The Effects of High-Intensity Intermittent Exercise Compared With Continuous Exercise on Voluntary Water Ingestion

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Water intake occurs following a period of high-intensity intermittent exercise (HIIE) due to sensations of thirst yet this does not always appear to be caused by body water losses. Thus, the aim was to assess voluntary water intake following HIIE. Ten healthy males (22 ± 2 y, 75.6 ± 6.9 kg, VO2peak 57.3 ± 11.4 m·kg⁻¹·min⁻¹; mean± SD) completed two trials (7–14 d apart). Subjects sat for 30 min then completed an exercise period involving 2 min of rest followed by 1 min at 100% VO2peak repeated for 60 min (HIIE) or 60 min continuously at 33% VO2peak (LO). Subjects then sat for 60 min and were allowed ad libitum water intake. Body mass was measured at start and end of trials. Serum osmolality, blood lactate, and sodium concentrations, sensations of thirst and mouth dryness were measured at baseline, postexercise and after 5, 15, 30, and 60 min of recovery. Vasopressin concentration was measured at baseline, postexercise, 5 min, and 30 min. Body mass loss over the whole trial was similar (HIIE: 0.77 ± 0.50; LO: 0.85 ± 0.55%; p = .124). Sweat lost during exercise (0.78 ± 0.22 vs. 0.66 ± 0.26 L) and voluntary water intake during recovery (0.416 ± 0.299 vs. 0.294 ± 0.295 L; p < .05) were greater in HIIE. Serum osmolality (297 ± 3 vs. 288 ± 4mOsmol·kg⁻¹), blood lactate (8.5 ± 2.7 vs. 0.7 ± 0.4 mmol·L⁻¹) and vasopressin (9.91 ± 3.36 vs. 4.43 ± 0.86 pg·ml⁻¹) concentrations were higher after HIIE (p < .05) and thirst (84 ± 7 vs. 60 ± 21) and mouth dryness (87 ± 7 vs. 64 ± 23) also tended to be higher (p = .060). Greater voluntary water intake after HIIE was mainly caused by increased sweat loss and the consequences of increased serum osmolality mainly resulting from higher blood lactate concentrations.

Keywords: osmolality, lactate, thirst

An increase in serum osmolality causing an increased release of vasopressin has been proposed as one of the mechanisms resulting in the sensation of thirst and water replacement (Stricker & Verbalis 1988). Following the onset of exercise, loss of water from the vascular space results in a rise in serum osmolality (Convertino et al., 1981). During and following continuous exercise, the resultant effect of increased osmolality and vasopressin release on voluntary water intake has been extensively studied (Cheuvront & Haymes 2001; Dugas et al., 2009; Wong et al., 1998), yet the effect on water intake following a bout of high-intensity intermittent exercise (HIIE) is less well known. During and following HIIE, there is an increase in blood lactate concentration, which has been linked to the prevention of serum sodium uptake from the vascular space to the intracellular space, resulting in an increased serum osmolality (Nose et al., 1991). Nose et al. explored the link between exercise intensity, plasma lactate and plasma sodium concentrations. As submaximal exercise intensity increased, a significant rise in plasma sodium concentrations was observed which correlated strongly with changes in plasma lactate concentrations.

Bouts of high-intensity exercise have also been shown to result in an increase in vasopressin release (Hew-Butler et al., 2008). For example, Hew-Butler et al. found that on completion of a maximal oxygen uptake test, vasopressin concentrations were significantly elevated compared with a submaximal bout of continuous exercise. However, subsequent water intake was not assessed so the effect of increased vasopressin release on sensations of thirst and voluntary water intake could not be established.

Following a period of high-intensity exercise there is a shift in water from the vascular to the interstitial and intracellular spaces (Convertino et al., 1981; Nose et al., 1991; Sjøgaard et al., 1985). The movement of hypotonic water out of the vascular space will contribute to the rise in plasma osmolality. Sjøgaard et al. analyzed extra- and intracellular muscle water shifts following one-legged dynamic knee-extensions in six males. They attributed the movement of water to the interstitial space due to an increase in blood pressure and to an increase in perfused...
Despite the known effect of HIIE on the rise in serum osmolality and an increase in vasopressin release, the effect on subsequent voluntary water intake is unknown. A rise in osmolality above the vasopressin release threshold of approximately 285 mOsmol·kg\(^{-1}\) (Thompson et al., 1986) will lead to maximal antidiuresis, resulting in an osmotically driven thirst signal, thus facilitating water intake. Following a bout of HIIE, increased serum osmolality above values experienced following continuous exercise of matched work, may result in a greater osmotic signal, ultimately leading to increased water intake. Excessive water intake may result in weight gain, which for weight bearing sports such as running may impair performance. In addition water intake may lead to increased urine output, which along with increased inconvenience may result in increased water losses (Wong et al., 1998).

Depending on the duration of the HIIE, sweat losses may not be large enough to result in sensations of thirst (Wolf, 1950) or may result in a level of dehydration that will impair performance (Sawka et al., 2007). Sensations of thirst have been shown to increase and result in voluntary water intake when body mass losses, reach and increase beyond approximately 0.8% (Wolf, 1950), while body mass losses of less than 2% can be tolerated without decrement in exercise performance (Sawka et al., 2007). However, despite this, water intake will usually occur after HIIE and suggests a mechanism independent of water losses is acting to increase sensations of thirst and subsequent voluntary water intake (Nose et al., 1991). As increased blood lactate concentration has been shown to affect serum sodium concentration and therefore serum osmolality (Nose et al., 1991), the question arises as to the influence of increased blood lactate concentrations on sensations of thirst and subsequent voluntary water intake.

It was hypothesized that the increase in lactate concentration, resulting from the high-intensity intermittent exercise, would increase serum sodium concentration and thus, serum osmolality, in turn causing increased sensations of thirst and subsequent voluntary water intake and also increased vasopressin release.

**Methods**

**Subjects**

Ten healthy male subjects (age 22 ± 2 years, mass 75.6 ± 6.9 kg, height 1.78 ± 0.08 m, \(\dot{V}O_{2\text{peak}}\) 57.3 ± 11.4 ml·kg\(^{-1}\)·min\(^{-1}\); mean ± SD) were recruited to take part in two trials, undertaken in a counterbalanced order. All subjects had the experimental protocol explained to them verbally and in writing. Subjects provided written informed consent and the experiment was approved by the Loughborough University Ethical Advisory Committee.

**Experimental Protocol**

Subjects were asked to visit the laboratory on four separate occasions for an \(\dot{V}O_{2\text{peak}}\) test, a familiarization trial and two experimental trials (high-intensity intermittent [HIIE] and continuous [LO] exercise). During the first visit, \(\dot{V}O_{2\text{peak}}\) was measured using a discontinuous incremental test to volitional fatigue on an electrically braked cycle ergometer (Lode Corival; Lode BV, Groningen, Netherlands). For later analysis and had nude body mass measured. Subjects visited the laboratory a further three times for the familiarization trial and two experimental trials. The familiarization trial was identical to the HIIE trial. Before each experimental trial subjects were asked to consume 500 ml of water two hours before arrival at the laboratory to ensure they were in a euhydrated state and to arrive after an overnight fast. In the 24 hr before the first experimental trial, subjects were asked to record their dietary intake (food and drink consumed, amount and method of preparation) and refrain from strenuous physical activity and consumption of alcohol. They were then asked to repeat this before each subsequent trial.

The experimental trials were separated by a period of 7–14 days and began in the morning at the same time for each subject. Experimental trials were identical apart from the exercise performed. A schematic outline of the experimental trial is presented in Figure 1. Experimental trial order was decided by incomplete Latin square design and subjects did not know which trial they were participating in when arriving at the laboratory for the first trial.

In each trial, on arrival, subjects voided and the whole urine volume was measured and a 5 ml sample retained for later analysis and had nude body mass measured. Subjects were asked to insert a rectal thermistor 10 cm past the anal sphincter. Skin thermistors were attached at the chest, tricep, thigh, and calf and a heart rate monitor was positioned (Polar Vantage; Kempele, Finland). Core (\(T_c\)) and skin temperature (\(T_s\)) were measured continuously throughout the trials and a minute average was taken every 10 min (BIOPAC MP100 System; BIOPAC, Santa Barbara, CA, USA). Mean weighted skin temperature was calculated using the formula outlined by Ramanathan (1964). Subjects sat for 30 min to account for postural alterations in blood volume at 19.7 ± 1.1 °C and 30.7 ± 10.5% relative humidity (RH). Baseline heart rate values every 10 min were recorded and a 100 mm visual analog subjective feelings questionnaire comprising of thirst and dry mouth scales was administered at the completion of the 30 min seated rest (0 mm = not at all thirsty/mouth.
During the rest period a 21g cannula (Surflo, Terumo, Leuven, Belgium) was inserted into a superficial vein on the forearm to allow venous blood sampling. The line was flushed with 2–3 ml of heparinized saline. A baseline (B) blood sample (7.5 ml) was collected at the end of the rest period. Subjects then cycled for a period of 60 min in 24.9 ± 0.7 °C and 51.1 ± 2.1% RH. In the HIIE trial, they rested for 2 min and then performed 1 min of cycling at a power output attempted to equal the maximum power achieved when recording VO₂peak (305 ± 55 W), however exact total work performed during the HIIE trial was not measured. This was repeated 20 times during the 60 min period. In the LO trial, subjects cycled continuously at 33% of their peak power output for 60 min (102 ± 18 W). Every 10 min in the LO trial, heart rate was recorded and subjects were asked to provide a rating of their perceived exertion (RPE) and thermal sensation. In the HIIE trial, this was performed at the end of a HIIE bout closest to the completion of a 10-min period. Immediately following completion of exercise (postexercise, PE), a blood sample (7.5 ml) was collected and thirst and dry mouth subjective feelings questionnaires were completed. Subjects voided, the volume was measured and a 5 ml sample was retained for later analysis and they then had nude body mass measured. After completion of the body mass measurement, subjects were allowed to leave the laboratory. Ambient temperature and relative humidity was measured at 10-min intervals (RH85 Digital Thermo-Hygrometer; Omega, Manchester, UK).

**Sample Analysis**

For each 7.5 ml venous blood sample, 1.0 ml was aliquoted and mixed with anticoagulant (K⁺ EDTA; 1.5 mg·ml⁻¹) for analysis of hemoglobin concentration, hematocrit and glucose concentration. A further 5.0 ml was aliquoted and mixed with anticoagulant (K⁺ EDTA; 1.5 mg·ml⁻¹) and from this, plasma was separated and frozen at −80 °C for later analysis of hormone concentration. The remaining blood (~2.0 ml) was allowed to clot and was centrifuged at 3,000 rpm for 15 min at 4 °C before the serum was removed and later analyzed for potassium and sodium concentration by flame photometry (Corning Clinical Flame Photometer 410C; Corning Ltd., Halstead, Essex, UK) and osmolality analysis by freezing point depression (Gonotec Osmomat auto Cryoscopic Osmometer; Gonotec, Berlin, Germany). Hemoglobin concentration was measured in duplicate using the cyanmethemoglobin method. Hematocrit was determined by microcentrifugation and measured in triplicate. Using the method of Dill and Costill (1974), blood and plasma volume changes were calculated from hemoglobin concentrations and hematocrit values. A 100 μl sample of anticoagulated blood was pipetted into 0.3 M perchloric acid in a ratio of 1:10 in duplicate for analysis.
of glucose by the GOD-PAP method (Randox Laboratories Ltd., Crumbin, UK) and lactate by fluorimetry using the method outlined by Maughan (1982). Plasma arginine vasopressin and aldosterone concentrations were measured by enzyme immunoassay (Enzyme Immunoassay; Enzo Life Sciences, Ann Arbor, MI, USA) using 100 μl samples. Samples were measured in duplicate.

The total volume of each urine sample was measured and a 5 ml sample was retained. This was analyzed for osmolality through freezing point depression (Gonotec Osmomat auto Cryoscopic Osmometer; Gonotec, Berlin, Germany).

Statistical Analysis

Data were checked for normality of distribution using Shapiro-Wilk tests. All samples were normally distributed and, subsequently, either paired samples t tests or repeated-measures ANOVA was performed. Post hoc paired samples t tests with Bonferroni correction were performed to identify where statistical differences occurred when significant main or interaction effects were observed. Linear regression values and Pearson’s product moment correlation coefficients were calculated when appropriate. Linear regression was used to examine the change in plasma vasopressin associated with the change in serum osmolality. Correlation analysis was calculated between variables deemed to be closely related in terms of physiological and behavioral mechanisms related to water balance. Statistical significance was accepted when p < .05. Data expressed as mean ± SD.

Results

Blood samples were collected from eight subjects due to cannulation problems in two subjects. Despite not being continuous data, the time points in Figures 3, 4, 5, and 6 are joined as the points represent progressive time points throughout the trials.

Baseline Values

There was no difference in baseline body mass between the HIIE (75.57 ± 7.28 kg) and LO trial (75.71 ± 6.98 kg; p = .496). Similar baseline values for urine osmolality (510 ± 248 vs. 507 ± 270 mOsmol·kg⁻¹ for HIIE and LO trials respectively), serum osmolality (285 ± 4 vs. 284 ± 3 mOsmol·kg⁻¹ for HIIE and LO trials respectively) and subjective feelings of thirst (53 ± 21 vs. 40 ± 15 for HIIE and LO trials respectively) and mouth dryness (52 ± 22 vs. 42 ± 16 for HIIE and LO trials respectively) were observed, suggesting subjects arrived in a similar state of euhydration (Sawka et al., 2007; p > .05).

Water Balance

Body mass loss from the beginning of the trial until after the recovery period following voluntary water intake was similar between trials (0.77 ± 0.50 vs. 0.85 ± 0.55% for HIIE and LO trials respectively; p = .124). Sweat loss was greater in the HIIE trial (0.78 ± 0.22 l) compared with the LO trial (0.66 ± 0.26 l; p = .009). In the HIIE trial, subjects consumed more water during the recovery period (p < .0001; Figure 2) but this difference was solely due to a higher water intake during the first 30 min of recovery (p = .006) while during the final 30 min of the recovery period, water intake was similar (p = .094). The increase in water intake between the LO and HIIE trials was positively correlated with the increased sweat losses that also occurred (r = .731, p = .534). Expressed as a percentage, the amount of water lost that was replaced was higher in the HIIE trial compared with the LO trial (44 ± 29 vs. 35 ± 34%; p = .012). In the HIIE trial, one subject drank more than the water lost (104%), and the next highest replacement value was 77%. In the LO trial, two subjects replaced 90–100% with the remaining subjects replacing less than 51% of the water lost during exercise. Negating water intake, body mass losses from baseline would have been similar: 1.34 ± 0.36% in the HIIE trial and 1.26 ± 0.39% in the LO trial (p = .205), with only one subject in both trials losing enough water to elicit a greater than 2% body mass loss. The difference in percentage body mass lost when water was included and negated in the calculation was greater in the HIIE trial (p < .0001). There was no difference in urine output at the end of the trial (0.23 ± 0.12 vs. 0.28 ± 0.12 l for HIIE and LO trials respectively; p = .203).

Serum osmolality was higher in the HIIE trial postexercise (p = .006) and after 5 (p = .048) and 30 min (p < .001) of the recovery period (Figure 3a). Serum osmolality values were similar across all sample points in the LO trial (p > .05) but were elevated above baseline and the recovery period samples postexercise in the HIIE trial (p ≤ .015). In the HIIE trial values had returned to baseline following 5 min of recovery (p > .05).

Figure 2 — Voluntary water intake during each trial. †Denotes difference between trials. *Denotes different from 30 to 60 min (p < .05).
Serum sodium concentrations postexercise were higher in the HIIE trial compared with the LO trial ($p = .018$; Figure 3b). In the HIIE trial, postexercise concentrations were greater compared with baseline and during the recovery period ($p \leq .015$) and had returned to baseline after 5 min of the recovery period ($p > .05$). In the LO trial serum sodium concentrations did not increase above baseline ($p > .05$). Serum potassium concentrations were the same at baseline and similar between trials and sample points (baseline: $4.4 \pm 0.3$ vs. $4.4 \pm 0.3$ mmol·L$^{-1}$; postexercise: $5.1 \pm 0.3$ vs. $4.9 \pm 0.4$ mmol·L$^{-1}$; 5 min: $4.4 \pm 0.3$ vs. $4.5 \pm 0.3$ mmol·L$^{-1}$; 15 min: $4.6 \pm 0.3$ vs. $4.5 \pm 0.3$ mmol·L$^{-1}$; 30 min: $4.5 \pm 0.3$ vs. $4.5 \pm 0.3$ mmol·L$^{-1}$ and 60 min: $4.6 \pm 0.3$ vs. $4.4 \pm 0.2$ mmol·L$^{-1}$; $p > .05$).

**Blood Analysis**

At baseline, blood lactate concentrations were similar between trials ($p = .914$) but increased during exercise and remained elevated throughout the recovery period ($p \leq .006$; Figure 4). In the HIIE trial, blood lactate concentrations peaked postexercise and remained elevated above baseline values until 30 min of the recovery period ($p \leq .015$).

Plasma vasopressin concentrations were higher in the HIIE trial at postexercise and after 5 and 30 min of the recovery period ($p < .05$; Figure 5a). In the HIIE trial, postexercise vasopressin concentrations increased from baseline ($p = .048$), had a tendency to remain elevated above baseline after 5 min of recovery ($p = .054$) and...
were elevated above baseline values after 30 min of the recovery period ($p < .05$). In the LO trial concentrations did not change from baseline ($p > .05$). In both the HIIE and LO trials, aldosterone concentration did not change from baseline ($p > .05$; Figure 5b) but after 30 min of the recovery period aldosterone concentrations were greater in the HIIE trial compared with the LO trial ($p = .048$).

Hemoglobin concentrations increased from baseline ($156 \pm 7$ and $158 \pm 7$ g·L$^{-1}$ for HIIE and LO respectively) to postexercise ($171 \pm 7$ and $163 \pm 8$ g·L$^{-1}$ for HIIE and LO respectively) in both trials ($p < .05$). Hemoglobin concentrations were higher postexercise in the HIIE trial ($p < .05$) but had returned to baseline concentrations after 15 min (HIIE) and 5 min (LO) of the recovery period. A similar response was found for hematocrit values with an increase from baseline to postexercise in the HIIE (44.0 ± 2.5 to 48.2 ± 2.3%; $p < .05$) and LO (44.5 ± 2.0 to 45.7 ± 2.4%; $p < .05$) trials. Hematocrit values were higher postexercise in the HIIE trial ($p < .05$) and returned to baseline at the same rate as hemoglobin concentrations.

Plasma volume change from baseline was greater in the HIIE trial compared with the LO trial at postexercise and after 5, 15, and 30 min of the recovery period ($p < .05$; Figure 6a). In the HIIE trial plasma volume was different...
compared with baseline at postexercise and after 5 and 15. In the LO trial plasma volume changes from baseline at each sample point were similar \((p > .05)\). Blood volume changes from baseline were greater in the HIIE trial compared with the LO trial at postexercise and after 5, 15, and 30 min of the recovery period \((p < .05)\; \text{Figure 6b}\). In the HIIE trial, blood volume decreased from baseline at postexercise and after 5 and 15 min of the recovery period \((p < .0001)\) before returning to baseline values. In the LO trial blood volume had decreased from baseline at postexercise \((p < .05)\) but had returned to baseline values by 5 min of the recovery period \((p > .05)\).

**Subjective Feeling Questionnaires**

Reported sensations of thirst peaked in both trials postexercise (Figure 7a) and tended to be higher in the HIIE trial compared with the LO trial at the postexercise sample \((p = .060)\). In the HIIE trial, postexercise reported sensations of thirst were greater than baseline and during the recovery period \((p < .0001)\). Reported sensations of mouth dryness peaked in both trials postexercise (Figure 7b) and tended to be higher in the HIIE trial compared with the LO trial at postexercise \((p = .060)\). There was no difference between trials at the other sample points \((p > .05)\). In the HIIE trial postexercise reported sensations of mouth dryness were greater than baseline and during the recovery period \((p < .0001)\).

**Correlations**

In the HIIE trial serum sodium concentrations were positively correlated to blood lactate concentrations and serum osmolality (Table 1). Serum osmolality was positively correlated to blood lactate concentrations, vasopressin and aldosterone concentrations, and sensations of thirst and mouth dryness. Vasopressin concentrations were positively correlated to blood lactate concentrations but there was no correlation with aldosterone concentrations. In the LO trial correlations were not found between serum osmolality, serum sodium concentrations, blood lactate concentrations and vasopressin and aldosterone concentrations.

**Core and Skin Temperature**

Core temperature peaked at the end of exercise in both the HIIE \((38.2 ± 0.3 \, ^\circ C)\) and LO \((37.6 ± 0.3 \, ^\circ C)\) trials. Core temperature was greater at 30, 40, 50 and 60 min of the exercise period and remained elevated after the first 10 min of the recovery period in the HIIE trial compared with the LO trial \((p < .05)\). Skin temperatures were similar between trials at all time points, and mean skin temperature over the duration of the trials was similar \((31.6 ± 1.1 \, vs. \, 31.6 ± 1.2 \, ^\circ C)\) for HIIE and LO trials respectively \((p > .05)\).

**Heart Rate, Rating of Perceived Exertion, and Thermal Sensation**

During the exercise period of the trials, heart rate was significantly higher during the HIIE trial \((158 ± 12 \, vs. \, 110 ± 10 \, \text{beats·min}^{-1}, p < .0001)\), with differences occurring at 10, 20, 30, 40, 50, and 60 min of exercise. Thermal sensation was higher after 20 \((5 ± 1 \, vs. \, 3 ± 1, p < .0001)\), 30 \((6 ± 1 \, vs. \, 4 ± 1, p < .0001)\), 40 \((6 ± 1 \, vs. \, 4 ± 1, p < .0001)\), 50 \((6 ± 1 \, vs. \, 4 ± 1, p < .0001)\) and 60 min \((6 ± 2 \, vs. \, 4 ± 1, p < .0001)\) of the exercise period in the HIIE trial. There was no difference in thermal sensation during the baseline and recovery periods \((p > .05)\). Ratings of perceived exertion were higher in the HIIE trial after 10 \((14 ± 2 \, vs. \, 10 ± 2, p = .006)\), 20 \((15 ± 1 \, vs. \, 11 ± 2, p < .0001)\), and 30 min \((16 ± 2 \, vs. \, 12 ± 2, p < .0001)\) of the recovery period.

![Figure 7](image-url) — Sensations of thirst (a) and mouth dryness (b) over the duration of each trial. *Different to baseline in the HIIE trial. #Different to post exercise in the HIIE trial. ^Different to 5 min in the HIIE trial \((p < .05)\). B = baseline sample. PE = postexercise sample.
High-Intensity Exercise and Voluntary Water Intake

Discussion

The aim of the study was to assess voluntary water intake following either a period of HIIE or continuous exercise. The exercise conditions were chosen to generate significant differences in blood lactate concentrations to examine the effects on the physiological mechanisms responsible for voluntary water intake. The 60 min of exercise under the two conditions achieved approximately the same workloads (366 kJ) and a clearly significant difference in blood lactate concentrations.

Water intake in the HIIE trial was greater during the first 30 min after exercise. Despite the greater water intake, body mass loss was similar between trials primarily due to the increased sweat loss recorded in the HIIE trial. It appeared that the increased water intake could be mainly related to the increased sweat losses. Body mass loss in both trials was greater than 0.8% and so would have stimulated sensations of thirst (Wolf, 1950) yet were not greater than 2% suggesting that rapid rehydration through water intake may have not been necessary (Sawka et al., 2007).

However, there was a wide range in the individual response to water intake with the amount of water replaced (12–104% in the HIIE trial and 0–94% in the LO trial), indicating that water intake replacement was highly variable. Nevertheless the water replacement helped alleviate thirst sensations in the HIIE trial indicated by the reduction in reported sensations of thirst following the onset of the water intake period. This adds strength to the notion that when ad libitum water is allowed then individuals consume sufficient amounts to alleviate sensations of thirst despite not replacing all of the water lost during exercise (Noakes, 2007). It must be noted that the sensation of thirst is complicated and can be affected and influenced by additional variables.

Table 1 Correlation Coefficients Between Measured Variables in Each Trial

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*p < .05 ; **p < .0001
not measured in the current study. In addition to those measured, oropharyngeal reflexes, absorption rate and fluid capacity of the gastrointestinal system (Greenleaf, 1992) and stomach distension (Rolls et al., 1980) have all been linked to sensations of thirst. Therefore, it would seem that in the current study, the variables measured would not completely explain all of the variance in thirst and voluntary water intake.

The sensation of thirst has been suggested to arise when serum osmolality is greater than 290 mOsmol·kg⁻¹ (Phillips et al., 1985). This may provide explanation as to why subjects in the current study consumed water despite body mass losses not greater than 2%. In the HIIE trial, osmolality first increased above the threshold value postexercise before decreasing below this value between 15 and 30 min of the recovery period, while in the LO trial, osmolality values did not increase above 290 mOsmol·kg⁻¹. In the HIIE trial, despite elevated serum osmolality after 5 and 15 min of the recovery period, sensations of thirst had decreased from peak postexercise values following the onset of drinking. It appears that the premise of a threshold for thirst works only to initiate drinking and once this has occurred then the effect of the threshold for thirst is diminished. As a result, water intake during the final 30 min of the HIIE trial was similar to the LO trial confirming that satiation of water intake occurs quickly following an initial bout of drinking (Rolls et al., 1980). Closer monitoring of the water intake period, particularly during the first 5 min would have allowed greater interrogation of water intake behavior in response to exercise.

It has been hypothesized that the efflux of sodium ions from the vascular space to the intracellular space is reduced by the negatively charged lactate ions produced following HIIE, thus causing an increase in serum sodium concentration and subsequently, serum osmolality. In the current study the difference in serum osmolality at postexercise between trials was approximately 10 mOsmol·kg⁻¹, while the difference in serum sodium concentration was approximately 3 mmol·L⁻¹. Using the formula assessed by Worthley et al. (1987), the change in serum osmolality (12 mOsmol·kg⁻¹) in the HIIE trial from baseline to after exercise was not completely accounted for by the change in serum sodium concentration (2 × 4 mmol·L⁻¹). Therefore it appeared that the change in blood lactate concentration was a direct contributing factor to the increase in serum osmolality (contribution of 4 mOsmol·kg⁻¹). The effect of blood lactate concentrations on serum osmolality values, both directly and indirectly through the increase of serum sodium concentrations, would have contributed to the osmotically driven release of vasopressin and potentially contributed to the increased consumption of water in the HIIE trial (Phillips et al., 1985).

Before the onset of thirst stimulated water intake, vasopressin is released to increase water reabsorption in the kidneys (Bankir, 2001). Vasopressin release will increase until maximum antidiuresis has been reached (Thompson et al., 1986). In the current study there was a large increase in plasma vasopressin concentration following the high-intensity exercise when serum osmolality values were above the reported threshold value. Vasopressin concentration remained elevated above baseline values throughout the HIIE trial, consistent with serum osmolality remaining above the threshold value outlined. Vasopressin concentration has been widely shown to decrease quickly (2.5–15 min) following initiation of drinking (Burrell et al., 1991; Figaro & Mack, 1997; Geelen et al., 1984; Seckl et al., 1986). However, in these latter studies serum osmolality decreased either at a similar rate (Burrell et al., 1991) or at a slightly delayed rate (30–60 min; Seckl et al., 1986). In the current study vasopressin concentration remained elevated, while thirst sensations decreased after the initial postexercise peak. Again, this is perhaps related to sensations of thirst becoming quickly satiated once water intake occurs and also suggests that vasopressin is not a direct stimulus of thirst. Increased serum osmolality increases vasopressin concentrations and sensations of thirst, however it appeared that in the current study, water intake satiated sensations of thirst quickly, whereas serum osmolality and vasopressin concentrations were more delayed in returning to baseline levels following water intake. In conjunction with the decrease in water intake during the final 30-min period it would also suggest that the increased blood lactate concentration and serum osmolality relationship may have had an effect on maintaining vasopressin concentrations.

Although the total work rates performed throughout the trials were similar they were not matched precisely. However, the study was effective in achieving its purpose of generating significant differences in blood lactate concentrations between exercise conditions.

During the recovery period it appeared that two main variables were influencing the decrease in serum osmolality from postexercise peak values. The reduction of blood lactate concentration and the intake of water both contributed to decreasing serum osmolality. As the effect of no water intake was not assessed, determining the relative contributing effect of each variable was difficult. However, when the effect of preventing or delaying water intake following HIIE was assessed, similar decreases in serum osmolality, blood lactate, and serum sodium concentrations were found (Mears & Shirreffs, unpublished data). Delaying water intake resulted in a similar voluntary water intake despite reduced serum osmolality values, suggesting that once the desire to drink arose, sensations remained until satiated.

**Conclusion**

In conclusion, water intake following a period of HIIE was greater than an exercise period of low intensity continuous exercise. The increased water intake in the HIIE trial was mainly attributed to the increased water losses. In addition, the result of an increase in serum osmolality and subsequent vasopressin release caused by an increased blood lactate concentration in combination
with an increased serum sodium concentration may have also contributed to the increased water intake.

Acknowledgments

This research was, in part, funded by a grant from the European Hydration Institute.

References


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