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Comparison of Sweat Rates and Sweat Electrolyte Concentrations in Collegiate Male and Female Athletes With and Without a History of Cramping

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Abstract

Context

Skeletal muscle cramps are one of the most common physical impairments for athletes during performance. Despite the high occurrence, little research has been done on this topic, and the cause of cramping is not well understood. There are two main theories explaining the onset of exercise associated muscle cramps (EAMCs): muscular fatigue or the electrolyte-dehydration theory.

Purpose

The purpose of this study was to explore these theories by comparing sweat rate and sweat-electrolyte concentrations of athletes with a reported history of EAMC to those with minimal cramping history to provide a potential link between sodium deficiency/sweat rate and EAMCs. The methodological consistency of analyses between two separate processes was also evaluated.

Methods

Forty-two student-athletes from the University of Arkansas voluntarily participated in this study. Sweat was collected from the forearm during exercise. Sweat samples were analyzed at two different Universities to quantify sweat-electrolyte concentration. Reliability statistics were compared between each University. Sweat rates were calculated for each athlete based on pre- and post-activity body mass. Sweat rates and sweat electrolyte values of athletes with a reported history of cramping were compared to those without.

Results

Sodium concentration between Universities was not significantly different ($p = .603$) and had an intraclass correlation of .887. 95% of data for sodium fit within the limits of agreement. Mean bias was less than 1 mmol/L for sweat sodium concentration. The cramping group (n=8) had a sweat rate of 2.55 ± 1.2 L/hr and a sweat sodium concentration of 39.2 ± 13.8 mmol/L. The non-cramping group (n=30) had an average sweat rate of 1.92 ± 1.03 L/hr and a sweat sodium concentration of 39.1 ± 26.6 mmol/L ($p = .113$).

Conclusions

Statistical analysis revealed moderate reliability between methods completed at two locations on two different electrolyte analyzers. In future studies, data can be pooled from these locations to create a larger sample size. The cramping group displayed trend toward a greater sweat rate than our non-cramping group, supporting the electrolyte-dehydration theory. However, a similar sweat sodium concentration and lack of statistical significance challenge this conclusion. EAMCs may have multiple causes and future research should be completed to identify an explanation for their onset.

Chapter 1: Introduction

Skeletal muscle cramps are one of the most common physical impairments for athletes during performance. In a study evaluating exertional heat illness (EHIs) among Division 1 football players in the southeast, 122 EHIs were identified in August alone, with 70% of those cases being skeletal muscle cramps.[1] Muscle cramps are characterized by a sudden, painful, involuntary contraction of muscle, proving to be debilitating. A cramp can be distinguished from an ordinary muscle spasm using the following criteria: they are acutely painful, they present an involuntary explosive onset and gradual spontaneous resolution or sudden termination with muscle stretching, one muscle or multiple muscle groups are involved, they are associated with both modest and forceful contractions, and they occur most often in calf and foot muscles or the hamstrings and quadriceps.[2] Proposed causes include fluid deficiencies (dehydration), electrolyte imbalances, neuromuscular fatigue, or any combination of these factors.[3]

Currently, there are two main theories explaining the onset of muscle cramping: muscular fatigue or the idea that athletes who sweat a lot and have salty sweat have a predisposition to cramping. First, muscle cramping may occur in overworked muscle fibers due to overuse or insufficient conditioning.[4] In performance or training, extended use on select muscle leads to local fatigue. The muscle fatigue hypothesis states that this strain causes an "excitatory alteration (increase) in muscle spindle afferent activity and an associated decrease in Golgi tendon organ inhibition" which leads to abnormal motor neuron control.[4] The combination of these events prohibits the contracted muscle to return to a relaxed state. This type of cramping remains localized to the overworked or fatigued muscle group and does not jump or wander around a muscle.[4] Football players have been shown to be most at risk for muscle cramping during the first three weeks of practice.[1] According to the muscular fatigue theory, athletes are more

vulnerable to cramping when performing exercise that the athlete is unaccustomed to because the muscle is easily overworked or fatigued.

The second common theory that attempts to explain cramping onset is the dehydration-electrolyte theory. Extensive sweating and a resulting whole-body exchangeable sodium deficit can lead to more widespread skeletal muscle cramping, with or without the presence of muscle overload or fatigue.[4] For athletes with high sweat sodium concentrations and a high sweating rate, plasma volume loss during exercise is accelerated, resulting in a more rapid shift of fluid from the interstitial compartment causing an onset of muscle cramping.[4] In other words, athletes who sweat a lot or have salty sweat are more at risk for exercise-induced cramps. Adequately replacing fluid and electrolyte loss has been shown to treat and prevent skeletal muscle cramps.

The amount of electrolyte or fluid loss required to prompt muscle cramping is not well described; however, "an estimated sweat-induced loss of 20% - 30% of the exchangeable sodium pool has been noted with severe muscle cramping."[4] Normal values of sweat rate and sodium loss for collegiate athletes have yet to be published. Without normal values, there is insufficient evidence to support sweat losses and/or sodium losses as contributing factors without association of these causing cramps. However, the dehydration-electrolyte theory cannot be ignored, as case reports have shown an elimination of muscle cramping with electrolyte supplementation.[5]

Researchers have offered explanations for the fatigue theory and electrolyte-dehydration theory. Studies have both supported and offered critique for these theories. Neither theory is able to physiologically explain the reason for cramping. Both studies can be and need to be challenged. Further research is required to establish the mechanisms behind muscle cramping.

The purpose of this experiment was to compare sweat rate and sweat-electrolyte values of athletes with a reported history of muscle cramping to those with minimal cramping history. This

was done to evaluate the potential link between sodium deficiency/sweat rate and skeletal muscle cramping risk.

Chapter 2: Literature Review

Muscle cramps

Skeletal muscle cramps have piqued the interest of researchers because of their prevalence and effect among people. Studies have been done in this subject for over 60 years, covering topics such as: the effect of Quinine on muscle cramping, calcium supplements in pregnant women, EMG activity during cramping, electrolyte deficiencies, etc.[6] The majority of research has involved the specifics of muscle and muscular function, while lacking a link between muscle cramping and exercise. Although researchers continue exploring this area, adequate information explaining a definite cause and recommended treatment for exercise associated muscle cramps is still lacking.

The word "cramp" has High German and Norse roots and is derived from the word "cram," meaning "squeezing, pressing, or pinching uncomfortably."[7] Today, we define muscle cramps as a sudden, spasmodic, painful, and involuntary contraction of muscle.[8,9] Muscular cramping is a widespread term that spans across several classifications - Table-1.[10] For the scope of this paper, exercise associated muscle cramps (EAMCs) will be the focus.

Table 1	
Paraphysiologic	• Occasional cramps • Exercise associated muscle cramps • Pregnancy
Idiopathic	• Familial • Sporadic • Others
Symptomatic	• Central and peripheral nervous system disorders • Muscular diseases • Cardiovascular disease • Endocrine-metabolic disease • Hydro-electrolyte disorders • Toxic and pharmacologic causes • Psychiatric disorders

The following questions can be used to differentiate between the types of cramping an athlete might be experiencing. If any of the questions are answered with a "yes," further medical treatment is required to eliminate other underlying causes.

- Is the cramping precipitated by physical exercise of very mild intensity and duration?
- Does the cramping occur at rest?
- Is the cramping associated with any other symptoms, such as paresthesia, pain, decreased sensation, or muscle weakness?
- Does the cramping episode occur during every exercise bout?
- Does passive stretching aggravate, rather than relieve the cramping?
- Is there a strong family history of cramping?
- Does the athlete use any drugs?
- Is cramping associated with a dark urine after exercise.[10]

If none of these symptoms exist, it can be assumed that the athlete is experiencing EAMCs.

It is very common for people to experience painful cramping, particularly after strenuous exercise or forceful contraction of muscle.[8] EAMC is prevalent in endurance events, such as triathlon, marathon, or ultramarathon distance running. Cramping has also been recorded in the following sports: American football, basketball, rugby, tennis, cricket, and cycling.[10] In one marathon, approximately 1 in 5 people were shown to have experienced EAMCs.[9] Cyclists are also prone to cramping, and another study revealed 30.2% of female and 69.8% of male cyclists reported cramping during a 100-mile race.[9]

The following characteristics can help distinguish true EAMC from other types of muscle pain or spasms.[7,9,11] Muscle cramps are involuntary painful contractions of muscle. They have a sudden onset with a quicker resolution, ranging from seconds to minutes. Cramps can occur at rest or after prolonged use of the muscle, and they normally occur while the muscle is in a shortened position. Lengthening or stretching the muscle tends to relieve cramping symptoms. True muscle cramps are associated with an electrical muscle-cramp discharge. They are acutely painful and may result in persistent soreness, with pain lasting anywhere between 48-72 hours.

Cramps usually happen in one muscle and are mostly seen in calves, feet, or large muscle groups. EAMC tends to occur in hot environments and when the athlete is partaking in endurance events.

A review evaluating studies using evidence-based medicine criteria revealed three risk factors for EAMC to be the most important. Does the athlete have a previous history of EAMC? Is the athlete exercising at a higher intensity or longer duration than normal? Is the athlete exercising in a hot and humid environment?[10] Exercise-induced muscle fatigue, body water loss, and high sweat sodium loss tend to be present in athletes with EAMC.[12] Based on these risk factors and studies relating exercise and cramping, two main theories explaining EAMC have emerged: muscular fatigue or electrolyte-dehydration.

Altered Neuromuscular Control Hypothesis

In the early 1990s, 1383 marathon runners were given a questionnaire. Of the 526 runners who had reported a past history of muscle cramping, 60% of these reported that muscular fatigue preceded the onset of cramping.[13] This study allowed researchers to begin investigating this link, and in March of 1996, a new theory was born. It is stated that muscle fatigue leads to altered neuromuscular control, which results in EAMC. Schwellnus proposed this theory based on two main observations. First, people are more vulnerable to EAMC after strenuous exercise. Continuous contraction of the biceps brachii in one study resulted in 18% muscle cramps before exercise and 26% muscle cramps after exercise.[13,14] Second, the onset of EAMC tends to occur in the final portion of a race, or competition.[13]

Muscle spindles and the Golgi tendon organ are muscle proprioceptors that play a role in this theory. When a muscle is stretched, muscle spindles send signals to the spinal cord, and in return, alpha motor neurons are stimulated causing the muscle to contract and resist the stretch.

The Golgi tendon organ has the opposite effect; when a muscle contracts and tension builds in the tendon, sensory neurons conduct action potentials to the spinal cord. The alpha motor neurons then cause the muscle to relax, which eliminates the tension in the tendon. The altered neuromuscular control hypothesis assumes that fatigue prompts an increase in muscle spindle afferent activity and a decrease in Golgi tendon organ inhibition.[4,9,15] Abnormal and sustained motor neuron activity results in increasing muscle contraction and decreasing muscle relaxation, leading to muscle cramps. Spinal reflex activity was studied in a response to a ramp stretch protocol under control conditions and after the induction of muscular fatigue.[16,17,18] Figure 1 shows the results from the muscle spindle afferent response.

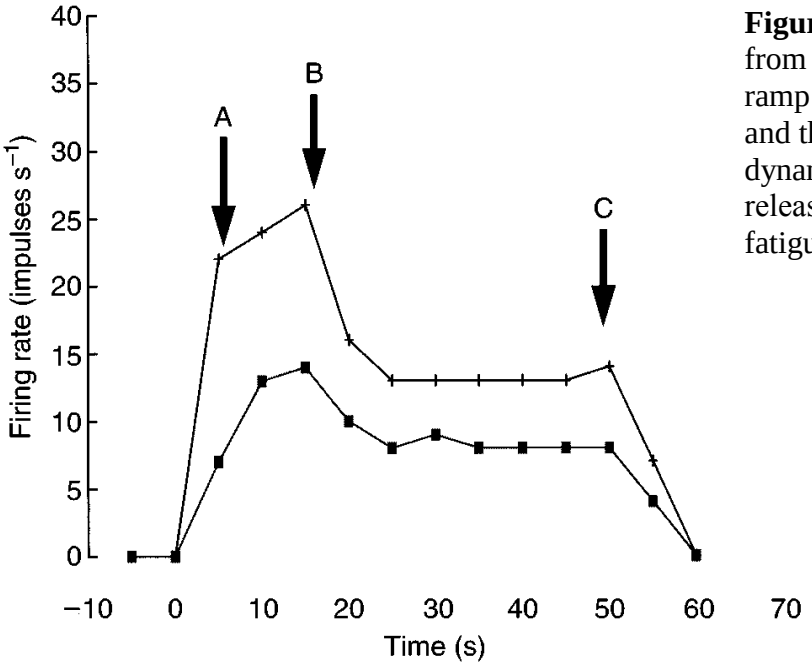

Figure 1 The Type Ia afferent firing rate from the muscle spindle in response to a ramp stretch protocol in the control muscle and the fatigued muscle. A = onset of the dynamic stretch, B = sustained tension, C = release of tension. ■, Control; +, fatigue.[16,17]

The data reveals that an increase in muscle spindle activity occurs as a result of muscular fatigue. A similar study showed the decrease in Golgi tendon firing rates in response to muscular fatigue.[16,18] Disruption in these peripheral muscle receptors have also been shown in animal models.[13] Electomyographic activity was used in studies to measure this activity. EMG activity

was shown to be greater in the cramping muscle than a non-cramping control muscle of the same athlete (a triathlete suffering from EAMC).[13] Researchers continue to use EMG readings to study neuromuscular activity in relation to muscle cramping.

If fatigue is the cause, it would make sense that EAMC occurs at high intensity exercise or exercise over a longer duration. A study among 210 triathletes revealed a correlation between faster predicted race times and faster actual race times with the development of EAMC.[19] A similar study also documented faster overall race times for triathletes in the EAMC group rather than the control group.[20] Athletes who competed at a faster pace were more prone to muscle fatigue, and therefore, more susceptible to EAMC.

Strengthening muscles has also been shown to reduce fatigue, resulting in a reduction of EAMC. A 42-year-old triathlete was unable to perform at his maximum due to recurring muscle cramping in his hamstrings. After implementing a program focused on strengthening and reeducating the gluteus maximus, EAMC was eliminated. It was determined that strengthening the agonistic gluteus maximus, reduced the effort required by the hamstrings, reducing EAMC.[21]

Stretching three times a day reduced the occurrence of nocturnal cramps in 44 patients.[7] In the same way, mild stretching is an effective immediate treatment for EAMC.[3,9] The fact that stretching eliminates EAMC is a case in itself. Stretching increases tension in the muscle, resulting in an increase in Golgi tendon organ activity.[10,13] This supports the claim that decreases in Golgi tendon organ firing rates (abnormal spinal reflex activity) contributes to EAMC. Passive stretching reduces EMG activity within 10 to 20 seconds and results in relief for the athlete.[10] According to the theory of altered neuromuscular control, EAMC should be reduced or prevented by reducing exercise duration or intensity and improving conditioning and range of motion through regular stretching programs.[4]

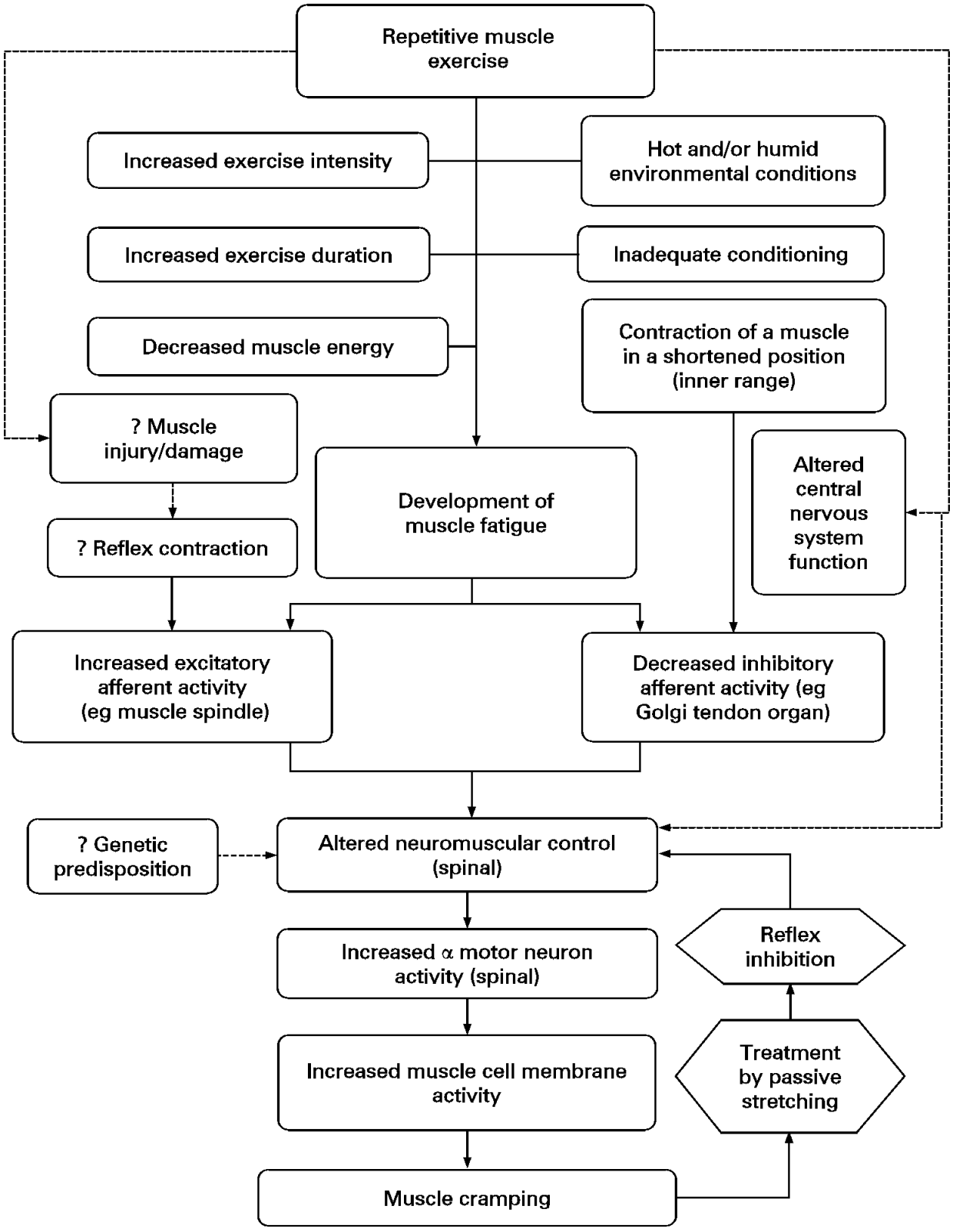

Figure 2 Altered Neuromuscular Control Hypothesis[13]

Serum Electrolyte and Dehydration Theory

In the early twentieth century, cases of muscle cramping were reported in those doing physical work in hot, humid environments.[12,13,16] Sugar cane cutters experiencing muscle cramping were shown to have lower urinary sodium levels than healthy laborers.[12] The authors concluded that workers had a whole body sodium deficit, which led to muscle cramping. Another case showed muscle cramping to be eliminated in steel mill workers who increased their consumption of table salt.[12] These cases along with others led people to believe that dehydration and sodium losses were the key causes of muscle cramping.

The human body has an advanced system in which it maintains proper fluid equilibrium. Sodium plays a large role in maintaining normal physiological function and providing an environment for optimal exercise performance.[22] In the body, water is distributed between the intracellular and extracellular space. The osmotic effect of sodium allows the body to control balance between the intravascular and interstitial fluids.[22] As an athlete sweats, water is shifted into different spaces of the body in order to maintain homeostasis. To compensate for plasma losses, water shifts from the interstitial fluid compartment to the intravascular space. As sweating continues or increases, the interstitial fluid compartment becomes more and more contracted.[4] "A contracted extracellular space (ECF) due to fluid and sodium loss may cause changes in the ionic concentrations in the ECF and mechanical deformation of motor nerve terminals during muscle shortening. These conditions may initiate hyperexcitability in selected motor nerve terminals."[22] As more water shifts from the interstitial to the intravascular space, adjacent nerve terminals and membranes could also be affected.[4] These data support the theory that EAMC can spread or jump to other muscle fibers. The effect of fluid loss on cramping can be partly explained by the mechanical effect of the contracted ECF on the nerve terminals.[23] Figure 3 shows that as sweat

rate increases, the concentration of sodium in the sweat also increases.[24] People with higher sweat rates could be more likely to be salty sweaters. This combination would cause a greater interruption of the fluid balance in the body, and potentially predispose the person to EAMC.

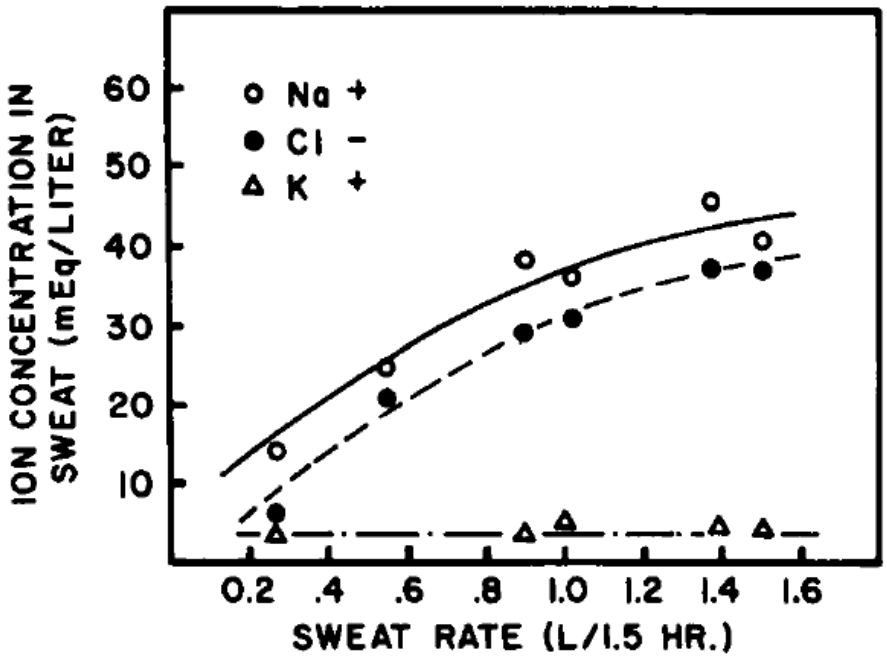

Figure 3

Relationship between sweat rate and the concentration of sweat sodium

Studies have been done to argue against the role that sweat rate and sweat electrolyte loss play in EAMC. Researchers claim that the electrolyte depletion and dehydration hypotheses are lacking physiological mechanisms and scientific evidence with thorough explanation.[13] A study looking at the effect of hydration on muscle cramping determined that hypohydrated men were no more susceptible to EAMC than hydrated men when fatigue and exercise intensity were controlled.[25] Two different studies measured serum sodium concentrations before and after a race and compared values between a cramping and non-cramping group. No significant differences were found, so the authors concluded that EAMC may not be associated with disturbances in fluid and electrolyte balances.[26,27] Whole-body exchangeable sodium deficit

usually is not detectable from measuring serum electrolytes, so these studies may be flawed in ruling out sodium loss as a contributor to EAMC.[4]

A high sweat sodium concentration has been linked to EAMC in recent studies.[13] One study found that evaluation and treatment of hydration status and electrolyte balance was able to eliminate EAMC.

> A 17-year-old, nationally ranked, male tennis player had been experiencing heat cramps during tennis match play. Medical exams proved to be normal, but an on-court evaluation displayed an extensive sweat rate (2.5 L/hr) and sweat sodium loss. Heat cramps were ultimately eliminated during competition and training by increasing his daily dietary intake of sodium.[5]

In other studies, an IV solution to replace fluids has successfully relieved cramping that was unresponsive to prolonged stretching.[28] In a study involving college-aged men with a history of muscle cramping, consumption of a carbohydrate-electrolyte beverage before and during exercise was shown to delay the onset of EAMC.[29] Studies have shown that monitoring and replacing fluids and electrolytes can decrease the occurrence of EAMC.

A study examining sweat rates and sweat concentrations in male athletes suggested that an increased sweat rate and sweat sodium concentration could predispose an athlete to EAMC.[30]

Table 2

Group	SR ($L \cdot h^{-1}$)	Na+ (mEq.L-1)	Cl- (mEq.L-1)	K+ (mEq.L-1)
Cramp-Prone	2.13 ±0.99	48.03 ±27.69	38.65 ±25.03	5.74 ±0.84
Matched-Control	1.82 ±0.80	26.09 ±15.28	20.48 ±14.32	6.49 ±1.40

Table 2 shows that higher sweat rates and sweat electrolyte concentrations were found in cramp-prone athletes compared to rates in matched-control athletes. One of the widely recognized treatments for EAMC is fluid replacement.[3,7,9,12,15] Adequate diets should also contain adequate supplies of nutrients.[31] If sweat loss and electrolyte loss is greater in cramp-prone athletes and fluid replacement is shown to treat EAMC, then it is apparent that the serum electrolyte and dehydration theory is worth exploring further. The purpose of this experiment was to explore this theory further by comparing sweat rate and sweat-electrolyte values of athletes with a reported history of muscle cramping to those with minimal cramping history to provide a potential link between sodium deficiency/sweat rate and skeletal muscle cramping. Further, we evaluated the methodological consistency of analysis between two electrolyte analyzers.

Methods: Sweat Collection

Two methods are commonly used to determine sweat loss: whole body washdown techniques (WBW) and regional skin surface collections (REG). The WBW method is more precise but requires a laboratory setting. The regional patch method is more practical for field testing. Unlike WBW, REG requires minimum supplies and allows for more freedom. Sweat can be collected from multiple body sites, and sites can be chosen based on convenience (taking sport, clothing, and site accessibility into consideration). Correction factors are used when only using one site.[32] For this study, the regional absorbent patch method was used to collect sweat and calculate sweat sodium losses. Studies have been done to compare the two methods due to rising concerns of the validity of the regional patch method. Limitations of the patch method include: “sweat composition and sweat rate vary across different regions of the body, covering the skin surface with a patch suppresses sweat evaporation in that specific region, and sweat

collected from within occlusive coverings may have falsely high electrolyte concentrations because of the electrolyte leaching from the stratum corneum of the skin."[32] The results of the studies showed that regression equations can be used to accurately determine WBW sweat from REG sweat collections.[32,33]

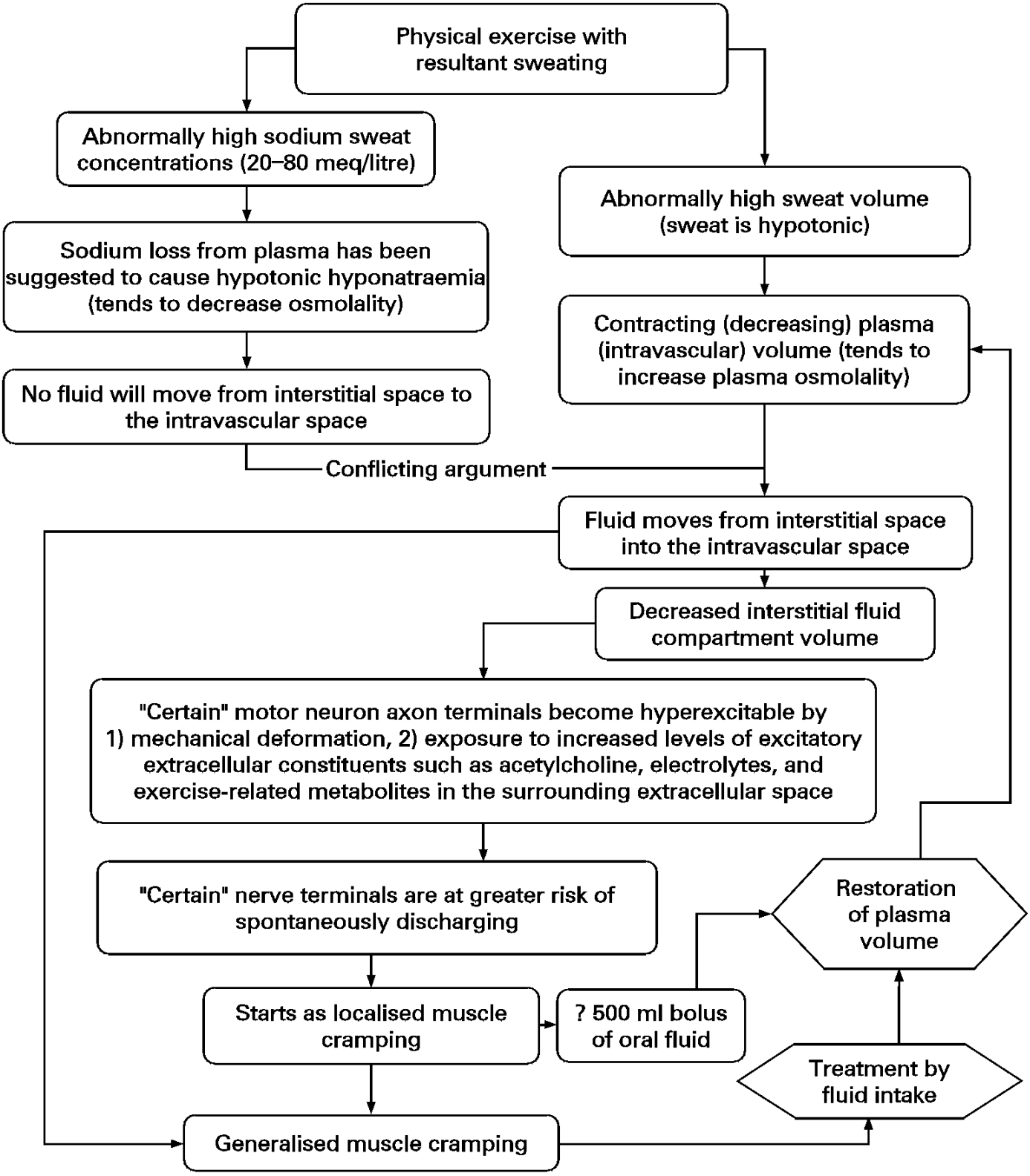

Figure 4 Serum Electrolyte and Dehydration Hypothesis

Chapter 3: Methods

Subjects

Forty-two medically cleared student-athletes from the University voluntarily participated in this study: twelve female basketball and twenty nine male track and field athletes. Subjects were between ages 18-24 (19.7 ± 1.5 years).

Table 3: Physical characteristics of subjects (n=41)

	Male (n=29)	Female (n=12)
Age (yrs)	**Mean:** 19.86 **Range:** 18 - 24	**Mean:** 19.27 **Range:** 18 - 22
Height (cm)	**Mean:** 184.06 **Range:** 165.1 – 193.04	**Mean:** 179.71 **Range:** 172.72 - 187.96
Body Mass (Kg)	**Mean:** 76.86 **Range:** 58.2 – 121.2	**Mean:** 75.81 **Range:** 67 – 90.6

Subjects signed a consent form (Appendix A) and were asked to fill out a short survey regarding demographics and muscle cramp history (Appendix B). Race demographics included 19 white/non-Hispanic, 13 African American, and 8 other. One athlete reported being sickle cell trait positive.

Collection

Subjects were asked to empty their bladder. The forearm was cleaned with an isopropyl alcohol pad , air dried, and then a sterile 3X3 gauze pad was placed on the athlete-selected forearm (Figure 5), adhered with transparent dressing, and wrapped with a small amount of tape.

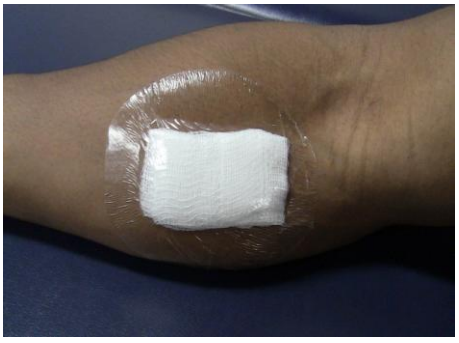

Figure 5

Pre exercise body mass was taken (if male, in shorts only; if female, in shorts and sports bra only). Then each subject participated in their scheduled practice or conditioning session. Subjects were allowed to drink as usual from labeled water bottles provided by researchers. This assured that researchers recorded accurate amounts of fluids consumed during activity. Each bottle was weighed pre- and post-exercise using the same Salter kitchen scale. After 30-45min, researchers wearing gloves removed the saturated sweat patches. Participants were asked to towel off and post-exercise body mass was recorded. The same body weight scale (Tanita) was used for both measurements. Researchers then placed the saturated sweat patches in a tube and transported them to the Human Performance Laboratory. Here, tubes were placed in a centrifuge (Sorvall) at 3000 rpm for ten minutes to extract whole sweat. This was transferred to storage tubes and frozen (-80°C).

Analysis

Samples were later thawed and pipetted into individual cuvettes. According to manufacturer recommendations, samples were diluted 9:1, vortexed, and analyzed in urine mode using an Easylyte Medica Electrolytes Analyzer. A correction factor was used because sweat was taken from only one body site.[32] Samples were then refrozen and shipped to Central Michigan University for concentration and analysis.

Central Michigan University used a 1:1 mix of sweat and 100 mmol NaCl. They took the Na and Cl reading from the NOVA 5 Electrolyte analyzer and subtracted half of what the NaCl solution concentration was. This value was then multiplied by 2 to find the sweat Na or Cl concentration for the sweat. Afterwards, the concentrations were corrected using Baker's equations.[32]

Chapter 4: Results

In order to pool data from multiple universities following the same methods, it was critical to determine whether electrolyte concentrations were consistent when calculated at two different universities. Analysis methods varied slightly, and samples were evaluated using two different analyzers: one at the University of Arkansas and one at Central Michigan University. Concentrations of chloride, sodium, and potassium in each sweat sample were compared across universities (Table 4). Statistical analysis assessing measurement error and reliability is shown in Table 5.[34]

Table 4: Descriptive Statistics (n=39)

		Mean	Minimum	Maximum	Std. Deviation
Pair 1	**ARK Na**	33.7828	17.78	78.97	13.52
	CMU Na	33.0900	16.75	77.74	12.19
Pair 2	**ARK Cl**	45.7551	28.90	100.30	15.99
	CMU Cl	33.7821	10.50	104.50	20.26
Pair 3	**ARK K**	4.7189	2.86	8.05	1.11
	CMU K	4.9527	2.10	10.31	1.51

Table 5: Measurement Error

	T-Test Sig. (2-tailed)	Spearman's rho	Intraclass Correlation	SEM	CV
	Sig. ≤ .05 Sig. Different	> 0.8 Strong < 0.5 Weak	0.7 - 0.8 Questionable	Small SEM = More Reliable	<10% Approximation
Sodium (Na)	.603	.602	.887	2.77	38.26%
Chloride (Cl)	.000	.833	.800	3.56	48.04%
Potassium (K)	.183	.666	.841	0.48	27.38%

The paired samples t-test and intraclass correlation (ICC) for sodium weren't questionable; however, Spearman's rho, SEM, and CV showed a weak correlation between Arkansas's and CMU's calculated Na values. Values for Chloride were questionable on every analysis except Spearman's rho. Chloride isn't a factor in muscle cramping, and literature rarely

takes it into consideration. Low correlation values for this electrolyte aren't a concern, as sodium and potassium are the electrolytes of focus. The potassium values were more comparable, with only Spearman's rho and CV showing inconsistency. Bland-Altman plots (Figures 6,7, and 8) visually show the correlation between values at Arkansas and CMU.

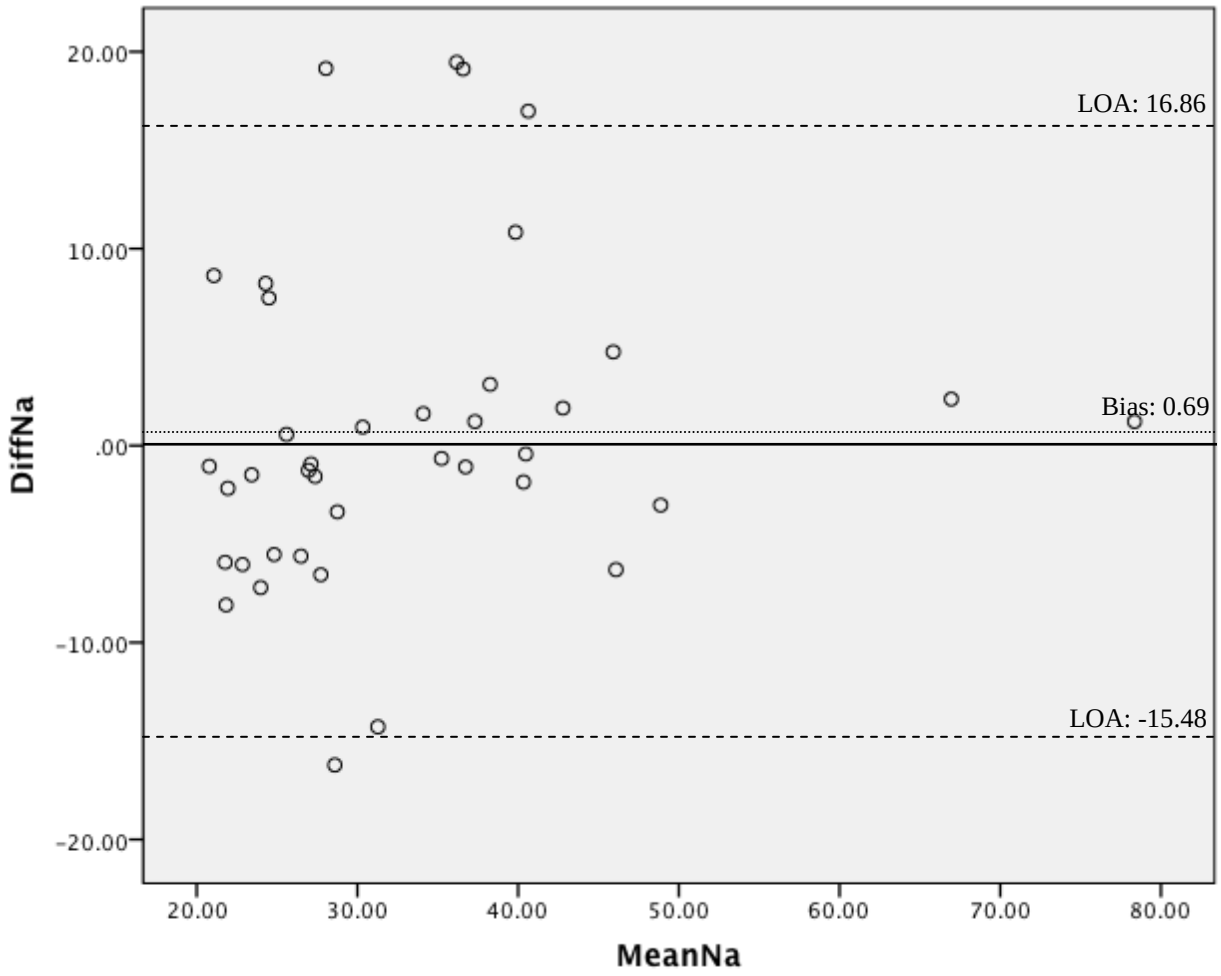

Figure 6: A Bland-Altman plot for the data presented in Table 4. The differences between sodium values are plotted against the mean values. The bias line and random error lines forming the 95% limits of agreement are shown in the figure. 13% of data may fall outside the LOA for Na+, but the large majority fits clinical LOA. Mean bias is less than 1mmol/L.

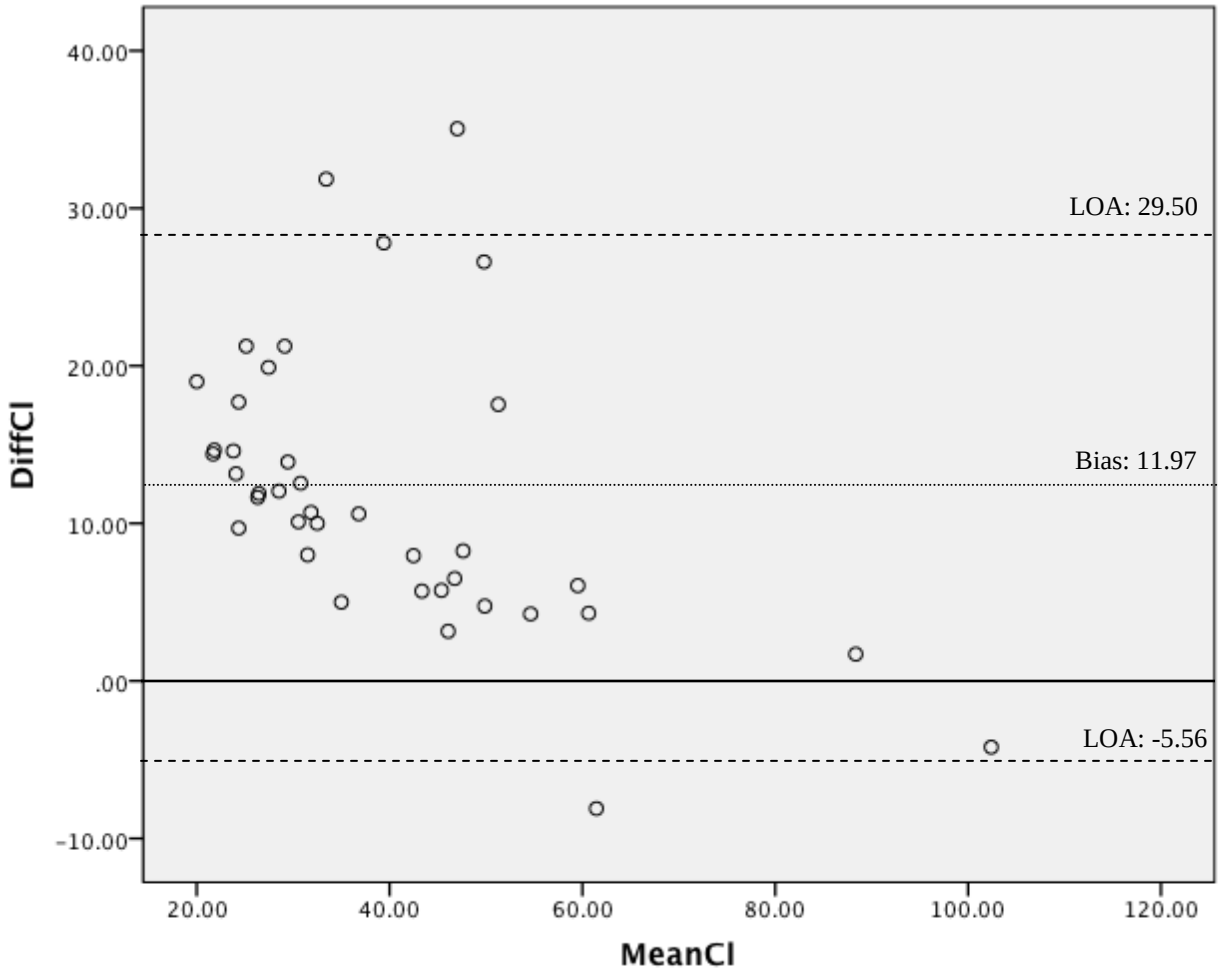

Figure 7: A Bland-Altman plot for the data presented in Table 4. The differences between chloride values are plotted against the mean values. The bias line and random error lines forming the 95% limits of agreement are shown in the figure. The plot suggests that 92% of data falls between the calculated LOA.

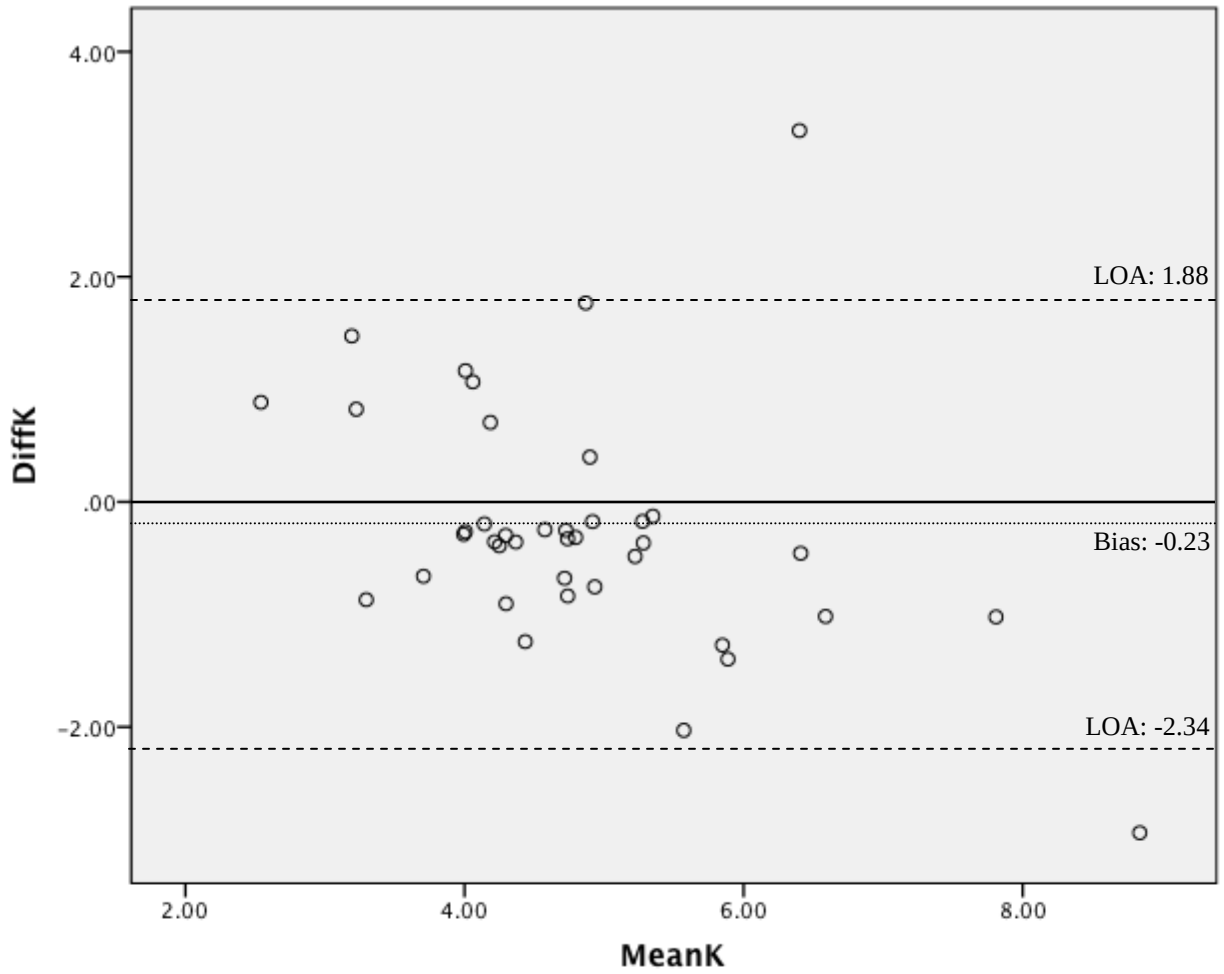

Figure 8: A Bland-Altman plot for the data presented in Table 4. The differences between potassium values are plotted against the mean values. The bias line and random error lines forming the 95% limits of agreement are shown in the figure. The majority of data fits within the LOA, with only 5% falling outside. Mean bias is relatively small.

The electrolyte values from Arkansas and CMU are correlated closely enough to pool data across universities in the future. A reliability >.8 is accepted and very good considering the use of two different machines at two different universities. Reliability for chloride was not high, but in terms of cramping, most people key in on sodium and potassium. Sodium values seemed

very reliable with a mean difference of less than 1 mmol/L. Clinically, limits of agreement could be altered in the future for more acceptable data points with less variance in electrolyte concentrations. For this study, Arkansas values collected to date were used to compare sweat rate and sweat-electrolyte values of athletes with a reported history of muscle cramping to those with minimal cramping history to provide a potential link between sodium deficiency/sweat rate and skeletal muscle cramping (Table 6). Athletes were considered crampers if they reported to have experienced muscle cramping at 3 or more times during their competitive season.

Table 6: Comparison of Sweat Rate and Sweat Electrolyte Values in Athletes with a History of Cramping to Those Without a History of Cramping

	Crampers (n=8)	Non-Crampers (n=30)	P-Value
Sweat Rate	2.55 ± 1.2 L/hr	1.92 ± 1.03 L/hr	.143
Sodium (Na)	39.2 ± 13.8 mmol/L	39.1 ± 26.6 mmol/L	.990
Chloride (Cl)	45.5 ± 7.7 mmol/L	44 ± 18.0 mmol/L	.812
Potassium (K)	6.9 ± 1.9 mmol/L	6.2 ± 2.0 mmol/L	.330

Table 7: Sensitivity, Specificity, and Relative Risk of Sweat Rate and Sweat Sodium

	Sensitivity	Specificity	Relative Risk
Sweat Rate	0.4000	0.8462	1.667
Sweat Sodium	0.1429	0.7667	0.604

Sweat rate averages were higher in student-athletes with a self reported history of muscle cramping. According to Arkansas values, athletes with high sweat rates are 1.6 times more likely to cramp than those without (relative risk). Sensitivity measured the proportion of actual positives (e.g. the percentage of athletes who were identified as being crampers and had high sweat rates or sweat sodium concentrations). Specificity measured the proportion of negatives (e.g. the percentage of athletes who were not crampers and had low sweat rates or sweat sodium concentrations). The average sweat sodium concentration was the same in both populations, and chloride and potassium differences were not significantly different.

Chapter 5: Discussion

The purpose of this experiment was to explore this electrolyte-dehydration theory further by comparing sweat rate and sweat-electrolyte values of athletes with a reported history of muscle cramping to those with minimal cramping history to provide a potential link between sodium deficiency/sweat rate and skeletal muscle cramping. The methodological consistency of analysis between two electrolyte analyzers was evaluated in order to determine whether data could be pooled across multiple campuses. Statistically, data that fit within the confines of the Bland-Altman plots in Figures 6, 7, and 8 could be used. The limits of agreement could be adjusted in order to pool data and cancel outliers. However, it might not be clinically accurate to combine data with a large error of difference. Further study needs to be done to standardize methods and assure that data analyzed at two different universities can be pooled into the same sample. The sample size of Arkansas athletes tested to this point was small. Our sweat rate and sweat-electrolyte values were taken from only 38 subjects, representing only two sports: men's track and field and women's basketball. 3 subjects were not factored in to the calculations due to incomplete data or too little sweat sample. Pooling data from other universities would increase the sample size and lead to more accurate assumptions when testing the two theories.

Athletes with a history of cramping had a higher average sweat rate than those who weren't considered crampers; however, sweat rates were not significantly different ($p = .603$). This leads to a possible conclusion that a high sweat rate is a risk factor for EAMC. Further data confirming these results would support Bergeron's theory that a high sweat rate or sweat sodium concentration is a predisposing factor for EAMC.

The average sweat sodium concentration in crampers was slightly above the average for noncrampers. This difference was very minimal and doesn't lead to the assumption that high

sweat sodium rates trigger muscle cramping. Previous studies supporting the muscle fatigue theory found no differences in sweat concentrations when testing athletes before and after a race and comparing the averages of crampers and noncrampers. It was concluded that high sweat sodium concentrations have no role in EAMC.[26,27] Our current findings strengthen this conclusion, and further testing may endorse the muscle fatigue theory of muscle cramping. However, similar research has shown significantly higher sodium concentrations in crampers than noncrampers.[30,35] A larger sample size and more defined separation between crampers and noncrampers is needed to evaluate whether athletes with a history of EAMC have higher sweat sodium concentrations than those who do not experience EAMC regularly.

The cramping history questionnaire needs to be revisited in order to more accurately distinguish crampers from non-crampers. Most athletes reported to have experienced cramping during their competitive season. We limited the cramping group to athletes who had reported three or more episodes of cramping. Educating the athletes on what is considered a true muscle cramp would lead to more accurate conclusions. In order to pool data, changes in the questionnaire would have to be made across all universities in order for methods to be standardized. Adding an educational piece or rewording cramping questions is suggested, but would need to be discussed among other campuses. Changes in the questionnaire or methods would exclude data collected up to this point from future analysis, which is something to be considered.

Athletes were tested during their off-season, which is one limitation for this study. Practices inducing less exertion could lead to lower sweat rates in the tested athletes. The time of year testing occurred also played a role. EAMC is more prevalent during the first few weeks of practice.[1] Heat acclimatization is a series of changes the body undergoes when placed under

stresses due to heat, exercise, etc. Heat acclimatization causes an increase in sweat rate and a decrease in sweat sodium concentration. Testing an athlete in the beginning of their normal season before acclimation might give more applicable numbers, providing a link to EAMC.

A small sample size was another limitation for this study. Our data was collected from two sports and did not represent the entire population of collegiate athletes. The results of this study are restricted to the small women's basketball and men's track and field populations tested. Due to the limited number of participants, it was hard to evaluate whether sweat rate and sweat concentration have an effect on cramping. More participants are needed to further evaluate differences in sweat rates and sweat-electrolyte concentrations in crampers and noncrampers. With a larger sample size, variances in sweat rates and concentrations among sports, gender, and demographics can be evaluated as well.

Conclusion

Due to a small sample size, a link between cramping and high sweat rates and sweat sodium concentrations could not accurately be seen. From our results, it appears that both theories might play a role in EAMC. Higher sweat rates in the cramping group support the electrolyte-dehydration theory; however, no differences in sodium concentrations among groups were found, backing the muscular fatigue theory. EAMC may have multiple causes. Continuation of this study is recommended to document normalized sweat rates for collegiate athletes and to examine the relationship between regularly cramping athletes and high sweat-electrolyte loss.

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Appendix A: Consent Form

Sweat rates and sweat electrolyte concentrations in collegiate male and female athletes
Consent to Participate in a Research Study
Principal Researcher: Brendon P. McDermott, PhD, ATC

INVITATION TO PARTICIPATE
You are invited to participate in a research study about sweat rates, sweat-electrolytes and muscle cramping. You are being asked to participate in this study because you are an athlete.

WHAT YOU SHOULD KNOW ABOUT THE RESEARCH STUDY

Who is the Principal Researcher?

Brendon P. McDermott, PhD, ATC
Assistant Professor in the Department of Health, Human Performance and Recreation
236B HPER (479) 575-4670

What is the purpose of this research study?
The purpose of this study is to compare sweat losses and sweat-electrolyte (i.e., sodium, potassium, and chloride) loss in athletes.

Who will participate in this study?
We are looking for 300 participants who normally engage in University athletics. Subjects will be ages 18-35, and will be medically cleared athletes.

What am I being asked to do?
Your participation will require the following:
You will be asked to fill out a short survey regarding demographics and muscle cramp history. You will also be asked to empty your bladder. Then, you will have a small sterile sweat patch (gauze pad and transparent dressing) applied to your forearm after it is cleaned with isopropyl alcohol and rinsed with deionized water. If necessary, this may require shaving a small patch for adhesiveness prior to cleaning. You may also be asked if we can apply a second sweat patch to your anterior forearm for data reliability questions. You will be weighed (if male, in shorts only; if female, in shorts in sports bra only). You will then participate in your scheduled practice or conditioning session. After 30-45min, researchers will remove saturated sweat patches.
You will be allowed to drink as usual from labeled water bottles provided by researchers. This will assure that researchers can record accurate amounts of fluids consumed during activity. Following exercise, you will be asked to towel off and be weighed again. Total time commitment for your participation is about 15-25min total.

Following removal of the sweat patch, researchers will place them in a tube and transport them to the Human Performance Laboratory. Here, they will be placed in a centrifuge for 15min. Following this, whole sweat will be transferred to labeled tubes for freezing/shipping. To assure accuracy of data, all sweat analysis will be conducted at North Dakota State University on the same machine with the same methods.

What are the possible risks or discomforts?

Due to the observational nature of this research, there is no increased risk to you as a participant.

What are the possible benefits of this study?

As a participant in this study, you will be provided with individual sweat rate data and sweat-electrolyte concentration that will be quantified during your participation. This can help you maintain hydration status and proper nutritional intake in future exercise participation.

How long will the study last?

This study will require roughly 15-25min. during one scheduled practice or conditioning session.

Will I receive compensation for my time and inconvenience if I choose to participate in this study?
There is no financial compensation for your participation in this study.

Will I have to pay for anything?
No, there is no financial obligation for participants completing the study.

What are the options if I do not want to be in the study?
If you do not want to be in this study, you may refuse to participate. Also, you may refuse to participate at any time during the study. There is no penalty for you to withdraw from participation at any point during the study, and your status within the University or as an athlete will not be affected in any way if you refuse to participate.

How will my confidentiality be protected?
All information will be kept confidential to the extent allowed by applicable State and Federal law. Your data from this study will be coded according to an assigned subject number. Furthermore, your data will remain under lock-and-key and the only researchers with access to the data will be the principal investigator (Brendon McDermott) and associated students and faculty of the University of Arkansas. Your information will be protected as much as possible if you are to volunteer for this study.

Will I know the results of the study?
At the conclusion of the study you will have the right to request feedback about the results. You may contact the faculty advisor, Brendon P. McDermott at brendonm@uark.edu. You will receive a copy of this form for your files.

What do I do if I have questions about the research study?
You have the right to contact the Principal Researcher or Faculty Advisor as listed below for any concerns that you may have.

Brendon P. McDermott
HPER 326B (479) 575-4670
brendonm@uark.edu

You may also contact the University of Arkansas Research Compliance office listed below if you have questions about your rights as a participant, or to discuss any concerns about, or problems with the research.

Ro Windwalker, CIP
Institutional Review Board Coordinator
Research Compliance
University of Arkansas
120 Ozark Hall
Fayetteville, AR 72701-1201
479-575-2208
irb@uark.edu

I have read the above statement and have been able to ask questions and express concerns, which have been satisfactorily responded to by the investigator. I understand the purpose of the study as well as the potential benefits and risks that are involved. I understand that participation is voluntary. I understand that significant new findings developed during this research will be shared with the participant. I understand that no rights have been waived by signing the consent form. I have been given a copy of the consent form.

_____

Appendix B: Demographics and Muscle Cramp History

Name:_____ **Subject #**: _____ **Date**: _____

****

QUALIFICATION QUESTIONS

	Answer	Decision
• Are you healthy (i.e., not sick or injured)?	Yes No	Disqualify if 'no'
• Are you taking any amphetamines?	Yes No	Disqualify if 'yes'
• Are you taking any salicylates?	Yes No	Disqualify if 'yes'
• Are you on thyroid medications?	Yes No	Disqualify if 'yes'
• Are you currently taking any antihistamines?	Yes No	Disqualify if 'yes'
• Are you taking atropine?	Yes No	Disqualify if 'yes'
• Are you taking any diuretics or B-adrenergic blockers?	Yes No	Disqualify if 'yes'
• Are you taking any supplements?	Yes No	Disqualify if 'yes'

****

BACKGROUND QUESTIONS

Age (y):_____ **Gender**: _____ **Height (m):**_____

Ethnicity (circle one):

A. African American
B. American Indian or Alaskan Native
C. Asian
D. Hispanic
E. Native Hawaiian/Pac. Islander
F. White/non-Hispanic

Are you within the first 10 days of your menstrual cycle?: Yes No

Have you exercised for at least 7 days in the heat in the last two weeks? Yes No

BMI (BMI = BM1 [kg]/Ht [m]): <18.5 18.6-24.9 25-29.9 >30 (obese)

****

SPORT QUESTIONS

Sport: _____ **Sport Position:**_____

Are you a starter?: Yes No

What level of competition do you participate in?

A. Division 1
B. Division 2
C. Division 3
D. Recreational/Intramural/Extramural

CRAMP HISTORY QUESTIONS
(these questions do not refer to menstrual cramps)

1. Do you have a history of muscle cramps during or after exercise?
   A. Yes
   B. No

If No to #1, please <u>do not</u> respond to any further questions.

If Yes to #1, please answer the following 6 questions:

1. Think of your competitive season. How many muscle cramps during or following exercise have you experienced <u>in your competitive season</u> (practices & games)?
   A. 1-2 cramps per month in-season
   B. 3-4 cramps per month in-season
   C. >5 cramps per month in-season

2. Think of the month in the last year where you cramped the most. In that month, how many times did you cramp <u>each week</u>, approximately?
   A. 1x/week
   B. 2x/week
   C. 3x/week
   D. 4x/week
   E. 5x/week
   F. 6x/week
   G. 7x/week

3. Have you ever experienced multiple cramps in the same exercise session?
   A. Yes
   B. No

4. When you cramp, which muscles are affected (circle all that apply)?
   A. Abdomen
   B. Arms
   C. Back
   D. Buttocks
   E. Calf
   F. Chest
   F. Foot
   G. Groin
   H. Hands
   I. Hamstrings
   J. Quadriceps

5. When during exercise do you typically experience muscle cramps?
   A. Beginning of game/match/practice
   B. Middle of the game/match/practice
   C. End of the game/match/practice
   D. After the game/match/practice

6. Are you currently doing anything *extra* to specifically prevent cramp episodes (e.g., doing any training program for cramping)?
   A. Yes
   B. No
   C. If yes, what are you doing? _____

Appendix C: Data Collection Sheet

Testing Environment Temperature & Humidity: _____°C _____%

WBGT: _____

Location of practice: Indoors Outdoors

Water bottle pre-exercise weight: _____ kg
Water bottle POST-exercise weight: _____ kg
Fluid consumed (∆ pre-weight & post-weight): _____ kg

Empty bladder completely.
Shave, clean, and dry forearms. DO NOT apply sweat patches.

Body Mass 1 (shorts only for men; shorts & sports bras for women): _____kg

Attach sweat patches. Remove after 30-40 minutes.

	1	2	AVG
Sweat [Na^+]			
Sweat [K^+]			
Sweat [Cl^-]			

End exercise. Towel dry.

Body Mass 2 (shorts only for men; shorts & sports bras for women): _____ kg

Exercise duration: _____ hours

Sweat Rate = (BM1 – BM2 + fluid volume consumed)/(exercise duration):

_____(L/h)

Appendix D: IRB Approval

January 15, 2013

MEMORANDUM

TO: Brendon McDermott
Kevin Miller
Susan Yeargin
Brittany Willard

FROM: Ro Windwalker
IRB Coordinator

RE: New Protocol Approval

IRB Protocol #: 13-01-394

Protocol Title: *Sweat Rates and Sweat Electrolyte Concentrations in Collegiate Male and Female Athletes*

Review Type: □ EXEMPT × EXPEDITED □FULL IRB

Approved Project Period: Start Date: 01/11/2013 Expiration Date: 01/10/2014

Your protocol has been approved by the IRB. Protocols are approved for a maximum period of one year. If you wish to continue the project past the approved project period (see above), you must submit a request, using the form Continuing Review for IRB Approved Projects, prior to the expiration date. This form is available from the IRB Coordinator or on the Research Compliance website (http://vpred.uark.edu/210.php). As a courtesy, you will be sent a reminder two months in advance of that date. However, failure to receive a reminder does not negate your obligation to make the request in sufficient time for review and approval. Federal regulations prohibit retroactive approval of continuation. Failure to receive approval to continue the project prior to the expiration date will result in Termination of the protocol approval. The IRB Coordinator can give you guidance on submission times.

This protocol has been approved for 750 participants. If you wish to make any modifications in the approved protocol, including enrolling more than this number, you must seek approval prior to implementing those changes. All modifications should be requested in writing (email is acceptable) and must provide sufficient detail to assess the impact of the change.

If you have questions or need any assistance from the IRB, please contact me at 210 Administration Building, 5-2208, or irb@uark.edu.