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Research article

EXERCISE-INDUCED HYPERVOLEMIA MAY NOT BE CONSEQUENTIAL TO DEHYDRATION DURING EXERCISE

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ABSTRACT

The purpose of this investigation was to determine whether the increase in plasma volume (PV) frequently observed 24 hours after exercise is proportional to the magnitude of dehydration occurring during exercise. Seven males (age 21.6 ± 4.4 y, body mass 71.5 ± 8.5 kg; $\dot{V}O_{2\ peak}$ 43 ± 7 mL.kg.minute^{-1}, peak 60-second cycling power output 282 ± 16 W) completed three cycling sessions at 50% of peak power output in an ambient environment of 35^{o}C, 50% relative humidity; with the exercise lasting either 30, 60 or 90 minutes (in random order) to elicit varying levels of dehydration (assessed by body mass changes). The percent change in PV was calculated 24 hours after each exercise session. All exercise sessions were separated by 7-days. Participants' body mass (means ± SD) decreased by 1.03 ± 0.22% in the 30-minute exercise protocol; 1.43 ± 0.26% in the 60-minute protocol; and 1.59 ± 0.37% in the 90-minute protocol. Significant PV expansions were not evident 24 hours after any protocol (0.76 ± 4.58% in the 30-minute protocol; 1.40 ± 4.58% in the 60-minute protocol, and 2.92 ± 3.2% in the 90-minute protocol). Regression analysis revealed a poor correlation between percent dehydration and percent change in plasma volume (r = 0.24). Our study revealed that the magnitude of dehydration elicited during this study was insufficient to stimulate a significant expansion in PV.

KEY WORDS: Exercise, dehydration, fluid volume, blood volume.

INTRODUCTION

A conspicuous physiological response that occurs immediately after completion of exercise, is auto-restoration of exercise-induced PV loss (Gillen et al., 1991; Mack et al., 1998; Nagashima et al., 1999). Even in the absence of oral fluid ingestion, PV is restored to baseline within minutes of exercise completion (Mack et al., 1998). The fluid flux into the vascular space occurs presumably to stabilize cardiovascular function, and is caused by alterations in Starling forces, elevations in plasma albumin mass, and increased renal tubule sodium absorption (Gillen et al., 1991; Hayes et al., 2000; Mack et al., 1998; Nagashima et al., 1999; Nagashima et al., 2001).

The PV post-restoration usually exceeds the original PV, resulting in the phenomenon of exercise-induced hypervolemia (Convertino, 1991; Gillen et al., 1991; Mack et al., 1998; Maw et al., 1996; Nagashima et al., 1999). If exercise is repeated over a number of days the resting PV may increase by up to 20% (Convertino, 1991; Convertino et al., 1980; Green et al., 1984). Furthermore, it appears that long-term training results in a chronic expansion of the extracellular volume (Maw et al., 1996). The exercise-induced hypervolemia appears to be an adaptation that results in lower relative loss of PV during succeeding bouts of exercise (Green et al., 1984), and will increase end-diastolic volume and ultimately maximal cardiac output (Krip et al., 1997; Warburton et al.,

1999). Subsequently, $\dot{V}O_{2\ peak}$ is increased consequential to an elevated PV, provided that the effects of the hypervolemia do not result in excessive hemodilution and compromise oxygen arterial pressure (Coyle et al., 1990; Warburton et al., 1999).

Knowledge of the stimulus initiating an increase in plasma volume would be relevant and beneficial for exercise physiologists assisting an athlete's preparation for competitions that require an elevated $\dot{V}O_{2\ peak}$ for success. Whilst exercise-induced hypervolemia appears to be a supra-compensatory response to the magnitude of dehydration occurring during prolonged running, cycling or rowing tasks (Green et al., 1984), this hypothesis is yet to be experimentally tested. Therefore, the purpose of this is investigation is to examine the hypothesis that the magnitude of exercise-induced hypervolemia is dependent upon, and proportional to, the magnitude of dehydration occurring during a continuous sub-maximal cycling bout in recreationally active males.

METHODS

Subjects

Seven recreationally active males (age 21.6 ± 4.4 years, body mass 71.5 ± 8.5 kg, peak 60 second cycling power output 282 ± 16 W) volunteered for this study after being informed of risks and giving their written informed consent. The study was approved by the Waikato Institute of Technology Human Research Ethics Committee. All exercise training and procedures were performed in the Human Performance Laboratory at the Waikato Institute of Technology.

Experimental protocol overview

Prior to the experimental sessions (7-14 days), the participants completed a standard incremental cycle ergometer protocol to determine their $\dot{V}O_{2\ peak}$ and peak 60 second cycling power output (PCPO). The experimental protocol consisted of three standard exercise sessions (50% PCPO continuous upright cycling in an ambient environment of 35°C, 50% relative humidity) with the duration of exercise varied (30, 60, or 90 minutes), in order to elicit a different level of relative dehydration on each occasion. Exercise sessions were separated by one week and completed in random order. The percent change in PV 24 hours after each exercise session was calculated from changes in haemoglobin concentration and haematocrit values using the method of Dill and Costill (1974).

Pre-experimental protocol

One week prior to the experimental period $\dot{V}O_{2\ peak}$ and PCPO were determined using a maximal incremental test on a Monark cycle ergometer (818E, Sweden). Expired air was analysed using open circuit spirometry (Sensormedics 2900 Metabolic Measurement System, USA) in mixing chamber mode. Work-rate was incremented after 5 minutes of cycling at 100 W in 25 W steps each minute until volitional exhaustion, defined as an inability to maintain pedal cadence above 60 rpm. $\dot{V}O_{2\ peak}$ was determined as the highest $\dot{V}O_2$ ($L.min^{-1}$) value recorded during the test. PCPO was defined as the average power output during the last fully completed minute of the incremental test.

Pre-control and blood sampling

The participants were asked to refrain from alcohol, caffeine or recreational drugs 48-hours prior to, and during the 24-hours of testing. Furthermore, we requested them to refrain from exercise 24 hours prior to, and 24 hours following the initial PV measurement. Dietary and hydration control occurred 24 hours prior to, and 24 hours following the initial PV measurement. During this time participants consumed a diet supplied by the investigators. Energy intake was 8350 ± 203.6 $kJ{\cdot}day^{-1}$ (mean ± SD), 3412 ± 127.3 mg $Na^{+}{\cdot}day^{-1}$. 1.5 L water was orally ingested in five equal aliquots (at 09:00; 13:00; 17:00; 21:00; 05:00) in each 24-hour period of hydration control.

Participants reported to the laboratory at 06:00, following the initial 24 hour period of dietary and hydration control. Nude body mass was recorded (Wedderburn Scales, Japan, data recorded to ± 5 g). The participants then underwent postural stasis (seated upright with hands palm down on knees, feet flat to the floor) for 20 minutes to allow for PV stabilisation (Nagashima et al., 2001), after which 5 mL of venous blood was sampled from the antecubital fossa. Blood samples were analysed for haemoglobin concentration and haematocrit using standard techniques (Lundvall and Lindgren, 1998). The participants then entered the environmental chamber, and cycled on the Monark ergometer at 50% PCPO, maintaining a cadence of 60 rpm. Food and fluid intake was disallowed during the exercise bout. Exercise continued until 30, 60, or 90 minutes of total exercise time had elapsed. Upon completion of exercise, nude body mass (after sweat removal) was recorded. 24 hours following exercise, all participants reported to the laboratory for the 20 minutes of postural stasis described earlier and had 5 ml of blood sampled to obtain the haemoglobin concentration and haematocrit values used to calculate the percent change in PV.

Statistical procedure

Percent changes in PV and body mass are reported as mean ± SD. Differences in the percent change in PV and body mass between treatments were analysed using simple analysis of variance (ANOVA). Tukey's honestly significant difference test was used for post hoc test comparison. Significance was defined as $p<0.05$.

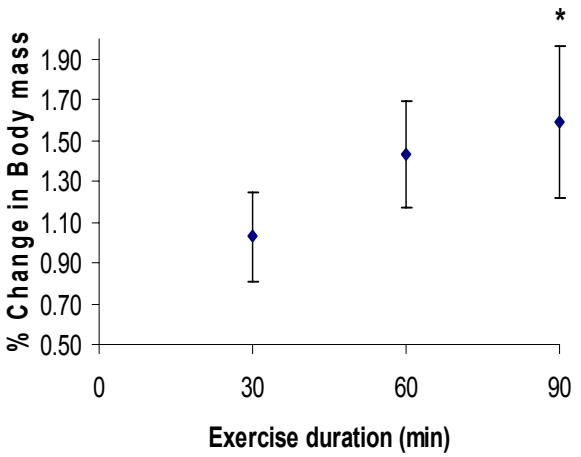

Figure 1. The percent change in body mass after participants completed the 30, 60 and 90 minute cycle bouts (mean ± SD) * $p < 0.01$.

RESULTS

There was a significant ($p < 0.01$) increase in the body mass lost after 90 minutes compared to after 30 minutes of exercise, however no other pair-wise comparison revealed a significant difference in body mass between treatments. The percentage loss of body mass was 1.03%, 1.43% and 1.59% after 30, 60 and 90 minutes of exercise respectively. The percent change in body mass is illustrated in Figure 1. The percent change in PV was however, not significantly affected by exercise of any duration ($p = 0.69$). The percent change in PV is illustrated in Figure 2. Finally, Figure 3 illustrates the correlation ($r = 0.24$) of percent dehydration experienced by all participants in all treatments, with the resulting PV changes 24 hours later.

DISCUSSION

The intention of the present study was to investigate whether the magnitude of exercise-induced hypervolemia is proportional to the level of dehydration incurred during exercise. We hypothesized that increased plasma volume 24 hours post-exercise would be well related and proportional to the percent dehydration experienced during exercise. Our results indicate that the relationship between dehydration (percent change in body mass) and percent change in PV 24 hours later was poor within the range of dehydration we investigated. Therefore, it is apparent the stimulus necessary for the induction of significant hypervolemia was not invoked in the present study. Subsequently, there is little support for our hypothesis that hypervolemia is a supra-compensatory response to dehydration, at least within the range of dehydration investigated in our study. However, the absence of any significant increases in PV despite significantly different levels of dehydration is a relevant finding of our investigation.

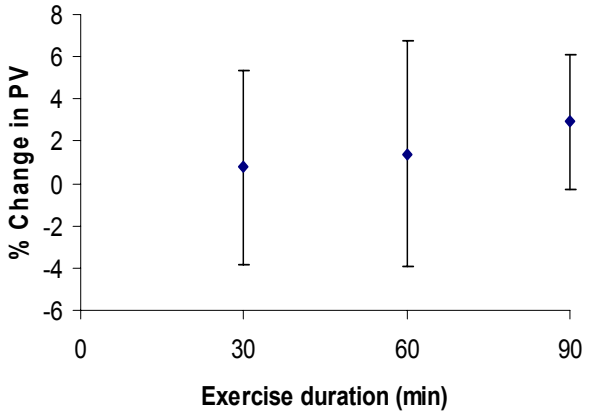

Figure 2. The percent change in plasma volume after participants completed the 30, 60 and 90 minute cycle bouts (mean ± SD).

The mechanisms of exercise-induced hypervolemia observed 24-hours after exercise are complex and could be consequential to first, an increase in plasma protein mass, secondly, a decrease in central venous pressure, or thirdly, an increase in renal fluid retention (Mack et al., 1998; Nagashima et al., 2001; Wu and Mack, 2001). An increase in plasma protein mass following exercise creates an osmotic gradient for water movement into the vascular space, and a decrease in central venous pressure would facilitate greater flux of fluid from the lymphatic system or interstitial space (Wu and Mack, 2001). Fluid conservatory hormones such as aldosterone are elevated greatly following exercise, and act to reduce urine output and consequently enhance fluid retention (Convertino, 1991). The present experiments resulted in a decrease in body mass ranging from 1-1.6%, which are similar to the values observed by Gillen et al., (1991), and would therefore be expected to have transiently lowered central venous pressure, increased aldosterone and stimulated an increase in plasma protein mass. It is therefore appropriate to discuss possible reasons why a significant hypervolemia was not observed in the present study, despite creating a disturbance in homeostasis that was anticipated to facilitate an increase in PV.

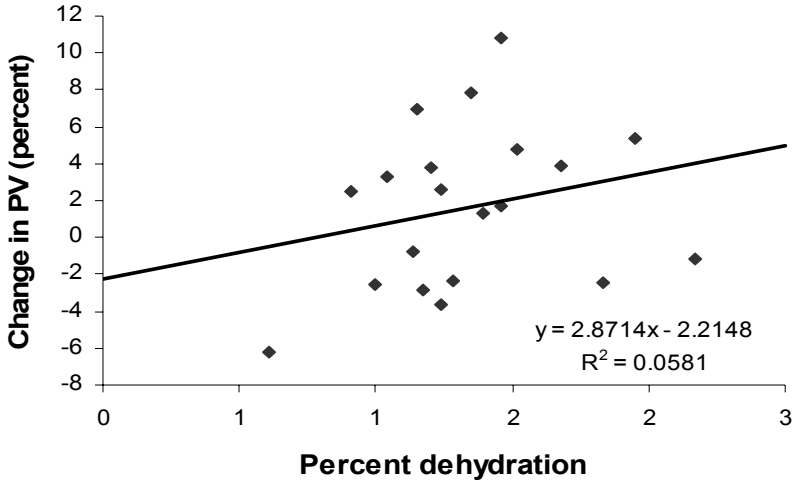

Figure 3. The regression plot for percent dehydration vs. change in plasma volume.

One possibility for the attenuated increase in PV, was that the magnitude of dehydration induced in the present study was not sufficient to result in exercise-induced hypervolemia 24 hours after exercise. Unfortunately, we were unable to induce greater levels of dehydration using the current exercise mode, as the participants were unable to continue beyond the 90 minutes of cycle ergometer exercise due to fatigue and discomfort. Future research may control for fatigue by eliciting greater levels of dehydration by non-exercise or "passive means", such as exposure to a sauna. Moreover, the failure of our study to detect an expansion in PV may be consequential to the participants engaging in only one and not several exercise sessions. The magnitude of hypervolemia appears to be consequential to the cumulative effect of several daily bouts of dehydration induced by exercise, as the greatest increases in hypervolemia are observed after several days of training (Green et al., 1984). Despite this concern, a single episode of exercise was anticipated to increase PV (Gillen et al., 1991). The dehydration experienced by the current participants was similar to that reported elsewhere (Gillen et al., 1991), and those subjects experienced an increase in PV of ~7%. Alternatively therefore, dehydration may not be the stimulus inducing hypervolemia, and some other covariate may be a more powerful mediator.

Indeed, Convertino et al. (1980) reported that 12% of exercise-induced hypervolemia could be attributed to exercise factors and 5% to thermal factors. One such "exercise factor" that may play a crucial role in the induction of hypervolemia is exercise intensity. Subjects in the study by Gillen et al. (1991) exercised intermittently at 85% of $VO_{2\,peak}$, which is higher than the ~50% we used. An observable increase in PV may be consequential to a specific exercise intensities effect on the body's fluid volume or distribution regulatory mechanisms. Nitric oxide production is proportional to exercise intensity (Chirpraz-Oddou et al., 1997), and in elevated concentrations may lower central venous pressure (Blackman et al., 2000), creating a favorable gradient for increased lymphatic drainage into the vascular space, or interstitial fluid to move directly into the vascular space. Intense exercise training also displays many of the biochemical features of the acute phase response (Tauler et al., 2002). An acute phase response elicited by exercise with high resistance loadings is associated with a disruption in muscle cell integrity, potentially allowing intracellular fluid to appear in the extracellular space (Kirwan and del Aguila, 2003). In particular, unaccustomed eccentric exercise elicits significant disturbance to muscle cell integrity (Kirwan and del Aguila, 2003), and is accompanied by a parallel increase in PV (Gleeson and Almey, 1994). We believe investigation into graded exercise intensities would elucidate whether the magnitude of the expansion is proportional to exercise intensity.

CONCLUSION

Finally, the role of post-exercise hypotension on PV should be investigated. Nagashima et al., (1999) has demonstrated that hypotension is crucial in the induction of hypervolemia as it creates a favorable gradient for fluid movement into the vascular space. It may be advantageous to prolong or accentuate the hypotension following exercise by postural manipulation or delaying hydration to evoke a significant and observable increase in PV. In conclusion, exercise physiologists aiming to

facilitate performance by inducing hypervolemia after exercise training should not use the protocols investigated in the present study. A greater understanding of the stimulus of exercise-induced hypervolemia is required by exercise physiologists if they are to prescribe appropriate strategies to evoke hypervolemia.

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KEY POINTS

- It may be advantageous to prolong or accentuate the hypotension following exercise by postural manipulation or delaying hydration to evoke a significant and observable increase in PV.
- A greater understanding of the stimulus of exercise-induced hypervolemia is required by exercise physiologists if they are to prescribe appropriate strategies to evoke hypervolemia.

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