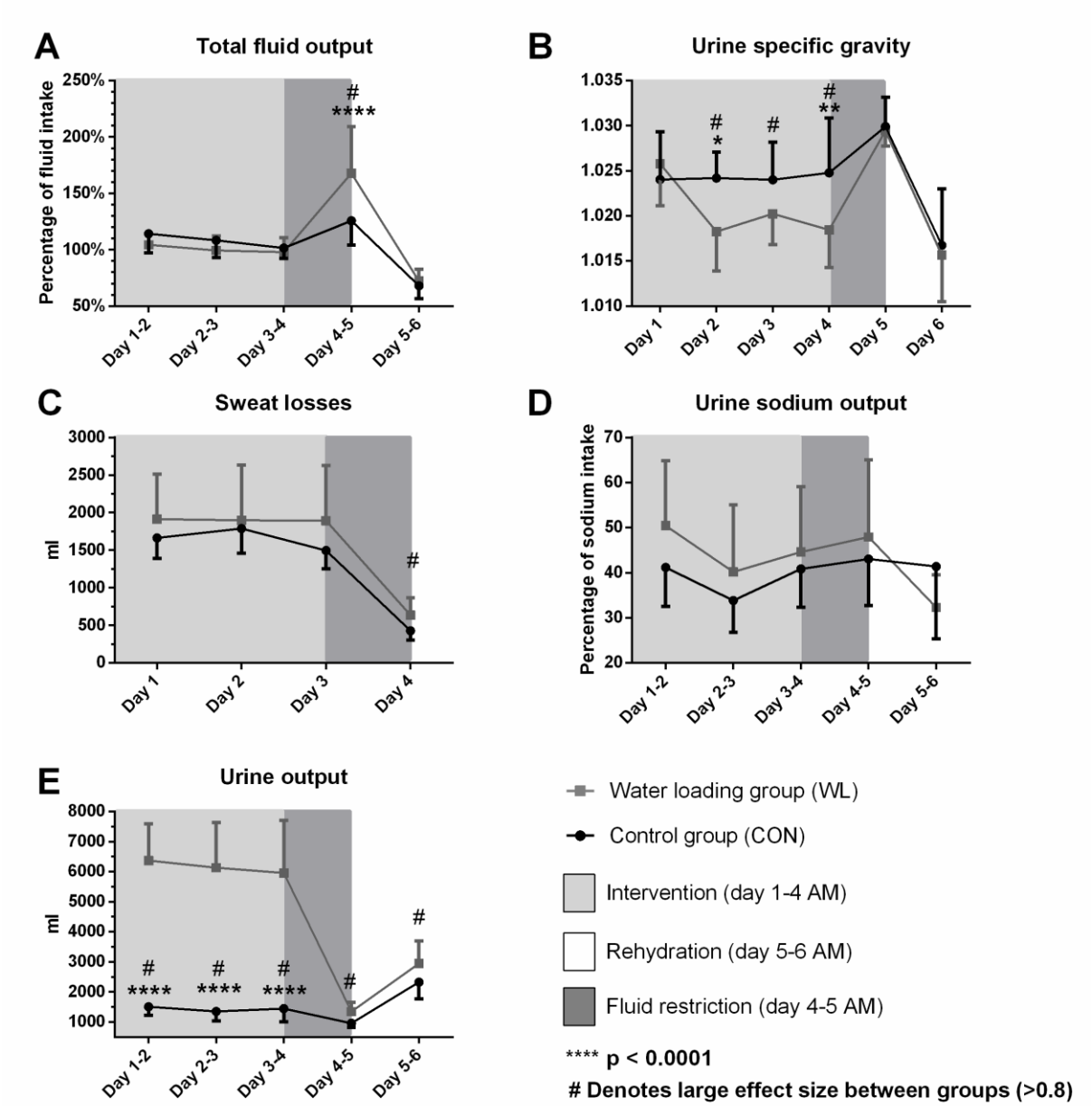

Figure 4. Urine and fluid balance analysis. Daily fluid output (urine + sweat), expressed as percentage of fluid intake ±SD (A). Daily waking urine specific gravity ±SD (B). Daily absolute sweat losses ±SD (C). Daily urine sodium output, expressed as percentage of sodium intake ±SD (D). Daily absolute urine output ±SD (E). 2 way ANOVAs revealed a main effect of time on; fluid output ($p<0.0001$), urine specific gravity ($p<0.0001$), sweat losses ($p<0.0001$), sodium output ($p = 0.035$), and urine output ($p<0.0001$), a main effect of fluid intake on urine specific gravity ($p<0.029$) and urine output ($p<0.0001$), and an interaction between time and fluid intake on; fluid output ($p<0.0001$), urine specific gravity ($p = 0.006$) and urine output ($p<0.0001$).

Renal hormone changes are displayed in Figure 5. Main effects of time were found for all measures, and an interaction was found between treatment and time for fold changes and absolute values in vasopressin.

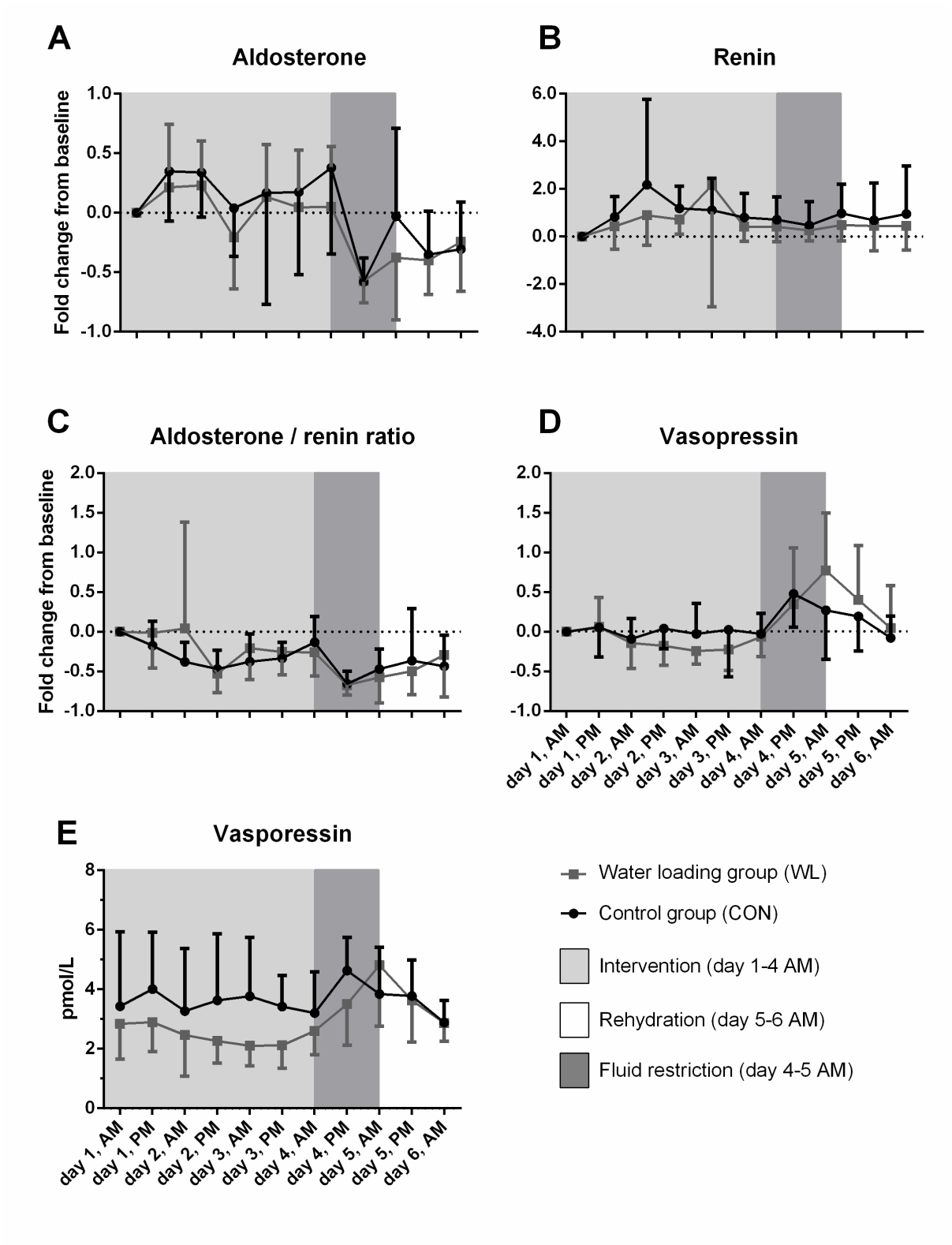

Figure 5. Changes in renal hormones across the 6-day intervention: aldosterone (A), renin (B), aldosterone/renin ratio (C) and vasopressin (D), expressed as fold change from baseline (mean ±SD). Main effect of time on aldosterone ($p<0.0001$), renin ($p=0.0187$), renin/aldosterone ratio ($p<0.0001$) and vasopressin ($p<0.0001$). Interaction between treatment and time on vasopressin.

Blood chemistry

Blood chemistry is displayed in Figure 6. Main effects were found for time with all measures, and an interaction was found between treatment and time for sodium, chloride and urea. Indices remained within critical values; none deviated significantly from typical clinical reference ranges for greater than one time point.

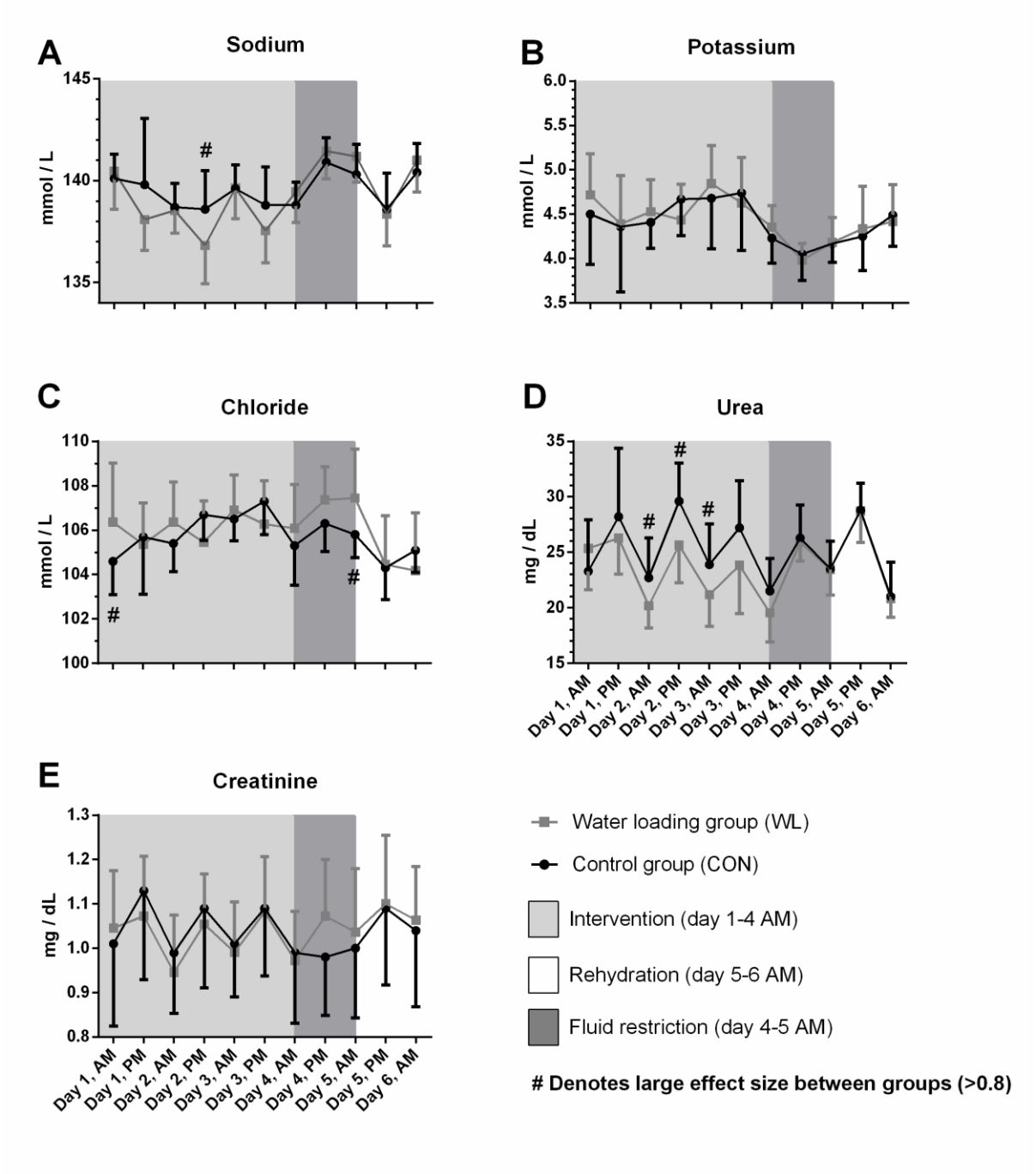

Figure 6. Blood chemistry across the 6-day intervention. Sodium (A), Potassium (B), Chloride (C), urea (D), and creatinine (E), expressed as mean ±SD. Main effect of time on sodium ($p < 0.0001$), potassium ($p<0.0001$), chloride ($p<0.0001$), urea ($p<0.0001$) and creatinine ($p<0.0001$). Main effect of treatment on urea ($p=0.0137$). Interaction between treatment and time on sodium ($p=0.0096$), chloride ($p=0.0137$) and urea ($p=0.0043$).

Gastro intestinal symptoms

A main effect of time for 'nausea' ($p=0.023$) and 'bloating' ($p=0.0005$) was revealed, with nausea peaking (mean 1.2±0.1) during fluid restriction and bloating peaking (mean 1.4±0.2) prior to dietary standardisation. 'Loss of appetite' was not affected by fluid intake or time.

Heart rate and blood pressure

A main effect of time for 'heart rate' ($p<0.0001$) was revealed; with the lowest values occurring on Day 0 and 6 (AM) and Day 5 (PM). No differences existed between groups. Blood pressure was not affected by fluid intake or time.

Physical testing

No differences between groups for physical performance tests existed. A main effect of time ($p=0.0354$) for total work completed during the RSA test and for peak displacement in the CMJ test ($p<0.0001$) was found. Subjects completed more total work during the RSA on Day 6 compared to Day 0 (pooled means; Day 0: 7542.8±371.5W vs Day 6: 7790.5±301.1W). Peak displacement was higher in the Day 6 CMJ post-test than the Day 0 pre-test (pooled means; pre-test 45.4±1.3cm vs post-test 47.6±0.8cm).

Discussion

This is the first investigation of the effectiveness and safety of 'water loading' as a means of manipulating BM in the context of weight category sports. The key findings were water loading was effective in increasing fluid and BM loss accompanying fluid restriction; this may potentially be mediated in part via the interventions effects on vasopressin. Water loading, as practiced in the current investigation (i.e. 100mL/kg dispersed evenly throughout the day), appears to be safe since there was no evidence of problematic blood chemistry changes or impairment of physical performance following rehydration.

These results support anecdotal outcomes described by athletes. We found the intake of large volumes (100 $mL \cdot kg \cdot d^{-1}$ or ~ 7-8L/d) of water for 3 days prior to one day of fluid restriction (15 $mL \cdot kg \cdot d^{-1}$) was associated with increased urine production, both during the days of high fluid consumption and fluid restriction. Specifically, diuresis continued during fluid restriction, leading to greater fluid losses relative to intake on the day as well as the losses recorded for a control group who had consumed 40 $mL \cdot kg \cdot d^{-1}$ (~ 3L/d) prior to this day. This was effective in achieving greater BM loss following the 5 d intervention in the WL group than the CON group. The combination of 5 days of a potentially mild energy deficit and reduced residue diet, including 1 day of fluid restriction, achieved total mean BM losses of 3.2 and 2.4% for WL and CON groups, respectively.

This acute BM loss was achieved in a scenario simulating the preparation for weigh-in and competition in combat sports, but without resorting to more extreme practices of severe energy restriction and active dehydration commonly observed (Franchini, Brito et al. 2012). However, before advocating water loading, investigation of safety concerns is necessary. It is well documented that excessive fluid intake is causative in hyponatremia (Adrogué and Madias 2000) with substantial lowering of blood sodium leading to negative outcomes, including death (Garigan and Ristedt 1999, Adrogué and Madias 2000). In the present investigation, however, no clinical meaningful blood chemistry changes

occurred with water loading, with perturbations following expected changes due to differences in fluid intake.

The present water loading protocol appeared to not increase hyponatremia risk; indeed cases in which fluid intake in healthy individuals has resulted in death, generally involved substantially greater intakes over much shorter time frames (e.g. >10 litres in 6 hours) (Garigan and Ristedt 1999, Adrogué and Madias 2000). Dilutional hyponatremia results when fluid ingestion rate exceeds excretion capacity (Adrogué and Madias 2000). Thus, in this intervention, it appears dispersing intake across the day, allowed renal adjustments to compensate. The hormone analysis provides some insight into a plausible mechanism providing blood chemistry maintenance and the water loading effect on fluid output. Although no main effect of fluid intake on vasopressin was evident, there was trend for lower vasopressin in the WL group and a significant interaction was present; that is mean vasopressin was decreased during the water loading phase in WL (and lower than in CON), before 'rebounding' to concentrations higher than baseline and higher than seen in the CON group following fluid restriction. Blood sodium decreased in the WL group during the water loading phase, but normalised in line with the CON group after water loading.

As vasopressin is under osmoregulation (Robertson, Shelton et al. 1976), blood sodium decreases in WL may explain the vasopressin suppression observed. Furthermore, vasopressin binds to vasopressin-2 receptors (V2R) found within the collecting ducts of the kidneys. This initiates a metabolic cascade increasing the permeability of the collecting ducts, and thus water reabsorption, via the insertion of aquaporin channels (Verbalis 2003), notably; aquaporin-2 (AQP2) channels. Conversely, in the absence of vasopressin, AQP2 channels (thus water reabsorption) are reduced (Verbalis 2003), assisting acute fluid regulation (Kwon et al. 2013). This mechanism has been directly observed in rodent models, with 24 hours of water loading associated with a reduction in intramembrane AQP2 channels and water permeability in the kidney collecting ducts (Lankford, Chou et al. 1991, Knepper 1997). Additionally, infusion of vasopressin has been shown to increase AQP2

channels mRNA expression (Knepper 1997). In rats unable to manufacture endogenous vasopressin, vasopressin infusion may take 3-5 days to 'return' mRNA expression of AQP2 channels to 'normal' levels (Kishore, Terris et al. 1996). Whilst the present data cannot confirm this hypothesis, this mechanism possibly explains persistent fluid losses evident following fluid restriction in WL.

Body mass losses prior to fluid restriction

Significant BM losses (~1-2%BM) occurred in both groups following days 1 and 2, before plateauing until fluid restriction. It is possible the mild energy deficit allowed a loss of fat mass and/or glycogen. However, the energy deficit required for this degree of fat loss is substantial and a major restriction of carbohydrate would be needed to create such glycogen depletion. Therefore, reduced gut content resulting from decreased fibre intake is the most plausible cause of the initial BM loss, especially considering the time frame. Low fibre/residue diets have been used by combat sport athletes and recommended by sports nutrition professionals (Reale, Slater et al. 2016) as a way to incur BM loss without the disadvantages associated with severe dehydration and energy restriction. Different foods possess different faecal bulking properties (Monro 2000), with those high in fibre drawing water into the intestinal space, increasing stool bulk. Reducing dietary fibre reduces undigested plant matter, equating to reduced gut contents and a lower overall BM. There is a linear relationship between fibre intake and bowel content (Wu, Rayner et al. 2011), with the adoption of a low fibre diet for even two days helping empty the bowel (Wu, Rayner et al. 2011) and seven days being as effective as pre-surgery bowel preparation formulas (Lijoi, Ferrero et al. 2009). Indeed, surgery preparation formulae have been shown to achieve BM reductions of 1.6% (Holte, Nielsen et al. 2004), in line with the ~1.5% BM loss in our study following 48 hours of lowered fibre intake. Considerable variability in whole gut transit times exists (~10-96 hours) (Lee, Erdogan et al. 2014), but in the absence of investigations of low fibre diets in the context of weight making for weight category sports, the present findings could be valuable in identifying the timeframe required to achieve significant BM loss using this technique.

The lack of a control group on a “normal” (higher fibre) intake is a limitation of our study, however, the measurement of fluid balance in our groups eliminates hypohydration as a confounding variable. The use of low residue diets in weight-making warrants further investigation.

Limitations

The major limitation of this study is the lack of a standardised ‘lead-in’ period prior to the commencement of the controlled diets. Achieving stable BM and increasing confidence in the prescription of appropriate energy and carbohydrate intakes would have allowed greater certainty in interpreting the source of BM losses in days 1-2 of our intervention. However, the standardisation which did take place prior to fluid restriction, combined with the careful observations of daily fluid input/output allows strong conclusions about the effect of water loading on fluid balance to be drawn. Additionally, since we only assessed blood sodium at specific time points, we cannot rule out the possibility that values were lower (and thus possibly indicated preliminary signs of hyponatremia) at other times points throughout the day.

Conclusions

Three days of dispersed consumption of large volumes of water (100 $mL \cdot kg \cdot d^{-1}$), prior to one day of fluid restriction, appears to be a safe and effective method of acutely reducing BM via a reduction in body water secondary to increased fluid losses. We suggest increased fluid consumption creates a small but potentially physiologically significant reduction in blood sodium concentration, which suppresses vasopressin release and downregulates the appearance of AQP2 channels in the collecting ducts in the kidneys. When this is employed immediately prior to fluid restriction, there is a continuation of increased fluid loss leading to greater losses relative to fluid restriction alone.

Novelty statement

This study is the first to investigate the acute weight loss method; 'water loading'. Under the conditions utilised in the present study, water loading was an effective and safe (no sign of hyponatremia) procedure to increase fluid losses during fluid restriction.

Practical application

Water loading represents another 'tool in the tool belt' which could be used alongside more traditional methods of acute weight loss. Nutrition professionals working with weight category sport athletes now have an evidence base from which to draw upon when educating athletes on the use of this method.

Acknowledgments, authorships, declarations of funding sources and conflicts of interest

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The study was designed by Reid Reale (RR), Gary Slater (GS), Louise Burke (LB), Ian Dunican (ID) and Gregory Cox (GC); data were collected by RR, ID, LB and analysed by RR and LB; data interpretation and manuscript preparation were undertaken by RR, GS, GC and LB. All authors approved the final version of the paper.

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All the authors declare that they have no conflict of interest derived from the outcomes of this study.

References

Adrogué, H. J. and N. E. Madias (2000). "Hyponatremia." New England Journal of Medicine **342**(21): 1581-1589.

Bovenschen, H. J., M. J. Janssen, M. G. van Oijen, R. J. Laheij, L. G. van Rossum and J. B. Jansen (2006). "Evaluation of a gastrointestinal symptoms questionnaire." Dig Dis Sci **51**(9): 1509-1515.

Cartledge, S. and N. Lawson (2000). "Aldosterone and renin measurements." Annals of clinical biochemistry **37**(3): 262-278.

Cohen, J. (1992). "A power primer." Psychological bulletin **112**(1): 155.

Crighton, B., G. L. Close and J. P. Morton (2015). "Alarming weight cutting behaviours in mixed martial arts: a cause for concern and a call for action." British journal of sports medicine **0**(0): 1-2.

Derkx, F., R. De Bruin, J. Van Gool, M. Van den Hoek, C. Beerendonk, F. Rosmalen, P. Haima and M. Schalekamp (1996). "Clinical validation of renin monoclonal antibody-based sandwich assays of renin and prorenin, and use of renin inhibitor to enhance prorenin immunoreactivity." Clinical chemistry **42**(7): 1051-1063.

Franchini, E., C. J. Brito and G. G. Artioli (2012). "Weight loss in combat sports: physiological, psychological and performance effects." J Int Soc Sports Nutr **9**(1): 52.

Franchini, E., C. J. Brito and G. G. Artioli (2012). "Weight loss in combat sports: physiological, psychological and performance effects." Journal of the International Society of Sports Nutrition **9**(1): 52.

Garigan, T. P. and D. E. Ristedt (1999). "Death from hyponatremia as a result of acute water intoxication in an Army basic trainee." Military medicine **164**(3): 234.

Glick, S. and A. Kagan (1979). "Radioimmunoassay of arginine vasopressin." Methods of Hormone Radioimmunoassay, eds Jaffe BM, Behrmann HR.(Academic, New York): 341-351.

Halperin, I., K. J. Williams, D. T. Martin and D. W. Chapman (2016). "The Effects of Attentional Focusing Instructions on Force Production During the Isometric Midthigh Pull." The Journal of Strength & Conditioning Research **30**(4): 919-923.

Holte, K., K. G. Nielsen, J. L. Madsen and H. Kehlet (2004). "Physiologic effects of bowel preparation." Diseases Of The Colon And Rectum **47**(8): 1397-1402.

Kishore, B. K., J. M. Terris and M. A. Knepper (1996). "Quantitation of aquaporin-2 abundance in microdissected collecting ducts: axial distribution and control by AVP." American Journal of Physiology-Renal Physiology **271**(1): F62-F70.

Knepper, M. A. (1997). "Molecular physiology of urinary concentrating mechanism: regulation of aquaporin water channels by vasopressin." American Journal of Physiology-Renal Physiology **272**(1): F3-F12.

Kwon, T.H., Frokiaer, J., & Nielsen, S. (2013). Regulation of aquaporin-2 in the kidney: a molecular mechanism of body-water homeostasis. *Kidney Research and Clinical Practice, 32*(3), 96-102.

Lankford, S. P., C. Chou, Y. Terada, S. M. Wall, J. B. Wade and M. A. Knepper (1991). "Regulation of collecting duct water permeability independent of cAMP-mediated AVP response." American Journal of Physiology-Renal Physiology **261**(3): F554-F566.

Lee, Y. Y., A. Erdogan and S. S. C. Rao (2014). "How to assess regional and whole gut transit time with wireless motility capsule." Journal Of Neurogastroenterology And Motility **20**(2): 265-270.

Lijoi, D., S. Ferrero, E. Mistrangelo, I. D. Casa, M. Crosa, V. Remorgida and F. Alessandri (2009). "Bowel preparation before laparoscopic gynaecological surgery in benign conditions using a 1-week low fibre diet: a surgeon blind, randomized and controlled trial." Archives Of Gynecology And Obstetrics **280**(5): 713-718.

Loucks, A. B. (2004). "Energy balance and body composition in sports and exercise." Journal of sports sciences **22**(1): 1-14.

Matthews, J.J and Nicholas, C. (2016). "Extreme rapid weight loss and rapid weight gain observed in UK mixed martial artists preparing for competition." International Journal of Sports Nutrition and Exercise Metabolism. **27**(2): 122-129

Monro, J. A. (2000). "Faecal bulking index: A physiological basis for dietary management of bulk in the distal colon." Asia Pacific Journal Of Clinical Nutrition **9**(2): 74-81.

Montoye, H. J. (2000). Energy costs of exercise and sport. Nutrition in sport. 7th ed. . London, Blackwell Sciences. **7:** 53-72.

Nana, A., G. J. Slater, W. G. Hopkins, S. L. Halson, D. T. Martin, N. P. West and L. M. Burke (2016). "Importance of Standardized DXA Protocol for Assessing Physique Changes in Athletes." International Journal of Sport Nutrition & Exercise Metabolism **26**(3).

Reale, R., G. Slater and L. M. Burke (2017). "Weight management practices of Australian Olympic combat sport athletes." International Journal of Sports Nutrition & Exercise Metabolism **0**(0): 1 -26 [Epub ahead of print]

Reale, R., G. Slater and L. M. Burke (2016). "Acute Weight Loss Strategies for Combat Sports and Applications to Olympic Success." Interntaional Journal of Sports Physiology & Perform **12**(2): 142-151.

Robertson, G. L., R. L. Shelton and S. Athar (1976). "The osmoregulation of vasopressin." Kidney international **10**(1): 25-37.

Verbalis, J. G. (2003). "Disorders of body water homeostasis." Best practice & research clinical endocrinology & metabolism **17**(4): 471-503.

Wu, K.-L., C. K. Rayner, S.-K. Chuah, K.-W. Chiu, C.-C. Lu and Y.-C. Chiu (2011). "Impact of low-residue diet on bowel preparation for colonoscopy." Diseases Of The Colon And Rectum **54**(1): 107-112.