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Muscle Cramping in the Marathon: Dehydration and Electrolyte Depletion vs. Muscle Damage

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Abstract

Martínez-Navarro, I, Montoya-Vieco, A, Collado, E, Hernando, B, Panizo, N, and Hernando, C. Muscle Cramping in the marathon: Dehydration and electrolyte depletion vs. muscle damage. *J Strength Cond Res* XX(X): 000–000, 2020—Our aim was to compare dehydration variables, serum electrolytes, and muscle damage serum markers between runners who suffered exercise-associated muscle cramps (EAMC) and runners who did not suffer EAMC in a road marathon. We were also interested in analyzing race pacing and training background. Nighty-eight marathoners took part in the study. Subjects were subjected to a cardiopulmonary exercise test. Before and after the race, blood and urine samples were collected and body mass (BM) was measured. Immediately after the race EAMC were diagnosed. Eighty-eight runners finished the marathon, and 20 of them developed EAMC (24%) during or immediately after the race. Body mass change, post-race urine specific gravity, and serum sodium and potassium concentrations were not different between crampers and noncrampers. Conversely, runners who suffered EAMC exhibited significantly greater post-race creatine kinase (464.17 ± 220.47 vs. 383.04 ± 253.41 UI/L, $p = 0.034$) and lactate dehydrogenase (LDH) (362.27 ± 72.10 vs. 307.87 ± 52.42 UI/L, $p = 0.002$). Twenty-four hours post-race also values of both biomarkers were higher among crampers (CK: 2,438.59 ± 2,625.24 vs. 1,166.66 ± 910.71 UI/L, $p = 0.014$; LDH: 277.05 ± 89.74 vs. 227.07 ± 37.15 UI/L, $p = 0.021$). The difference in the percentage of runners who included strength conditioning in their race training approached statistical significance (EAMC: 25%, non-EAMC: 47.6%; $p = 0.074$). Eventually, relative speed between crampers and noncrampers only differed from the 25th km onward ($p < 0.05$). Therefore, runners who suffered EAMC did not exhibit a greater degree of dehydration and electrolyte depletion after the marathon but displayed significantly higher concentrations of muscle damage biomarkers.

Key Words: sodium depletion, pacing, athletic performance, strength training

Introduction

Exercise-associated muscle cramps (EAMC) are defined as "painful, spasmodic, and involuntary contractions of skeletal muscle during or immediately after physical exercise" (30). Muscle cramps are one of the most important performance-limiting factors in long-distance races and one of the main causes given for withdrawing from those competitions (11). Previous studies have reported an EAMC prevalence of 14% during a 166-km ultramarathon (12), 18% during a road marathon (20), 23% during an Ironman-distance triathlon and a 100-km ultramarathon (14,32), and 41% during a 56-km ultramarathon (29).

Exercise-associated muscle cramps have a typical clinical presentation resulting from intense and prolonged physical exercise, and they usually occur in muscles subjected to a high contractile demand during exercise exertion (28). The first and most popular hypothesis about the etiology of EAMC was the dehydration and electrolyte depletion theory (13); in fact, most runners still believe that sodium intake during endurance exercise prevents the occurrence of muscle cramps (21). However, scientific evidence is inconsistent with this theory, and it does not offer pathophysiological mechanisms by which this could occur (9,24). More recent studies suggest that the origin of these alterations in neuromuscular control are primarily caused by the action of excessive muscular fatigue linked to vigorous physical exercise, this being the main factor associated with the appearance of muscle cramps (12,28). To date, observational studies have failed to show differences in either dehydration (assessed as body mass [BM] loss or by means of urine specific gravity [USG]) or post-race serum electrolyte concentrations between athletes experiencing EAMC and those who do not experience EAMC (12,14,20,29,32,35). Controlled laboratory studies, using an electrical stimulation cramping model, also failed to link dehydration to muscle cramp threshold frequency (2,22). Conversely, in a recent study by Hoffman and Stuempfle (12), significantly higher values of muscle damage were found in those runners who had suffered EAMC. Authors interpreted these results suggesting that these runners had subjected their muscles to an excessive demand according to their current state of training thus generating an alteration in neuromuscular control that finally triggered muscle cramping. Therefore, considering that the "altered neuromuscular control theory" seems to be the most scientifically acceptable theory of EAMC (7,27), the focus to determine their etiology now should shift to the identification of the factors associated with their appearance (25,33).

Therefore, the main purpose of our study was to observe whether runners who suffered from EAMC exhibited differences

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in serum electrolytes, dehydration markers, and enzyme biomarkers of muscle damage, compared with runners who did not experience EAMC. In addition, to assess whether those athletes experiencing EAMC adopted a different pacing strategy during the race or exhibited differences in training-related variables (i.e., strength training, previous running experience, weekly running volume, etc.), compared with those who did not experience EAMC. Interestingly, as far as we are aware, no previous study has compared objectively measured relative intensity (i.e., percentage of maximal speed and speed associated with second ventilatory threshold (VT2) measured in a cardiopulmonary exercise test [CPET]) and strength training background between crampers and noncrampers, although strengthening has been postulated as a suitable intervention to reduce EAMC incidence (37). Our hypothesis is that runners who experienced EAMC displayed greater concentrations of muscle damage biomarkers without differences in serum electrolytes and dehydration markers. Moreover, we believe that crampers ran at a higher relative intensity during the first half of the race and did not perform strength training during their preparation for the marathon.

Methods

Experimental Approach to the Problem

The study was performed in the Valencia Trinidad Alfonso EDP 2016 Marathon. Average temperature and relative humidity during the race were 19° C and 61%, respectively. Water aid stations were located every 5 km. Before the race, training-related and competition-related data were obtained, and subjects were subjected to a CPET. Before and after the race, subjects' BM was measured, urine samples were collected, and blood samples were drawn by experienced nurses. Exercise-associated muscle cramps were diagnosed immediately after the race.

Subjects

All subjects of the race received an invitation email to participate in the study. Two information seminars were organized to fully explain the study design (aims, measurements, etc.) to those subjects who accepted the invitation ($N = 456$). A total of 98 runners (83 men and 15 women) were selected to participate in this study, according to the following inclusion criteria: age between 30 and 45 years; BM index between 16 and 24.99 $kg \cdot m^{-2}$; having a performance best time in marathon between 3 and 4 hours for men and 3:30 and 4:30 hours for women; and healthy subjects who were free from cardiac or renal disease and from taking any medication on a regular basis. Subject characteristics are presented in Table 1. All subjects included in this study were fully informed and gave their written consent to participate. The research was conducted according to the Declaration of Helsinki, and it was approved by the Research Ethics Committee of the Jaume I University of Castellon. This study is enrolled in the ClinicalTrails.gov database, with the code number NCT03155633 (www.clinicaltrials.gov).

Procedures

Training-Related and Competition-Related Data. A standardized questionnaire was used to collect demographic and medical information as well as training-related and competition-related data (10). The following variables were considered for analysis in this study: number of years running, number of completed marathons, mean weekly training days, mean weekly training hours, mean weekly training volume (km), recovery hours from the last run (either specific or not to their training program) before the race, strength training (i.e., having performed at least one weekly lower-body resistance training in the previous 3 months), and injuries (i.e., having sustained any injury that results in time loss from training in the previous 3 months).

Cardiopulmonary Exercise Test and Pacing. Cardiopulmonary exercise tests were performed on a treadmill (H/P/cosmos pulsar; H/P/cosmos sports & medical GmbH, Nussdorf-Traunstein, Germany) between 2 and 4 weeks before the marathon. Pulmonary $\dot{V}O_2$ and $\dot{V}CO_2$ were measured breath-by-breath using an automated online system (Oxycon Pro system, Jaeger, Würzburg, Germany). Gas analysis system was calibrated for ambient temperature and humidity, air flow, and $\dot{V}O_2$ and $\dot{V}CO_2$ concentrations (with a 4.96% CO_2–12.10% O_2 gas mixture), before each testing session according to manufacturer's instructions. A CPET protocol consisted of 3 minutes warm-up at 6 $km \cdot h^{-1}$, followed by ramp speed increases of 0.25 $km \cdot h^{-1}$ every 15 seconds until volitional exhaustion (8,23). A 3-minute constant speed stage at 11 $km \cdot h^{-1}$ for women and 12 $km \cdot h^{-1}$ for men was included in the protocol so as to enable running economy measurements. $\dot{V}O_2max$ values were accepted when a plateau (an increase of <2 $ml \cdot kg \cdot min^{-1}$) or a decline in $\dot{V}O_2$ was reached despite increasing workloads and a respiratory exchange ratio above 1.15 was achieved. If these criteria were not met, a $\dot{V}O_2peak$ value was taken, defined as the highest $\dot{V}O_2$ measured over a 30 seconds period. Second ventilatory threshold was estimated from gas exchange data by 2 independent researchers following a validated standard methodology previously described (18). Five-kilometer split times were extracted from the official race results and then relativized according to each runner speed at VT2 ($\%V_{VT2}$) and maximal speed ($\%V_{MAX}$) achieved during the CPET.

Hydration Status. Hydration status was estimated in duplicate from USG and from changes in BM. The USG was measured from a first-morning void urine sample (the day of the race) and the first–post-race void urine sample. The BM was measured within 1 hour before race started and immediately after crossing the finishing line. The BM measurements were made with calibrated electronic scales with precision 0.1 kg (Seca 813; Vogel and Halke, Hamburg, Germany). Following a previous study (19), both pre-race and post-race 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 at baseline (the day before the race), after finishing the marathon and 24 hours post-race using BD Vacutainer PST II tubes by experienced nurses. Samples were centrifuged at 3,500 rpm for 10 minutes and kept at 4° C during transport to the Vithas-Nisa 9 de Octubre Hospital (Valencia), where they were processed using the modular platform Roche/Hitachi clinical chemistry analyzer Cobas c311 (Roche Diagnostics, Penzberg, Germany), as previously published (1). The following blood variables were considered for analysis: lactate dehydrogenase (LDH), creatine kinase (CK), sodium [Na+], and potassium [K+]. For the blood sample obtained immediately after the race, values of the aforementioned biomarkers were corrected using Dill and Costill formula (6). Briefly, when considering pre-post comparisons in biomarkers after an exercise

Table 1
Sample main characteristics (mean ± *SD*).*

	All sample ($n = 98$)	Males ($n = 83$)	Females ($n = 15$)
Age (y)	38.72 ± 3.63	38.76 ± 3.65	38.50 ± 3.63
BMI ($kg \cdot m^{-2}$)	22.87 ± 1.71	23.18 ± 1.48	21.32 ± 2.01
$\dot{V}_{O_2}peak$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	54.53 ± 5.63	55.74 ± 5.14	48.27 ± 3.60
V_{MAX} ($km \cdot h^{-1}$)	16.89 ± 1.28	17.26 ± 1.01	15.01 ± 0.76
V_{VT2} ($km \cdot h^{-1}$)	13.92 ± 0.97	14.14 ± 0.83	12.78 ± 0.88
No. of years running	6.49 ± 2.81	6.58 ± 2.91	5.38 ± 1.80
No. of previous marathons	3.28 ± 3	3.56 ± 3.09	1.92 ± 2.08
Weekly training days	4.81 ± 0.86	4.90 ± 0.85	4.33 ± 0.81
Weekly running volume (km)	63.16 ± 13.42	64.45 ± 13.21	55.66 ± 12.79
Weekly training hours	7.30 ± 2.67	7.46 ± 2.69	6.21 ± 2.27
Strength training (%)	39.8%	42.2%	26.7%

*BMI = body mass index; $\dot{V}_{O_2}peak$ = peak oxygen uptake; V_{MAX} = peak speed reached at the cardiopulmonar exercise test; V_{VT2} = speed associated with the second ventilatory threshold in the cardiopulmonar exercise test; Strength training (%), percentage of subjects who performed at least one weekly strength training in the previous 3 months.

bout, changes in plasma volume and hemoconcentration caused by dehydration should be considered. To accomplish that purpose, a correction factor based on hemoglobin and hematocrit values is applied.

Diagnosis of Exercise-Associated Muscle Cramps. During pre-race assessments, all runners were informed about the symptoms and signs of EAMC. After race completion, an experienced sports physician asked finishers whether they have suffered EAMC during or immediately after 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 (31).

Statistical Analyses

Statistical analyses were conducted using SPSS software (IBM SPSS Statistics for Windows, version 22.0, IBM Corp., Armonk, NY). Normal distribution of the variables was a priori verified through the Kolmogorov-Smirnov test. Subsequently, non-normally distributed variables (BM change, pre-race USG, post-race USG, pre-race CK and LDH, post-race and 24 hours post-race LDH and CK, and training-related variables) were compared between crampers and noncrampers using Mann-Whitney *U*-tests, whereas normally distributed variables (post-race [Na+] and [K+]) were compared using Student's *t*-tests. Categorical data (strength training and injuries) were analyzed by means of Chi-square tests. The same procedure was used to examine possible sex differences in cramping incidence. A repeated measures multivariate analysis of variance (ANOVA) was used to assess the effects of a marathon and cramping (EAMC vs. non-EAMC) and their interaction on race pacing (i.e., 5-km split speeds relativized for V_{VT2} and V_{MAX}). For each ANOVA, if a significant main effect or interaction was identified, pairwise comparisons were adjusted using Bonferroni's correction. The meaningfulness of the outcomes was estimated through the effect size (ES, mean divided by the *SD*) as follows: an ES <0.5 was considered small; between 0.5 and 0.8, moderate; and greater than 0.8, large (36). The significance level was set at p value <0.05, and data are presented as mean ± *SD*.

Results

From the initial sample of 98 subjects, 88 runners finished the marathon and we could obtain whole data from 84, 72 men (86%) and 12 women (14%), who constitute the final sample of the study. Their average finishing time was 3 hours:34 minutes:20 seconds ± 20 minutes:55 seconds, ranging from 2 hours:58 minutes:25 seconds to 4 hours:36 minutes:03 seconds. A total of 20 runners developed EAMC (24%) during or immediately after the race. No sex differences were identified in EAMC incidence (women: 25%, men: 23.6%; $p = 0.917$).

In relation to the etiological nature of EAMC, no significant differences were found in hydration status variables (pre-race and post-race USG and BM change) or serum [Na+] and [K+] between those who did or did not experience EAMC (Table 2). Conversely, CK immediately after the race (464.17 ± 220.47 vs. 383.04 ± 253.41 UI/L; $p = 0.034$) and 24 hours after (2,438.59 ± 2,625.24 vs. 1,166.66 ± 910.71 UI/L; $p = 0.014$) were significantly greater in subjects who experienced EAMC than those who did not. Likewise, crampers displayed significantly greater values of LDH both immediately after the race (362.27 ± 72.10 vs. 307.87 ± 52.42 UI/L; $p = 0.002$) and 24 hours after (277.05 ± 89.74 vs. 227.07 ± 37.15 UI/L; $p = 0.021$). Considering *Cohen's D* ESs, those differences ranged from moderate (CK post-race) to large (LDH post-race, CK 24 hours post-race and LDH 24 hours post-race) (see Table 2).

Unlike post-race values, pre-race CK and LDH were not significantly different between those athletes who suffered EAMC and those who did not (Table 2). Neither the number of hours from the last training to the race nor the percentage of runners who have sustained an injury in the past 3 months before the marathon differ between crampers and noncrampers. Regarding training-related and experience-related variables, the number of previous marathons, the number of years running, the weekly training days, the hours, and running volume (i.e., kilometer) were not different between crampers and noncrampers (Table 3). However, the difference in the percentage of runners who undertook regular strength training approached statistical significance between those who experienced EAMC and those who did not (EAMC: 25%, non-EAMC: 47.6%; $p = 0.074$).

Pacing data by 5-km splits of the race are presented in Figure 1 (panel A: $\%V_{MAX}$; panel B: $\%V_{VT2}$). Repeated measures multivariate ANOVA revealed a significant effect of a "marathon" on both $\%V_{VT2}$ ($F = 69.85$; $p = 0.001$; η^2 partial = 0.46, 95% confidence interval [CI]: 0.40–0.50) and $\%V_{MAX}$ ($F = 68.36$; $p = 0.001$; η^2 partial = 0.46, 95% CI: 0.39–0.50). The "cramping" factor significantly affected $\%V_{MAX}$ ($F = 5.15$; $p = 0.026$; η^2 partial = 0.06, 95% CI: 0–0.18) whereas "fatigue × cramping" interaction affected both $\%V_{VT2}$ ($F = 6.85$; $p = 0.001$; η^2 partial

Table 2
Hydration status and serological variables in crampers and noncrampers (mean ± *SD*).*

	Crampers ($n = 20$)	Noncrampers ($n = 64$)	p	ES	95% CI for ES	
					Upper bound	Lower bound
Pre-race USG ($g \cdot ml^{-1}$)	1.018 ± 0.005	1.018 ± 0.006	0.748	−0.09	0.41	−0.59
Post-race USG ($g \cdot ml^{-1}$)	1.019 ± 0.006	1.018 ± 0.008	0.509	−0.15	0.37	−0.66
ΔBM (%)	3.06 ± 1.01	2.87 ± 1.10	0.549	−0.17	0.35	−0.70
Post-race Na^+ ($mmol \cdot L^{-1}$)	139.6 ± 2.1	140.7 ± 2.6	0.107	0.44	0.96	−0.07
Post-race K^+ ($mmol \cdot L^{-1}$)	4.69 ± 0.39	4.70 ± 0.44	0.962	0.01	0.52	−0.50
Pre-race CK ($UI \cdot L^{-1}$)	171 ± 70	156 ± 81	0.250	−0.20	0.30	−0.70
Pre-race LDH ($UI \cdot L^{-1}$)	213 ± 108	184 ± 57	0.135	−0.40	0.10	−0.91
Post-race CK ($UI \cdot L^{-1}$)	464 ± 220	383 ± 253	**0.034**	−0.33	0.18	−0.85
Post-race LDH ($UI \cdot L^{-1}$)	362 ± 72	308 ± 52	**0.002**	−0.96	−0.43	−1.49
24 hours post-race CK ($UI \cdot L^{-1}$)	2,439 ± 2,625	1,167 ± 911	**0.014**	−0.86	−0.34	−1.38
24 hours post-race LDH ($UI \cdot L^{-1}$)	277 ± 90	227 ± 37	**0.021**	−0.93	−0.41	−1.45

Entries in bold indicate significant differences between crampers and non-crampers.
*ES = effect size; CI = confidence interval; USG = urine specific gravity; ΔBM = body mass change; Na = sodium; K = potassium; CK = creatine kinase; LDH = lactate dehydrogenase.

= 0.07, 95% CI: 0.03–0.11) and $\%V_{MAX}$ ($F = 6.72$; $p = 0.001$; η^2 partial = 0.07, 95% CI: 0.03–0.11). Pairwise comparisons showed that both crampers and noncrampers significantly increased $\%V_{VT2}$ and $\%V_{MAX}$ from the first to the second 5-km split. Then, both groups maintained $\%V_{VT2}$ and $\%V_{MAX}$ in the following 5-km splits until 21st km (i.e., half marathon). From this point onward, crampers significantly decreased $\%V_{VT2}$ and $\%V_{MAX}$ in the subsequent 5-km splits until the finishing line, whereas noncrampers decreased $\%V_{VT2}$ and $\%V_{MAX}$ in the 21–25-, 25–30-, and 30–35-km splits but not in the 35–42-km split. Regarding intergroup comparisons, from the 25th km onward (i.e., 25–30-, 30–35-, and 35–42-km splits) athletes who suffered EAMC ran at a significantly lower $\%V_{MAX}$ (67.95 ± 4.91% vs. 70.98 ± 4.96%, 64.45 ± 8.08% vs. 68.98 ± 5.77% and 61.40 ± 8.93% vs. 68.05 ± 6.80% respectively; *p value* between 0.001 and 0.019). Likewise, from the 30th km onward (i.e., 30–35- and 35–42-km splits), crampers ran at a significantly lower $\%V_{VT2}$ (78.75 ± 9.14% vs. 83.44 ± 8.05% and 75.01 ± 10.47% vs. 82.25 ± 8.91% respectively; p value between 0.003 and 0.031). Considering *Cohen's D* ESs, those intergroup differences ranged from moderate ($\%V_{MAX}$ at 25–30- and 30–35-km splits; $\%V_{VT2}$ at 30–35- and 35–42-km splits) to large ($\%V_{MAX}$ at 35–42-km split).

Discussion

The prevalence of cramps in our sample (24%) was somewhat higher than previously reported in a marathon (18%) (27) yet very similar to the data collected during Ironman-distance triathlon and a 100-km ultramarathon (23%) (14,32). Regarding EAMC etiology, our results show that runners who suffered muscle cramps during or immediately after the marathon did not show a greater loss of BM or a lower value of post-race USG. Likewise, crampers did not exhibit a lower post-race serum [Na+] or [K+] concentration. Therefore, EAMC seem not to be related to dehydration or electrolyte depletion. These results confirm previous findings from studies performed both in a road marathon (20) and other athletic events (Ironman-distance triathlon and ultramarathons) (12,29,32,35).

On the other hand, the higher concentration of muscle damage biomarkers (LDH and CK) observed in crampers 24 hours and immediately after the race adds further evidence to the "altered neuromuscular control theory" of EAMC (27,28) and coincides with a previous study performed in a 161-km ultratrail (12). Conversely, the absence of differences in pre-race muscle damage biomarkers do not agree with previous studies in which EAMC were hypothesized to be related to a greater degree of subclinical pre-race muscle damage (29). Post-race values of CK are similar to those previously reported in amateur runners after a marathon (3,5,17,26,38); whereas, LDH values were similar to those depicted by Lijnen et al. (17) but somewhat lower than those reported by Del Coso et al. (5). Interestingly, in the latter study, subjects whose running pace decreased more than 15% from the first to the last 5-km split displayed significantly higher concentrations of LDH and CK but not a greater BM loss (5). Bearing in mind these outcomes, it seems that the group of runners who suffered cramps during the

Table 3
Experience-related and training-related variables in crampers and noncrampers (mean ± *SD*).*

	Crampers ($n = 20$)	Noncrampers ($n = 64$)	p	ES	95% CI for ES	
					Upper bound	Lower bound
No. of years running	7.25 ± 3.39	6.4 ± 3.03	0.289	−0.28	0.23	−0.78
No. of previous marathons	4.2 ± 2.8	3.19 ± 3.12	0.201	−0.34	0.17	−0.84
Weekly training days	4.75 ± 0.85	4.95 ± 0.85	0.357	0.24	0.75	−0.26
Weekly running volume (km)	60.5 ± 12.24	62.84 ± 14.05	0.506	0.17	0.68	−0.33
Weekly training hours	7.35 ± 3.23	7.33 ± 2.19	0.969	−0.01	0.49	−0.51
Pre-race recovery time (h)	56.4 ± 23.71	58.67 ± 23.91	0.712	0.10	0.60	−0.41
Previous injuries (%)	40%	36.5%	0.779			
Strength training (%)	25%	47.6%	0.074			

*ES = effect size; CI = confidence interval; Pre-race recovery time = number of hours from the last training to the race; Previous injuries = having sustained any injury in the previous 3 months; Strength training (%), percentage of subjects who performed at least one weekly strength training in the previous 3 months.

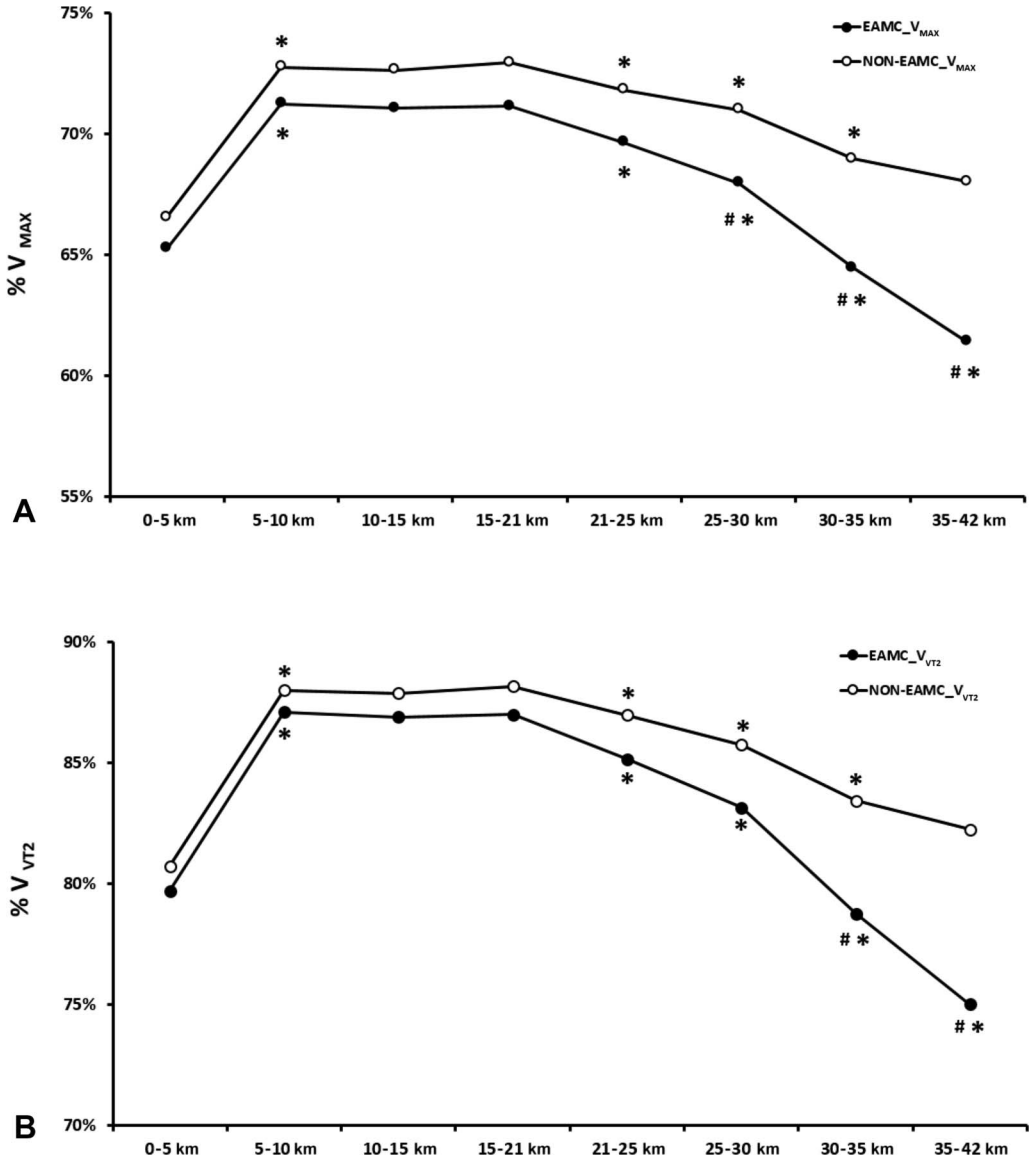

Figure 1. Five-kilometer split pace of runners who did and did not experience EAMC during the marathon expressed as $\%V_{MAX}$ (panel A) and $\%V_{VT2}$ (panel B). Solid circles represent data from EAMC runners and open circles represent data from non-EAMC runners. *Significantly different from the preceding 5-km split ($p < 0.05$); #Significantly different from non-EAMC runners ($p < 0.05$). EAMC = exercise-associated muscle cramps.

marathon effectively subjected their muscles to an excessive intensity demand in relation to their fitness level, giving rise to the inference that the type of training developed during the preparation for long-distance races has a relationship with the appearance of EAMC. Regarding this hypothesis, we set out to explore whether training-related variables differ between crampers and noncrampers. Interestingly, Wagner et al. (37) showed that a triathlete with a complaint of recurrent cramping was able to complete 3 triathlons without cramping after the completion of an 8-month strengthening and neuromuscular reeducation program. Conversely, previous studies have found no relationship between flexibility training and EAMC prevalence (29,32,34) and even a tendency to expend more time stretching among crampers (29). This discrepancy could be explained by the fact that strength training, unlike flexibility training, has been largely demonstrated to be the most effective conditioning strategy to minimize overuse injuries (15,16). Moreover, a strength training intervention was demonstrated to delay fatigue and enable an improved 10-km running overall performance through a higher speed during the middle-to-last phases of the time trial (4). Therefore, it seems plausible that strength training could also exert a protective effect against EAMC in a road marathon as our results suggest.

On the other hand, previous research showed that muscle cramping was associated with a faster initial speed (29). However, in the aforementioned study absolute but not relative speed was considered and therefore those results and ours could not be compared. We observed no differences in relative speed (neither $\%V_{MAX}$ nor $\%V_{VT2}$) between crampers and noncrampers until the 25th km. Conversely, athletes who suffered EAMC displayed lower relative $\%V_{MAX}$ and $\%V_{VT2}$ in the last 15 km of the race compared with their noncramping counterparts. Moreover, noncrampers, unlike crampers, did not lower their speed in the final 7-km split of the race. This result confirms that muscle cramping constitutes one of the most important factors limiting performance in long-distance races (11) but contrasts with

previous studies performed in Ironman-distance triathletes (32,34). In those studies, subjects who suffered EAMC were capable of achieving better cycling leg and overall times. Notwithstanding, as previously discussed for initial speed, absolute but not relative speeds were considered in former studies. Summing up, considering also the abovementioned difference in EAMC prevalence between those athletes who performed strength training and those who did not during their preparation for the marathon, it could be suggested that a proper strength conditioning, rather than a greater endurance or total training volume, would enable a more regular pacing during long-distance races (4).

In summary, muscle damage, unlike dehydration and electrolyte depletion, was consistently greater immediately after and 24 hours after the marathon among crampers compared with noncrampers, thus confirming our first hypothesis. Meanwhile, contrary to our expectations, crampers did not run at a higher relative velocity in the first half of the race (i.e., compared with noncrampers), as previously described in the literature. Finally, in relation to our second hypothesis, although the percentage of runners who undertook regular strength training in their preparation for the marathon was not significantly different between crampers and noncrampers, the fact that the difference nearly approached the statistical significance threshold lead us to cautiously suggest that strengthening could aid in the reduction of a muscle cramping incidence.

Nevertheless some limitations of the study should be acknowledged. One limitation concerns post-race BM measurement, in which sweat accumulation on clothing was not accounted for. Second, we did not ask subjects about a previous history of cramping in the questionnaire so we cannot rule out that this was a leading factor to having suffered EAMC again. Third, regarding strength training performed by the subjects, we only collected the weekly frequency of lower-body resistance training performed in the previous 3 months. Further experimental studies comparing different modes of strength training are encouraged to verify whether they exert or not exert a protective effect against EAMC.

Practical Applications

In light of the abovementioned findings, it could be suggested that runners suffering from muscle cramping during a marathon should be aware that they have subjected their muscles to an excessive demand according to their current state of training, thus provoking greater muscle damage, and should consider a longer post-race recovery. Furthermore, our results suggest that strength training, rather than a greater endurance or total training volume, could exert a protective effect against EAMC and enable in turn a more regular pacing during long-distance races. Therefore, both runners and coaches are encouraged to include this kind of training in their preparation for the marathon.

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