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Muscle Cramping in Ultra-Trail: Dehydration and Electrolyte Depletion versus Muscle Damage

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Abstract

Martinez-Navarro, I, Vicente-Mampel, J, López-Grueso, R, Collado-Boira, E, Recacha-Ponce, P, Gómez-Seré, T, and Hernando, C. Muscle cramping in ultra-trail: Dehydration and electrolyte depletion versus muscle damage. *J Strength Cond Res* XX(X): 000–000, 2026—Our aim was to compare dehydration variables, electrolyte and muscle damage serum markers, race pacing, and training background between runners who suffered exercise-associated muscle cramps (EAMC) and those who not in an ultra-trail race. Fifty-eight finishers (37 men and 21 women) from 2 editions of the same race (Penyagolosa Trails CSP) were analyzed. Blood and urine samples were collected, and body mass (BM) was measured before and after the race. Exercise-associated muscle cramps were diagnosed immediately after the race. Nine subjects developed EAMC (16%). Although it was not significantly different, the incidence in men was 4 times greater than in women (males: 22%, females: 5%). Body mass change, postrace urine specific gravity, and serum sodium levels did not differ between crampers and noncrampers. Conversely, postrace potassium was higher among crampers (5.04 ± 0.39 mmol·L^{-1} vs. 4.66 ± 0.48 mmol·L^{-1}; $p = 0.029$; $d = 0.83$). At 24 h and 48 h postrace, creatine kinase ($5{,}166 \pm 3067$ U·L^{-1} vs. 1940 ± 1523 U·L^{-1}, $p = 0.002$, $d = 1.80$; $3{,}194 \pm 2340$ U·L^{-1} vs. 894 ± 775 U·L^{-1}, $p = 0.001$, $d = 2.05$) and lactate dehydrogenase (565 ± 118 U·L^{-1} vs. 420 ± 185 U·L^{-1}, $p = 0.030$, $d = 0.83$; 582 ± 140 U·L^{-1} vs. 401 ± 179 U·L^{-1}, $p = 0.006$, $d = 1.06$) were greater among crampers. Pacing indices were not different between crampers and noncrampers. The percentage of runners who underwent regular lower-limb strength training was different (crampers: 55.6%, noncrampers: 87.5%; $p = 0.021$). Therefore, runners who suffered EAMC did not exhibit a greater degree of postrace dehydration and sodium depletion but displayed higher concentrations of muscle damage biomarkers from 24 to 48 h after the race.

Key Words: sodium, potassium, pacing, strength training, sex differences

Introduction

Exercise-associated muscle cramps (EAMC) may be defined as painful, spasmodic, and involuntary contractions of a skeletal muscle during or shortly after exercise. Exercise-associated muscle cramps are one of the most common injuries in trail runners (45), but their pathophysiology has remained a matter of debate for 2 decades (3,28,35,37). The first and most popular hypothesis about the etiology of EAMC was the dehydration and electrolyte imbalance theory; in fact, a great majority of runners still believe that sodium intake during endurance and ultra-endurance exercise impedes the occurrence of muscle cramps (17,25). However, scientific evidence has demonstrated that the pathophysiological mechanisms originally advocated by this theory are inconsistent (14,28,30).

Alternatively, the altered neuromuscular control theory, first proposed in 1997 and updated in 2009, states that EAMC occur when excessive muscle fatigue linked to vigorous physical exercise generates an imbalance between excitatory and inhibitory stimuli at the α motor nerve (35,37). Consistent with this hypothesis, several studies have failed to show differences in either dehydration (assessed as body mass [BM] loss or by means of urine specific gravity [USG]) or postrace serum electrolyte concentrations between crampers and noncrampers (16,23,24,27,36,39,41). Likewise, controlled laboratory studies, using an electrical stimulation cramping model, also failed to link dehydration to muscle cramp threshold frequency (8,26). Conversely, we and others have shown significantly higher values of muscle damage biomarkers in those runners who had suffered EAMC after a 161-km ultra-trail (UT) and a road marathon. Accordingly, EAMC appear to be the consequence of subjecting the muscles to an excessive demand relative to their current state of training, thus exacerbating muscle damage (16,23).

Hence, considering that the altered neuromuscular control theory seems to be the most scientifically acceptable theory of EAMC (11,28,35), the focus should not only be to ascertain EAMC etiology but also to identify the factors associated with their prevention (31,40). Moreover in the case of UT athletes, given that muscle-related factors and not so much the cardiovascular and energetic components seem to be the main impediment to performance in long UT races (6,13,44). Regarding in-race strategies, a faster first half of the race (i.e., a more positive pacing) has been associated with a higher probability of suffering EAMC in a 56-km ultramarathon (36), whereas no differences in

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relative exercise intensity were noted between crampers and noncrampers during the first half of a full marathon (23). On the other hand, concerning prerace strategies, a recent review showed that stretching has no prophylactic effect in reducing EAMC incidence (12). Conversely, strength training appears to exert a protective effect against cramping (23,46).

Thereby, the main purpose of our study was to assess whether runners who suffered from EAMC exhibited differences in serum electrolytes, dehydration markers, and muscle damage biomarkers, compared with runners who did not experience EAMC during an UT race. In addition, we wanted to explore whether crampers followed a more aggressive pacing in the first segment of the race or exhibited differences in training-related variables (i.e., strength training, recent injuries, running experience, weekly running volume, and cumulative positive/negative elevation gain, etc.) compared with noncrampers. Following former studies in the field (16,23,36), we hypothesized that runners who experienced EAMC displayed greater concentrations of muscle damage biomarkers without differences in serum electrolytes and dehydration markers. In addition, we thought that a lower percentage among crampers regularly performed lower-limb strength training; on the other hand, we believe that crampers ran faster (i.e., relative to average speed during the whole race) the first segment of the race and largely slowed in the last segment of the race.

Methods

Experimental Approach to the Problem

The study was a cross-sectional investigation that used data from 2 different editions (2019 and 2025) of the same race (Penyagolosa Trails CSP). Data from the 2019 sample are a secondary analysis from a published paper assessing the effects of full body compression garment on UT recovery (20). Race characteristics varied minimally between the 2 editions. Track of 2019 edition consisted of 107.4 km, starting at an altitude of 40 m and finishing at 1,280 m above the sea level, with a total positive and negative elevation of 5,604 and 4,356 m, respectively. Track of 2025 edition consisted of 106.1 km, starting at an altitude of 40 m and finishing at 1,280 m above the sea level, with a total positive and negative elevation of 5,584 and 4,369 m, respectively. In 2019, the temperature at the start was 17.2° C, and it ranged between 18 and 10.6° C at mid-race (km 66), and between 20.1 and 1.5° C at the finish line. In 2025, the temperature at the start was 14° C, and it ranged between 18.9 and 7.4° C at mid-race (km 66), and between 16.2 and 0.5° C at the finish line. Competition- and training-related data were obtained using a standardized questionnaire. Before and after the race, the subjects were weighed and urine and blood samples were collected. Exercise-associated muscle cramps were diagnosed immediately after the race.

Subjects

Data from 58 experienced trail runners (37 men and 21 women; age range from 31 to 60 years), finishers of the race in 2 different years (31 athletes from the 2019 edition and 27 athletes from the 2025 edition) were analyzed in this study. An email invitation to participate in the study was sent to all race entrants. The volunteers were excluded if they were previously diagnosed with cardiac or renal diseases or taking medication regularly, or did not finish at least 2 races longer than 65 km. Exclusion criteria were verified by subjects self-report through a first-contact questionnaire, and 5 volunteers were excluded from the study. The subjects' characteristics are presented in Table 1. All subjects were fully informed of the procedure and provided written consent to participate. They were also allowed to withdraw from the study at will. Both research projects were conducted according to the Declaration of Helsinki and approved by the Research Ethics Committee of the Jaume I University of Castellon (reference numbers CD/007/2019 and CEISH/103/2024, respectively). They were also enrolled in the ClinicalTrails.gov database, with the code numbers NCT03990259 and NCT06969898, respectively (www.clinicaltrials.gov).

Procedures

Training and Competition-Related Data and Pacing Indexes. A standardized questionnaire was used in both research projects to collect demographic and medical information, and competition and training-related data of the 3-month period leading to the race. The following variables were considered for analysis in the present study: number of years running, number of completed races >100 km, mean weekly training days, mean weekly training hours, mean weekly training volume (km), mean weekly cumulative positive/negative elevation gain, strength training (i.e., having performed at least 2 weekly lower-limb strength training sessions), and injuries (i.e., having sustained any injury that resulted in time-loss from training). The race was divided into 4 sections based on the 3 main aid stations established by the organization (33rd km, 66th km, and 84th km; 0–33 km, 33–66 km, 66–84 km, and 84th km finishing line), partial times were obtained from race results (LiveTrail, LiveTrail SARL, France), and flat-equivalent mean running speed was calculated according to the procedure established by Saugy et al. (34). Two pacing indices were calculated: IP_{1stQ}, race average speed divided by the average speed on the first race segment, as a measure of athletes' degree of contention/aggressivity in their race strategy, and IP_{4thQ}, percentage speed decrease in the last race segment as compared with the first race segment (2,22).

Hydration Status. Hydration status was estimated in duplicate from USG and from changes in BM. Urine specific gravity was measured from the last urine sample before race started (within the hour before) and the first postrace void urine sample (sediMax conTrust, 77 Elektonika Kft, Budapest, Hungary). Body mass was measured within 1 hour before the race started and immediately after crossing the finishing line. The BM measurements were made using calibrated electronic scales with a precision of 0.1 kg (Seca 813; Vogel and Halke, Hamburg, Germany). Following a previous study (23), both prerace and postrace measurements were made with the runner clothed in running wear and shoes, but other items such as waist packs and hydration vests were removed, and nothing was permitted in the runner's hands.

Blood Sampling and Analysis. Blood samples were collected from an antecubital vein by venipuncture the day before the race, after crossing the finishing line, and 24 and 48 hours postrace using BD Vacutainer PST II tubes. The samples were centrifuged at 3,500 rpm for 10 minutes and kept at 4° C during transport to the hospital. Samples were processed in 2 different hospitals of Castellon (2019 samples at the Vithas Rey Don Jaime Hospital and samples from 2025 edition at the Provincial Hospital Consortium) using a modular platform analyzer Cobas c311 (Roche Diagnostics, Penzberg, Germany) and a Beckman Coulter DXC 700 AU analyzer (Beckman Coulter, Inc., Brea, CA), respectively. The following blood variables were analyzed: lactate dehydrogenase (LDH) and creatine kinase (CK) from prerace to

Table 1
Sample main characteristics.*

	All sample (n = 58)	Males (n = 37)	Females (n = 21)
Age (y)	44 ± 7	42 ± 6	46 ± 8
Number of years running	11 ± 6	12 ± 7	10 ± 5
Number of races >100 km	2 ± 4	3 ± 4	2 ± 3
Weekly training days	5 ± 1	5 ± 1	5 ± 1
Weekly training hours	11 ± 6	11 ± 6	11 ± 5
Weekly running volume (km)	70 ± 22	73 ± 23	65 ± 19
Weekly cumulative positive/negative elevation (m)	2,145 ± 1,026	2,305 ± 1,139	1851 ± 711
Previous injuries (%)	82.5%	75.7%	95%
Strength training (%)	29.3%	29.7%	28.6%

*Values are expressed as mean ± *SD*. Previous injuries (%) = percentage of subjects who had sustained any injury in the previous 3 months; strength training (%) = percentage of subjects who performed at least 2 weekly lower-limb strength training sessions.

48 hours postrace (9); sodium [Na+] and potassium [K+] from prerace to postrace. For the blood samples obtained after the race, values of the aforementioned biomarkers were corrected using the Dill and Costill formula (10). Briefly, when considering pre-post comparisons of biomarkers after long-lasting strenuous exercise, changes in plasma volume (ΔPV) and hemoconcentration caused by dehydration should be considered, and for that purpose, a correction factor based on hemoglobin (Hb) and hematocrit (Htc) values is applied: ΔPV (%): $100 \times ([Hb_{pre}/Hb_{post}] \times [100\text{-}Htc_{post}]/[100\text{-}Htc_{pre}] - 1)$ (1,10).

Diagnosis of Exercise-Associated Muscle Cramps. During the prerace assessments, all runners were informed of the symptoms and signs of EAMC. After race completion, an experienced sports physician asked finishers whether they had suffered EAMC during the race and verified that cramping was located in a very active muscle group during the race (i.e., lower-limb muscles) with no history of an acute muscle tear, following established clinical criteria (38) and previous studies in the field (23).

Statistical Analysis

Statistical analyses were carried out using the Statistical Package for the Social Sciences software (IBM SPSS Statistics for Windows, version 30, IBM Corp., Armonk, NY). The normal distribution of the variables was a priori verified through the Kolmogorov-Smirnov test (K-S test). Subsequently, non-normally distributed variables (prerace [Na+], prerace and postrace USG, all CK-derived absolute and relative parameters, and training-related variables except for mean weekly training volume and cumulative positive/negative elevation) were compared between crampers and noncrampers using nonparametric tests: Friedman and Wilcoxon tests for paired samples, and Mann-Whitney *U*-tests for unpaired samples. Normally distributed variables were compared using repeated measures mixed-effects ANOVA and Student's *t*-tests. Homogeneity of variance was verified by Levene's test, and Greenhouse-Geisser adjustment was performed when sphericity was violated. Pairwise post-hoc comparisons in both parametric and nonparametric tests were adjusted by Bonferroni correction. Categorical data (frequency of strength training and injuries in crampers and noncrampers; incidence of EAMC in men and woman) were analyzed by means of chi-square tests. The meaningfulness of the outcomes was estimated through the partial estimated effect size (η^2 partial) for ANOVA and Cohen's D effect size for pairwise comparisons. In the latter case, a Cohen's $D < 0.5$ was considered small; between 0.5 and 0.8, moderate; and greater than 0.8, large (43). The significance level was set at $p < 0.05$, and data are presented as means and $\pm SD$.

Results

Nine runners developed EAMC (16%) during or immediately after the race. The difference in the incidence between men and women (females: 5%; males: 22%) was not statistically significant ($p = 0.088$). In relation to the etiological nature of EAMC, no significant differences were found in hydration status variables (prerace and postrace USG and BM change), prerace and postrace serum [Na+] between those who did or did not experience EAMC. Conversely, postrace [K+] was significantly higher among crampers (5.04 ± 0.39 mmol·L^{-1} vs. 4.66 ± 0.48 mmol·L^{-1}; $p = 0.029$; $d = 0.83$) (see Table 2). Regarding muscle damage biomarkers, Friedman analysis revealed significant differences both in crampers and noncrampers in the evolution of CK from prerace to 48 hours after the race (crampers: $\chi^{2(3)} = 22.73$, $p < 0.001$; noncrampers: $\chi^{2(3)} = 145.82$, $p < 0.001$) (see Figure 1 and Table 3). Both in crampers and noncrampers, CK concentration largely increased from prerace to postrace and largely decreased from 24 hours postrace to 48 hours postrace. However, from postrace to 24 hours postrace, CK levels only decreased among crampers. In addition, crampers showed significantly and largely higher CK levels than noncrampers at 24 hours postrace and 48 hours postrace. Levene's test showed no significant difference in variances between crampers and noncrampers in LDH concentrations across measurement points ($p > 0.05$), and the resulting ANOVA analysis showed a significant effect on LDH for "Time" [$F = 78.01$; $p < 0.001$; η^2 partial = 0.59] and "Time × Group (crampers vs. noncrampers)" interaction [$F = 5.61$; $p = 0.012$; η^2 partial = 0.09] (see Figure 2 and Table 3). Both in crampers and noncrampers, post-hoc analysis showed that LDH concentration largely increased from prerace to postrace. However, from postrace to 24 hours postrace, LDH levels only decreased among noncrampers. In addition, crampers showed significantly and largely higher LDH levels than noncrampers at 24 hours postrace and 48 hours postrace.

On the other hand, pacing indices were not different between crampers and noncrampers. The percentage of runners who have sustained an injury in the last 3 months before the race, the number of years running, the number of completed races >100 km, the mean weekly training days and hours, and the mean weekly training volume and cumulative positive/negative elevation gain were neither different between crampers and noncrampers (see Table 4). However, a significant difference in the percentage of runners who undertook regular lower-limb

Table 2
Hydration status and electrolyte variables prerace and postrace in crampers and noncrampers.*†

	NONCRAMPERS (n = 49)	CRAMPERS (n = 9)	p	ES	95% CI for ES	
					Upper bound	Lower bound
Prerace USG ($mg \cdot ml^{-1}$)	1,016 ± 6	1,018 ± 6	0.238	0.43	1.15	−0.28
Postrace USG ($mg \cdot ml^{-1}$)	1,023 ± 6	1,024 ± 5	0.711	0.19	0.90	−0.53
Δ BM (%)	−2.98 ± 1.96	−4.33 ± 2.55	0.162	−0.67	0.05	−1.39
Prerace Na^+ ($mmol \cdot L^{-1}$)	139.4 ± 2.7	139.6 ± 2.5	0.957	0.08	0.79	−0.63
Postrace Na^+ ($mmol \cdot L^{-1}$)	139.7 ± 4.2	140.7 ± 3.6	0.494	0.25	0.96	−0.47
Prerace K^+ ($mmol \cdot L^{-1}$)	4.43 ± 0.52	4.63 ± 0.67	0.304	0.37	1.09	−0.34
Postrace K^+ ($mmol \cdot L^{-1}$)	4.66 ± 0.48	5.04 ± 0.39	0.029‡	0.83	1.55	0.10

*USG = urine specific gravity; ΔBM = body mass change; NA = sodium; K = potassium.
†Values are expressed as mean ± *SD*.
‡$p < 0.05$.

strength training was observed between those who experienced EAMC and those who did not (crampers: 55.6%, noncrampers: 87.5%; $p = 0.021$).

Discussion

The prevalence of cramps in our sample (16%) was similar to that reported after a 161-km UT race (14.3%) (16), although it was lower than that recorded after shorter races (i.e., road marathons, flat 56-km and 100-km ultramarathons; from 18 to 40%) (18,23,24,36). Although it was not significantly different, the cramping incidence in men was 4 times greater than in women (males: 22%, females: 5%). This finding is similar to a previous study by Schwellnus et al. (39) in an Ironman distance triathlon, where the cramping incidence in men was 2.5 times greater than in women (males: 24%, females: 9%; $p = 0.089$). In another study from this group (36), although the difference for sex was neither statistically significant, among 7 women, only 1 suffered cramps (14.3%), whereas 19 out of 42 men experienced cramps (45.2%). Moreover, the prevalence of a history of EAMC among race entrants of the Comrades Marathon (90 km) was significantly higher among men (16 vs. 8%) (19). Therefore, although our results did not confirm it, previous literature suggest that women appear to be less prone than men to suffer EAMC as race distance increases. This might be related to their larger proportion of type I fibers and their greater resistance to neuromuscular fatigue (4,5,42).

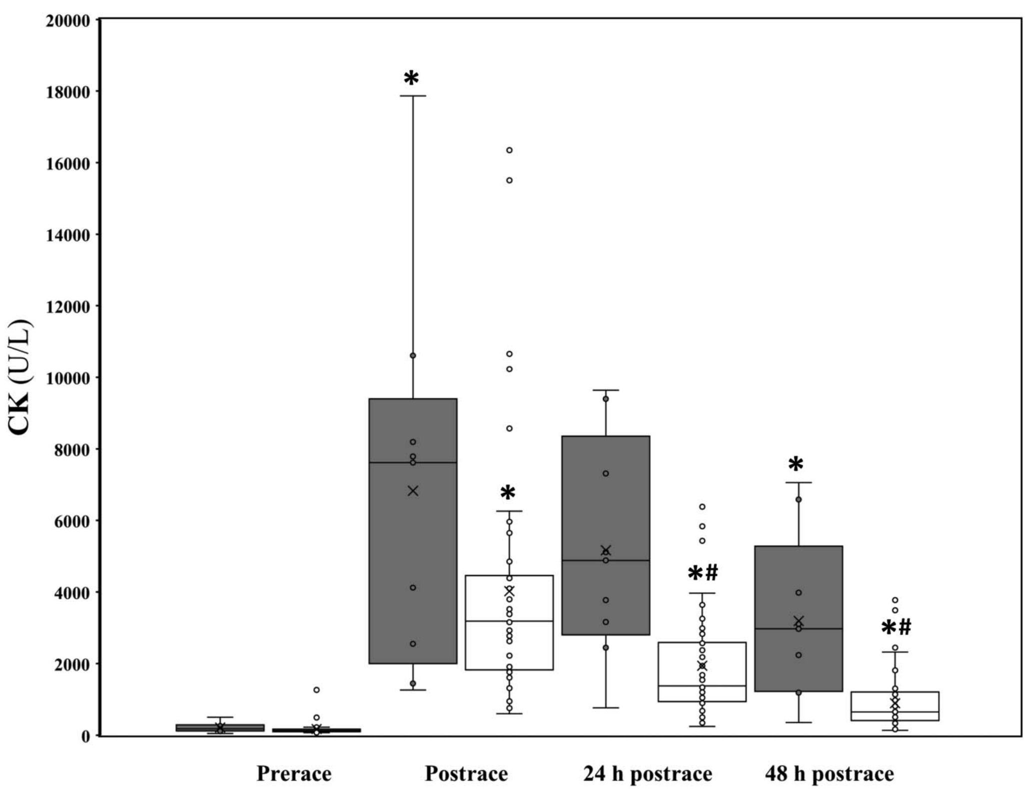

Figure 1. Creatine kinase evolution from prerace to 48 h postrace in crampers (gray boxes) and noncrampers (white boxes). Paired samples were analyzed using Friedman and Wilcoxon tests, whereas unpaired samples were compared by Mann-Whitney *U*-test. Noncrampers = 49; crampers = 9. The box includes the values between the first and the third quartiles (i.e., interquartile range, IQR), and the whiskers the lowest and the highest data points within 1.5 × IQR below Q1 and above Q3, respectively. *Significantly different from the preceding time point ($p < 0.05$). #Significantly different from crampers ($p < 0.05$).

Table 3

Creatine kinase and lactate dehydrogenase evolution from prerace to 48 h postrace in crampers and noncrampers (mean ± *SD*).*†

			Time factor pairwise comparisons (noncrampers; crampers)		Group factor pairwise comparisons	
	Noncrampers (n = 49)	Crampers (n = 9)	p	Cohen's D [95% CI]	p	Cohen's D [95% CI]
CK ($U \cdot L^{-1}$)						
Prerace	173 ± 177	212 ± 136	—	—	0.127	0.23 (−0.48 to 0.94)
Postrace	4,019 ± 3,381	6,828 ± 5,303	<0.001‡; 0.008‡	1.62 (1.17 to 2.08); 1.87 (0.76 to 2.98)	0.141	0.77 (0.04 to 1.49)
24 h postrace	1940 ± 1,523	5,166 ± 3,067	<0.001‡; 0.374	−0.80 (−1.21 to −0.39); −0.41 (−1.34; 0.53)	0.002‡	1.80 (1.02 to 2.58)
48 h postrace	894 ± 775	3,194 ± 2,340	<0.001‡; 0.008‡	−0.87 (−1.29 to −0.46); −0.77 (−1.72 to 0.19)	0.001‡	2.06 (1.25 to 2.86)
LDH ($U \cdot L^{-1}$)						
Prerace	259 ± 91	287 ± 68	—	—	0.383	0.32 (−0.39 to 1.04)
Postrace	527 ± 248	603 ± 149	<0.001‡; <0.001‡	1.45 (1 to 1.89); 2.89 (1.57 to 4.22)	0.359	0.33 (−0.39 to 1.04)
24 h postrace	420 ± 185	565 ± 118	<0.001‡; 0.887	−0.49 (−0.90 to −0.09); −0.30 (−1.23 to 0.63)	0.030‡	0.83 (0.11 to 1.56)
48 h postrace	401 ± 179	582 ± 140	0.098; >0.99	−0.11 (−0.50 to 0.29); 0.14 (−0.79 to 1.06)	0.006‡	1.06 (0.32 to 1.80)

*CK = creatine kinase; LDH = lactate dehydrogenase. For the time factor, pairwise comparisons referred to the preceding time point.
†Values are expressed as mean ± *SD*.
‡$p < 0.05$.

Regarding EAMC etiology, our results showed that runners who suffered muscle cramps did not evidence neither greater BM losses nor lower postrace USG values. Nevertheless, although not statistically significant, the difference in ΔBM loss between crampers and noncrampers was of moderate magnitude ($-4.33 \pm 2.55\%$ vs. $-2.98 \pm 1.96\%$; $p = 0.162$; $d = -0.67$). This discrepancy between ΔBM and USG as indicators of dehydration might be explained by the fact that an important percentage of BM loss during ultra-endurance events is because of factors other than pure fluid loss (i.e., loss of glycogen, as fuel for energy production, and the corresponding loss of body water) (29,33). Accordingly, greater weight losses during self-paced ultra-endurance events could be also the reflection of a greater energy expenditure (21).

Crampers did neither exhibit a lower postrace serum [Na+] concentration. Therefore, EAMC were not related to dehydration or sodium depletion. These results add further evidence to previous studies performed in road marathon (23,24), Ironman

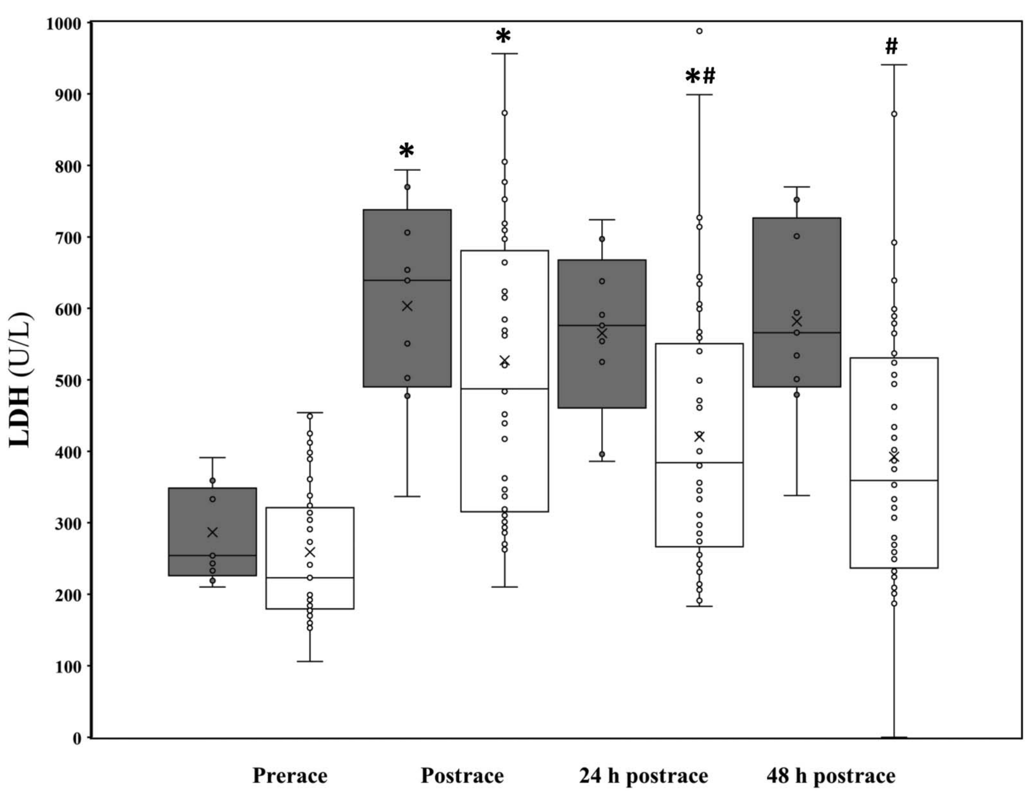

Figure 2. Lactate dehydrogenase evolution from prerace to 48 h postrace in crampers (gray boxes) and noncrampers (white boxes). A repeated measures mixed-effects ANOVA with Bonferroni-adjusted post-hoc pairwise comparisons was used as a statistical procedure. Noncrampers = 49; crampers = 9. The box includes the values between the first and the third quartiles (i.e., interquartile range, IQR), and the whiskers the lowest and the highest data points within 1.5 × IQR below Q1 and above Q3, respectively. Time × group (crampers vs. noncrampers) interaction [$F = 5.61$; $p = 0.012$; η^2 partial = 0.09]. *Significantly different from the preceding time point ($p < 0.05$). #Significantly different from crampers ($p < 0.05$).

Table 4
Pacing, competition, and training-related variables in crampers and noncrampers.*†

	Noncrampers (n = 49)	Crampers (n = 9)	p	ES	95% CI for ES Upper bound	95% CI for ES Lower bound
IP_{1stQ} (%)	83 ± 6	82 ± 7	0.550	−0.22	0.49	−0.93
IP_{4thQ} (%)	−33 ± 9	−34 ± 11	0.613	−0.19	0.52	−0.90
Number of years running	11 ± 6	15 ± 10	0.283	0.61	1.33	−0.11
Number of races >100 km	2 ± 4	4 ± 4	0.199	0.51	1.23	−0.21
Weekly training days	6 ± 1	6 ± 1	0.711	0.11	1.75	0.28
Weekly training hours	11 ± 6	12 ± 4	0.107	0.18	0.89	−0.53
Weekly running volume (km)	69 ± 22	77 ± 19	0.287	0.38	1.09	−0.34
Weekly cumulative positive/negative elevation (m)	2073 ± 1,060	2,530 ± 757	0.223	0.45	1.17	−0.26

*IP_{1stQ}= race average speed divided by the average speed on the first race segment; IP_{4thQ} = percentage speed decrease in the last race segment as compared with the first race segment.
†Values are expressed as mean ± *SD.*

distance triathlon (39,41), ultramarathon, and UT races (16,36). The higher [K+] among crampers may be because of a disproportionate replacement of sweat K+ losses through sports drinks and supplements (28). It could also be the consequence of a larger liberation of intramuscular K+, as crampers sustained greater muscle damage (15). A similar result was observed in cramping Ironman triathletes (32). Thereby, proper electrolyte replacement strategies and supplement use should be further emphasized, mainly among recurrent crampers who still believe that electrolyte imbalance is the cause of EAMC (15,17,25,28).

Meanwhile, we observed largely higher concentrations in CK and LDH at 24 and 48 hours after the race among those runners experiencing EAMC. Our findings thus endorse the "altered neuromuscular control theory" of EAMC (28,35) and coincide with 2 previous studies conducted in a road marathon and a 161-km UT race (16,23). In contrast, the absence of differences in prerace muscle damage biomarkers refutes previous assumptions regarding the relationship between EAMC and the degree of subclinical prerace muscle damage (36). Consequently, pacing strategies aimed at avoiding excessive premature muscle damage and training-related strategies targeted at diminishing the susceptibility to muscle damage may play a role in EAMC prevention.

A faster first half of the race (i.e., a more positive pacing) was associated with a higher probability of suffering EAMC in a 56-km ultramarathon (36), whereas we found no differences in relative speed (i.e., expressed as a percentage of either the peak speed in a cardiopulmonary exercise test or the speed associated with the second ventilatory threshold) between crampers and noncrampers until the 25th km of a marathon (23). Contrary to our hypothesis, the results of the current study showed that neither a faster initial race segment nor a slower last race segment were traits of crampers as compared with noncrampers. In other words, no pacing differences were noted between those who suffered EAMC and those who did not. It is possible that the cramping influence on pacing is lower in longer (and slower) UT races than in flat road marathons (23). Nevertheless, further studies performing an in-depth assessment of race pace (i.e., using GPS or accelerometry-derived data) are required to ascertain the influence of cramping and muscle damage in level, downhill, and uphill UT pacing. On the other hand, increasing weekly running distance and cumulative negative elevation gain have been proposed as a training strategy to reduce muscle damage responsiveness in UT races (44). However, our results showed no differences between crampers and noncrampers regarding mean weekly mileage and cumulative negative elevation gain. Previous studies on ultramarathons also showed no differences in weekly running distance between those subjects who reported EAMC and those who did not (16,36). Conversely, as in our previous research on marathon runners (23), UT athletes who experienced EAMC were less likely to regularly perform lower-limb strength training than their noncramping counterparts (crampers: 55.6%, noncrampers: 87.5%). Taken together, these results should encourage UT runners to improve their adherence to strength training to, among other benefits (7,47), reduce their proneness to suffer EAMC.

In summary, muscle damage, unlike dehydration and electrolyte depletion, was consistently greater from 24 to 48 h after the race among crampers than among noncrampers, thus confirming our first hypothesis. In addition, women appear to be less prone to suffer EAMC in UT races than are men. On the other hand, contrary to our expectations, crampers and noncrampers did not differ by the pacing adopted during the race. Finally, the results of the present study reinforce the notion that strength straining, rather than increasing weekly running distance and cumulative negative elevation gain, could exert a protective role against EAMC occurrence.

Nevertheless, we must acknowledge that the EAMC group was relatively small as a limitation of the study. Taking as a reference the effect size found in a previous work (large, >0.8) for crampers versus noncrampers pairwise comparison regarding post-marathon CK and LDH (23), between 35 and 37 subjects (parametric/nonparametric comparisons) would have been the appropriate sample size (1-tailed $\alpha < 0.05$, 1-β >0.95) (Gpower, version 3.1.9.7, Universität Düsseldorf, Germany). This could have resulted in inaccurate effect size estimates, and thus the results of our study should be interpreted with caution until replicated in larger studies.

Practical Applications

In light of the abovementioned findings, runners suffering from EAMC should be aware that they have subjected their muscles to an excessive demand according to their current state of training, thus provoking exacerbated muscle damage, and should consider a longer postrace recovery period. At the same time, clinicians and nutritionists should further emphasized proper electrolyte replacement strategies and supplement use in those athletes. Last, coaches are urged to prioritize strength training in the training regime of their UT athletes.

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