Original Article

Maintained total body water content and serum sodium concentrations despite body mass loss in female ultrarunners drinking ad libitum during a 100 km race

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We investigated in 11 female ultra-runners during a 100 km ultra-run, the association between fluid intake and prevalence of exercise-associated hyponatremia in a cross-sectional study. Athletes drank *ad libitum* and recorded their fluid intake. They competed at 8.0 (1.0) km/h and finished within 762 (91) min. Fluid intake was 4.1 (1.3) L during the race, equal to 0.3 (0.1) L/h. Body mass decreased by 1.5 kg (p<0.01); pre race body mass was related to speed in the race (r = -0.78, p<0.05); and change (Δ) in body mass was not associated with speed in the race. Change in body mass was positively (r = 0.70; p<0.05), and Δ urinary specific gravity negatively (r = -0.67; p<0.05), correlated to Δ percent total body water. Changes in body mass were not related to fluid intake during the race. Fluid intake was not correlated to running speed and showed no association with either Δ percent total body water nor Δ [Na] in plasma. Fluid intake showed no relationship with both Δ haematocrit and Δ plasma volume. No exercise-associated hyponatremia occurred. Female ultra- runners consuming fluids *ad libitum* during the race experienced no fluid overload, and *ad libitum* drinking protects against exercise-associated hyponatremia. The reported higher incidence of exercise-associated hyponatremia in women is not really a gender effect but due to women being more prone to overdrink.

Key Words: body composition, dehydration, ultra-endurance, water, performance

INTRODUCTION

Ultra-endurance runs are of increasing popularity. Apart from the classic marathon run over 42.195 km, ultramarathons as a single run^{1,2} or multi-day runs^{3,4} attract more and more athletes. Abundant literature is available about marathon running, however, little is known about the effects on the human body after running hundreds or thousands of kilometres.¹⁻⁴

Marathon and especially ultra-marathon running is associated with different problems such as a decrease in body mass and dehydration,⁵ loss of skeletal muscle mass^{1,3,4} and an increase in total body water.^{1,4,6} The increase in total body water could be due to several different mechanisms such as protein catabolism⁷ and the consequent development of hypoproteinemic oedema, increase in plasma proteins and subsequent increase in plasma volume,⁸ increased plasma volume due to increased plasma sodium concentration,⁹ retention of sodium due to increased activity of aldosterone,^{6,10} increase in plasma volume due to an increased activity in vasopressin.¹¹

The increase in total body water might also be due to fluid overload. In studies in long-distance triathlons and marathons, an association between excessive fluid intake and plasma sodium concentration has been demonstrated.^{12,13} In cases of dehydration – as has been found in

ultra-marathon running⁵ – body mass decreased and urinary specific gravity increased. 14,15

Since women hydrated more during a marathon,¹⁶ developed a positive mean fluid balance during a marathon,¹⁷ had significant lower post-race plasma sodium concentration after an Ironman triathlon¹⁸ and exercise-associated hyponatremia was more frequently found in females;¹⁹⁻²¹ we investigated in female ultra-runners fluid intake and change in total body mass, plasma sodium and total body water.

Risk factors for exercise-associated hyponatremia due to fluid overload are, a slow running pace and a high frequency of fluid intake.¹⁹ Since ultra-runners compete rather slowly and have many aid stations during an ultrarun,¹ fluid overload might occur. We focused on female ultra-runners and investigated their risks of exerciseassociated hyponatremia and overdrinking.

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MATERIALS AND METHODS

Subjects and race

The organiser of the 50th annual event of the 100 km run in Biel, Switzerland, contacted all participants of the race in 2008 via a separate newsletter, 3 months before the race, in which they were asked to participate in the study. About 2,500 male and female runners started in the race. The race took place during the night of 13 to 14 June 2008. The runners started on 13 June at 10:00 p.m. to run the 100 km, with a difference in altitude of 645 metres. During this 100 km they had 17 aid stations, approximately every 5 to 10 km, with different food and beverages. The organiser offered isotonic sports drinks, tea, soup, caffeinated drinks, water, bananas, oranges, energy bars and bread. The athletes were allowed to be supported by a cyclist in order to have additional food and clothing, if necessary. At the start at 10:00 p.m., the temperature was 15 °Celsius. During the night the temperature dropped to 8 °Celsius and rose to 18 °Celsius the next morning by 10:00 a.m. A total of 14 female nonprofessional experienced ultra-runners were interested in our investigation. They all gave their informed written consent. The study was approved by the Institutional Ethics Committee of St. Gallen, Switzerland. Eleven female athletes out of our study group finished the race within the time limit. The anthropometric and training parameters of the successful finishers are represented in Table 1.

Measurements and calculations

From 5:00 p.m. to 10:00 p.m. before the start of the race, and immediately after arrival at the finish line, every participant underwent anthropometric measurements, the collecting of blood samples and urinary sampling as well as bioelectrical impedance analysis in order to determine body mass, skeletal muscle mass, percent body fat and percent total body water. Samples of urine were collected for determination of creatinine, urea, sodium, potassium, urinary specific gravity and protein. Urinary specific gravity was analysed using Clinitek Atlas® Automated Urine Chemistry Analyzer (Siemens Healthcare Diagnostics, Deerfield, IL, USA). Creatinine and urea of urinary samples were measured using COBAS INTEGRA® 800. Electrolytes in the urinary samples were determined using ISE IL 943 Flame Photometer (GMI, Inc., Ramsey, MN, USA). Urea, sodium, potassium and protein in urine were normalised for creatinine in urine. At the same time, blood samples were taken to determine haematocrit, plasma urea, plasma potassium concentration and plasma sodium concentration using i-STAT® 1 System (Abbott

Table 1. Parameters of anthropometry and training volume (n = 11)

variables	mean (SD)
Age (y)	44.9 (11.6)
Body mass (kg)	61.8 (10.8)
Body height (m)	1.67 (0.09)
BMI (kg/m^2)	21.9 (2.4)
Years as active runner	8.7 (7.7)
Kilometres run per week	66.5 (19.1)
Hours run per week	6.5 (2.0)
Number of finished marathons $(n = 11)$	16 (10.7)
Number of finished 100 km runs $(n = 6)$	2.8 (3.9)

Laboratories, Abbott Park, IL, USA). Body mass was measured to the nearest 0.1 kg using an electronic balance (Beurer, Ulm, Germany). Percentage of body fat was calculated using the anthropometric method according to Ball et al.,²² and skeletal muscle mass was determined using the anthropometric method following Lee et al.²³ Percent total body water was measured using InBody 3.0 (Biospace, Seoul, Korea) following Bedogni et al.²⁴ Since body mass and skeletal muscle mass were expressed in kg, fat mass was calculated in kg from body mass and percent body fat. Change in plasma volume was determined following Beaumont.²⁵ Throughout the run the athletes reported their intake of fluids and solid nutrition at each aid station. At these aid stations, liquids and food were prepared in a standardised manner. Ingestion of fluids and solid food were determined according to the reports of the athletes using a food table.²⁶ Energy expenditure was estimated according to the table of energy expenditure for different sports disciplines and intensities.²⁷ As an example: A women with a 57 km body mass and completing the race within 681 min (average speed of 8.8 km/h equal to an estimated energy expenditure of 7.9 kcal/min) expended 5,380 kcal.

Statistical analysis

The Shapiro-Wilk test was applied to check for Gaussian distribution of the variables. Normally distributed data is presented as mean (SD), whereas non-normally distributed data is presented as median (interguartile ranges). Ttests or Wilcoxon signed rank tests were used to compare anthropometric and laboratory parameters before and after the race. The Hodges-Lehmann method was used to estimate median (interquartile range) changes of nonnormally distributed parameters during the race. The coefficient of variation ($CV\% = 100 \times SD/mean$) of total race time in minutes was calculated. The changes in parameters during the race were correlated using Pearson and Spearman's rank correlation analysis to check for associations. The significance level was set at p < 0.05. To account for multiple testing, Bonferroni-adjustments were applied for the correlation analysis.

RESULTS

Performance and energy turnover

The athletes finished within 762 (91) min (CV = 12.0 %). Pre-race body mass was negatively and highly significantly associated with speed in the race (Figure 1). They ingested 570 (230) kcal during the race and competed at 8.0 (1.0) km/h expending 6,310 (1,340) kcal in total. An energy deficit of 5,750 (1,170) kcal resulted. The energy deficit was not related to Δ total body mass, but significantly to Δ fat mass (Figure 2).

Change in body composition

Body mass decreased by 1.5 kg (p<0.01) (Table 2); skeletal muscle mass and fat mass showed no changes. Percent total body water increased by 2.2 % (p<0.01). The change in body mass was not related to speed in the race. The changes in fat mass and skeletal muscle mass were not related to Δ total body mass. The change in body mass (Figure 3) and Δ urinary specific gravity (Figure 4) were both correlated with Δ percent total body water.



Figure 1. Pre-race body mass was highly significantly associated with speed during the race (n=11) (r = -0.78, p < 0.05)



Figure 2. Δ fat mass was positively related to the energy deficit (n=11) (r = 0.64, p<0.05)

Table 2. Anthropometric and laboratory parameters in 11 female runners, values are presented as mean (SD) or median (IQR) and comparisons were performed by t-test or Wilcoxon sign rank test for paired groups as appropriate, Hodges-Lehmann method was used to estimate median (IQR) differences; a = anthropometric method, bia = bioelectrical impedance analysis, p = plasma, u = urine. (* = p < 0.05, ** = p < 0.01)

	Unit	Pre-race	Post-race	Change (post-pre race)
Body mass	kg	61.8 (10.8)	60.3 (10.6)	- 1.5 (1.1) **
Skeletal muscle mass (a)	kg	27.4 (4.8)	27.4 (5.1)	-0.0(1.7)
Fat mass (a)	kg	16.7 (6.7)	15.9 (6.1)	-0.8(1.2)
Percent total body water (bia)	%	58.9 (2.7)	61.2 (3.2)	2.2 (2.0) **
Haematocrit (p)	%	41.5 (2.3)	40.0 (3.2)	- 1.5 (3.5)
Sodium (p)	mmoL/L	138.3 (1.7)	137.4 (2.4)	-0.8(2.2)
Potassium (p)	mmoL/L	4.8 (4.5-4.9)	4.5 (4.4-4.7)	-0.1 (-0.4;0.1)
Urea (p)	mmoL/L	6.3 (1.4)	10.1 (2.8)	3.7 (2.4) **
Sodium/Creatinine (u)	mmoL/µmoL	0.03 (0.02)	0.005 (0.003)	-0.02 (0.02)**
Potassium/Creatinine (u)	mmoL/µmoL	0.008 (0.006-0.01)	0.008 (0.006-0.01)	-0.0007 (-0.005;0.001)
Urea/Creatinine (u)	mmoL/µmoL	0.05 (0.01)	0.04 (0.02)	-0.01 (0.02)
Protein/Creatinine (u)	mg/mmoL	11.9 (8.8-14.7)	13.2 (11.2-14.5)	1.3 (-3.0;4.3)
Urinary specific gravity	g/mL	1.011 (0.00)	1.024 (0.00)	0.013 (0.007) **



Figure 3. Δ Total body mass was positively related to Δ percent total body water (n = 11) (r = 0.70; p < 0.05).



Figure 4. Δ Urinary specific gravity was negatively associated with Δ percent total body water (n = 11) (r = -0.67, p < 0.05).

Urea in plasma and urine showed no association with Δ skeletal muscle mass. The change in percent total body water was neither related to post-race plasma sodium concentration (*p*>0.05) nor to Δ plasma sodium concentration (*p*>0.05).

Change in blood parameters

Haematocrit, plasma sodium and plasma potassium did not change (Table 2), plasma urea increased significantly (p<0.01). No hyponatremia occurred. Plasma volume increased by 7.6 %. The change in plasma volume showed no relationship to both Δ percent total body water and fluid intake. Post-race plasma sodium concentration was not correlated to Δ body mass. The change in plasma sodium concentration was not associated with Δ body mass. Race time was neither correlated to Δ plasma sodium concentration nor to post-race plasma sodium concentration. Post-race sodium concentration was not associated with post-race total body water (Figure 5).

Change in urinary parameters

Sodium decreased significantly (p<0.01) by 0.02 (0.02) mmoL/µmoL from 0.03 (0.02) mmoL/µmoL to 0.005 (0.003) mmoL/µmoL, potassium showed no changes and remained stable at 0.008 (0.006-0.01) mmoL/µmoL. The potassium-to-sodium ratio was 0.26 pre-race and rose to 1.6 post-race. Urea and protein did not change; urinary specific gravity significantly increased (p<0.01).

Fluid intake

Fluid intake was 4.1 (1.3) L during the run, equal to 0.3 (0.1) L/h. Fluid intake was not related to average running speed, showed no association with Δ percent total body water and there was no association between fluid intake and the change in plasma sodium in plasma. The change in body mass was not related to total fluid intake during the race. Fluid intake showed no association with both Δ haematocrit and Δ plasma volume.



Figure 5. Post-race plasma sodium concentration was not related to post-race total body water (n=11) (r=-0.26, p>0.05)

DISCUSSION

We hypothesised finding an association between fluid intake and the increase in total body water in female ultra-endurance runners. Indeed, we found an increase in percent total body water; however, fluid intake showed no association with Δ percent total body water.

In cases of excessive fluid intake with fluid overload,²⁸ we would have expected an increase in total body mass,^{28,29} a decrease in plasma sodium concentration,^{16,21,28,29} an increase in plasma volume²¹ and a decrease in haematocrit due to haemodilution¹³ since hyponatremic runners drink excessive amounts of fluids.¹⁶ In our study of female ultra-endurance runners, body mass decreased, plasma sodium and haematocrit remained unchanged and plasma volume increased. The decrease in body mass showed no relationship to both the change in plasma sodium and fluid intake.

We interpret the decrease in body mass as being due to dehydration.¹⁴ In cases of dehydration – as has been found in ultra-marathon running⁵ – body mass should decrease and urinary specific gravity increase.^{14,15} We found – apart from the significant decrease in total body mass – a significant increase in urinary specific gravity. A loss of body mass and an increase of urinary specific gravity should indicate dehydration according to Kavouras.¹⁴

Fluid intake showed no relationship with Δ haematocrit, Δ plasma sodium concentration, Δ percent total body water, Δ plasma volume and Δ total body mass. For a real fluid overload, fluid intake must have been far higher and the athletes would have to gain weight as described in Speedy *et al.*,²⁸ where one Ironman triathlete with exercise-associated hyponatremia (Na 130 mEq/L) drank 16 L over the course of the Ironman and gained 2.5 kg in total body mass.

Average fluid intake was at 0.32 (0.10) L/h. We assume that this amount was entirely appropriate and that no fluid overload occurred. In general, higher amounts of 0.8 to 1.6 L/h are recommended to maintain hydration status.³⁰ However, amounts of about 0.5 L/h might al-

ready lead to fluid overload and a decrease in serum sodium concentration. Stumpfle *et al.*¹³ reported fluid consumptions of 0.3 (0.1) L/h in an ultra-distance race, and Speedy *et al.*²¹ a median hourly fluid intake of 0.7 L/h in Ironman triathletes. In both studies, however, subjects who developed hyponatremia had evidence of fluid overload despite moderate fluid intake. Our runners were running slowly and the speed of running is the key determinant of the sweat rate and hence the fluid replacement rate.

Since fluid intake was not related to the increase in percent total body water (p>0.05), a possible explanation for the increase in percent total body water could be an increase in plasma volume due to sodium retention, as a consequence of increased aldosterone activity.^{12,13} After intense exercise, aldosterone is increased and rises with the growing intensity of exercise.³¹ An increased activity in aldosterone should lead to an increase in plasma sodium according to the findings of Wade et al.¹⁰ in a 20day 500-km race. We found no changes in plasma sodium; however, urinary sodium declined significantly. Potassium was significantly elevated in both plasma and urine. We see the increase in urinary potassium as a reaction of a stimulation of the RAAS (renin-angiotensin-aldosteronesystem). The ratio potassium-to-sodium in urine was 0.25 pre race and increased to 1.6 post-race. This means that during the race more potassium than sodium was excreted and a positive ratio for potassium to sodium in urine is an indicator of an increased aldosterone activity. The ratio potassium-to-sodium in urine > 1.0 reflects a contraction of the effective extra-cellular volume leading to a hyperreninemic hyperaldosteronemia. The ratio for potassiumto-sodium in urine is a physiological reflection of the potassium secretion in the distal tubulus and in comparison to the sodium re-absorption is an estimation of aldosterone activity in serum.

Transient expansion in plasma volume is reported after endurance events.⁶ The activation of the RAAS leads to an enhanced retention of sodium and free water, consequently resulting in an increase in plasma volume. We would therefore expect an increase in both plasma volume and plasma sodium concentration. Furthermore, both increases should be positively correlated. However, we found no increase in plasma sodium and no association between plasma volume increase and plasma sodium. An increase in plasma volume and extracellular fluid volume, respectively, should cause a fall in plasma sodium concentration unless there is mobilisation of sodium from some other source.

The increase in plasma volume in total body water could also be due to fluid retention because of inadequate suppression of the antidiuretic hormone secretion.²⁹ Unfortunately, osmolality was not determined in this investigation, which is a limitation. So we cannot estimate a potential relationship between plasma volume and total body water. However, although plasma volume was increased by 7.6 %, Δ plasma volume showed no relationship with Δ percent total body water.

The investigation is limited due to the small sample size and the design. In ultra-endurance races, only a few women start. In the 2008 race, a total of 1,993 men and 354 women finished within the time limit. Our sample of 11 subjects represents 3 % of the female finishers. Furthermore, a cross-sectional study is descriptive in nature where condition and potentially related factors are measured at a specific point in time for a defined population. Causal statements should not be made from this type of study.

CONLUSION

We found in these female 100 km runners consuming fluids *ad libitum* during the race that there was no fluid overload, and *ad libitum* drinking protects against exercise-associated hyponatremia in women. The reported higher incidence of exercise-associated hyponatremia in women is not really a gender effect but is due to women being more prone to overdrink. In future studies in ultraruns, osmolality in both plasma and urine should be determined and the activity of both aldosterone and the antiduretic hormone before and after an ultra-endurance run should be measured.

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AUTHOR DISCLOSURES

The authors have no conflict of interest and received no external funding.

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Original Article

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女性長跑者在100公里競賽中自由攝取液體可維持體內 水份及血鈉濃度

本橫斷性研究調查在 100 公里的長跑競賽中,11 名女性選手其液體攝取與運動 低血鈉的相關性。運動選手可自由攝取液體並且記錄喝了多少。他們以時速 8.0(1.0)公里進行比賽,並在 762(91)分鐘內完成。在比賽中,液體的總攝取量為 4.1 (1.3)公升,相當於每小時攝取 0.3 (0.1)公升的液體。平均總身體質量減少了 1.5 公斤(p < 0.01);而賽前的身體質量是與賽中的速度呈負相關 (r = -0.78, p < 0.05);然而總身體質量的改變則和賽中的速度不相關。總身體質量的改變和身 體水份百分率改變呈正相關(r = 0.70; p < 0.05),相反地,尿液比重改變則與身 體水份百分率改變呈負相關 (r = -0.67; p < 0.05)。身體質量的改變與賽中的液體 攝取量無關。液體攝取量也與競跑速度、身體水份百分率改變、血漿中鈉離子 濃度改變、血比容積及血漿體積的變化無關。在賽後,沒有低血鈉的發生。在 本篇研究中,女性長跑者在競賽中自由攝取液體,並無液體過度負荷的情形, 且避免運動引起的低血鈉。過去報導女性有較高的運動相關低血鈉發生率,可 能不是性別的效應,而是女性較傾向於過度攝取液體。

關鍵字:身體組成、脫水、超耐力、水份、競賽表現