Significant and serious dehydration does not affect skeletal muscle cramp threshold frequency

Kyle W Braulick,[1] Kevin C Miller,[1] Jay M Albrecht,[2] Jared M Tucker,[3] James E Deal[4]

[1]Departments of Health, Nutrition, and Exercise Sciences, North Dakota State University, Fargo, North Dakota, USA
[2]Department of Health and Physical Education, Minnesota State University Moorhead, Moorhead, Minnesota, USA
[3]Healthy Weight Center, Helen DeVos Children's Hospital, Grand Rapids, Michigan, USA
[4]Human Development and Family Science, North Dakota State University, Fargo, North Dakota, USA

Correspondence to
Dr Kevin C Miller, Department of Health, Nutrition, and Exercise Sciences, North Dakota State University, NDSU Dept #2620, PO Box 6050, Fargo ND 58108-6050, USA; Kevin.C.Miller@ndsu.edu

Received 26 June 2012
Revised 8 October 2012
Accepted 10 November 2012
Published Online First 6 December 2012

ABSTRACT

Objective Many clinicians believe that exercise-associated muscle cramps (EAMC) occur because of dehydration. Experimental research supporting this theory is lacking. Mild hypohydration (3% body mass loss) does not alter threshold frequency (TF), a measure of cramp susceptibility, when fatigue and exercise intensity are controlled. No experimental research has examined TF following significant (3–5% body mass loss) or serious hypohydration (>5% body mass loss). Determine if significant or serious hypohydration, with moderate electrolyte losses, decreases TF.

Design A prepost experimental design was used. Dominant limb flexor hallucis brevis cramp TF, cramp electromyography (EMG) amplitude and cramp intensity were measured in 10 euhydrated, unacclimated men (age=24±4 years, height=184.2±4.8 cm, mass=84.8±11.4 kg). Subjects alternated exercising with their non-dominant limb or upper body on a cycle ergometer every 15 min at a moderate intensity until 5% body mass loss or volitional exhaustion (3.8±0.8 h; 39.1±1.5°C; humidity 18.4±3%). Cramp variables were reassessed posthypohydration.

Results Subjects were well hydrated at the study's onset (urine specific gravity=1.005±0.002). They lost 4.7±0.5% of their body mass (3.9±0.5 litres of fluid), 4.0±1.5 g of Na^+ and 0.6±0.1 g K^+ via exercise-induced sweating. Significant (n=5) or serious hypohydration (n=5) did not alter cramp TF (euhydrated=15±5 Hz, hypohydrated=13±6 Hz; $F_{1,9}$=3.0, p=0.12), cramp intensity (euhydrated=94.2±41%, hypohydrated=115.9±73%; $F_{1,9}$=1.9, p=0.2) or cramp EMG amplitude (euhydrated=0.18±0.06 μV, hypohydrated=0.18±0.09 μV; $F_{1,9}$=0.1, p=0.79).

Conclusions Significant and serious hypohydration with moderate electrolyte losses does not alter cramp susceptibility when fatigue and exercise intensity are controlled. Neuromuscular control may be more important in the onset of muscle cramps than dehydration or electrolyte losses.

INTRODUCTION

Exercise-associated muscle cramps (EAMC) are painful involuntary contractions of skeletal muscle occurring during or shortly following exercise.[1] EAMC can impair athletic performance,[2] and symptoms can last up to 8 h postactivity.[3] EAMC typically occur in multijoint muscles and are common.[4 5] Despite their prevalence, EAMC cause remains unknown.

The dehydration and electrolyte theory is the most popular theory to explain the onset of EAMC.[6] The theory states that an exercise-induced sweating causes fluid to shift from the interstitium to the intravascular space.[7 8] The interstitium then contracts increasing pressure on select nerves and altering excitability.[7] EAMC then ensue.[8] Support for this theory comes from research comparing fluid and electrolytes losses in crampers and non-crampers.[9–11]

Others have observed altered muscle spindle[12] and golgi tendon organ[13] activity in fatigued muscle. Thus, some scientists proposed that EAMC occur when neuromuscular control becomes altered and afferent activity causes a reflexive excitation at the α-motor neuron pool.[1] Support for this theory comes from investigations linking EAMC risk with faster race times (and possibly greater fatigue) rather than dehydration.[14] Moreover, crampers have lower golgi tendon organ inhibition than non-crampers[15] and afferents activated by hypertonic saline reduced cramp threshold frequency (TF, minimum electrical stimulation needed to induce cramp).[16]

Not controlling exercise intensity or fatigue is a limitation of studies examining EAMC.[9–11 17 18] Some scientists have attempted to isolate the effects of dehydration and electrolyte losses from fatigue by electrically inducing cramps and measuring cramp TF.[19] These scientists[19] observed mild hypohydration (3% body mass reduction) did not alter TF. Cramp TF is thought to be a quantitative measure of cramp risk where lower TF indicates increased risk and vice versa.[20] Since some athletes have high fluid and electrolyte losses,[9–11] determining cramp risk when individuals are significantly or seriously hypohydrated is needed. The National Athletic Trainers Association[21] defines significant and serious hypohydration as 3–5% and >5% body mass losses, respectively. No research has examined cramp TF following significant or serious hypohydration. Based on previous observations,[19] we hypothesised that TF would be similar when euhydrated or significantly or seriously hypohydrated and peripheral fatigue and exercise intensity are controlled.

MATERIALS AND METHODS

Subjects

Nine subjects were needed to achieve 80% power at α=0.05. A convenience sample of 13 men between 18 and 30 years old volunteered; three were excluded because cramps could not be induced in the flexor hallucis brevis (FHB) on the familiarisation day. Ten men completed testing (age=24±4 years, ht=184.2±4.8 cm, pre-exercise mass=84.8±11.4 kg).

Volunteers were excluded if: (1) they experienced injury to their legs in the 6 months predata collection; (2) they self-reported any cardiovascular, neurological or blood borne diseases; (3) they did

To cite: Braulick KW, Miller KC, Albrecht JM, *et al. Br J Sports Med* 2013;**47**:710–714.

not self-report a history of leg cramps within the 12 months predata collection; (4) FHB cramps could not be induced; (5) they were not active (≥30 min of activity on most days);[22] (7) they were taking any medications and (8) they had a history of cardiac events, heat exhaustion or heat stroke. All procedures were approved by our university's ethics board and subjects provided informed consent pretesting.

Procedures

Subjects reported for one familiarisation session and one testing day. For the familiarisation session, subjects were asked to arrive hydrated and avoid caffeine, alcohol and exercise for 24 h. They reported to a laboratory and had their leg dominance determined.

Subjects lay supine and had their dominant leg prepped for electromyography (EMG) analysis.[19] Subjects' medial ankle, lower leg, tibial tuberosity and midbelly of the gastrocnemius were shaved (if necessary), debrided with fine sandpaper and cleaned with alcohol. Two Ag-AgCl EMG electrodes (EL502, Biopac Systems Inc, Aero Camino, California, USA) were placed over the midbelly of the FHB with a 2 cm centre-to-centre distance. A single ground EMG electrode was placed over the ipsilateral tibial tuberosity. A single Ag-AgCl electrode (6750, Prometheus Group, Dover, New Hampshire, USA) was placed over the middle of the gastrocnemius and connected to a biofeedback unit (Pathway TR-10C; Prometheus Group).

The ankle was placed in a foam block with a foot pad angled at 120°. The big toe was placed into a toe harness. Subjects performed 20, 2 s duration FHB maximum voluntary isometric contractions (MVIC) with 1 min of rest separating each MVIC. Following these MVICs, subjects rested 5 min and performed three, 2 s MVICs. The mean EMG amplitude of these contractions was averaged and used to determine cramp intensity.

To ensure subjects were using the correct muscle during MVICs, gastrocnemius activity was monitored; activity exceeding 8 mV indicated a failed attempt. If subjects performed MVIC incorrectly, a 1 min rest period was given and MVIC was reattempted. Researchers have reported high MVIC intratester reliability using this method (intraclass correlation coefficient (ICC) 3.3>0.81).[23] Following MVICs, subjects big toe and ankle were removed from the toe harness and foam block, respectively, and subjects were prepped for cramp induction.

An 8 mm Ag-AgCl shielded electrode (EL258S; Biopac) was placed over the medial ankle distal to the medial malleolus where the tibial pulse was felt. An 8 cm, square dispersive electrode was placed over the lateral malleolus. To determine proper placement of the stimulating electrode, the tibial nerve was stimulated with 1 ms electrical stimuli at 80 V (Grass S88 stimulator with SIU5 stimulus isolation unit, Astro-Med Inc, West Warwick, Rhode Island, USA). The proper electrode location was the site causing the greatest hallux flexion.

Subjects were instructed to relax during cramp induction. Subjects received 1 s of rest followed by two consecutive bursts of 80 V electrical stimulation. Initial burst frequency was 4 Hz. If cramp was not induced, subjects rested 1 min and stimulation frequency was increased by 2 Hz until FHB cramped. These procedures have been used previously and have high intratester (ICC 3,1=0.84), and intertester reliability (ICC 3,1=0.96).[24]

A muscle cramp was defined as an involuntary contraction of the FHB immediately following the end of the electrical stimuli and must have met three criteria. First, poststimulus EMG amplitude must have been ≥50% of MVIC EMG amplitude, and must have maintained this intensity for >5 s. Second, subjects verified the cramp. Finally, sustained flexion of the first ray must have been observed postelectrical stimulation. The frequency used to induce a cramp meeting these criteria was deemed cramp TF. If a cramp did not spontaneously resolve after 5 s, the FHB was passively stretched. The electrode sites were marked for future testing sessions. Subjects reported for their testing session ≥36 h following the familiarisation day.

Twenty-four hours before their testing day, subjects avoided exercise. Twelve hours before the testing day, subjects were instructed to fast for 6 h, drink water consistently, and avoid consuming any beverage besides water. On the testing day, subjects emptied their bladders completely and urine specific gravity was measured using a refractometer (Sur-NE, Atago VSA Inc, Bellevue, Washington, USA). If hypohydrated (urine specific gravity >1.010),[21] subjects testing day was rescheduled. If euhydrated, subjects lay supine for 30 min for fluid compartment equilibration.[25] The dominant leg was prepped for cramp induction using the procedures described above. A sterile catheter was inserted into the subject's arm; a 5 ml blood sample was collected (euhydrated sample).

Subjects performed 10 practice MVICs (1 min rest between each MVIC), rested for 5 min, and performed three consecutive 2 s duration MVICs. After 15 min rest, subject's euhydrated TF was determined. Postcramp induction, the electrodes were removed. Subjects donned a heart rate monitor (Polar Electric Inc, Lake Success, New York, USA), inserted a rectal thermistor (Yellow Spring Instruments 4600, Advanced Industrial Systems Inc, Prospect, Kentucky, USA) 10 cm past the anal sphincter, and were weighed nude. Both posterior mid-forearms were shaved, cleaned with deionised water and dried. Sterile sweat patches (occlusive dressing and sterile gauze) were placed at these sites.

Subjects donned sweat pants, socks, shoes and a t-shirt and entered an environmental chamber (39.1±1.5°C; 18.4±3% humidity). They alternated exercising with an upper body ergometer or non-dominant cycling every 15 min at 70% of their age-predicted maximal heart rate. Sweat patches were removed after 30 min of exercise. Following sweat patch collection, subjects donned a hooded sweatshirt for the remainder of the exercise protocol. After 60 min, subjects exercised for 5 min at a self-selected lower intensity to cool down. They exited the environmental chamber, removed their clothes, towel dried, emptied their bladders completely and were weighed nude. Subjects dressed and rested for 10 min in a climate-controlled room. They resumed exercising for another 60 min using the above protocol. These procedures continued until subjects lost 5% of their body mass or were too exhausted to continue. Upon 5% hypohydration or volitional exhaustion, subjects exited the environmental chamber, removed the heart rate monitor and rectal thermistor and lay supine for 30 min.

During the rest period, EMG and stimulation electrodes were placed over the previously marked locations. After the rest period, a 5 ml blood sample was collected (hypohydrated sample). Subjects performed 10 practice MVICs (1 min rest between each MVIC), rested for 5 min, and performed three consecutive 2-s duration MVICs. Cramp TF was reassessed 15 min following MVIC. Following cramp induction, the electrodes were removed and the subjects were excused. No fluids were given during testing.

Cramp and MVIC EMG procedures

FHB muscle action potentials were sampled at 2000 Hz, amplified (gain=2000) and filtered (band pass, low frequency=10 Hz, high frequency=500 Hz) using the BioNomadix analogue-to-digital system operated by Acqknowledge 4.0 software (Biopac). Amplifier impedance was 2 mΩ with a common mode rejection ratio of 11 dB and a signal-to-noise ratio of 0.75 dB.

Blood analysis procedures

Plasma osmolality (OSM_p), plasma sodium concentration ($[Na^+]_p$), plasma potassium concentration ($[K^+]_p$), haematocrit and haemoglobin concentration ([Hb]) were measured to describe the extracellular compartment pre-exercise and postexercise.

Blood was collected into 6 ml lithium heparin vacutainers. Haematocrit and [Hb] were determined in triplicate immediately after sampling. To determine haematocrit, blood was drawn into heparinised microcapillary tubes and centrifuged at 3000 rpm (IEC Micro-MB; International Equipment Co., Needham Heights, Massachusetts, USA) for 5 min. Haematocrit was read using a microcapillary reader (model IEC 2201; Damon/IEC, Needham Heights). To measure [Hb], the cyanomethemoglobin technique was used. Per cent change in plasma volume was estimated using the Dill and Costill equation.[26]

Any remaining blood was centrifuged at 3000 rpm at 3°C for 15 min. Plasma was removed and stored (−80°C) for later analysis of OSM_p (freezing-point depression osmometry; model 3D3 Osmometer, Advanced instruments, Inc Norwood, Massachusetts, USA), $[Na^+]_p$, and $[K^+]_p$ (analysed in duplicate, NOVA 16, NOVA Biomedical, Waltham, Massachusetts, USA).

Sweat analysis procedure

Sweat $[K^+]$, sweat $[Na^+]$ and sweat volume were measured to estimate fluid and electrolyte losses. Sweat patches were centrifuged for 10 min at 5000 rpm at 3°C and analysed in duplicate for sweat $[Na^+]$ and $[K^+]$. Sweat $[Na^+]$ and $[K^+]$ were corrected using Baker *et al*'s[27] equations which have high reliability ($r=0.96$ for Na^+ and $r=0.9$ for K^+) with the whole body wash down technique.

Sweat volume was estimated by subtracting the final postexercise body mass from subject's pre-exercise mass and correcting for urine volume produced.[21] It was assumed 1 kg of body mass lost equalled 1litre of fluid lost.

Statistical analysis

Repeated measures analysis of variances were used to determine differences between euhydrated and hypohydrated TF, cramp intensity, cramp EMG amplitude and MVIC as well as calculate reliability of the MVIC ($ICC_{2,3}$) and TF ($ICC_{2,1}$) data. Shapiro-Wilk tests confirmed normality. Significance was accepted when $p<0.05$ (NCSS 2007, Kaysville, Utah, USA).

RESULTS

Subjects self-reported compliance with pretest instructions. Subjects began exercise well hydrated and became significantly or seriously hypohydrated (exercise duration=3.9±0.8 h; table 1). Five subjects experienced volitional exhaustion before achieving 5% body mass reduction; thus, they were only significantly hypohydrated.

The familiarisation day's MVIC EMG amplitude, cramp TF, and cramp EMG amplitude were compared to the testing day's euhydrated condition to calculate reliability. MVIC EMG amplitude ($ICC_{2,3}=0.85$), cramp TF ($ICC_{2,1}=0.91$) and cramp EMG amplitude ($ICC_{2,1}=0.79$) were reliable. However, cramp intensity was inconsistent ($ICC_{2,1}=0.10$). Blood and sweat data are reported descriptively in tables 2 and 3, respectively.

Significant or serious hypohydration did not alter TF ($F_{1,9}=3.0$, $p=0.12$, 95% CI of mean differences=−4.6 to 0.61 Hz), cramp intensity ($F_{1,9}=1.9$, $p=0.2$) or cramp EMG amplitude ($F_{1,9}=0.07$, $p=0.79$; table 1). However, MVIC EMG amplitude was higher when euhydrated ($F_{1,9}=9.04$, $p=0.02$).

DISCUSSION

Significant or serious hypohydration did not alter cramp TF. Cramp TF is often used as an indicator of cramp susceptibility in scientific studies where changes reflect increased or decreased cramp risk.[16 19 28] Miller *et al*[19] observed an insignificant 2 Hz reduction in TF when subjects were euhydrated (24±2 Hz) or hypohydrated (20±2 Hz) and subjects lost 3% of their body mass and ~3 g Na^+. Our study expands Miller *et al*'s[19] results by having subjects' hypohydrated to 4.7±0.5%, exercising for ~2 h longer in similar conditions, and losing 4 g Na^+. The strength of the current and previous study[19] is the induction of cramps in a rested muscle before and after dehydration. Other studies examining cramping investigate differences in haematological or other variables pre-exercise and postexercise in athletes.[9 10 14 17 18 29] These experimental designs do not allow scientists to make valid conclusions regarding cramp susceptibility because fatigue and dehydration occur concomitantly.

Table 1 MVIC and cramp variables while euhydrated or hypohydrated (n=10)

Subject	Pre-exercise U_{sg}	H (% body mass lost)	Cramp TF (Hz)		MVIC EMG amplitude (μV)		Cramp EMG amplitude (μV)		Cramp intensity (% of MVIC activity)	
			E	H	E	H	E	H	E	H
1	1.005	5.1	8	4	0.15	0.12	0.23	0.34	153.3	275.8
2	1.004	5.0	14	8	0.22	0.17	0.16	0.26	74.4	152.9
3	1.005	4.1	16	10	0.22	0.16	0.13	0.12	58.4	74.4
4	1.003	5.0	12	14	0.17	0.15	0.16	0.08	97.2	50.7
5	1.008	4.0	18	20	0.33	0.24	0.23	0.19	70.3	80.0
6	1.004	4.1	24	18	0.29	0.20	0.15	0.14	50.0	71.8
7	1.003	4.5	10	12	0.22	0.11	0.31	0.20	138.6	176.8
8	1.004	5.3	10	8	0.14	0.15	0.22	0.25	158.9	163.3
9	1.004	5.0	16	12	0.19	0.19	0.15	0.10	78.8	50.0
10	1.008	4.6	22	24	0.17	0.17	0.10	0.11	62.2	63.7
Mean	1.005	4.7	15	13	0.21*	0.17	0.18	0.18	94.2	115.9
SD	0.002	0.5	5	6	0.06	0.04	0.06	0.09	41.0	73.9

*Euhydrated>hypohydrated ($p<0.05$).
E, euhydrated; EMG, electromyography; H, hypohydrated; MVIC, maximum voluntary isometric contraction; TF, threshold frequency; U_{sg}, Urine specific gravity.

Table 2 Descriptive data of blood variables while euhydrated or hypohydrated

	Euhydrated	Hypohydrated
$[Na^+]_p$ (mmol/l)	141.9±3.1	149.5±1.8
$[K^+]_p$ (mmol/l)	4.9±0.4	5.0±0.4
OSM_p (mOsm/kg H_2O)	287±7	301±5
Δ PV (% from baseline)	0±0	−11.8±4.9
Hct (%)	45±4	47±4
[Hb] (g/dl)	15.9±1.0	17.4±1

Values are Means±SD (n=10).
[Hb], haemoglobin concentration; Hct, haematocrit; $[K^+]_p$, plasma potassium concentration; $[Na^+]_p$, plasma sodium concentration; OSM_p, plasma osmolality; ΔPV, change in plasma volume.

Our data do not support the theory that dehydration and electrolyte losses cause cramping. A lack of fluid or electrolyte losses cannot explain our observations. Our plasma data confirm significant haemoconcentration of the extracellular space postexercise. Moreover, our subjects lost similar or more fluid and electrolytes than other studies examining crampers and non-crampers. Bergeron[10] observed tennis players with a history of EAMC lost 2.7 g of Na^+ and 2.6% of their body mass via exercise-induced sweating during match play. Similarly, Stofan *et al*[11] observed total sweat Na^+ losses of 5.1 g and fluid losses of ~3% in athletes with a history of cramp following 5 h of American football. Our subjects lost 48% more Na^+ and 81% more fluid than other authors[10] observing fluid and electrolyte losses in cramp-prone athletes. Differences in Na^+ and fluid losses are likely due to other authors[10 11] allowing subjects to consume fluids during data collection where our subjects were fluid restricted for the duration of testing.

Though the majority of clinicians believe that dehydration and electrolyte losses cause EAMC,[6] many observations outside of the current study argue against dehydration and electrolyte losses causing EAMC. First, $[Na^+]_p$, $[K^+]_p$, serum magnesium concentration and serum calcium concentration are often similar in crampers and non-crampers precompetition and post-competition.[17 29] In comparison with others' cramping athletes postexercise blood,[17] our subjects $[Na^+]_p$ and $[K^+]_p$ were 6% and 2% higher, respectively. Second, even when $[Na^+]_p$ was lower in runners suffering from EAMC than non-crampers,[18] $[Na^+]_p$ were within normal clinical ranges. Third, crampers often have similar body mass reductions following exercise as non-crampers.[17 18 29] Furthermore, sweat $[Na^+]$ of crampers is often within the normal clinical range.[11] Fourth, stretching rapidly relieves cramp,[30] yet adds no fluids or electrolytes to the body. Finally, subjects experienced cramping even when subjects replaced their sweat losses with a carbohydrate–electrolyte solution.[31] Furthermore, cramps occurred ~15 min into the protocol equating to ~500 ml of fluid lost (1% body mass lost).[31]

Table 3 Descriptive data of sweat variables and total fluid lost

$[Na^+]_{sw}$ (mmol/l)	53.2±16.7
$[K^+]_{sw}$ (mmol/l)	4.6±0.6
Sweat Volume (litre)	3.2±0.5
Sweat Rate (L/h)	0.87±0.3
Total Fluid Lost (litre)	3.9±0.5

Values are means±SD (n=9); one subject's sweat electrolyte concentrations could not be measured due to technical difficulties. $[Na^+]_{sw}$ sweat sodium concentration; $[K^+]_{sw}$ sweat potassium concentration. Sweat $[Na^+]$ and $[K^+]$ values were corrected using Baker *et al*'s equations.[27]

Our data are more aligned with the theory that cramps are the result of neuromuscular changes. Scientists[1] propose a combination of increased excitation from Ia and decreased inhibition from Ib afferents cause altered α-motor neuron activity leading to the onset of cramps. Both quasi-experimental[2 32] and experimental research support this theory.[12 13 15 33–35] For example, a triathlete's chronic hamstring cramping was relieved following eight neuromuscular re-education sessions when fluid and Na^+ intake proved to be unsuccessful.[2] Pretherapy, hamstring activation during terminal swing and first half of the stance phase of running was ~36% of MVIC (normal=19%) suggesting that cramps were due overactivation of the hamstrings.[2] By increasing gluteal activation, hamstring activity decreased and the athlete reported three triathlons without cramp incidence. Furthermore, Nelson and Hutton[12] observed increases in Ia firing frequency when stretches were applied to a fatigued muscle. In a follow-up study,[13] Ib activity was lower when fatigued. Scientists[15] have also demonstrated cramps can be inhibited by tendon afferent stimulation and TENS application at a site away from cramping.[32] Moreover, cramp TF was significantly greater when the tibial nerve was blocked (18±3 vs 13 ±3 Hz)[35] suggesting afferents play an important role in cramp genesis. Since TF remained unchanged following significant and severe dehydration, it is possible afferent activity was unaffected in the rested leg and similar electrical stimuli were required to induce cramp.

Three limitations must be addressed. First, cramps were induced with percutaneous electrical stimulation rather than exercise. However, authors have observed high correlation with FHB cramp TF when comparing subjects with a history of EAMC and subjects with no history.[20] Second, not all subjects achieved 5% hypohydration. However, even the subjects who were significantly hypohydrated lost amounts of fluid and electrolytes similar to those reported in the literature for crampers.[9 11] Finally, the effect of hypohydration may be dependent on baseline TF or proportional to baseline TF. The cramp-prone subjects in our study had low cramp TF relative to non-cramp prone individuals.[20] The cramp TF observed here are similar to those reported by other authors [20 24 36 37] but may not have allowed for large changes in TF to occur. Repeating the current investigation in cramp-prone individuals or non-cramp prone individuals with a higher baseline TF would clarify this point.

In summary, significant and/or serious hypohydration does not increase cramp risk, as indicated by TF. Therefore, cramps occurring in hypohydrated individuals may be more related to neuromuscular control changes. Strategies to increase neuromuscular endurance or correct muscle imbalances may be more successful at minimising the onset of EAMC than rehydration or electrolyte replenishment strategies.

What this study adds

- No experimental research has examined the effects of significant (3–5% body mass loss) or serious hypohydration (>5% body mass loss) on muscle cramp threshold frequency (TF).
- Significant or serious hypohydration did not alter TF.
- Muscle cramps may be more due to neuromuscular fatigue than dehydration or electrolyte losses.

Acknowledgements We thank NDSU's College of Human Development and Education, NDSU's Department of Health, Nutrition, and Exercise Sciences, and NDSU's Athletic Training Education Program for partially funding this research.

Contributors KWB was involved in the conception and design of the study, data collection, analysis, and interpretation, and drafting, revision and final approval of the manuscript. KCM was involved in the conception and design of the study, data collection, analysis, and interpretation, and drafting, revision and final approval of the manuscript. JMA was involved in the conception and design of the study, data interpretation, and revision and final approval of the manuscript. JMT was involved in the conception and design of the study, data interpretation, and revision and final approval of the manuscript. JED was involved in the conception and design of the study, data interpretation, and revision and final approval of the manuscript. All authors take responsibility for the data and text reported.

Funding None.

Competing interests None.

Ethics approval North Dakota State University Institutional Review Board.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Schwellnus M, Derman E, Noakes T. Aetiology of skeletal muscle 'cramps' during exercise: a novel hypothesis. *J Sports Sci* 1997;15:277–85.
2 Wagner T, Behnia N, Ancheta W, *et al*. Strengthening and neuromuscular reeducation of the gluteus maximus in a triathlete with exercise associated cramping of the hamstrings. *J Orthop Sports Phys Ther* 2010;40:112–19.
3 Dickhuth H, Rocker K, Niess A, *et al*. Exercise-induced, persistent and generalized muscle cramps. *J Sports Med Phys Fitness* 2002;42:92–4.
4 Cooper E, Ferrara M, Broglio S. Exertional heat illness and environmental conditions during a single football season in the Southeast. *J Athl Train* 2006;41:332–6.
5 Kantarowski P, Hiller W, Garrett W. Cramping studies in 2600 endurance athletes. *Med Sci Sports Exerc* 1990;22:S104.
6 Stone M, Edwards J, Stemmans C, *et al*. Certified athletic trainers' perceptions of exercise associated muscle cramps. *J Sport Rehabil* 2003;12:333–42.
7 Jansen P, Joosten E, Vinerhoets H. Muscle cramp: main theories as to aetiology. *Eur Arch Psychiatr Neurol Sci* 1990;239:337–42.
8 Bergeron M. Muscle cramps during exercise—is it fatigue or electrolyte deficit? *Curr Sports Med Rep* 2008;7:S50–5.
9 Bergeron M. Heat cramps during tennis: a case report. *Int J Sport Nutr* 1996;6:62–8.
10 Bergeron M. Heat cramps: fluid and electrolyte challenges during tennis in the heat. *J Sci Med Sport* 2003;6:19–27.
11 Stofan J, Zachwieja J, Horswill C, *et al*. Sweat and sodium losses in NCAA football players: a precursor to heat cramps? *Int J Sport Nutr Exerc Metab* 2005;15:641–52.
12 Nelson D, Hutton R. Dynamic and static stretch responses in muscle spindle receptors in fatigued muscle. *Med Sci Sports Exerc* 1985;17:445–50.
13 Hutton R, Nelson D. Stretch sensitivity of golgi tendon organs in fatigued gastrocnemius muscle. *Med Sci Sports Exerc* 1986;18:69–74.
14 Schwellnus M, Drew N, Collins M. Increased running speed and previous cramps rather than dehydration or serum sodium changes predict exercise associated muscle cramping: a prospective cohort study in 210 Ironman triathletes. *Br J Sports Med* 2011;45:650–6.
15 Khan S, Burne J. Reflex inhibition of normal cramp following electrical stimulation of the muscle tendon. *J Neurophysiol* 2007;98:1102–7.
16 Serrao M, Arendt-Nielsen L, Ge H, *et al*. Experimental muscle pain decreases the frequency threshold of electrically elicited muscle cramps. *Exp Brain Res* 2007;182:301–8.
17 Schwellnus M, Nicol J, Laubscher R, *et al*. Serum electrolyte concentrations and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners. *Br J Sports Med* 2004;38:488–92.
18 Sulzer N, Schwellnus M, Noakes T. Serum electrolytes in Ironman triathletes with exercise associated muscle cramping. *Med Sci Sports Exerc* 2005;37:1081–5.
19 Miller K, Knight K, Mack G, *et al*. Three percent hypohydration does not affect the threshold frequency of electrically-induced muscle cramps. *Med Sci Sports Exerc* 2010;42:2056–63.
20 Miller K, Knight K. Electrical stimulation cramp threshold frequency correlates well with the occurrence of skeletal muscle cramps. *Muscle Nerve* 2009;39:364–8.
21 Casa D, Armstrong L, Hillman S, *et al*. National Athletic Trainers Association position statement: fluid replacement for athletes. *J Athl Train* 2000;35:212–24.
22 Whaley M. *ACSM's guidelines for exercise testing and prescription*. 7th edn. Philadelphia: Lippincott Williams & Wilkins, 2006:4.
23 Miller K, Knight K. Initial electrical stimulation frequency's effect on cramp threshold frequency and force. *J Athl Train* 2012;47:643–7.
24 Stone M, Edwards J, Babington J, *et al*. Reliability of an electrical method to induce muscle cramp. *Muscle Nerve* 2003;27:122–3.
25 Hagan R, Diaz F, Horvath S. Plasma volume changes with movement to supine and standing positions. *J Appl Physiol* 1978;45:414–18.
26 Dill D, Costill D. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 1974;37:247–8.
27 Baker L, Stofan J, Hamilton A, *et al*. Comparison of regional patch collection vs. whole body washdown for measuring sweat sodium and potassium loss during exercise. *J Appl Physiol* 2009;107:887–95.
28 Stone M, Edwards J, Huxel K, *et al*. Threshold frequency of an electrically induced cramp increases following a repeated, localized fatiguing exercise. *J Sports Sci* 2010;28:399–405.
29 Maughan R. Exercise induced muscle cramp: a prospective biochemical study in marathon runners. *J Sports Sci* 1986;4:31–4.
30 Bertolasi L, De Grandis D, Bongiovanni L, *et al*. The influence of muscular lengthening on cramps. *Ann Neurol* 1993;33:176–80.
31 Jung A, Bishop P, Al-Nawwas A, *et al*. Influence of hydration and electrolyte supplementation on incidence and time to onset of exercise-associated muscle cramps. *J Athl Train* 2005;40:71–5.
32 Mills K, Newham D, Edwards R. Severe muscle cramps relieved by transcutaneous nerve stimulation: a case report. *J Neurol Neurosurg Psychiatry* 1982;45:539–42.
33 Ge H, Zhang Y, Boudreau S, *et al*. Induction of muscle cramps by nociceptive stimulation of latent myofascial trigger points. *Exp Brain Res* 2008;187:623–9.
34 Miller K, Mack G, Knight K, *et al*. Reflex inhibition of electrically-induced muscle cramps in hypohydrated humans. *Med Sci Sports Exerc* 2010;42:953–61.
35 Minetto M, Holobar A, Botter A, *et al*. Mechanisms of cramp contractions: peripheral or central generation. *J Physiol* 2011;23:5759–3.
36 Miller K, Knight K, Wilding S, *et al*. Duration of electrically induced muscle cramp increased by increasing stimulation frequency. *J Sport Rehab* 2012;21:182–5.
37 Minetto M, Botter A, Ravenni R, *et al*. Reliability of a novel neurostimulation method to study involuntary muscle phenomena. *Muscle Nerve* 2007;37:90–100.