HUMAN MOVEMENT
2012, vol. 13 (2), 94–101

IS THE PREVALENCE OF EXERCISE-ASSOCIATED HYPONATREMIA HIGHER IN FEMALE THAN IN MALE 100-KM ULTRA-MARATHONERS?

doi: 10.2478/v10038-012-0009-2

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ABSTRACT
Purpose. The prevalence of exercise-associated hyponatremia (EAH) has mainly been investigated in male endurance athletes. The aim of the present study was to investigate the prevalence of EAH in female 100-km ultra-marathoners and to compare them to male ultra-runners since females are considered more at risk of EAH. **Methods.** Changes in body mass, hematocrit, [Na+] and [K+] levels in both plasma and urine, plasma volume, urine specific gravity, and the intake of energy, fluids and electrolytes was determined in 24 male and 19 female 100-km ultra-marathoners. **Results.** Three male (11%) and one female (5%) ultra-marathoners developed asymptomatic EAH. Body mass decreased, while plasma [Na+], plasma [K+] and hematocrit remained stable in either gender. Plasma volume, urine specific gravity and the potassium-to-sodium ratio in urine increased in either gender. In males, fluid intake was related to running speed ($r = 0.50$, $p = 0.0081$), but not to the change in body mass, in post-race plasma [Na+], in the change in hematocrit and in the change in plasma volume. Also in males, the change in hematocrit was related to both the change in plasma [Na+] ($r = 0.45$, $p = 0.0187$) and the change in the potassium-to-sodium ratio in urine ($r = 0.39$, $p = 0.044$). Sodium intake was neither related to post-race plasma [Na+] nor to the change in plasma volume. **Conclusions.** The prevalence of EAH was not higher in female compared to male 100-km ultra-marathoners. Plasma volume and plasma [Na+] were maintained and not related to fluid intake, most probably due to an activation of the renin-angiotensin-aldosterone-system.

Key words: ultra-endurance, electrolyte disorder, fluid overload, sport nutrition

Introduction

Exercise-associated hyponatremia (EAH) is defined as a serum sodium concentration of ($[Na^+]$) < 135 mmol/L and was described first in scientific literature in 1985 by Noakes et al. [1] in male ultra-marathoners in South Africa as being due to "water intoxication". EAH is a well-known and a well-described fluid and electrolyte disorder in marathoners [2–8]. The prevalence of EAH varies between 3% and 22% in marathoners depending upon the number of studied athletes, their gender and fitness level [2–6]. There is abundant literature about the prevalence of EAH in marathoners [2, 4–7]. Studies investigating EAH in ultra-marathoners are rare, in which exclusively male athletes have been investigated [9–11]. In marathoners presenting EAH, an association between excessive fluid intake and both an increase in body mass and a decrease in plasma sodium [Na^+] has been demonstrated [2, 5, 6, 12, 13]. In ultra-marathoners, however, dehydration is a more common finding [14], resulting in a decrease of body mass and an increase in urine specific gravity [15, 16]. In cases of excessive fluid intake with fluid overload during endurance performance [17], we would also expect in ultra-runners a stable or increased body mass [13, 17], a decrease in plasma [Na^+] [12, 13, 18], an increase in plasma volume [18] and a decrease in hematocrit due to haemodilution [12].

Risk factors for fluid overload and subsequent EAH are the female gender, a slow running pace and a high frequency of fluid intake [2, 3, 19]. Following Noakes, three independent mechanisms explain why some athletes develop EAH during and after prolonged exercise: (*i*) overdrinking due to biological or psychological factors; (*ii*) an inappropriate secretion of antidiuretic hormone (ADH), in particular, the failure to suppress ADH-secretion in the face of an increase in total body water; and (*iii*) a failure to mobilize Na^+ from osmotically inactive sodium stores or the alternatively inappropriate osmotic inactivation of circulating Na^+ [13]. Because the mechanisms causing factors (*i*) and (*iii*) are unknown, it follows that the prevention of EAH requires that athletes be encouraged to avoid overdrinking during exercise [13]. Since ultra-marathoners run at a rather slow pace [20, 21], they may be at an especially high risk of fluid overload.

The aim of the present study was to investigate the prevalence of EAH in both female and male ultra-marathoners in the "100 km Lauf Biel" in Biel, Switzer-

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land. This race is the most famous 100-km ultra-marathon in Europe. The organizers offer a total of 17 aid stations and the athletes may be accompanied by a cyclist providing continuous fluid and nutrition support while running. Since the female gender, a slow running pace and excessive drinking behaviour [13, 19], combined with a high frequency of fluid consumption [2, 13], are considered as the main risk factors for fluid overload, and subsequently developing EAH, we hypothesized that (*i*) the prevalence of EAH would be higher in 100-km ultra-marathoners compared to existing reports on marathoners and that (*ii*) the prevalence of EAH would be significantly higher in female than in male ultra-marathoners.

Material and methods

After receiving approval by the Institutional Review Board for use of Human Subjects of St. Gallen, Switzerland, all the participants of the 50th annual "100 km Lauf Biel" in Biel, Switzerland in 2008 were contacted via a separate newsletter, three months before the race, where they were asked to participate in the current study. Out of about 2,000 runners who were to start in the race, 31 male and 19 female, non-professional, experienced ultra-runners agreed to take part in this study, with all of them providing their informed written consent. The race began in the night of 13 to 14 June 2008, with the runners beginning on 13 June at 10:00 p.m. and had to finish the 100 km distance with a total climb in altitude of 645 metres within a time limit of 21 hours. Two-thirds of the course was on asphalt with the remaining third on unpaved roads. Throughout the 100 km there were 17 aid stations at intervals of ~5 km that provided a variety of food and beverages. The organizers 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 access to food and clothing, if necessary. At the start of the race the temperature was 15° Celsius. During the night, the temperature dropped to 8° Celsius and then rose to 18° Celsius the next morning by 10:00 a.m. A maximum temperature of 31°C was reached at 01:00 p.m. on 14 June 2008.

Out of the initial group of participants, twenty-seven male and all female participants finished the race within the 21 h time limit, with one male runner finishing in the top three. Table 1 shows the age, anthropometric characteristics, training and pre-race experience of the subjects. Before the start of the race and after arrival at the finish line, every participant underwent analysis to determine body mass, take blood samples and be subject to urinary sampling. Body mass was measured to the nearest 0.1 kg using an electronic balance (Beurer, Germany) after voiding the urinary bladder. The athletes were weighed pre- and post-race in an identical manner in their running wear excluding shoes. Samples of urine were collected for the determination of urine creatinine, urine [Na^+], urine [K^+] and urine specific gravity. Urine specific gravity was analysed using a Clinitek Atlas® Automated Urine Chemistry Analyser (Siemens Healthcare Diagnostics, USA). Creatinine in urinary samples was measured using a COBAS INTEGRA® 800 (Roche Diagnostics, Switzer-

Table 1. Comparison of age, anthropometric characteristics, training and pre-race experience between male and female subjects. Results are presented as mean (SD)

	Male finishers (N = 27)	Female finishers (N = 19)
Age (years)	46.7 (8.0)	44.0 (10.4)
Body height (m)	1.78 (0.06)	1.67 (0.09)**
Body mass (kg)	74.3 (10.2)	61.0 (10.1)**
Body mass index (kg/m^2)	23.3 (2.2)	21.5 (2.3)*
Number of years participating in running (years)	11.2 (8.4)	10.3 (8.3)
Weekly distance ran (km)	73.7 (28.7)	66.3 (19.5)
Hours ran per week (h)	7.8 (3.2)	6.9 (2.3)
Number of weekly training units (n)	4.3 (1.5)	4.0 (0.7)
Minimal distance per week (km)	26.6 (21.4)	29.4 (19.0)
Maximal distance per week (km)	85.6 (56.7)	74.0 (35.4)
Distance per run training session (km)	18.8 (12.8)	14.9 (3.0)
Duration of run training sessions (min)	88.0 (24.9)	87.9 (24.5)
Mean speed of the training sessions (km/h)	10.7 (1.5)	9.5 (1.6)**
Yearly running distance (km)	3,158.9 (1,568.1)	2,185.8 (924.1)
Yearly hours ran (h)	307.1 (171.5)	222.4 (80.8)
Number of finished marathons (n)	30.9 (38.5) (n = 27)	20.0 (14.3) (n = 17)
Personal best time in a marathon (min)	207.8 (31.3)	231.2 (20.4)**
Number of finished 100 km ultra-marathons (n)	4.9 (6.9) (n = 18)	2.8 (3.5) (n = 5)
Personal best time in a 100 km ultra-marathon (min)	621.6 (250.2)	831.8 (173.3)

* $p < 0.05$; ** $p < 0.01$

land). Electrolytes in the urine samples were determined using an ISE IL 943 Flame Photometer (GMI, Inc., USA). [Na^+] and [K^+] in urine were normalised for creatinine in urine. At the same time, blood was sampled to determine hematocrit, plasma [Na^+] and plasma [K^+] using an i-STAT® 1 System (Abbott Laboratories, USA). The changes in plasma volume were calculated according Beaumont's equation [22].

While running, the athletes consumed food and drinks *ad libitum* and recorded their intake of fluid and solid nutrition using paper and pencil at each aid station. At every station, beverages and food were provided in same size portions. The ingestion of fluids, electrolytes and solid food between pre- and post-race measurements were determined by the reports of the athletes using a food table [23]. Energy expenditure was estimated using a stepwise calculation using body mass, mean velocity and the time spent during performance [24].

Pre-race, the participants were asked to maintain a comprehensive training diary consisting of their daily workouts, their distance and duration in preparation for the race. The training record consisted of the number of training units showing duration, kilometres, pace, the amount of kilometres ran per week, the amount of hours ran, the minimal and maximal amount of kilometres ran per week as well as the running speed during training in min/km were also recorded. Additionally, they reported on the number of years they had actively participated in running, the number of marathons and 100-km ultra-marathons they successfully completed and the best times that were achieved in these races. Following their arrival at the finish line, the subjects were asked if they felt the symptoms of EAH [19].

Data are presented as mean and standard deviation (SD). The measured parameters of both males and females were compared using the Kruskal-Wallis test. The Student's *t*-test was used to compare the parameters before and after the race. Correlations in the changes in the parameters during the race were evaluated using the Pearson's Product-Moment Correlational Analysis. The significance level was set at $p < 0.05$.

Results

Three male (11%) and one female (5%) finishers were diagnosed with asymptomatic EAH, where one male and one female athlete showed post-race plasma [Na^+] of 131 mmol/L, and two male athletes were found with plasma [Na^+] of 134 mmol/L. Throughout the race, females ran slower, consumed less energy, expended less energy, ingested less fluid and less electrolytes than the males (see Tab. 2). Body mass decreased ($p < 0.01$) while plasma [Na^+] and plasma [K^+] remained unchanged ($p > 0.05$) in either gender. For both genders, the decrease in body mass was found not to be related to an energy deficit ($p > 0.05$). Also, the decrease in body mass was not related to running speed ($p > 0.05$). Hematocrit levels decreased non-significantly in the males ($p > 0.05$) and significantly in the females ($p < 0.05$), plasma volume increased by 5.5% in the males and by 6.4% in the females. For both the males and the females, race time was not correlated to post-race plasma [Na^+] ($p > 0.05$). In the three male athletes with EAH, body mass decreased by – 3.5 (1.2) kg.

Fluid intake was significantly and positively related to the running speed of males (see Fig. 1), but not for females (see Fig. 2). Running speed, however, was neither related to post-race plasma [Na^+] ($p > 0.05$) nor to the change in plasma [Na^+] ($p > 0.05$) in either gender. For both males and females, there was no association between fluid intake and the following: the change in body mass, post-race plasma [Na^+], the change in hematocrit and the change in plasma volume ($p > 0.05$). Sodium intake was not related to post-race plasma [Na^+] and potassium intake was not related to post-race plas-

Table 2. Comparison of race time, energy turnover, fluid and electrolyte intake and body mass between male and female subjects. Results are presented as mean (SD)

	Male finishers ($N = 27$)	Female finishers ($N = 19$)
Race time (min)	689.9 (119.9)	770.5 (103.4)*
Running speed (km/h)	8.9 (1.6)	7.9 (1.1)*
Energy intake (kcal)	758.5 (302.3)	566.8 (229.3)*
Energy expenditure (kcal)	7,424.5 (1,666.4)	6,198.2 (1,366.8)*
Energy balance (kcal)	–6,666.0 (1,648.5)	–5,631.4 (1,187.6)*
Fluid intake (L/h)	0.52 (0.18)	0.32 (0.11)**
Fluid intake (L/kg body mass)	0.08 (0.02)	0.21 (0.03)**
Sodium intake (mg/h)	445 (471)	364 (250)**
Potassium intake (mg/h)	146 (176)	62 (24)**
Body mass pre-race (kg)	74.3 (10.2)	61.0 (10.1)**
Body mass post-race (kg)	72.4 (10.1)	59.6 (10.0)**
Body mass change (kg)	–1.9 (1.5)##	–1.4 (0.9)##

* $p < 0.05$, ** $p < 0.01$ (between genders); ## $p < 0.01$ (within gender)

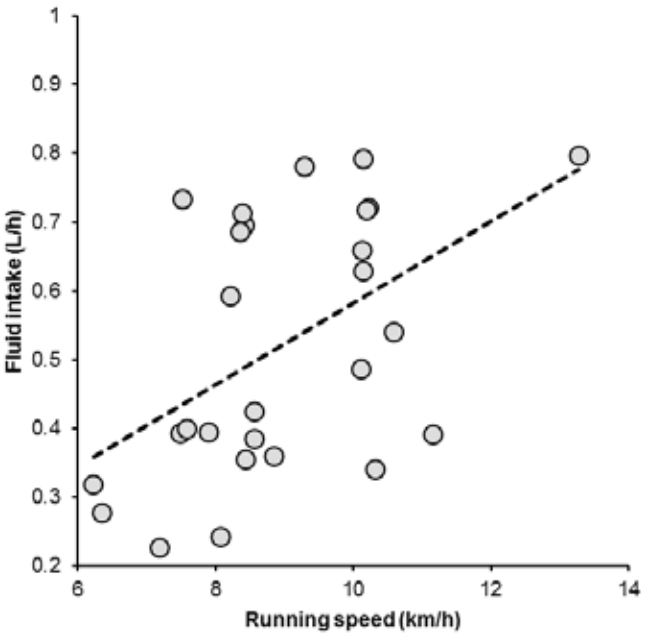

Figure 1. Hourly fluid intake during the race was significantly and positively related to running speed in males ($N = 27$) ($r = 0.50$; $p = 0.0081$)

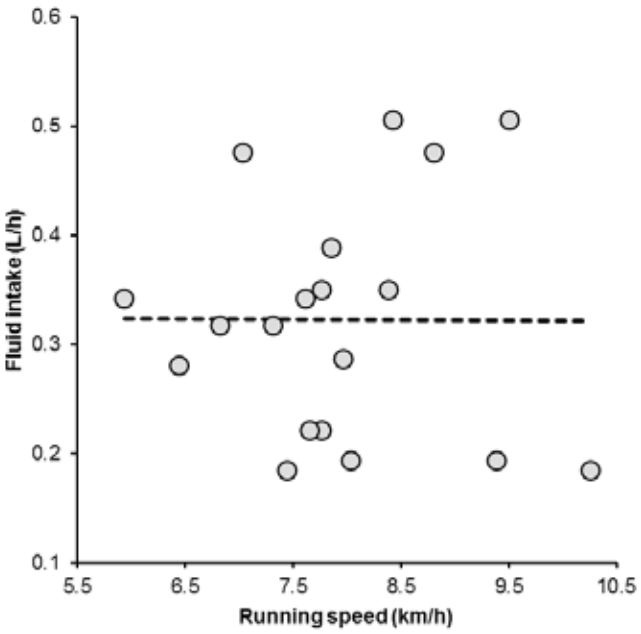

Figure 2. In females, fluid intake and running speed had no association ($N = 19$) ($r = 0.00$, $p = 0.98$)

Table 3. Comparison of race performance and results obtained during the race between male and female subjects. Results are presented as mean (SD)

	Male finishers ($N = 27$)	Female finishers ($N = 19$)
Hematocrit pre-race (%)	44.1 (2.8)	41.5 (2.4)**
Hematocrit post-race (%)	43.0 (2.9)	40.3 (3.4)**
Hematocrit change (%)	–1.1 (3.3)	–1.2 (3.5)#
Change in plasma volume (%)	+5.5 (13.9)	+6.4 (13.7)
Plasma sodium pre-race (mmol/L)	139.5 (1.4)	138.4 (1.7)*
Plasma sodium post-race (mmol/L)	139.6 (3.8)	137.7 (2.3)*
Plasma sodium change (mmol/L)	0.15 (4.13)	–0.74 (2.23)
Plasma potassium pre-race (mmol/L)	4.9 (0.7)	4.7 (0.5)
Plasma potassium post-race (mmol/L)	5.3 (1.0)	4.7 (0.6)*
Plasma potassium change (mmol/L)	0.5 (1.2)	0.05 (1.0)
Urine sodium/Creatinine pre-race (mmol/mmoL)	0.022 (0.009)	0.031 (0.016)*
Urine sodium/Creatinine post-race (mmol/mmoL)	0.006 (0.004)	0.010 (0.002)
Urine sodium/Creatinine change (mmol/mmoL)	–0.016 (0.009)##	–0.027 (0.015)*##
Urine potassium/Creatinine pre-race (mmol/mmoL)	0.008 (0.006)	0.011 (0.006)*
Urine potassium/Creatinine post-race (mmol/mmoL)	0.010 (0.004)	0.009 (0.006)
Urine potassium/Creatinine change (mmol/mmoL)	0.002 (0.006)	–0.002 (0.008)
Potassium-to-sodium ratio pre-race	0.36 (0.19)	0.39 (0.21)
Potassium-to-sodium ratio post-race	2.10 (1.12)	2.25 (1.03)
Potassium-to-sodium ratio change	1.72 (1.15) ##	1.86 (1.08)##
Urine specific gravity pre-race (g/mL)	1.013 (0.008)	1.011 (0.007)
Urine specific gravity post-race (g/mL)	1.026 (0.005)	1.024 (0.004)
Urine specific gravity change (g/mL)	0.012 (0.007) ##	0.013 (0.007)##

* $p < 0.05$, ** $p < 0.01$ (between genders); # $p < 0.05$, ## $p < 0.01$ (within gender)

ma [K^+] in either gender ($p > 0.05$). In males, the change in plasma [Na^+] was related to the change in hematocrit (see Fig. 3). Urine specific gravity increased in both male and female subjects ($p < 0.01$), urine [Na^+] decreased ($p < 0.01$) and urine [K^+] remained unchanged ($p > 0.05$) (see Tab. 3). The potassium-to-sodium ratio in urine increased in both males and females ($p < 0.01$). The change in post-race potassium-to-sodium ratio in urine was significantly and positively related to the change in hematocrit in males (see Fig. 4), but not in females (see Fig. 5).

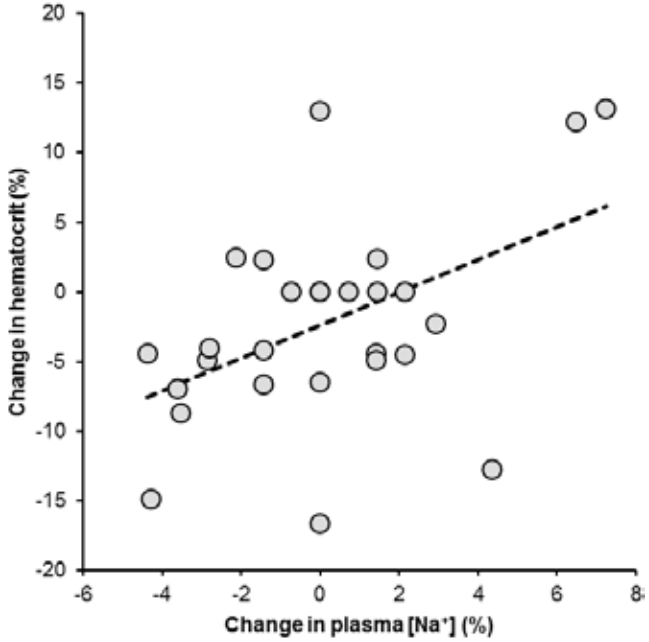

Figure 3. The change in plasma [Na^+] was significantly and positively related to the change in hematocrit in males (N = 27) (r = 0.48, p = 0.015)

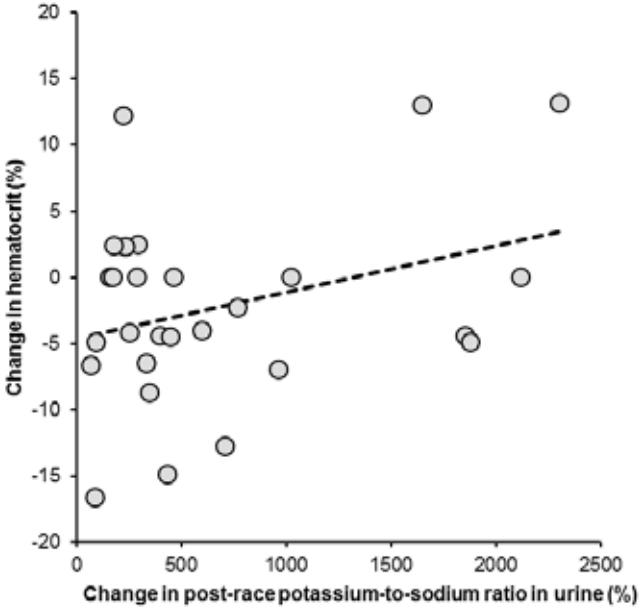

Figure 4. The change in the post-race potassium-to-sodium ratio in urine was significantly and positively related to the change in hematocrit in males (N = 27) (r = 0.32, p = 0.01)

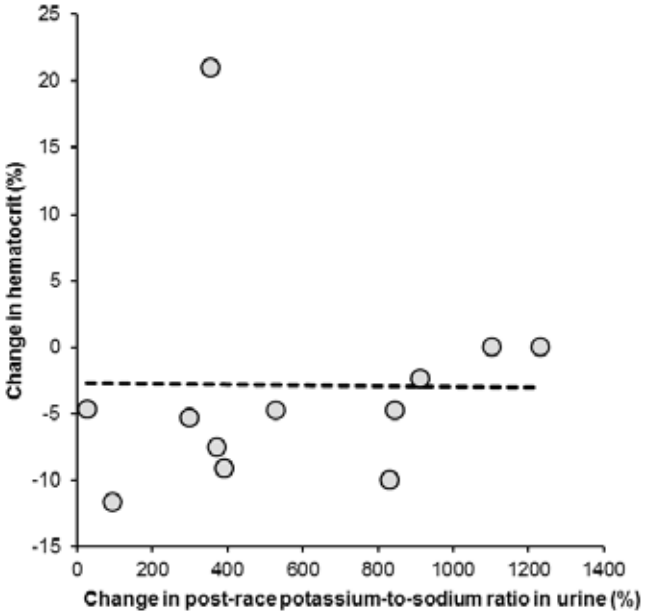

Figure 5. In females, the change in the post-race potassium-to-sodium ratio in urine showed no association with the change in hematocrit (N = 19) (r = –0.01, p = 0.96)

The weekly running distance (r = –0.48, p = 0.0122), the mean running speed during training (r = –0.52, p = 0.0053), the personal best time in a marathon (r = 0.62, p = 0.0005) and the personal best time in a 100-km ultra-marathon (r = 0.79, p = 0.0002) were related to the achieved race time in the group of males. In females, the training variables were not related to race time ($p > 0.05$), however, the personal best time in a marathon (r = 0.59, p = 0.0136) and the personal best time in a 100-km ultra-marathon (r = 0.82, p = 0.0091) were associated with their race time.

Discussion

The aim of the present study was to investigate the prevalence of EAH in both female and male ultra-marathoners in a 100-km ultra-marathon. Since the female gender, a slow running pace and excessive drinking behaviour with a high frequency of fluid consumption were considered as the main risk factors for fluid overload, we hypothesized (*i*) that the prevalence of EAH would be higher in 100-km ultra-marathoners as based on the available reports on marathoners and (*ii*) be especially higher in females than in male ultra-marathoners. Three males (11%) and one female (5%) developed asymptomatic EAH. The 11% prevalence of EAH in the male ultra-marathoners was the same rate as had been recently found in marathoners in the London Marathon [5]. The prevalence rates for EAH for marathoners seem, however, to vary between 3% [6] to 22% [3] depending upon weather and temperature [6] and the fitness level of the subjects [3]. For the female ultra-marathoners, the 5% prevalence of EAH was considerably lower compared to the males.

Excessive fluid intake leading to fluid overload is considered to be the most important risk factor for EAH [2, 13, 19]. Fluid intake was significantly and positively related to running speed for males (see Fig. 1), where faster male athletes were drinking more compared to slower ones. However, fluid intake was not associated with the decrease in body mass, post-race plasma [Na^+],

the change in hematocrit and the change in plasma volume. For fluid overload, fluid intake would have to have been far greater and the athletes would have had to gain weight as described by Speedy et al., where one Ironman triathlete with EAH and who presented plasma [Na^+] < 130 mmol/L drank 16 L over the course of the event and gained 2.5 kg in body mass [17]. The athletes in this race, compared to a classical marathon, had the opportunity to be supported by a cyclist. This cyclist could carry food and drinks as well as additional clothing. We assume that the faster runners were followed by a cyclist who provided fluids between the aid stations, so they did not have to stop at each aid station to replenish their fluid level. However, the increased availability of fluids did not lead to fluid overload and EAH.

Apart from the female gender, event inexperience and a slow running pace are also considered as risk factor for EAH [19]. In the present subjects, training volume regarding the distance ran each year and the amount of hours ran was not different between genders. The female ultra-marathoners ran slower during training and had a slower personal best marathon time; however, the personal best time in a 100-km ultra-marathon was not different between genders. 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 pre-race experience is an important factor in preventing EAH in ultra-marathoners. In these subjects, weekly running distance, mean running speed during training, personal best time in a marathon and personal best time in a 100-km ultra-marathon were all related to race time. Recent reports on 100-km ultra-marathoners reported that pre-race experience such as a high training volume in the distance ran in a week, a fast running speed during training and a fast personal best time in a marathon were associated with race time in a 100-km ultra-marathon [25–27]. A high training volume [25, 26] and a fast running speed while training [25–27] were especially significant indicators for a fast 100-km race time. We presume that these subjects were both highly trained and highly experienced ultra-runners which might explain that the prevalence of EAH was lower in these athletes compared to existing reports on marathoners.

The mean hourly fluid intake was 0.52 (0.18) L for males and 0.32 (0.11) L for females, where males were consuming more fluids compared to females. Faster male runners drank more than slower runners (see Fig. 1), whereas no association between running speed and fluid intake existed in females (see Fig. 2). We speculated that a slower running pace during the race coupled with a frequent fluid intake would lead to fluid overload and EAH. In contrast, the faster runners drank more when compared to slower ones while running speed showed no association with either post-race plasma [Na^+] or the change in plasma [Na^+]. The fact that no fluid overload occurred in the faster runners although they drank more might be explained by a higher perspiration rate in these runners. We assume that the rather low amount of fluids despite *ad libitum* fluid consumption was responsible for the fact that no fluid overload occurred. Although aid stations were provided every ~5 km and athletes could be followed by a support crew to provide fluids, both male and female athletes were found to not overdrink. In general, amounts greater than 0.8 L per hour to 1.6 L per hour are recommended to maintain hydrated in performances lasting 1–3 h [25]. However, hourly amounts of ~0.5 L could also lead to fluid overload and a decrease in serum [Na^+] concentration [12, 18]. Stuempfle et al. reported fluid consumptions of 0.3 (0.1) L per hour in an ultra-distance race [12], and Speedy et al. described a mean hourly fluid intake of 0.7 L in Ironman triathletes [18]. In both studies, subjects developing EAH had evidence of fluid overload despite a moderate fluid intake. Stuempfle et al. concluded that the current recommendations for ultra-distance athletes to consume 0.5 L to 1.0 L per hour may be too high [12], and Speedy et al. summarised that subjects developing EAH had evidence of fluid overload despite modest fluid intakes [18]. Therefore, recommendations for fluid intake, especially in ultra-endurance performances, should be adapted to take into account these recent findings, where Gisolfi and Duchman have already recommended reducing hourly fluid intake to 0.5 L to 1.0 L for endurance performances lasting longer than 3 h [28]. Their recommendations for fluid intake are as follows: a possible starting point suggested for marathon runners (who are hydrated from the outset) is they drink *ad libitum* from 0.4 L per hour to 0.8 L per hour, with the higher rate suggested for faster, heavier individuals competing in warm environments while the lower rate for the slower, lighter persons competing in cooler environments [29–32].

In a state of fluid overload, we would expect a stable or rather increased body mass [19]. We found, however, a significant decrease in body mass and a significant increase in urine specific gravity in the studied ultra-runners. Since the energy deficit during the race was not related to the change in body mass, the decrease in body mass must be therefore associated with dehydration. In cases of dehydration resulting from ultra-marathon running [14], body mass should decrease and urine specific gravity should increase [15, 16]. Regarding our results, a loss of ~2.5% in body mass and an increase in urine specific gravity to ~1.025 g/mL indicated severe dehydration, according to Kavouras [15].

Hematocrit decreased non-significantly in males and significantly in females, plasma volume increased by 5.5% in males and by 6.4% in females. A transient expansion in plasma volume is reported after endurance events [33]. The increase in plasma volume, however, was not related to fluid intake. A possible explanation for the increase in plasma volume could be a retention

of [Na^+] as a consequence of increased aldosterone activity since both fluid and sodium intake were not related to post-race plasma [Na^+] [12]. After intense exercise, aldosterone increases and rises with growing exercise intensity [34]. The activation of the renin-angiotensin-aldosterone system (RAAS) leads to an enhanced retention of plasma [Na^+] and water, consequently resulting in an increase in plasma volume. An increased activity in aldosterone should lead to an increase in plasma [Na^+] according to the findings of Wade et al. from a 20-day 500-km race [35]. We found, however, no change in plasma [Na^+] while urine [Na^+] declined. The potassium-to-sodium ratio in urine was, however, increased. The potassium-to-sodium ratio in urine is a physiological reflection of the [K^+] excretion in the distal tubulus and when compared to [Na^+] re-absorption as an estimate of the aldosterone activity in serum. We see the increase in the potassium-to-sodium ratio in urine as a stimulation of the RAAS. During the race, more urine [K^+] than urine [Na^+] was excreted and a positive ratio for urine [K^+] to urine [Na^+] suggests an increased activity of aldosterone. A recent study on male 100-km ultra-marathoners showed a significant and positive association between the change in aldosterone and both the change in the potassium-to-sodium ratio in urine and the post-race transtubular potassium gradient [36]. A potassium-to-sodium ratio in urine > 1.0 reflects a contraction of the effective extra-cellular volume leading to a hyperreninemic hyperaldosteronemia. Since the change in hematocrit was positively related with both the change in plasma [Na^+] (see Fig. 3) and the post-race potassium-to-sodium ratio in urine (see Fig. 4) for males, we assume that both the change in hematocrit and the increase in plasma volume was due to an increased activity of aldosterone and not due to fluid intake. However, the decrease in hematocrit could also be a result of intravascular hemolysis while running.

One limitation of this study is that we did not record the urine output of the athletes during the race. Fluid balance might be estimated better with fluid intake and urine output. Future studies should include fluid balance with an estimation of urines loss.

Conclusion

To summarize, the prevalence of EAH in these 100-km ultra-marathoners was not higher compared to existing reports on marathoners and EAH was not more frequent in female than in male ultra-marathoners. Although body mass decreased, plasma volume and plasma [Na^+] were maintained. Fluid intake showed neither an association with the decrease in body mass, nor with post-race plasma [Na^+] and the increase in plasma volume. We assume that the rather low fluid intake was responsible for the low prevalence of EAH. The potassium-to-sodium ratio in urine increased post-race to >1.0 and showed a significant and positive association with the change in hematocrit. Maintained fluid homeostasis in these ultra-runners was most probably due to a stimulation of the RAAS. Future studies investigating EAH in ultra-marathoners should determine the activity of aldosterone and include larger samples of female ultra-marathoners.

Acknowledgments
We wish to thank the organisers of '100 km Lauf Biel' and the athletes for their help in collecting data. In addition we wish to thank Mary Miller from England who helped us with translating this manuscript.

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Paper received by the Editors: March 12, 2011
Paper accepted for publication: December 5, 2011

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