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Do male 100 km ultra-marathoners overdrink?

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Running head: Hyponatremia in ultra-runners

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Abstract

**Purpose:** Fluid overload is considered a main risk factor for exercise-associated hyponatremia (EAH). The aim of this study was to investigate the incidence of EAH in ultra-runners at the 100 km ultra-run in Biel, Switzerland.

**Methods:** Pre and post race, body mass, urinary specific gravity, hemoglobin, hematocrit, plasma [Na⁺], and plasma volume were determined.

**Results:** Of the 145 finishers, seven runners (4.8 %) developed asymptomatic EAH. While running, the athletes consumed a total of (median and IQR) 6.9 (5.1-8.8) L over the 100 km distance, equal to 0.58 (0.41-0.79) L/h. Fluid intake correlated negatively and significantly with race time (r = -.50, P < .0001). Body mass decreased, plasma [Na⁺] remained unchanged, hematocrit and hemoglobin decreased, urinary specific gravity increased. Plasma volume increased by 4.6 (- 2.3 – 12.8) %. Δ body mass correlated with both post race plasma [Na⁺] and Δ plasma [Na⁺]. Post race plasma [Na⁺] correlated to Δ plasma [Na⁺]. Fluid intake was neither associated with post race plasma [Na⁺] nor with Δ plasma [Na⁺]. Fluid intake was related to Δ body mass (r = .21, P = .012), but not to post race body mass. Fluid intake showed no correlation to Δ plasma volume. Δ plasma volume was associated with post race [Na⁺].

**Conclusions:** Incidences of EAH in 100 km ultra-marathoners were lower compared to reports on marathoners. Body mass decreased, plasma volume increased and plasma [Na⁺] was maintained. Since fluid intake was neither related to Δ plasma volume nor to Δ plasma [Na⁺], we assume that factors other than fluid intake maintained body fluid homeostasis.

**Key words:** hyponatremia - body weight – sodium – hydration status – fluid
INTRODUCTION

Exercise-associated hyponatremia (EAH) - defined as serum [Na⁺] < 135 mmol/L and described first in 1985 by Noakes et al.¹ as being due to ‘water intoxication’ - is a well-known and a well described fluid and electrolyte disorder in marathoners, where the incidence of EAH amounts to about 22% depending upon the number of investigated athletes, their gender and fitness level.²⁻⁷ Risk factors for EAH are exercise duration of more than four hours, slow exercise pace, female gender, low body mass, excessive drinking of more than 1.5 L/h while racing, pre-exercise hyperhydration, abundant availability of drinking fluids at the event, nonsteroidal anti-inflammatory drugs, and an extremely hot or extremely cold environment.⁸⁻¹¹ In recent years, non-osmotic secretion of arginine-vasopressin, combined with high fluid availability plus sustained fluid intake, has been hypothesized as leading to an increase in EAH.⁹,¹² Other potential mechanisms in the pathogenesis of EAH include sweat sodium loss, inability to mobilize exchangeable sodium stores, metabolic water production, impaired renal blood flow and glomerular filtration rate.⁹

While there is abundant literature about EAH in marathoners,²⁻⁷ studies investigating EAH in ultra-marathoners are rare.¹³⁻¹⁶ Since ultra-marathoners run at a slow pace¹⁶,¹⁷ they may be at an especially high risk of fluid overload.⁸⁻¹¹ In a recent study of 45 male ultra-marathoners in a 161-km ultra-marathon, 51.2% of the finishers presented with EAH.¹⁸ The authors discussed the significantly longer nature of a 161-km ultra-marathon compared to a classic marathon as a risk factor for EAH.

The aim of this present study was to investigate the incidence of EAH in male ultra-marathoners in the 100 km ultra-run in Biel, Switzerland. This race is the most famous 100 km run in Europe with about 1,200 finishers each year from all over Europe. The organizers offer a total of 17 aid stations and the athletes may be accompanied by a cyclist providing fluid and nutrition. Since a slow running pace and excessive drinking behavior⁸⁻¹¹, with high frequency of fluid consumption², is considered as the main risk factor for fluid overload, and thus subsequently developing EAH, we hypothesized that the incidence of EAH would be higher in these 100 km ultra-marathoners compared to the existing reports on marathoners. In cases of fluid overload, we would expect, post race, an increase in body mass and a decrease in plasma sodium.
METHODS

Subjects
Data was collected over four consecutive years in a 100 km ultra-run; the 100 km run in Biel, Switzerland, in order to increase the sample size. The Race Director contacted all the participants, in the years 2007 to 2010, via a separate newsletter at the time of inscription, in which they were asked to participate in the study. About 1,500 male ultra-runners started in the race each year; a total of 157 male ultra-runners volunteered to participate in our investigation over this four year period. The athletes were informed of the procedures and gave their informed written consent. The study was approved by the Institutional Review Board for use of Human Subjects of St. Gallen, Switzerland. Age, body mass, body mass index, training and pre race experience of the subjects is represented in Table 1.

Design
The 100 km run in Biel, Berne, Switzerland, generally takes place during the night of the first weekend in June. The athletes start the 100 km run at 10:00 p.m. They have to climb a total altitude of 645 meters. During these 100 km, the organizer provides a total of 17 aid stations offering an abundant variety of food and beverages such as hypotonic sports drinks, tea, soup, Coca Cola® and water. The athletes are allowed to be supported by a cyclist in order to have additional food and clothing, if necessary. In all four years, the general weather conditions were comparable, with the temperature at the start being 15 to 18 °C, night lows of 8 to 10 °C, and daily highs of 25 to 28 °C the following day. There was no rain or wind.

Methodology
Before the start of the race and after arrival at the finish line, every subject underwent determination of body mass, and the collection of capillary blood and urinary samples. Body mass was measured using a commercial scale (Beurer BF 15, Beurer GmbH, Ulm, Germany) to the nearest 0.1 kg. Body height was determined using a stadiometer to the nearest 0.01 m. Capillary blood samples were taken from the fingertip; plasma [Na⁺], hemoglobin and hematocrit were analyzed using the i-STAT® 1 System (Abbott Laboratories, Abbott Park, IL, USA). Standardization of posture prior to blood collection was respected since postural changes can influence blood volume and therefore hemoglobin concentration and hematocrit. Percentage changes in plasma volume (%ΔPV) were calculated from pre- and post-exercise levels of hematocrit (Hct) and hemoglobin (Hb), following the equation of Strauss et al.19 Urinary specific gravity was analyzed using Clinitek Atlas® Automated Urine Chemistry Analyzer (Siemens Healthcare Diagnostics, Deerfield, IL, USA). Fluid intake was estimated according to the reports of the athletes. During the race, the athletes recorded their intake of fluids on a sheet of paper which they carried with them during the run. At each aid station, they marked the number of consumed cups. In addition, all supplemental fluid intake provided by the support crew was recorded. Upon inscription to the investigation participants were instructed to keep a comprehensive training diary until the start of the race. All training units in running were recorded showing the distance in kilometers and the duration. In addition, every athlete indicated his number of finished marathons on a flat course and his personal best time in marathon running achieved on a flat course, including the year when this time was achieved. Furthermore, the number of finished 100 km runs and the personal best time was also recorded. At arrival for the post race measurements, the athletes were asked for symptoms of EAH.9-11
Statistical Analysis
The Shapiro-Wilk test was applied to check for normal distribution of data. Data is presented as mean and interquartile ranges (IQR) and mean and standard deviation (SD) as appropriate. Student’s t-tests or the Wilcoxon signed rank tests were used to compare anthropometric and laboratory parameters before and after the race as appropriate. The correlations of the changes in the parameters during the race were evaluated using Pearson Product-Moment and Spearman rank-order correlation analysis as appropriate. For all statistical tests, significance was set at a level of 0.05.
RESULTS

Among these 157 ultra-runners, 145 athletes finished the 100 km ultra-run within 699 (620-775) min, competing at an average running speed of 8.7 (1.4) km/h. Twelve participants did not finish within the time limit of 21 hours. One athlete dropped out due to low back pain, four non-finishers suffered overuse injuries of the lower limbs, and seven runners complained about exhaustion. Of the 145 finishers, seven runners (4.8 %) developed asymptomatic EAH.

While running, the athletes consumed a total of (median and IQR) 6.9 (5.1-8.8) L over the 100 km distance, equal to 0.58 (0.41-0.79) L/h. Fluid intake varied between 0.27 L/h to 1.62 L/h and correlated negatively and significantly to race time (Figure 1). Body mass decreased, plasma [Na+] remained stable, hematocrit and hemoglobin decreased, urinary specific gravity increased (Table 2). Plasma volume increased by 4.6 (-2.3 – 12.8) %. Δ body mass correlated with both post race plasma [Na+] (Figure 2) and Δ plasma [Na+] (Figure 3). Post race [Na+] correlated positively and significantly with Δ [Na+] (r = .73, P < .0001). Fluid intake was neither associated with post race plasma [Na+] nor with Δ plasma [Na+]. Fluid intake was related to Δ body mass (Figure 4), but not to post race body mass. Fluid intake showed no correlation to post race urinary specific gravity, Δ urinary specific gravity or Δ plasma volume. Δ plasma volume was associated with post race [Na+] (Figure 5), but not with Δ [Na+], post race body mass, or Δ body mass. Race time showed no association with post race body mass, Δ body mass, post race [Na+], Δ [Na+], post race urinary specific gravity or Δ urinary specific gravity.

The number of completed marathons (r = .23, P = .0077), the personal best marathon time (r = .71, P < .0001) and the personal best 100 km time (r = .77, P < .0001) were related to race time.
DISCUSSION

The aim of this study was to investigate the incidence of EAH in male ultra-marathoners and we hypothesized that the incidence of EAH would be higher in these 100 km ultra-marathoners compared to marathoners. Studies investigating the incidence of EAH in marathoners reported data between 0 % and 28 %. However, the incidence of ~5 % EAH was lower in these 145 ultra-marathoners compared to most reports on marathoners. These different findings of incidence rates might, however, be due to sampling bias. Regarding existing literature on EAH in ultra-marathoners, the incidence of EAH seems to be lower in ultra-marathoners compared to marathoners. In line with our low incidence rate of ~5 %, both Reid and King and Glace et al. found no EAH in ultra-marathoners. In ultra-marathoners, plasma [Na+] and plasma osmolality are well regulated and do not change while drinking ad libitum.

Fluid overload is considered to be a main risk factor for EAH. We hypothesized that the incidence of EAH would be higher in these 100-km ultra-marathoners compared to marathoners, and that fluid intake would be related to both post race body mass and post race plasma [Na+]. In this race, the ultra-marathoners were offered 17 refreshment points over the 100 km, on average every 5.9 km. Our ultra-marathoners consumed – despite the increased availability of aid stations – on average 0.58 (0.41-0.79) L/h. In the ‘Position Statement’ of IMMDA, marathoners should drink no more than 0.4 to 0.8 L/hour ad libitum, according to their thirst. The mean rate of 0.58 (0.41-0.79) L/h of our ultra-marathoners was exactly in line with these recommendations.

An interesting finding was the fact that fluid intake was significantly and negatively related to race time; faster runners were consuming more fluids than slower runners. In marathoners, EAH was associated with increased fluid consumption during the race because slower athletes showed a higher drinking frequency. Although the faster athletes consumed significantly more fluids, race time was neither associated with post race plasma [Na+] nor with Δ plasma [Na+]. This is in contrast to the opinion that high fluid consumption leads to dilutional hyponatremia. The athletes in this race, compared to a marathon race, had the opportunity of being followed by a support crew member, mainly a cyclist. This cyclist can carry food and drinks as well as clothes. We assume that the faster runners also drank fluids between the aid stations, provided by the cyclist. Presumably, a runner finishing in the top 10 would not stop at the refreshment points, but rather consume fluids from the support crew. For example, an athlete finishing within 504 min (8 h 24 min) drank 9.8 L of fluids, equal to 1.34 L/h. Experienced ultra-runners with a fast race time were obviously able to consume rather large amounts of fluids so that neither dehydration nor fluid overload occurred. We assume that the faster runners were followed by a support crew providing fluids, so that their athlete did not have to stop at an aid station in order to drink. Alternatively, we might speculate that the faster runners had greater sweat rates than the slower runners and, therefore, consumed more fluids.

In cases of fluid overload, we expected an increase in body mass and a decrease in plasma [Na+]. Fluid intake was related to Δ body mass in these ultra-marathoners, but not to post
race body mass. Furthermore, fluid intake was neither related to post race [Na⁺] nor to Δ plasma [Na⁺]. In contrast to marathoners, ultra-marathoners seem to be at a lower risk of developing EAH,15,16 although Stuempfle et al. described an incidence of 44% of EAH in ultra-marathoners.25 In a 161-km race no case of hyponatremia occurred, although decreased plasma [Na⁺] after the race was due to fluid overload caused by excessive fluid consumption.26 Also in a 160-km foot race, no case of hyponatremia was found.16 Glace et al. described a significant and negative relationship between fluid intake and post race plasma [Na⁺] in their ultra-marathoners; high fluid intakes were correlated with lower post race plasma [Na⁺].16 Their ultra-runners consumed 19.4 (5.6) L during the 26.2 (0.4) hours, on average 0.74 L/h. This was slightly more compared to our runners with 0.58 (0.41–0.79) L/h, and might be the reason for the lower post race plasma [Na⁺] concentration. In a very recent study of runners in a 161-km ultra-marathon, the incidence of EAH amounted to ~50%.18 The 45 athletes in that study competed for ~26 hours, considerably longer compared to our ultra-runners who completed the 100 km within ~12 hours. Lebus et al. discussed the significantly longer nature of a 161-km ultra-marathon compared to a classic marathon as a risk factor for the increased rate of EAH.18

Weight gain during endurance exercise is associated with EAH.11 The determination of Δ body mass is a useful measure of both fluid intake and fluid retention.27 These 100 km ultra-marathoners, however, lost ~1.8 kg of body mass. A decrease in body mass is – apart from other parameters – a marker of dehydration28,29 and we assume that these 100 km ultra-marathoners were rather dehydrated than overhydrated regarding the change in body mass and urinary specific gravity. Our ultra-marathoners lost ~2.4% of body mass. Following the ‘Statement of the Consensus Conference’, loss of ~2% body mass seems to be preventive against EAH.11 Urinary specific gravity decreased highly significantly post race. Body mass and urinary specific gravity are considered as reliable markers of hydration status.28,29 The post race urinary specific gravity of > 1.020 mg/L indicated significant dehydration; the loss of ~2.4% body mass (~1.8 kg body mass) indicated minimal dehydration.29 The decrease in body mass might, however, also be due to a decrease in solid mass such as fat mass and skeletal muscle mass, as has been shown in ultra-runners.30,31 Body mass changes correlated with both post race plasma [Na⁺] and Δ plasma [Na⁺]. This is in line with the recent findings in marathon runners by Mettler et al., where Δ body mass correlated with both post race plasma [Na⁺] and Δ plasma [Na⁺].6 Post race [Na⁺] correlated to Δ [Na⁺] in their marathoners, as has been found in our 100 km ultra-marathoners. Mettler et al. demonstrated a significant association between post race plasma [Na⁺] and post race plasma osmolality, and they speculated that the increased plasma osmolality might be due to an increased activity of vasopressin.6

Hematocrit and hemoglobin decreased during this ultra-marathon and plasma volume increased by 4.6 (2.3 – 12.8) %. During a marathon, however, plasma volume decreased32,33, as has also been found in an ultra-marathon over 67 km.34 In longer ultra-endurance races, however, plasma volume increased.26,35 Hew-Butler et al. assumed that intensity was responsible for these disparate findings since marathoners compete faster compared to ultra-marathoners. Ultra-endurance athletes may preserve a ‘fluid reserve’ in the interstitial fluid of the extracellular fluid compartment.35 The increase in plasma volume might, however, also be due to an increased activity of aldosterone during a 100 km run.36 Fellmann et al. concluded that the increase in plasma volume after an ultra-endurance race was mainly due to plasma sodium retention.37 In a 24-hour race, they found an increase in plasma volume, aldosterone and vasopressin.38 Stuempfle et al. showed an increased activity of both aldosterone and vasopressin after an ultra-endurance race.26 Presumably, the increase in plasma volume is due to increased activity of both vasopressin and aldosterone.
Event inexperience is one of the athlete-related risk factors for EAH. In marathoners, the number of pre race completed marathons varied between one and eight races. Non-hyponatremic finishers in a marathon had completed more marathons compared to non-hyponatremic ones. In these studies, the incidence rate of EAH amounted to 22%. Our 100 km ultra-runners had completed ~30 marathons pre race, and 98 athletes (67%) had already completed ~6 100 km ultra-runs. Due to the low incidence of EAH in our ultra-marathoners, we assume that pre race experience is an important determinant in minimizing the risk of EAH. The personal best time in a 100 km run showed about the same correlation coefficient compared to a personal best marathon time (r = .71) and 100 km race time (r = .77). A personal best marathon time might be an independent predictor variable for an ultra-running performance; in a 24-hour run, the personal best marathon time was related to total running kilometers.

We focused our study primarily on EAH being due to fluid overload. Apart from an increased activity of aldosterone to maintain plasma [\(\text{Na}^+\)] and plasma volume, some athletes might be able to mobilize sodium from internal stores that are otherwise osmotically inactive. For instance up to one fourth of the total body sodium may exist in bone and cartilage stores, and is not osmotically active but potentially recruitable into an osmotically active form. The exchangeable sodium pool may serve as a buffer for losses of sodium that occur through sweat or urine losses, and can also buffer changes in serum sodium levels that occur with changes in total body water. Apart from these factors, absorption of ingested water in the gastrointestinal tract could lower the plasma [\(\text{Na}^+\)] without changing body weight, and suggest osmotic inactivation of sodium. During exercise, the breakdown of glucose produces an increase in the intracellular concentration of osmotically active particles and may cause transient movement of water into cells with an increase in plasma [\(\text{Na}^+\)].

A limitation of this study was that we did not ask about the intake of nonsteroidal anti-inflammatory medication, which is also considered as a risk factor for EAH. The use of nonsteroidal anti-inflammatory drugs might influence renal function and increase the risk of exercise-associated hyponatremia. Furthermore, we did not ask about additional oral salt supplementation. However, two studies of Ironman triathletes showed that oral sodium supplementation was not necessary to prevent hyponatremia.

**Practical Applications**

These ultra-runners were dehydrated with a decrease in body mass and with post race urinary specific gravity > 1.020 mg/L according to the concept of determination of hydration status. However, regarding the increase in plasma volume, the associations of \(\Delta\) body mass with \(\Delta[\text{Na}^+]\) and respecting recent literature, body fluid must have been increased. Fluid intake was not related to \(\Delta[\text{Na}^+]\) and other factors might have been responsible for fluid regulation in these ultra-endurance runners. We assume that other factors maintained body fluid homeostasis during this ultra-marathon such as a hormonal regulation by vasopressin and aldosterone. In recent studies of marathoners and ultra-marathoners over 56 km, the activity of vasopressin was measured in addition to body mass, plasma [\(\text{Na}^+\)], osmolality and fluid intake. Recent findings suggest that EAH was not only due to fluid overload but also to an increased activity in vasopressin. In future studies of 100 km ultra-runners the activity of vasopressin should also be investigated. Since plasma sodium retention was the major factor in the increase in PV and aldosterone was increased after an ultra-endurance race, the activity of aldosterone should also be determined. This might provide more insight into fluid and electrolyte regulation in ultra-marathoners.
Conclusions
To summarize, the incidence of EAH in these 100 km ultra-marathoners was lower compared to reports on marathoners. Plasma volume increased and plasma [Na⁺] was maintained although body mass decreased. Fluid intake showed no association with the increase in plasma volume, post race [Na⁺] and Δ [Na⁺]. Hydration status was adequately maintained as has been found in other ultra-endurance races.22,35,44

REFERENCES


44. Rose SC, Peters EM. Ad libitum adjustments to fluid intake in cool environmental conditions maintain hydration status in a three-day mountain bike race. **Br J Sports Med.** doi:10.1136/bjsm.2008.049551
Table 1: Age, anthropometry, training and pre race experience of the finishers. Results are presented as median (IQR) and mean (SD) as appropriate.
<table>
<thead>
<tr>
<th></th>
<th>Pre race</th>
<th>Post race</th>
<th>Change (absolute)</th>
<th>Change (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body mass (kg)</strong></td>
<td>73.9 (68.6-79.1)</td>
<td>71.6 (67.0-77.3)</td>
<td>- 1.8 (1.4) *</td>
<td>- 2.4 (1.8)</td>
</tr>
<tr>
<td><strong>Plasma [Na⁺] (mmol/L)</strong></td>
<td>138.0 (137.0-139.8)</td>
<td>138.0 (137.0-140.0)</td>
<td>+ 0.5 (3.4)</td>
<td>+ 0.4 (2.5)</td>
</tr>
<tr>
<td><strong>Hematocrit (%)</strong></td>
<td>45 (43-47)</td>
<td>44 (42-46)</td>
<td>- 1 (-3 - +1) *</td>
<td>- 1.6 (15.5)</td>
</tr>
<tr>
<td><strong>Hemoglobin (g/dL)</strong></td>
<td>15.1 (14.4-16.0)</td>
<td>14.7 (14.1-15.6)</td>
<td>- 0.4 (1.0) *</td>
<td>- 2.4 (6.6)</td>
</tr>
<tr>
<td><strong>Urinary specific gravity (g/mL)</strong></td>
<td>1.015 (1.005-1.020)</td>
<td>1.025 (1.020-1.030)</td>
<td>+ 0.010 (0.005-0.015) *</td>
<td>+ 0.98 (0.74)</td>
</tr>
</tbody>
</table>

* = P < .001

Table 2: Change of body mass, hematologic and urinary parameters. Results are presented as median (IQR) and mean (SD) as appropriate. * = P < .001
Figure 1: Fluid intake correlated with race time ($n = 145$) ($r = .50$, $P < .0001$).
Figure 2: The change in body mass was associated with post race [Na⁺] (n = 145) (r = -.35, P < .0001).
Figure 3: The change in body mass was associated with the change in $[\text{Na}^+]$ (n = 145) ($r = - .34$, $P < .0001$).
Figure 4: Fluid intake was significantly and positively related to the change in body mass (n = 145) (r = .21, P = 0.012).
Figure 5: The increase in plasma volume was associated with post race [Na$^+$] ($r = -0.29$, $P = 0.0375$) ($n = 145$).