Effect of a multistage ultraendurance triathlon on aldosterone, vasopressin, extracellular water and urine electrolytes

B Knechtle*†, N P Hernández Morales‡, E Ruvalcaba González‡, A A Aguirre Gutierrez‡, J Noriega Sevilla‡, R Amézquita Gómez‡, A R Estrada Robledo‡, A L Marroquín Rodríguez‡, O Salas Fraire‡, J L Andonie§, L C Lopez§, G Kohler and T Rosemann†**

*Gesundheitszentrum St Gallen, St Gallen, Switzerland; †Institute of General Practice and for Health Services Research, University of Zurich, Zurich, Switzerland; ‡Universidad Autónoma de Nuevo León, Monterrey, Nuevo León, Mexico; §Multisport Andonie, Monterrey, Nuevo León, Mexico; **Radio-Oncology, University Hospital, Basel, Switzerland
E-mail: beat.knechtle@hispeed.ch

Abstract

Prolonged endurance exercise over several days induces increase in extracellular water (ECW). We aimed to investigate an association between the increase in ECW and the change in aldosterone and vasopressin in a multistage ultraendurance triathlon, the 'World Challenge Deca Iron Triathlon' with 10 Ironman triathlons within 10 days. Before and after each Ironman, body mass, ECW, urinary [Na^+], urinary [K^+], urinary specific gravity, urinary osmolality and aldosterone and vasopressin in plasma were measured. The 11 finishers completed the total distance of 38 km swimming, 1800 km cycling and 422 km running within 145.5 (18.8) hours and 25 (22) minutes. ECW increased by 0.9 (1.1) L from 14.6 (1.5) L prerace to 15.5 (1.9) L postrace ($P < 0.0001$). Aldosterone increased from 70.8 (104.5) pg/mL to 102.6 (104.6) pg/mL ($P = 0.033$); vasopressin remained unchanged. The increase in ECW was related neither to postrace aldosterone nor to postrace vasopressin. In conclusion, ECW and aldosterone increased after this multistage ultraendurance triathlon, but vasopressin did not. The increase in ECW and the increase in aldosterone were not associated.

Keywords: body mass, dehydration, fluid, sodium, potassium

Introduction

Long-distance triathlons such as the Ironman triathlon are highly popular and have been held since the first event in 1978 in Hawaii.[1] In 2006, for the first time, a multistage long-distance triathlon was held in Mexico, where athletes had to complete one Ironman distance per day for 10 days.[2]

In single Ironman triathlons, exercise-associated hyponatraemia (EAH) is considered a frequent and serious problem.[3,4] EAH is defined as a decrease in plasma [Na^+] concentration <135 mmol/L[5] and fluid overload is the likely aetiology.[4,6] However, regarding recent findings, an increased activity of vasopressin may contribute to the pathogenesis of EAH in runners.[7,8]

An interesting finding in multistage ultraendurance performances is the increase in total body water.[9,10] It has been well documented that prolonged strenuous exercise over several consecutive days induces a progressive increase in extracellular water (ECW), plasma volume and total body water.[11–15] Sodium retention seemed to be the major factor in the increase in plasma volume.[9] In multistage ultraendurance runs, plasma volume increased,[13–15] plasma [Na^+] concentration was maintained[15,16] and aldosterone increased.[13,14] Presumably, fluid homeostasis during an ultraendurance performance is regulated by both aldosterone and vasopressin.

Also for Ironman triathletes, an increase in plasma volume has been described.[4,17] Ironman triathletes with symptomatic EAH showed abnormal fluid retention with an increased extracellular volume.[4] Fluid retention, however, was not associated with an increased activity of vasopressin in Ironman triathletes.[4]

The association of vasopressin with effect on renal function and association with EAH has been investigated in marathon runners.[8] Marathon runners developing EAH showed an increase in body mass,[18,19] and it was assumed that inappropriate antidiuretic hormone release during exercise altered renal function,[19] which may lead to water retention and EAH.[20]

The aim of this study was to investigate the association between the increase in both total body water and ECW with changes in both aldosterone and vasopressin during a multistage ultraendurance triathlon, in order to

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determine whether the increase in total body water and ECW would be related to an increased activity in aldosterone or in vasopressin. We hypothesized that both vasopressin and aldosterone would be increased after a multistage ultraendurance race, and that rather the increase in aldosterone would be related to the increase in both total body water and ECW than the increase in vasopressin.

Materials and methods

Participants

The organizer of the World Challenge Deca Iron Triathlon in 2009 contacted all participants and asked them to participate in the study. The study was approved by the Ethical Committee of St Gallen, Switzerland. All participants were informed about the experimental procedures and gave their informed written consent. All 17 participating athletes were interested and took part in the investigation. Of the 17 starters, 11 athletes with (mean and standard deviation) 44.0 (10.5) years, 76.0 (7.3) kg body mass, 1.73 (0.06) m body height and a body mass index of 25.4 (2.0) kg/m^2 finished the race and were included in the study.

The race

The 'World Challenge Deca Iron Triathlon' was held in 2009 for the third time. The competition took place in the City of Monterrey in the Province of Nuevo León in northern Mexico. The race started on 9 November 2009. Seventeen male and one female ultraendurance triathletes entered the race. Every day they had to complete the distance of one Ironman triathlon of 3.8 km swimming, 180 km cycling and 42.195 km running with a time limit of 24 hours. Every morning at 9:00 the event started with a swim in the 25 m indoor pool at the 'Centro Acuatico Olimpico Universitario' of 'Universidad Autonoma de Nuevo Leòn' in Monterrey. The pool was heated and held at a constant temperature of 28°C. Laps of 25 m were counted by personal lap counters. After completing the swim, the athletes changed in the transition area and cycled to the 'Parque Niños Héroes'. This park was closed to traffic, completely illuminated and had a cycling track that was 95% flat, but included an inclination of 5%. The cycling consisted of 94 laps of 1.915 km each. After changing for the running course, the athletes first had to run a short lap of 703 m and then 22 laps of 1.886 km. In total, the athletes had to climb an altitude of 1650 m per Ironman triathlon. Drafting during cycling was strictly prohibited. Laps in the cycling and running course were counted electronically with a chip system. Athletes could be helped by their own support crew for nutrition and changes of equipment and clothes. During the whole race, accommodation was offered in the 'Parque Niños Héroes'. The athletes and their support crew had a room with bed, toilet and shower. For nutrition, the organizer offered a variety of food in a restaurant that was open for 24 hours.

Analysis of body composition

Before and after each stage, body composition was measured using the InBody 3.0 body composition analyser with direct segmental multifrequency bioelectrical impedance method (InBody 3.0; Biospace, Seoul, Korea) following Bedogni *et al.*[21] This method is reliable for determination of body water under both laboratory situations[21] and field conditions.[9] However, hydration status must be unchanged[22] and extracellular osmolality must be stable.[23] InBody 3.0 has a tetrapolar 8-point tactile electrode system and performs 20 impedance measurements at each session, using four different frequencies (5, 50, 250 and 500 kHz) at each of five segments (right arm, left arm, trunk, right leg and left leg). The subjects stood barefoot in an upright position on foot electrodes, with the legs and thighs not touching and the arms not touching the torso. Four foot electrodes were used and the subjects were asked to grip the two palm-and-thumb electrodes. They did this without shoes or excess clothing. The skin and electrodes were cleaned and dried before testing. Body mass, lean body mass, total body water, intracellular water, ECW, fat mass and protein mass were directly determined.

Laboratory analysis

Before the start of the race, aldosterone and vasopressin were determined at rest. After venipuncture of an antecubital vein, the blood was centrifuged at 3000 g for 10 minutes at 4°C; the serum was then separated, stored on ice and then processed to determine both aldosterone and vasopressin using a radioimmunoassay method. The radioimmunoassay method quantitatively measured the concentration of aldosterone in the blood within a reference range of 10.0–300 pg/mL, the activity of vasopressin within a reference range of 1.0–2.0 μU/mL. The same procedure was repeated immediately upon arrival at the finish line after the last stage on day 10. Immediately before and immediately after each stage, samples of urine were collected. In the urine samples, [Na^+], [K^+], specific gravity and osmolality were determined. The samples [Na^+] and [K^+] were determined using an ion selective electrode method. Specific gravity was measured manually using a NE Uricon refractometer which has a scale from 1000 to 1050 g/mL. To determine osmolality, urea was measured in the urine samples using a HITACHI 911 automated equipment using an enzymatic method; the formula urinary osmolality = (Na + K × 2) + urea/5.6 was used to calculate osmolality with a reference range of 300–1000 mosm/kg.

Statistical analysis

For finishers and non-finishers, overall changes of the variables over time were tested by MANOVA-procedure. In case of a significant change during the race, paired *t*-tests were applied to detect a significant change from one time period to the other. For non-finishers, paired-*t*-tests were applied to detect changes from one time period to the other as long as the athlete was in the race and had

completed a full stage. For finishers, Pearson correlation analysis was used to check for associations between parameters with statistically significant changes. Statistical significance was accepted with $P < 0.05$.

Results

Performance

A total of 17 male competitors started on 9 November 2009; six athletes did not reach the finish line. Of those who did not finish, the reasons cited were accident, overuse injury of the lower limb, fatigue, loss of motivation and respiratory problems. The 11 finishers completed the total distance of 38 km swimming, 1800 km cycling and 422 km running within 145.5 (18.8) hours and 25 (22) minutes, equal to 8757 (1129) minutes. Table 1 represents the daily total race times per Ironman distance and the daily highest temperature. The time per Ironman increased throughout the race ($P < 0.0001$) (Figure 1); the daily highest temperature was not related to the daily race time.

Changes in body composition during the race

For finishers, body mass decreased by 1.7 (2.4) kg from 76.3 (7.7) kg prerace to 74.6 (7.1) kg postrace ($P < 0.0001$) (Figure 2). Fat mass decreased by 3.2 (1.6) kg from 13.1 (4.0) kg prerace to 10.1 (3.6) kg postrace ($P < 0.0001$). The decrease in body mass was significantly and positively related to the decrease in fat mass ($r = 0.71$, $P = 0.022$). Race time was neither correlated to prerace body mass nor to prerace fat mass. Lean body mass increased by 1.1 (2.5) kg from 62.9 (4.6) kg prerace to 64.0 (6.5) kg postrace ($P < 0.01$). Protein mass showed no changes. Total body water increased by 1.5 (1.6) L from 46.3 (3.5) L prerace to 47.8 (4.1) L postrace ($P < 0.0001$) (Figure 3). ECW increased by 0.9 (1.1) L from 14.6 (1.5) L prerace to 15.5 (1.9) L postrace ($P < 0.0001$). Intracellular water showed no changes. The increase in total body water was related to the increase in ECW ($r = 0.64$, $P = 0.047$). The increase in lean body mass was neither correlated to the increase in total body water ($r = -0.36$, $P = 0.33$) nor to the increase in ECW ($r = -0.36$, $P = 0.33$).

Table 1 Daily finish times per Ironman distance and daily highest temperature

Day	Finish time (min)	Temperature (°C)
1	760 (106)	27
2	849 (166)	26
3	815 (95)	28
4	855 (108)	27
5	897 (133)	29
6	882 (127)	31
7	906 (119)	31
8	889 (139)	23
9	953 (155)	19
10	940 (203)	23

Race times are presented as mean (standard deviation)

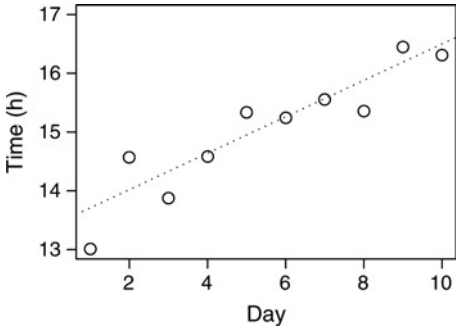

Figure 1 Daily finish times throughout the race. Time per Ironman increased significantly throughout the race ($P < 0.0001$)

Postrace lean body mass was highly significantly correlated to both postrace total body water ($r = 0.94$, $P < 0.0001$) and postrace ECW ($r = 0.81$, $P = 0.0044$).

Changes in urinary parameters, aldosterone and vasopressin

Urinary [Na^+] showed no changes (Figure 4), urinary [K^+] and the potassium-to-sodium ratio in urine increased ($P < 0.0001$) for finishers. Urinary specific gravity and urinary osmolality showed no changes. Aldosterone increased from 70.8 (104.5) pg/mL prerace to 102.6 (104.6) pg/mL postrace ($P = 0.033$); vasopressin remained

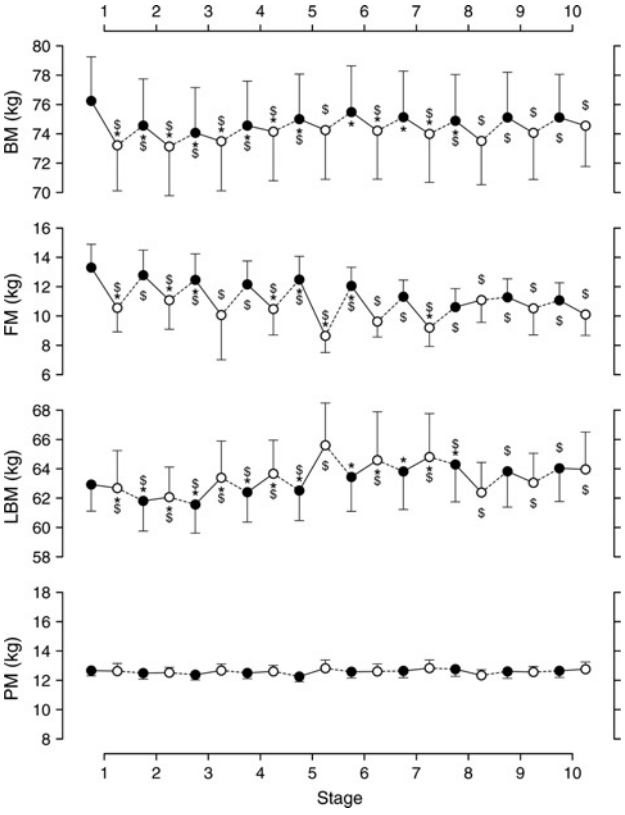

Figure 2 Changes in body mass (BM), fat mass (FM), lean body mass (LBM) and protein mass (PM) during the 10 days. $^{\$}P < 0.05$ versus baseline value; $^{*}P < 0.05$ versus previous value

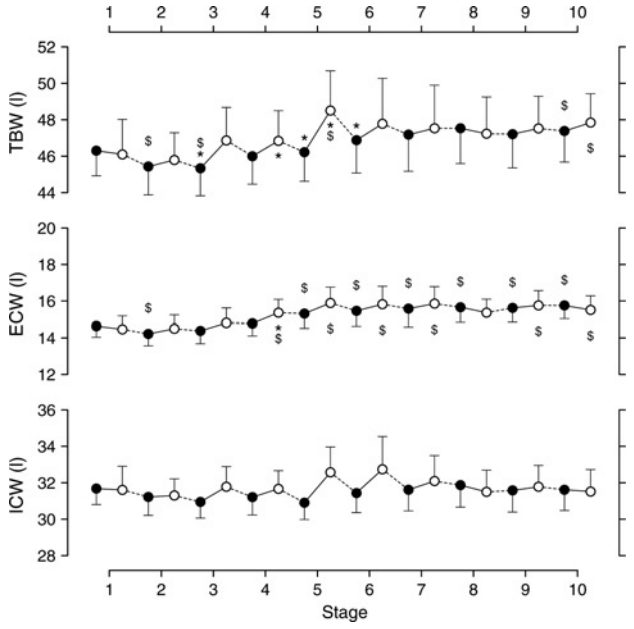

Figure 3 Changes in total body water (TBW), extracellular water (ECW) and intracellular water (ICW) during the 10 stages. $^{\$}P < 0.05$ versus baseline value; $^{*}P < 0.05$ versus previous value

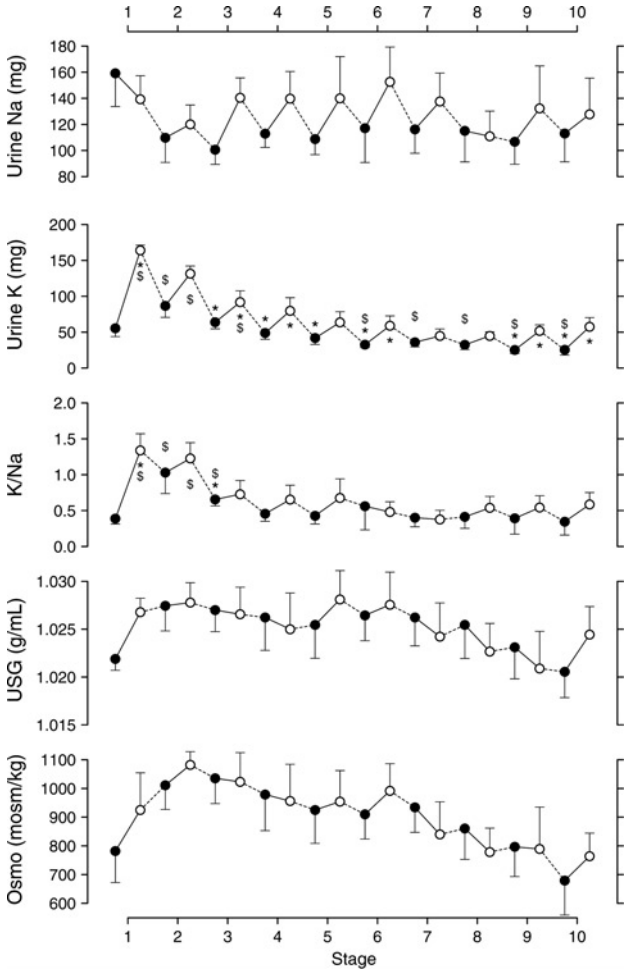

Figure 4 Changes in urinary sodium (urine Na), urinary potassium (urine K), the potassium-to-sodium ratio (K/Na), urinary specific gravity (USG) and urinary osmolality (osmo) during the 10 days. $^{\$}P < 0.05$ versus baseline value; $^{*}P < 0.05$ versus previous value

unchanged at 1.34 (0.46) μU/mL prerace to 1.68 (0.73) μU/mL postrace ($P = 0.252$). The change in aldosterone and the change in and the potassium-to-sodium ratio in urine were not associated ($r = -0.40$, $P = 0.28$); also postrace aldosterone and postrace potassium-to-sodium ratio in urine showed no association ($r = 0.05$, $P = 0.89$). The change in vasopressin was not related to the change in urinary osmolality ($r = -0.22$, $P = 0.59$), and postrace vasopressin was not associated to postrace urinary osmolality ($r = 0.10$, $P = 0.80$). The increase in ECW was neither related to postrace aldosterone ($r = -0.36$, $P = 0.33$) nor to postrace vasopressin ($r = -0.33$, $P = 0.38$). The changes in aldosterone ($r = -0.32$, $P = 0.40$) and vasopressin ($r = -0.28$, $P = 0.47$) were not related to the increase in ECW. Postrace ECW was neither related to postrace aldosterone ($r = -0.40$, $P = 0.28$) nor to postrace vasopressin ($r = -0.26$, $P = 0.49$).

Results of the non-finishers

Table 2 represents the results of the non-finishers. After three stages, four non-finishers were still in the race. Body mass and fat mass decreased significantly whereas total body water and ECW showed no changes. While urinary sodium and urinary potassium remained stable, the potassium-to-sodium ratio increased. Urinary specific gravity and urinary osmolality showed no changes.

Discussion

The aim of this study was to investigate the association between the increase in both total body water and ECW with changes in both aldosterone and vasopressin in a multistage ultraendurance triathlon, in order to determine whether the increase in total body water and ECW would be related to an increased activity in aldosterone or in vasopressin.

A first hypothesis was that aldosterone would be increased after this multistage ultraendurance triathlon. In line with our hypothesis, serum aldosterone increased significantly from 70.8 (104.5) ng/dL prerace to 102.6 (104.6) ng/dL postrace. We see the increase in the potassium-to-sodium ratio as being due to an increased activity of aldosterone. The potassium-to-sodium ratio in urine was <1.0 before the start of the race and increased to >1.0 after day 1 and before and after day 2. This suggests that during this time more potassium than sodium was excreted through the kidney, and a positive ratio for potassium-to-sodium in urine suggests an increased activity of aldosterone. We interpret the increase in urinary potassium as a reaction to a stimulation of the renin–angiotensin–aldosterone-system (RAAS). This suggests that during these days more potassium than

Table 2 Results of the non-finishers were only included when an athlete finished the stage and results from both pre- and poststage were available

Parameter	Before stage 1 ($n = 5$)	After stage 1 ($n = 5$)	Before stage 2 ($n = 4$)	After stage 2 ($n = 4$)	Before stage 3 ($n = 4$)	After stage 3 ($n = 4$)
Body mass (kg)	76.6 (5.7)	72.7 (4.9)*†	74.0 (5.2)*	72.8 (5.1)*†	73.1 (4.6)*†	72.2 (4.6)*
Fat mass (kg)	11.1 (2.7)	8.6 (1.9)	10.7 (1.9)*	8.8 (1.0)*	10.7 (0.9)*†	9.7 (1.3)*†
Lean body mass (kg)	65.6 (5.0)	64.1 (3.3)	63.3 (5.1)	64.0 (4.6)	62.4 (5.1)	62.6 (5.3)
Protein mass (kg)	13.1 (0.9)	12.9 (0.7)	12.8 (1.0)	12.9 (1.0)	12.5 (1.0)	12.5 (1.0)
Total body water (L)	48.4 (3.8)	47.2 (2.5)	46.5 (3.8)	47.1 (3.5)	46.0 (3.8)	46.1 (4.0)
Extracellular water (L)	15.6 (1.5)	14.8 (0.9)	14.5 (1.4)	14.7 (1.2)	14.7 (1.4)	14.8 (1.5)
Intracellular water (L)	32.8 (2.3)	32.4 (1.7)	32.0 (2.5)	32.4 (2.4)	31.3 (2.4)	31.3 (2.5)
Urinary sodium (mg)	176.4 (43.1)	95.8 (46.0)	83.4 (30.5)	82.8 (15.7)	102.5 (41.0)	116.3 (44.4)
Urinary potassium (mg)	73.6 (72.9)	132.4 (57.3)	51.5 (14.8)	135.1 (51.8)	43.1 (11.4)	137.2 (38.7)
Potassium-to-sodium ratio	0.50 (0.65)	1.61 (0.92)	0.70 (0.40)	1.60 (0.43)*	0.49 (0.27)†	1.36 (0.75)
Urinary specific gravity (g/mL)	1.016 (0.004)	1.028 (0.004)	1.025 (0.004)	1.030 (0.001)	1.034 (0.004)	1.022 (0.010)
Urinary osmolality (mosmoL/kg)	746.3 (146.4)	940.4 (177.3)	909.5 (134.9)	1,086.3 (203.4)	1,016.9 (179.7)	1,138.1 (143.0)

*$P < 0.05$ versus baseline value
†$P < 0.05$ versus previous value

sodium was excreted through the kidneys and a positive ratio for potassium to sodium in urine suggests an increased activity of aldosterone. A potassium-to-sodium ratio in urine >1.0 reflects a contraction of the effective extracellular volume leading to a hyper-reninaemic hyperaldosteronaemia. The potassium-to-sodium ratio in urine is a physiological reflection of the potassium excretion in the distal tubulus and in comparison to the sodium re-absorption, an estimate of aldosterone activity in serum. This is also backed up by the increased activity of aldosterone postrace. In the first two days, the potassium-to-sodium ratio was >1.0, and we must assume that aldosterone activity was higher in these days compared with the rest of the race since the RAAS-axis is stimulated in an intensity-dependent fashion.[24] This might also be backed up with the faster stage times in the beginning of the race and the finding that both postrace aldosterone and the change in aldosterone were not related to both the postrace potassium-to-sodium ratio and the change in potassium-to-sodium ratio in urine. The timeframe over 10 days was probably too long that aldosterone would have been increased highly enough.

A second hypothesis was that the increase in aldosterone would be related to the increase in ECW. Although ECW increased, the increase in ECW was neither related to both postrace serum aldosterone nor to the change in serum aldosterone. Speedy *et al.*[4] measured both vasopressin and aldosterone in five subjects and 11 controls after an Ironman triathlon. The median postrace aldosterone in the subjects did not differ significantly from the controls. They found no evidence that aldosterone contributed to fluid retention. Regarding recent findings, an increased activity of vasopressin may contribute to the pathogenesis of EAH.[7,8] However, vasopressin showed no change in this race and the increase in ECW was neither related to postrace vasopressin nor to the change in vasopressin. Likewise, vasopressin and urinary osmolality showed no relationship. Speedy *et al.*[4] measured vasopressin in addition to aldosterone in their subjects and controls after an Ironman triathlon. The median postrace vasopressin was significantly lower in the subjects compared to the controls. They concluded that fluid retention was not due to vasopressin. Also, Williams *et al.*[14] found during a multistage performance an increase in plasma volume, but no increase in vasopressin. One problem in the determination of the activity of vasopressin during a field study is its short half-life time of 24.1 minutes.[25] Considering that the athletes got slower and slower during the race, the activity of vasopressin will also be reduced. The fastest Ironman was on day 1, and the slowest Ironman was on day 9. This might also explain why vasopressin was not increased postrace since vasopressin release is intensity dependent.[26] There is also a delay between the arrival at the finish line and the blood sampling under field conditions.[4] The determination of Copeptin as a stable peptide and precursor of vasopressin might be more useful to detect hormonal changes.[27–29]

Another problem regarding the determination of the fluid retention could be the applied method of bioelectrical impedance analysis. We measured total body water, ECW and intracellular water using the bioelectrical impedance analysis method. This method is reliable under both laboratory situations[21] and field conditions.[9] However, hydration status[22] and extracellular osmolality[23] must be stable. In our athletes, urinary specific gravity, urinary osmolality and urinary sodium remained unchanged throughout the race. Since urinary indices are reliable indices of hydration status[30] with urine osmolality as the most promising marker available,[31] we consider the results of bioelectrical impedance analysis for both total body water and ECW reliable. In a recent study in a multistage mountain bike race, the time course of the decrease in body mass and the increase in ECW could be described in the same manner using the same method.[11] In another recent study in a multistage cycling race, an increase in lean body mass was described while using dual energy X-ray absorptiometry.[32] The meaning of this increase in lean body mass remains unclear, and the development of

an oedema might be postulated.[33] However, we found no association between the increase in lean body mass and the increase in both total body water ($r = -0.36$, $P = 0.33$) and ECW ($r = -0.36$, $P = 0.33$).

One might assume that the non-finishers may have dropped out due to disorders in fluid and electrolyte metabolism since EAH in Ironman triathletes may amount to 18%.[34] However, EAH in Ironman triathletes is usually asymptomatic[34] and the non-finishers in this race gave up due to accident (1), overuse injuries of the lower limbs (1), fatigue and overuse injuries of the lower limbs (2), respiratory problems (1) and loss of motivation (1). These symptoms were not typical for EAH.[5]

This investigation is limited in that haemoglobin, haematocrit, plasma sodium and plasma potassium were not determined before and after each stage in order to show changes between performance and recovery. Also, the data of fluid intake and urinary output would give more insight into fluid regulation.[11] In athletes at the edge of physical performance and without a support crew; however, it is not possible to burden them unnecessarily. The non-invasive investigation such as measuring body composition using bioelectrical impedance analysis[2] and urinary analysis[35] takes little time per day, does not bother ultraendurance athletes while racing and resting, and provides reliable data. However, analysis of haemoglobin, haematocrit and plasma electrolytes could be performed in ultraendurance athletes using a point-of-care (POC) analyser such as the i-STAT,[36] where only capillary samples would be needed.[37]

To summarize, we found an increase in aldosterone and ECW after this multistage ultraendurance triathlon. However, we could not establish an association between the increase in ECW and the increase in aldosterone. Furthermore, we found neither an increase in vasopressin nor an association between both the change in vasopressin and postrace vasopressin with the increase in ECW. The increase in total body water and ECW could not be explained by hormonal changes. In future studies, the marker Copeptin should be determined as a stable peptide and precursor of vasopressin that might be more useful to detect hormonal changes during ultraendurance performance and a potential association between the increase in ECW and hormonal changes. Renal function might have changed during these 10 days and excretion of water might have been reduced. Furthermore, intake of fluid during the heat of the day might have led to fluid overload.

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