

## Research article

# PLASMA VOLUME EXPANSION 24-HOURS POST-EXERCISE: EFFECT OF DOUBLING THE VOLUME OF REPLACEMENT FLUID

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### ABSTRACT

The effects of two volumes (1.5 L or 3.0 L) of commercially available electrolyte beverage (1.44 mM·L<sup>-1</sup> Na<sup>+</sup>) taken during a 24-hour recovery period post-exercise, on plasma volume (PV) expansion 24-hours post-exercise were assessed. A simple random-order crossover research design was used. Subjects (n = 9 males: age 21 ± 4 years, body mass 80.0 ± 9.0 kg, peak incremental 60-second cycling power output 297 ± 45 W [means ± SD]) completed an identical exercise protocol conducted in hot ambient conditions (35°C, 50% relative humidity) on two occasions; separated by 7-days. On each occasion, subjects received a different volume of 24-hour fluid intake (commercial beverage) in random order. In each case, the fluid was taken in five equal aliquots over 24-hours. PV expansions 24-hours post-exercise were estimated from changes in haemoglobin and haematocrit. Dependent t-testing revealed no significant differences in PV expansions between trials, however a significant expansion with respect to zero was identified in the 3.0 L trial only. Specifically, PV expansions (%) were; 1.5 L trial: (mean ± SE) 2.3 ± 2.0 (not significant with respect to zero), 3.0 L trial: 5.0 ± 2.0 (p < 0.05, with respect to zero). Under the conditions imposed in the current study, ingesting the greater volume of the beverage lead to larger mean PV expansion.

**KEY WORDS:** Hypervolemia, dehydration, re-hydration.

### INTRODUCTION

As a consequence of exercise, an acute loss of plasma volume (PV) occurs (Costill and Fink, 1974; Dill and Costill, 1974). A reduction in PV challenges the maintenance of homeostasis, as it results in destabilization of the cardiovascular system (Hanel et al., 1997; Krip et al., 1997; Neuhaus and Gaehtgens, 1994). A conspicuous physiological response that begins almost immediately upon exercise cessation is auto-restoration of lost PV (Convertino, 1991; Fellmann, 1992; Green et al., 1984; Mack et al., 1998). Even in the absence of fluid ingestion, PV is restored to baseline within 60-

minutes of exercise cessation (Gillen et al., 1991; Lundvall and Lanne 1989). This fluid-flux into the vascular space from other fluid pools, notably the lymph, (Nagashima et al., 2001) occurs due to alterations in Starling forces and due to elevations in plasma albumin mass (Gillen et al., 1991; Hayes et al., 2000; Mack et al., 1998; Nagashima et al., 2000). Plasma osmolarity may also play a mediatory role (Nose et al., 1988), as well as the specific stimulus for influx (Jiminez et al., 2002; Kay et al., 2004). Specifically, it has been recently shown in our laboratory (Kay et al., 2004) that the relative loss of body fluid itself (in the range of 0-2% body mass) is not likely to be an important mediator of the

response (i.e. PV restoration): rather, that other coincident factors are likely to be primary mediators.

The restoration of PV usually “overshoots” the original PV, and is retained at 24-hours post-exercise (Gillen et al., 1991; Haskell et al., 1997; Nagashima et al., 1999; 2001): resulting in the phenomenon of exercise-induced hypervolemia. The exercise-induced hypervolemia may be further enhanced by increased renal sodium and water retention in the 24-hours following exercise, potentially as a consequence of an increase in aldosterone concentration (Nagashima et al., 1999) or reciprocally, a decrease in ANP concentration (Hanel et al., 1997). Further, there is an increase in the rate of albumin transcription (Yang et al., 1998), and a decrease in the trans-capillary escape-rate of albumin (Haskell et al., 1997; Lang et al., 1987). If exercise is repeated over a number of days, the resting PV “resets” to a higher level and PV may exceed pre-exercise levels by approximately 20% (Convertino et al., 1980). Indeed, in the endurance-trained condition, chronic hypervolemia has been demonstrated in cross-sectional (Maw et al., 1996) and in longitudinal studies (Convertino, 1991; Costill and Fink, 1974). In recreationally active individuals, expanded PV increases end-diastolic volume and consequently cardiac output (Hopper et al., 1998; Kanstrup and Ekblom, 1982; Krip et al., 1997). The increase in cardiac output in turn increases  $\text{VO}_2$  peak (Coyle et al., 1990), and results in lowered blood viscosity through the effects of the Fahraeus-Lindqvist effect (Neuhaus and Gaetgens, 1994). These factors theoretically lead to lowered cardiovascular stress during exercise at any given intensity. Indeed, PV expansion appears to result in lowered physiological stress as assessed via numerous physiological markers during subsequent exercise (Fellmann, 1992; Hopper et al., 1998; Kanstrup and Ekblom, 1982; Krip et al., 1997; Maw et al., 1996; Mitchell et al., 2000; Neuhaus and Gaetgens, 1994; Watt et al., 1999).

Little is understood however, about the various re-hydration strategies commonly used to help maximise this physiological response over short (0-3 hours), and longer (up to 24-hours) periods post-exercise. It is understood that over a three-hour recovery and re-hydration period, greater fluid volume consumed (150% volume lost vs. 100% volume lost during exercise) is beneficial to PV expansion ( $p < 0.05$ ) and whole body re-hydration ( $p < 0.05$ ). However, few previous researchers have investigated re-hydration strategy with respect to PV expansion over a 24-hour post-exercise period. Possible mediators over this longer time-frame include (a) the volume of fluid consumed (O'Brien, 2001), (b) the rate of fluid consumption (Kovacs et al., 2002), (c) the sodium content in oral fluid (Hanel

et al., 1997; Nagashima et al., 2001), and (d) the dietary intake of sodium (Luetkemeier, 1995). Therefore, the intention of the current investigation was to determine whether increased consumption of a commonly used commercially available re-hydration beverage during the 24-hours following a standard exercise bout, is associated with increased exercise-induced hypervolemia 24-hours post-exercise (when factors such as those above are controlled). It was hypothesised that the greater beverage volume trial would be associated with greater mean PV expansion 24-hours post-exercise.

## METHODS

### *Subjects*

Subjects ( $n = 9$  recreationally active males; age  $21 \pm 4$  years, body mass  $80.0 \pm 9.0$  kg, peak cycling power output  $297 \pm 15$  W, [mean  $\pm$  SD]) were recruited from within the Waikato Institute of Technology, Centre for Sport and Exercise Science. All subjects volunteered to take part, and provided written informed consent. The Waikato Institute of Technology Research Ethics Committee approved all procedures. Subjects underwent pre-screening for exercise contraindications, specifically resting blood pressure in excess of 145 / 90 mmHg, and resting heart rate in excess of 100 beats·minute<sup>-1</sup>. Subjects also completed a medical history questionnaire so that individuals with other contraindicating factors could be identified and excluded.

### *Experimental design overview*

The effects of two different re-hydration strategies, on PV expansion 24-hours post exercise were assessed. A randomised crossover design was used. During the 24-hours before and the 24-hours following exercise, all subjects underwent dietary and hydration control. Subjects attended the laboratory on three separate occasions (each separated by 7 days). The first session comprised the determination of individual peak 60-sec cycling power output using a standardised continuous-incremental protocol (5-min at 100W, then +25W·min<sup>-1</sup> to exhaustion). During the subsequent two sessions, an intense-intermittent exercise bout in the heat (75% peak 60-sec cycling power output, 8 repetitions of 4-min work/5-min rest, 35°C, 50% relative humidity) was used to elicit exercise induced hypervolemia on each occasion. After each exercise session, subjects consumed one of two volumes (1.5 or 3.0L) of re-hydration beverage (commercially available beverage) during the 24-hours post exercise, in random order. In each case, the beverage was taken by all subjects in five equal aliquots at set times during the 24-hours post exercise (0800; 1200; 1600; 2000; 0400). The

volume of beverage to be consumed was not adjusted for fluid lost during the exercise sessions, as fluid loss within the range expected is not likely to play an important role in mediating eventual PV expansion (Kay et al, 2004). Venous blood samples were obtained prior to exercise, and at 24-hours post exercise. Haemoglobin concentration ( $\text{g}\cdot\text{dL}^{-1}$ ) and haematocrit (%) were measured, and these parameters used to estimate percentage changes in PV (Dill and Costill, 1974).

### ***Experimental protocol***

Subjects were asked to refrain from consuming any nutritional supplement, ergogenic aid (or proposed ergogenic aid), alcohol, or other recreational drug during the entire experimental period. Further, subjects were asked to refrain from caffeine in food and drink for 48-hours prior to, and during the 24-hours of testing. Finally, subjects were asked to maintain their normal sporting and exercise activities, except to refrain from exercise for 24-hours prior-to testing, and during the 24-hours post-testing. Compliance was assessed via each subject completing a nutritional and training diary, which was supplied by the researchers. Each testing week began 24-hours prior-to exercise, with a period of dietary and hydration control, which continued for 24-hours post-exercise. During the second 24-hour period, there was a slight increase in both energy intake, and dietary sodium intake, which was in order to prevent negative energy balance or sodium status, due to the exercise and dehydration imposed. All subjects ate the same foods, except that dinner (commercially available frozen meals) was available in several flavours, hence the variability in intakes. Specifically, day one intakes were (mean  $\pm$  SE)  $8350 \pm 72$  kJ ( $1988 \pm 17$  kcal),  $3412 \pm 45$  mg  $\text{Na}^+$ , 1.5 L water (5 equal aliquots). Day two intakes were  $9721 \pm 108$  kJ ( $2315 \pm 26$  kcal),  $4354 \pm 67$  mg  $\text{Na}^+$ , the re-hydration fluid volume was varied by week of testing, as determined by random assignment. The researchers have assumed the water content of the different commercial meals was the same. The sodium mass provided in the re-hydration fluid was additional to that in the diet, discussed above. This sodium mass was (159 and 318 mg respectively): i.e. an overall increase in total sodium intake of  $\sim 4\%$  in the 3.0 L trial as compared with the 1.5 L trial. The SE ( $\pm 67\text{mg}$ ) from the dietary variation equated to  $\sim 1.5\%$  of total sodium mass intake.

Following the first 24-hour period of dietary and hydration control (which stipulated the timing of all food and fluid intakes), subjects reported to the laboratory at the appointed time of day (0600), which was held constant throughout the testing period. Testing began following the last meal and fluid intake (breakfast) by two hours. After subjects'

journals were checked, all subjects were cleared to proceed with testing. Subjects first voided, and nude body mass was recorded. Subjects then entered the environmental chamber ( $35^\circ\text{C}$ , 50% relative humidity), and sat on a chair with feet flat on the floor and palms hands down on the knees for a period of 20-minutes. This postural control was to allow for stabilisation of PV (Kargotich et al., 1998). Following this, a 5mL sample of blood was collected from a superficial vein in the antecubital fossa of either arm. Samples were immediately refrigerated at  $3^\circ\text{C}$ , and analysed within 120-minutes (see below). Subjects then completed the required exercise. Subjects were required to remain in the sealed environmental chamber throughout the protocol, and food / drinking during the exercise session was disallowed. Following the exercise, subjects warmed down (5-minutes at 100 W), and then towelled off, ensuring that they were as dry as possible. Nude weighing was then repeated, in order to assess the degree of whole-body dehydration experienced by each subject during the session.

Finally, subjects were also asked to refrain from adopting the supine posture for at least 5-hours post-exercise (Nagashima et al., 2000), and to refrain from water immersion for at least 5-hours post-exercise (Hayes et al., 2000; Hinghofer-Szalskay et al., 1987). Subjects remained under observation for 30-minutes post-exercise, in case of any ill effect from the exercise / heat stress. After this, subjects were given the appropriate re-hydration beverage and a drinking schedule. Subjects next reported at 0600 the following day; for repeated weighing, postural control, and blood sampling as above. The procedure was repeated 7-days later, such that all subjects received both beverage volumes in random order.

### ***Blood sampling and analysis***

Blood was collected using a standard 'complete blood count' (CBC) lithium / heparin vacuum container, and a 20-gauge hypodermic needle. Haematocrit percentage (Hct) was determined in triplicate using capillary tubes filled from the sample containers after they had been inverted ten times to homogenise the blood. Capillary tubes were then centrifuged at  $3600$  revolutions. $\text{minute}^{-1}$ , for a period of 5-minutes. A ruler marked with 0.5 mm increments was used to determine the percentage of packed red cells after centrifuging, according to (*packed cell length / total sample length*)  $\times 100$ . Haemoglobin (Hb) concentration ( $\text{g}\cdot\text{dL}^{-1}$ ) in the samples was determined in duplicate using a Ciba-Corning Co-oximeter (860 series, USA), following homogenisation (as above).

### ***Determination of estimated plasma volume changes***

Estimated changes in PV (mean  $\pm$  SE of the difference, %) were determined from Hct and Hb in blood samples taken at rest (Dill and Costill, 1974). In order to determine 'baseline  $\pm$  SE'; on three occasions prior to the investigation (separated by 24-hours, and under controls as above), Hct and Hb was determined from blood samples taken at rest (Dill and Costill, 1974). As the method of Dill and Costill (1974) requires two values for both Hb and Hct, comparing sets of repeated measures in every permutation (3 x 2 x 1; or six of), necessarily yielded a mean change of zero, with a known SE of measurement. From this we have conducted analyses comparing the known baseline value (zero  $\pm$  SE) with the post-intervention values for each treatment condition during the experimental period (means  $\pm$  SE). SE of the difference for reporting, was then calculated in the usual fashion.

### **Statistical analysis**

Changes in body mass are reported as mean  $\pm$  SE of the difference, after absolute values for body mass change were converted to a percentage change from baseline (i.e. zero  $\pm$  SE; calculated as for PV above) in order to allow agreement in terms between assessment of body mass changes and PV changes (i.e. percent). Differences in mean percent PV changes, and in mean percent body mass changes between trials were subjected to dependent *t* testing. Alpha was set at 0.05.

## **RESULTS**

The 1.5 L re-hydration volume trial was associated with PV expansions of  $2.3 \pm 2.0\%$  (not a statistically significant increase with respect to zero), and the 3.0 L re-hydration volume trial was associated with PV expansions of  $5.0 \pm 2.0\%$  ( $p < 0.05$ , with respect to zero). The mean expansions for each trial noted during the current study however, did not statistically differ from one-another. The 1.5 L re-hydration volume trial was associated with body mass changes over 24-hours of  $-0.2 \pm 0.1\%$ , and the 3.0 L re-hydration volume trial was associated with body mass changes over 24-hours of  $0.2\% \pm 0.3\%$ . Neither of these body mass changes were statistically different from zero, nor were these values statistically different from each other. Whole-body dehydration sustained by the current subjects during the exercise sessions was  $1.0 \pm 0.2\%$  by body mass lost during the 1.5L trial, and  $1.0 \pm 0.1\%$  by body mass lost during the 3.0L trial. These two values were not significantly different from one another. Absolute fluid loss (as determined from changes in body mass) was  $875 \pm 148$  mL (1.5L trial), and  $820 \pm 97$  mL (3.0L trial). These values

were also not significantly different from one another.

## **DISCUSSION**

### ***Effects of re-hydration beverage volume consumed on PV expansions***

The major finding of this study was that under the conditions we imposed, 3.0 L of the specific re-hydration fluid we used was associated with a statistically significant mean PV expansion, while 1.5 L of the same re-hydration fluid was not. It was originally hypothesised that ingestion of a greater volume of the re-hydration fluid in the 24-hours post-exercise would be beneficial to PV expansion 24-hours post-exercise. The mean values we have provided appear to support this hypothesis.

### ***Magnitude of PV changes with respect to body mass changes***

The mean estimated PV change 24-hours post-exercise noted during this study, was approximately half ( $3.7 \pm 2.0\%$ ; pooled results vs.  $7.3 \pm 1.3\%$ ; pooled results) of that reported previously by other researchers (Gillen et al., 1991; Haskell et al., 1997; Nagashima et al., 1999) who have used an almost identical exercise protocol as the stimulus, and who have used the same PV estimation technique (Dill and Costill, 1974). The degree of dehydration noted previously (Mack et al., 1998) following the use of the same exercise protocol we have used (except that intensity was set at 85%  $\text{VO}_2$  peak); was  $10.2 \pm 1.1$   $\text{ml}\cdot\text{kg}^{-1}$  body mass. From the subject demographics given by those authors, the mean percent loss of body mass during that study was  $1.0 \pm 0.1\%$ . This compares closely to the percentage loss of body mass noted by the current investigators ( $1.0 \pm 0.2\%$ ). It appears therefore, that percent dehydration was unlikely to be a major factor in this discrepancy with previous research. The current researchers chose 75% peak power as the workload, because previous subjects were often unable to complete the protocol due to exhaustion (Gillen et al., 1991; Haskell et al., 1997; Nagashima et al., 1999).

### ***Exercise intensity as a possible mediator***

Previous research (Gillen et al., 1991; Hayes et al., 2000; Nagashima et al., 1999) suggests that the movement of albumin from the lymph to the plasma is the most likely primary mediator of fluid influx to the plasma pool. This movement of lymph is dependant upon a period of post-exercise hypotension (Hayes et al., 2000; Nagashima et al., 1999). Hypotension indeed occurs for several hours post-exercise (Hayes et al., 2000), and is mediated primarily by the accumulation of chemicals such as nitric oxide, and other metabolites (Blackman et al.,

2000; Ferrari et al., 2001; Planitzer et al., 2001). The increase in PV may therefore occur because the hypotension creates a favourable gradient for increased lymphatic drainage into the vascular space (hence increased delivery of albumin), and/or for interstitial fluid to move directly into the vascular space regardless. These arguments provide for the possibility that relative exercise intensity may play an important mediatory role in PV expansion post-exercise, as nitric oxide production is proportional to exercise intensity (Chirpaz-Oddou et al., 1997). Future research is required to elucidate.

## CONCLUSIONS

It was originally hypothesised that under the conditions imposed during this study, a greater volume of oral re-hydration fluid would be associated with increased mean PV expansion 24-hours post-exercise. The results of this study support this hypothesis.

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## KEY POINTS

- Greater volume of re-hydration beverage is beneficial to mean PV expansion 3-hours post-exercise, however this relationship has not been previously tested under adequate controls over 24-hours to our knowledge.
- This study indicates that under the conditions we imposed, over 24-hours increased volume (1.5 Vs. 3.0 L) of oral re-hydration fluid is associated with increased mean PV expansion.
- Although we used an almost identical exercise stimulus as previous researchers, relative intensity was slightly lower in the current study. Coincidentally, PV expansions noted during this study were approximately half those reported by others.

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