Higher Prevalence of Exercise-Associated Hyponatremia in Triple Iron Ultra-Triathletes Than Reported for Ironman Triathletes

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Abstract

In a recent study of male and female ultra-marathoners in a 161-km ultra-marathon, the prevalence of exercise-associated hyponatremia (EAH) was higher than reported for marathoners. Regarding triathletes, the prevalence of EAH has been investigated in Ironman triathletes, but not in Triple Iron ultra-triathletes. The aim of this study was to investigate the prevalence of EAH in male ultra-triathletes competing in a Triple Iron ultra-triathlon over 11.4 km swimming, 540 km cycling, and 126.6 km running. Changes in body mass, fat mass, skeletal muscle mass, total body water, haematocrit, plasma volume, plasma sodium concentration ([Na$^+$]) and urine specific gravity were determined in 31 male athletes with (means $\pm$ standard deviation) 42.1 $\pm$ 8.1 years of age, 77.0 $\pm$ 7.0 kg body mass, 1.78 $\pm$ 0.06 m body height and a BMI of 24.3 $\pm$ 1.7 kg/m2 in the 'Triple Iron Triathlon Germany'. Of the 31 finishers, eight athletes (26%) developed asymptomatic EAH. Body mass, fat mass, skeletal muscle mass, and haematocrit decreased, plasma volume increased ($P < 0.05$), plasma [Na$^+$], total body water and urine specific gravity remained stable. The decrease in body mass was related to both the decrease in fat mass and skeletal muscle mass ($P < 0.05$), but was not related to overall race time, the change in plasma [Na$^+$], post-race plasma [Na$^+$], or urine specific gravity. The prevalence of EAH was higher in these Triple Iron ultra-triathletes compared to existing reports on Ironman triathletes. Body fluid homeostasis remained stable in these ultra-triathletes although body mass decreased.

Key Words: ultra-endurance, electrolyte, plasma sodium, dehydration

Introduction

Exercise-associated hyponatremia (EAH) is defined as a serum sodium concentration ([Na$^+$]) < 135 M during or within 24 hours of exercise, and was first described in the scientific literature in 1985 by Noakes *et al*. (36) in ultra-marathoners in South Africa as being due to 'water intoxication'. EAH is a well-known and well described fluid and electrolyte disorder in marathon runners (1, 5, 6, 11, 18, 32). The prevalence of EAH has been observed to be as high as ~22% in marathoners, depending on the number of investigated athletes, their gender and fitness level (1, 5, 6, 11, 18, 32).

Three main factors are responsible for the occurrence of EAH in endurance athletes: [1] overdrinking due to biological or psychological factors; [2] inappropriate secretion of the antidiuretic hormone (ADH), in particular, the failure to suppress ADH-secretion in the face of an increase in total body water (TBW); and [3] a failure to mobilize Na$^+$ from the osmotically inactive sodium stores or alternatively inappropriate osmotic inactivation of circulating Na$^+$ (38). The main reason for developing EAH is the

behaviour of overdrinking during an endurance performance (35, 38). Studies have shown that when athletes were encouraged to limit their fluid intakes, drinking only in response to thirst, there were no cases of EAH (37, 50). To date, no study has found that drinking more than *ad libitum* during exercise produced any biological advantage, but it could cause EAH (34). We know that this form of behaviour is the only risk factor for EAH since when athletes are told to drink to thirst during exercise the prevalence of EAH becomes negligible as described in Ironman races held in South Africa and New Zealand (35). Fluid overload leads to EAH, and a correlation between an increase in body weight due to fluid overload and a decrease of serum $[Na^+]$ was described in several studies (16, 38, 48). The primary cause of such weight gain can only be the overconsumption of fluid during exercise because of an exaggerated thirst drive during exercise or because of behavioural conditioning (38). In Ironman triathletes, large changes in body weight during a triathlon were not associated with a greater prevalence of medical complications or higher rectal temperatures but were associated with higher post-race serum $[Na^+]$ (43). In another study on Ironman triathletes, there was an indirect relationship between serum $[Na^+]$ and body weight changes during the race; the greater the body weight loss, the higher the serum $[Na^+]$ (47). An indirect relationship between post-race serum $[Na^+]$ and percentage change in body weight was observed in a further study on Ironman triathletes (46).

Apart from studies on marathoners, several studies investigating EAH in ultra-marathoners do exist (7, 10, 23, 29, 39, 41, 42, 52, 53). Since ultra-marathoners run at a slow pace (19, 27, 28), they might be at an especially high risk of fluid overload and subsequent EAH. In a recent study of male and female ultra-marathoners in a 161-km ultra-marathon, ~51% of the finishers developed EAH (29). The prevalence of EAH has also been investigated in Ironman triathletes (43, 46, 48, 56). The prevalence of EAH for Ironman triathletes competing over 3.8 km swimming, 180 km cycling and 42.195 km running ranged between 1.8 % and 18 % (43, 46, 48, 56). To date, no study has investigated the prevalence of EAH in Triple Iron ultra-triathletes, competing over 11.4 km swimming, 540 km cycling, and 126.6 km running (20, 25). In these races, athletes have to be followed by their own support crew providing nutrition and material, and speed in the race is considerably slower compared to an Ironman triathlon (20, 25). These circumstances might increase the prevalence of EAH.

The aim of this study was to investigate the prevalence of EAH in male Triple Iron ultra-triathletes. Since a slow running pace together with excessive

drinking behaviour (12, 13), meaning a high frequency of fluid consumption (1), is considered as the main risk behaviour for fluid overload, and subsequently developing EAH, we hypothesized that the prevalence of EAH would be higher in these ultra-triathletes compared to existing reports on Ironman triathletes. Lebus *et al.* (29) considered, as a possible explanation for their high prevalence of ~ 51% of EAH in a 161-km ultra-marathon, the race time of ~26 h and the high maximum temperature of 37.6°C. This race time is considerably longer compared to Ironman triathletes competing for ~12 h where the prevalence of EAH is considerably lower (14, 43, 46, 48, 56).

Materials and Methods

The Subjects

All 53 athletes that had registered for the 'Triple Iron Triathlon Germany' in 2007, which took place in Lensahn, Schleswig-Holstein, Germany, were contacted by a separate newsletter from the organizer three months before the start of the race and asked to participate in our investigation. Fifty-three athletes (three women and 50 men) expressed an interest, however only 45 male athletes entered our investigation. The other eight athletes denied participation. The 45 participants gave their informed written consent. The study was approved by the Institutional Ethics Committee of St. Gallen, Switzerland. No criteria for inclusion/exclusion were used except that the participants had to complete the Triple Iron ultra-triathlon within the overall time limit of 58 h. Thirty-one recreational male ultra-triathletes with (means ± standard deviation) 42.1 ± 8.1 years of age, 77.0 ± 7.0 kg body mass, 1.78 ± 0.06 m body height and a BMI of 24.3 ± 1.7 kg/m² finished the race successfully within the time limit. The other 14 athletes dropped out due to medical problems such as exhaustion and overuse injuries of the lower limbs. Pre-race experience and training parameters for all finishers, including hyponatremic and non-hyponatremic finishers, are presented in Table 1. Subjects were identical to those subjects used in a previous study where changes in hydration status and solid masses were investigated (22).

The Race

From July 27th to July 29th 2007, the 16th edition of the 'Triple Iron Triathlon Germany' in Lensahn, Schleswig-Holstein, Germany took place covering 11.6 km swimming, 540 km cycling and 126.6 km running. On Friday 27th at 07:00 a.m., the race started. The race site is ~10 m above sea level. Swimming was held in a heated 50 m outdoor pool with a constant

temperature of 25 °Celsius and wetsuits were allowed. After passing through the transition area, 67 laps of 8 km each on a nearly flat course with ~30 m of both climb and descent per lap had to be cycled in the surroundings of the town. After the cycling section, the athletes moved onto the flat run course, consisting of 96 laps of 1.31 km per lap, in the town of Lensahn. All the athletes had their own support crew to provide nutrition and changes of clothing and equipment. The athletes were free to manage their own nutrition. The weather was dry with temperatures varying from 11.9 °Celsius (July 29th at 00:00) to 20.2 °Celsius (July 27th at 02:00 p.m.). Relative humidity varied from 57% (July 27th at 03:00 p.m.) to 96% (July 29th at 03:00 a.m.) with an average of 82% to 90% during daytime.

Measurements and Calculations

The evening before the start of the race early next morning and directly after the race, every participant underwent the determination of anthropometric characteristics as well as the collection of blood and urinary samples in order to determine body mass, fat mass, skeletal muscle mass, total body water changes, haematocrit, plasma volume, plasma [Na$^+$] and urine specific gravity. The circumferences of the limbs and the thicknesses of eight skin-folds (pectoralis, axillar, triceps, subscapular, abdomen, suprailiacal, front thigh and medial calf) were measured on the right side of the body. With these anthropometric measurements, skeletal muscle mass and fat mass were estimated. Body mass was measured after voiding of the urinary bladder 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. The circumferences of upper arm, thigh and calf were measured using a non-elastic tape measure (cm) (KaWe CE, Kirchner und Welhelm, Germany). The skin-fold data were obtained using a skin-fold calliper (GPM-Hautfaltenmessgerät, Siber and Hegner, Zurich, Switzerland) and recorded to the nearest 0.2 mm. One trained investigator took all the measurements. The skin-fold measurements were taken three times and the mean was then used for the analyses. The skin-fold measurements were standardized to ensure reliability and readings were performed 4 s after applying the calliper, according to Becque *et al.* (3). An intra-tester reliability check was conducted on 27 male athletes prior to testing (21). Intraclass correlation (ICC) within the two judges was excellent for all anatomical measurement sites, and various summary measurements of skin-fold thicknesses (ICC > 0.9). Agreement tended to be higher within measurers than between measurers but still reached excellent reliability (ICC > 0.9) for the sum-

mary measurements of the skin-fold thicknesses. The circumference of the upper arm was measured at mid-upper arm, the circumference of the thigh at mid-thigh and the circumference of the calf at mid-calf. Skeletal muscle mass was estimated using the following formula following Lee *et al.* (30) with skeletal muscle mass = Ht * (0.00744 * CAG^2 + 0.00088 * CTG2 + 0.00441 * CCG2) + 2.4 * sex – 0.048 * age + race + 7.8 where Ht = body height, CAG = skin-fold-corrected upper arm girth, CTG = skin-fold-corrected thigh girth, CCG = skin-fold-corrected calf girth, sex = 0 for female and 1 for male; age in years; race = 0 for white and 1 for non-white men. Fat mass was estimated using the anthropometric method of Stewart and Hannan (51) with fat mass (g) = 331.5 * (abdominal) + 356.2 * (thigh) + 111.9 * *m* – 9,108 where abdominal is the thickness of abdominal skin-fold in mm, thigh is the thickness of thigh skin-fold in mm and *m* is body mass in kg. Changes (Δ) in total body water were estimated using the equation Δ total body water = Δ body mass – (Δ skeletal muscle mass + Δ fat mass) + endogenously produced water where endogenously produced water = water produced by oxidation of fuel + glycogen-complexed water following Weschler (55).

Samples of urine were collected for the determination of urine specific gravity using URYXXON$^®$ 300 (Macherey-Nagel, GmbH, Düren, Germany). After collection of the urine sample, capillary blood samples were drawn from the fingertip to determine haematocrit and the concentration of plasma sodium ([Na$^+$]) using i-STAT$^®$ 1 System (Abbott Laboratories, Abbott Park, IL, USA). The accuracy of the i-STAT$^®$ 1 System was validated in laboratory studies (4, 33). All capillary blood samplings were performed in an upright standing position. Standardization of posture prior to blood collection was respected since postural changes can influence blood volume and therefore haemoglobin concentration and haematocrit (44). The changes in plasma volume were determined from the pre- and post-race haematocrit (H) values according to Beaumont (2) using the equation % Δ plasma volume = $[100/(100 - H_{pre})] \times [100(H_{pre} - H_{post})/H_{post}]$.

From the time of entering the race with their inscription the athletes kept a comprehensive training diary, recording their training units in swimming, cycling and running, showing the distance (km) and duration (h) completed each training session, and discipline, up to the start of the race. In addition, every athlete indicated his number of finished Ironman and Triple Iron triathlons as well as his personal best time in each type of race. Upon arrival at the finish line, athletes were asked for symptoms of EAH such as weakness, confusion, headache, nausea or vomiting.

Table 1. Pre race experience, personal best times and training variables for all finishers and comparison between non-hyponatremic and hyponatremic finishers

Pre race experience and training	All finishers (n = 31)	Non-hyponatremic finishers (n = 23)	Hyponatremic finishers (n = 8)
Number of finished Ironman Triathlon races (n)	10.8 ± 13.0 (n = 28)	9.3 ± 12.7 (n = 21)	15.1 ± 14.7 (n = 7)
Personal best time in an Ironman Triathlon (min)	661 ± 48	659 ± 48	667 ± 50
Number of finished Triple Iron Triathlon races (n)	2.2 ± 3.4 (n = 18)	1.7 ± 2.2 (n = 13)	3.8 ± 5.5 (n = 5)
Personal best time in a Triple Iron Triathlon (min)	2,759 ± 432	2,823 ± 389	2,593 ± 541
Weekly training volume (h)	19.4 ± 9.7	18.1 ± 5.6	22.9 ± 16.6
Weekly swimming volume (h)	3.4 ± 2.1	3.1 ± 1.5	4.3 ± 3.2
Weekly cycling volume (h)	10.2 ± 6.5	9.6 ± 3.6	12.1 ± 11.4
Weekly running volume (h)	5.7 ± 2.0	5.4 ± 1.6	6.6 ± 2.8
Weekly swimming volume (km)	8.7 ± 4.7	8.4 ± 4.2	9.7 ± 6.2
Weekly cycling volume (km)	270 ± 136	248 ± 89	328 ± 219
Weekly running volume (km)	58.7 ± 21.0	56.6 ± 17.7	64.6 ± 28.7
Speed in swim training (km/h)	2.6 ± 0.7	2.6 ± 0.6	2.5 ± 0.8
Speed in cycle training (km/h)	27.5 ± 5.5	26.4 ± 3.5	30.3 ± 8.9
Speed in run training (km/h)	10.3 ± 1.5	10.5 ± 1.4	9.8 ± 1.5

Results are presented as means ± SD. Significance was set at a significance level of $P < 0.05$. No differences were found between non-hyponatremic and hyponatremic finishers.

Statistical Analysis

The Shapiro–Wilk test was used to check for normal distribution of the data. The results are presented as means ± standard deviation (SD). The paired *t*-test was used to check for significant changes in the parameters before and after the race for all athletes. Changes from pre- to post-race as well as differences between hyponatremic and non-hyponatremic finishers in body mass, hematologic and urinary parameters were analysed using a 2-way (group x time) repeated ANOVA with subsequent Tukey-Kramer post-hoc test. Correlation analysis was performed to assess the univariate association between the changes in the anthropometric variables and the changes in the laboratory data. For all statistical tests, significance was set at a level of $P < 0.05$.

Results

The Table 1 represents the pre-race experience and training for all finishers, separated for both non-hyponatremic and hyponatremic finishers. No differences were found between non-hyponatremic and hyponatremic finishers. The successful athletes finished the race within 2,874 ± 401 min. Of the 31 finishers, eight athletes (26%) developed asymptomatic EAH with post-race plasma [Na$^+$] between 129 M and 134 M. Non-hyponatremic finishers com-

pleted within a time of 2,887 ± 373 min, no faster than the hyponatremic finishers with a total race time of 2,863 ± 498 min ($P > 0.05$).

For both EAH and non-EAH, there were mean decreases in body mass, skeletal muscle mass and fat mass (see Table 2) ($P < 0.01$). Total body water showed no changes. The decrease in body mass was associated with both the decrease in skeletal muscle mass (r = 0.44; $P < 0.05$) and the decrease in fat mass (r = 0.51; $P < 0.01$). The change in body mass varied between – 10% and + 7.5% (see Fig. 1). In five athletes, body mass increased between 0.2 kg and 2.0 kg; in 26 athletes, body mass decreased between 0.2 and 6.6 kg. When the 23 non-hyponatremic and the eight hyponatremic finishers were compared, the changes in body mass were no different (see Table 3). Hematocrit decreased in all finishers (see Table 2); no differences were found when non-hyponatremic and hyponatremic finishers were compared (see Table 3). Plasma volume increased by 14.7 ± 16.2% (n.s.). When all 31 finishers were considered, plasma [Na$^+$] remained stable (see Table 2). In the non-hyponatremic finishers, plasma [Na$^+$] remained stable, whereas plasma [Na$^+$] decreased highly significantly in the hyponatremic finishers (see Table 3). Urine specific gravity remained stable for all finishers (see Table 2) as well as for both the non-hyponatremic and the hyponatremic finishers (see Table 3). The decrease in body mass was not related to race time (see Fig.

Table 2. Mean values and standard deviation (SD) of body mass hematocrit, plasma sodium and urine specific gravity before and after the race, for all finishers (n = 31)

	Pre-race	Post-race	Change (absolute)	Change (%)
Body mass (kg)	77.2 ± 7.7	75.7 ± 7.7	-1.4 ± 1.7**	-2.2 ± 2.5
Skeletal muscle mass (kg)	40.5 ± 3.6	39.5 ± 3.7	-1.0 ± 0.9**	-2.4 ± 2.3
Fat mass (kg)	9.2 ± 3.9	8.6 ± 3.7	-0.6 ± 1.3**	-4.9 ± 16.5
Total body water (kg)	27.6 ± 2.0	27.6 ± 2.7	+0.05 ± 1.4	+0.09 ± 4.9
Hematocrit (%)	47.2 ± 5.0	44.3 ± 3.8	-2.9 ± 5.0*	-5.4 ± 9.4
Plasma [Na$^+$] (M)	137.9 ± 1.9	137.2 ± 3.4	-0.7 ± 3.2	-0.5 ± 2.3
Urine specific gravity (g/mL)	1.013 ± 0.007	1.017 ± 0.007	+0.004 ± 0.01	+0.4 ± 0.9

Significance was set at a significance level of $P < 0.05$. * = $P < 0.05$; ** = $P < 0.01$.

Table 3. Comparison of changes in body mass, hematologic and urinary parameters for both hyponatremic (n = 8) and non-hyponatremic finishers (n = 23)

	Non-hyponatremic finishers (n = 23)			Hyponatremic finishers (n = 8)		
	Pre-race	Post-race	Δ (post – pre)	Pre-race	Post-race	Δ (post – pre)
Body mass (kg)	77.2 ± 6.8	75.8 ± 7.0*	-1.4 ± 1.4	77.4 ± 8.3	75.0 ± 9.9*	-2.4 ± 3.2
Hematocrit (%)	46.1 ± 4.5	43.1 ± 2.8*	-3.0 ± 4.2	50.2 ± 5.4	47.8 ± 4.4*	-2.4 ± 7.2
Plasma [Na$^+$] (M)	138.2 ± 1.9	138.7 ± 2.5	+0.5 ± 2.4[#]	137.1 ± 1.7	132.8 ± 1.6*	-4.2 ± 2.7
Urine specific gravity (g/mL)	1.012 ± 0.009	1.017 ± 0.008	+0.005 ± 0.009	1.018 ± 0.008	1.018 ± 0.009	-0.0005 ± 0.01

Results are presented as mean (SD). Significance was set at a significance level of $P < 0.05$. * = significant different within the group, # = significant different between the groups.
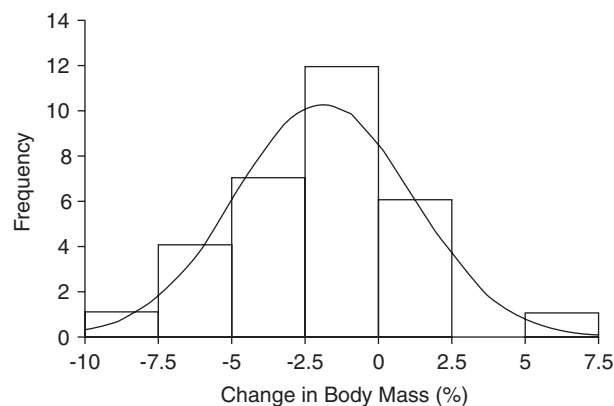

Fig. 1. The change in body mass varied between – 10% and + 7.5%.

2A), the change in plasma [Na$^+$] (see Fig. 2B), post-race plasma [Na$^+$] (see Fig. 2C) or the change in urine specific gravity. Race time was neither associated with pre-race body mass nor with post-race plasma [Na$^+$]; pre-race body mass was not related to post-race plasma [Na$^+$]. Post-race plasma [Na$^+$] was, however, highly significantly related to the change in plasma [Na$^+$] (r = 0.84, $P < 0.0001$).

Discussion

The aim of this study was to investigate the prevalence of EAH in male Triple Iron ultra-triathletes. We hypothesized that the prevalence of EAH would be higher in these ultra-triathletes compared to existing reports on Ironman triathletes, and in cases of EAH we expected an increase in body mass and a decrease in plasma [Na$^+$]. In accordance with the hypothesis and in line with the recent findings of Lebus et al. (29) showing a prevalence rate of EAH of ~51% in ultra-marathoners, we found asymptomatic EAH in eight of our 31 ultra-triathletes, corresponding to 26% EAH of the finishers. EAH seems obviously to occur more frequently in ultra-triathletes compared to existing reports on Ironman triathletes (43, 46, 48, 56). However, EAH was asymptomatic and no athlete developed exercise-associated hypotonic enceph-alopathy.

Considering the risk behaviours for EAH, event inexperience is one of the athlete-related risk be-haviours (12, 13). In marathoners, the number of pre-race completed marathons varied between one and eight races (1, 5, 6, 32), and non-hyponatremic marathoners had completed more marathons compared
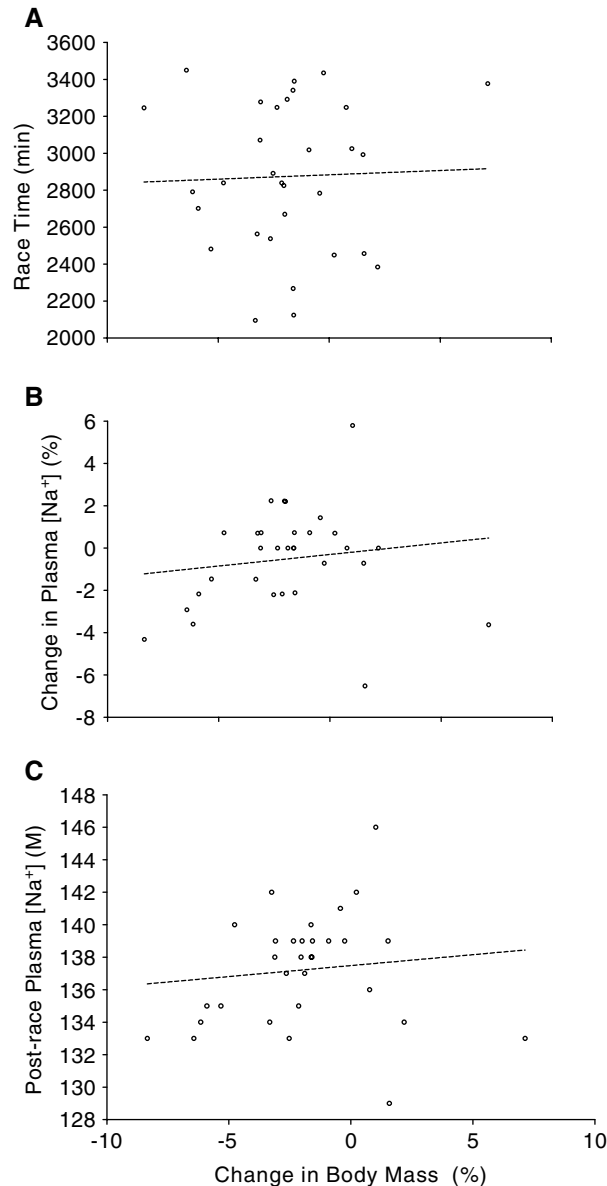
A

B

C

Fig. 2. The change in body mass showed no association with race time ($r = 0.03$, $P = 0.8518$) (Panel A), the change in plasma $[Na^+]$ ($r = 0.14$, $P = 0.4522$) (Panel B), and post-race plasma $[Na^+]$ ($r = 0.12$, $P = 0.5321$) (Panel C) for the 31 athletes.

the non-hyponatremic finishers ~1 Triple Iron ultra-triathlon, respectively. These data do not agree that limited experience is associated with an increased prevalence of EAH.

Speedy *et al.* (49) investigated the response to a fluid load in Ironman athletes with a history of EAH. There seemed to be any unique pathophysiological characteristic that explained why some athletes developed EAH in response to fluid overload during prolonged exercise. Inexperienced triathletes are nonetheless more likely to believe the 'drink to stay ahead of thirst' dogma. In future studies, experienced and in-experienced ultra-endurance athletes should be asked in these ultra-endurance races about their beliefs of fluid consumption.

Low body weight is a further risk for EAH (12, 13), however, pre-race body mass was not related to post-race plasma $[Na^+]$ in these ultra-triathletes. Exercise duration of four hours or more is considered as a further risk for EAH (12, 13); however, race time was not related to post-race plasma $[Na^+]$ in these athletes. Unfortunately, we did not record fluid intake in these athletes, since fluid overload is considered as the main risk behaviour for developing EAH (12, 13). The correct determination of fluid intake during an ultra-endurance event of this dimension is rather difficult (29). Also Lebus *et al.* were not able to record fluid intake in their 161-km ultra-marathoners (29). However, recent studies showed that ultra-marathoners in a 100-km ultra-marathon did not over-drink (24, 26).

In cases of fluid overload, we would expect an increase in body mass and a decrease in plasma $[Na^+]$. Weight gain during an endurance performance is associated with overdrinking thus leading to EAH (12, 13, 38). Noakes *et al.* (38) reported that weight gain consequent to excessive fluid consumption was the principal cause of a reduced serum $[Na^+]$ after endurance exercise. Subjects who gained weight maintained or increased serum $[Na^+]$ required the addition of significant amounts of Na^+ (> 500 M) into an expanded volume of total body water. This Na^+ likely originated from osmotically inactive, exchange-able stores. The determination of changes in body mass is a useful measure of both fluid intake (1) and fluid retention (45). Body mass decreased signif-icantly in these athletes and the decrease in body mass was related to the decrease in both skeletal muscle mass and fat mass, as has recently been shown in ultra-marathoners (27, 28). A decrease in body mass is – apart from other parameters such as changes urine specific gravity, urine osmolality, plasma osmolality, bioelectrical impedance, skin-fold thickness, heart rate and blood pressure – a marker of dehydration (17, 44). Urine specific gravity is considered as a reliable marker of hydration status (17, 44). In cases of

to hyponatremic ones (1). In these studies, the prevalence of EAH amounted to ~22% (1, 5, 6, 32). Our ultra-triathletes had completed ~11 Ironman triathlons and ~2 Triple Iron ultra-triathlons but we found no statistically significant difference between hyponatremic and non-hyponatremic finishers regarding pre-race experience. However, in details, hyponatremic finishers had completed ~15 Ironman triathlons and non-hyponatremic finishers ~9 Ironman triathlons, respectively. Regarding the number of finished Triple Iron ultra-triathlons, the hyponatremic finishers had completed ~3 Triple Iron ultra-triathlons,

dehydration during an ultra-endurance performance, urine specific gravity should be increased post-race compared to pre-race (27). However, the athletes in the present study did not seem dehydrated, but rather euhydrated with a stable urine specific gravity, a stable plasma [Na$^+$], an unchanged total body water and a non-significant increase in plasma volume (17, 44).

In marathoners, Mettler *et al.* (32) observed an association between a change in body mass and both the post-race plasma [Na$^+$] and the change in plasma [Na$^+$]. In 161-km ultra-marathoners, however, Lebus *et al.* (29) found no association between the changes in body mass and the changes in plasma [Na$^+$]. Also, in these ultra-triathletes, the change in body mass showed no association with post-race plasma [Na$^+$] or with the change in plasma [Na$^+$]. This finding is not in accordance with the recent findings in marathoners reported by Mettler *et al.* (32). These authors 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 ADH.

Plasma volume increased by $14.7 \pm 16.2\%$ (n.s.). During a marathon, plasma volume decreased (31, 57), as has also been found in ultra-marathoners competing over 67 km (40). In longer ultra-endurance races, plasma volume increased (14, 26, 53). Increased plasma volume may be due purely to a shift rather than an increase in total body water, from interstitial fluid to plasma, as happens when plasma [protein] increased. Hew-Butler *et al.* (14) assumed that the intensity of performance was responsible for these disparate findings, since marathoners compete faster compared to ultra-endurance athletes. Ultra-endurance athletes may preserve a 'fluid reserve' in the interstitial fluid of the extracellular fluid compartment.

A limitation of this study is that we did not record fluid intake and did not ask about the intake of non-steroidal anti-inflammatory drugs (NSAIDs), which is also considered as a risk behaviour for EAH (12, 13, 56). The use of NSAIDs might influence renal function (42) and thus increase the prevalence of EAH (41). A further limitation is that we did not determine plasma [Na$^+$] in the non-finishers, so if any of these 14 non-finishers did suffer from EAH, the prevalence of EAH would have considerably increased. Regarding the increase in plasma volume, the stable plasma [Na$^+$] and the stable urine specific gravity, body fluid homeostasis must have been maintained. We assume that other factors than fluid intake maintained body fluid homeostasis during this ultra-triathlon, such as a hormonal regulation by ADH and aldosterone (8, 53). In recent studies of marathoners (45) and ultra-marathoners over 56 km (15),

the activity of ADH was measured in addition to changes in body mass, plasma [Na$^+$], plasma osmolality and fluid intake. Recent findings suggested that EAH was not only due to fluid overload, but also to an increased activity in ADH (54). In future studies of ultra-endurance athletes such as ultra-runners and ultra-triathletes, the activity of ADH should be investigated. As mentioned, the three independent mechanisms [1] overdrinking due to biological or psychological factors, [2] inappropriate ADH-secretion, in particular, the failure to suppress ADH-secretion in the face of an increase in TBW, and [3] a failure to mobilize Na$^+$ from the osmotically inactive sodium stores or alternatively inappropriate osmotic inactivation of circulating Na$^+$ explain why some athletes develop EAH during and after prolonged exercise (38). Because the mechanisms causing factors [2] and [3] are currently unknown, it follows that the prevention of EAH requires that athletes be encouraged to avoid overdrinking during exercise. The inadequate suppression of ADH-secretion would produce the fluid retention that causes subjects who drink to excess during exercise to gain weight (38). Since sodium retention was the major factor in the increase in plasma volume (9), and aldosterone was increased after an ultra-endurance race (53), the activity of aldosterone should also be determined. This might provide more insight into fluid and electrolyte regulation in ultra-endurance athletes.

To summarize, the prevalence of EAH in these Triple Iron ultra-triathletes was higher when compared to existing reports of Ironman triathletes. In accordance with recent findings of a higher prevalence of EAH in ultra-marathoners compared to marathoners, ultra-endurance athletes competing for > 24 h seem to be at a higher risk to develop EAH. In future studies, the fluid intake during an ultra-endurance performance of > 24 h should be recorded and the intake of NSAIDs should be investigated. Also, the athletes should be asked in these ultra-endurance races about their beliefs of fluid consumption. Furthermore, the activity of both ADH and aldosterone should be determined in ultra-endurance athletes such as ultra-triathletes and ultra-marathoners. Presumably an increased activity of both hormones is the reason for maintained fluid homeostasis in ultra-endurance athletes competing for > 24 h.

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