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Sweat and Sodium Losses in NCAA Football Players: A Precursor to Heat Cramps?

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This observational study was designed to determine whether football players with a history of heat cramps have elevated fluid and sodium losses during training. During a "two-a-day" training camp, five Division I collegiate football players (20.2 ± 1.6 y, 113 ± 20 kg) with history of heat cramps (C) were matched (weight, age, race and position) with a cohort of teammates (19.6 ± 0.6 y, 110 ± 20 kg) who had never cramped (NC). Change in body weight (adjusted by fluid intake) determined gross sweat loss. Sweat samples (forearm patch) were analyzed for sodium and potassium concentrations. Ad libitum fluid intake was measured by recording pre- and post-practice bottle weights. Average sweat sodium loss for a 2.5-h practice was projected at 5.1 ± 2.3 g (C) vs. 2.2 ± 1.7 g (NC). When averaged across two practices within the day, fluid intake was similar between groups (C: 2.6 ± 0.8 L vs. NC: 2.8 ± 0.7 L), as was gross sweat loss (C: 4.0 ± 1.1 L vs. NC: 3.5 ± 1.6 L). There was wide variability in the fluid deficit incurred for both C and NC (1.3 ± 0.9 vs. 0.7 ± 1.2%) due to fluid intake. Sweat potassium was similar between groups, but sweat sodium was two times higher in C versus NC (54.6 ± 16.2 vs. 25.3 ± 10.0 mmol/L). These data indicate that sweat sodium losses were comparatively larger in cramp-prone football players than in NC. Although both groups consumed sodium-containing fluids (on-field) and food (off-field), both appeared to experience an acute sodium deficit at the end of practices based on sweat sodium losses. Large acute sodium and fluid losses (in sweat) may be characteristic of football players with a history of heat cramping.

Key Words: heat illness, fluids, salt, electrolytes, cramping

Heat-related muscle cramps (heat cramps) have been reported in the literature for decades (6, 14, 17, 24). The association between heat cramps and salt (sodium) losses is not a novel idea, as other occupations have shown that the heat cramp incidence can be reduced by the addition of salt replacement (6, 14, 17, 24). Over the past 100 y, stokers on ocean steamers, underground miners, desert soldiers, and summer laborers learned to add salt to their drinks to prevent heat cramping (9). Likely, football players share some similarities (e.g., sweat loss, heat exposure) with

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those occupations suffering heat cramps. The association between heat cramps and salt loss has, however, never been formally reported in any level of the American football population. Further, the use of salt replacement to minimize the incidence of heat cramps in football has not been documented, except in the treatment of heat cramps with administration of intravenous saline solutions.

Heat cramping in football can sideline players. Common to tennis and triathlons, this form of cramping that involves involuntary, painful spasms of large muscle groups—spurs ongoing debate on etiology, prevention, and therapy within American football. Also puzzling is why some players are predisposed to heat cramps, yet others on the same team with similar physical characteristics and in the same environment are seemingly not. A recent survey of 997 athletic trainers regarding the cause and prevention of exercise-associated heat cramps centered on factors related to dehydration and electrolyte imbalance—heat, humidity, fluid and electrolyte replacement (23).

Muscle fatigue is purported to be a factor in heat cramping (19). Copious sweating due to intense exercise in the heat also seems to contribute, by causing large losses of fluid and sodium and a consequent contraction of the extracellular fluid space. Muscle function and physiology under an altered extracellular environment has implications for normal cellular and nerve function. The theories compete and therapies still vary, however. Potassium, calcium, and magnesium mineral deficiencies have been implicated in the etiology of heat cramping, even though these minerals are proportionately scarce in sweat. This has led to specious sideline therapies like eating bananas or injecting calcium gluconate or magnesium sulfate. Other unsupported "remedies" have included quinine, amino acids, and quaffing water (4).

Sodium depletion could be the predominant factor in heat cramping. For some athletes, even when fit and heat-acclimatized, sweat sodium and chloride concentrations can be high and can rise further as sweat rate increases and sweat gland reabsorption decreases (4). We have observed that football players prone to heat cramps have the appearance of sweating heavily and occasionally show signs of salty sweat (salt caking on skin and clothes) compared to those athletes who have no history of heat cramps. Our pilot data documenting large fluid and sodium losses during practice in professional football players, some prone to heat cramping, suggests sodium is an important contributing factor (21). That study, however, had no control group. The main purpose of this study, then, was to test our hypothesis that American football players prone to heat cramps lose more fluid and sodium (salt) via sweating (either high water output or high salt concentration) than teammates who have never cramped.

Methods

Subjects

The university institutional review board approved this study. Subjects were told the aim and risks, signed an approved informed consent form, and voluntarily agreed to participate. Ten football players in a National Collegiate Athletic Association (NCAA) Division I program were recruited for the study. All were first-team members who had played throughout high school and at least one season at the

intercollegiate level. Five players, identified by the head certified athletic trainer, had a history of recent (episodes within 1 y) and severe heat cramping. Severe heat cramps are defined as a whole-body cramping experience symmetrical in terms of the muscle groups affected, with the episode requiring intravenous fluid treatment and the player to discontinue practice or competition. This definition is consistent with the literature description of the phenomenon (3, 24). On average, the group identified as crampers (C) experienced three (range 1 to 5) episodes in the previous season. Five others, matched for age, size, race, and position, had never experienced such cramping and were considered the non-cramping controls (NC). Physical characteristics are shown in Table 1.

Conditions

Players were in pre-season training camp in August of 2002. We measured sweat loss, fluid intake, and sweat sodium and potassium during the morning (~ 8 AM) and evening (~ 6 PM) of a two-a-day practice session. Practices, 2.5 h each, were held outdoors with the players in full uniform (i.e., helmets and pads) and involved a variety of skill drills, playbook execution, game situations, and conditioning. Environmental heat stress was assessed with a WBGT meter (QuesTemp 36, Quest Diagnostics, Oconomowoc, WI) that recorded wet, dry, and globe bulb temperatures at 15-min intervals.

Measurements and Sample Collection

Before each practice, a sample of urine was collected to gauge specific gravity (USG) using a clinical refractometer (Atago, model A300CL). Sweat loss was determined by measuring pre-practice and post-practice bodyweight to the nearest 0.1 kg on a calibrated scale (PW200, Mettler-Toledo, Inc., Columbus, OH). Players were weighed in undershorts or athletic supporter only, and those who required ankle taping were weighed after taping. A single (dry) ankle tape was later weighed and the resulting value multiplied by two and used to correct initial bodyweight. At the end of practice, players returned to the locker room, wiped down with a

Table 1 Physical Characteristics of the Two Groups of Subjects

Characteristic	Crampers (*N* = 5)	Non-crampers (*N* = 5)
Age (y)	20.2 ± 1.6	19.6 ± 0.6
Weight (kg)	112.9 ± 19.9	110.2 ± 20.4
Height (m)	1.9 ± 0.1	1.9 ± 0.1
Race*	3 B, 2 W	3 B, 2 W
Positions**	WR, TE, DL, DL, OL	WR, TE, DT, DE, OL

Note. Values are means ± standard deviation. * Race designations are B, black; W, white. ** Positions are WR (wide receiver), TE (tight end), DT (defensive tackle), DL (defensive lineman), DE (defensive end), and OL (offensive lineman).

towel, and were weighed without ankle tape while wearing the same shorts/athletic supporter as before. Change in bodyweight, corrected for fluid intake and urine loss during the practice, was used to estimate gross sweat loss. Sweat rate was calculated per hour of practice.

Players received several individual, 32-ounce squeeze bottles that were weighed full of water, a sports drink (Gatorade, The Gatorade Co., Chicago, IL), or a sports drink with additional electrolytes and minerals (GatorLYTES, The Gatorade Co., Chicago, IL). Electrolyte content was determined for the sports drink beverages and is shown in Table 2. Players drank as much or as little as they wanted (ad libitum) and were free to choose among the three types of drink. After practice, bottles were re-weighed and pre-post weight differences were totaled to estimate overall fluid intake and intake of each of the three drinks. Consumed electrolyte content was determined from intake data. Student athletic trainers "shadowed" players through practice, so fluids were readily available. They held racks of six bottles and assured that the players did not spit, spill, or share the fluid and that each player drank only from his own bottles.

Sterile sweat patches (Osteopatch, PacificBiometrics, Inc., Irvine, CA) were used to collect samples of regional sweat. The patches were tested in our lab for background sodium and potassium content and were found to not contribute detectable levels of these electrolytes using flame photometry. The forearm site, well correlated with whole-body values obtained in laboratory studies, was used as a surrogate measure for whole body sweat electrolytes (18) for this on-field study in which we needed quick and easy access for retrieving the sweat samples in the midst of practice. Just before players entered the practice field, patches were placed on the right forearm, after residual minerals were removed from the skin by wiping the forearm with sterile gauze soaked in distilled water and drying with a mineral-free paper towel. During natural breaks in practice, patches were removed with clean forceps and put in a sterile tube. One sweat sample was collected during each practice. Patches were removed after the first 30 min of practice, having allowed adequate time for the onset of sweating to occur.

All foods that players consumed during the daily observation period were recorded and photographed to estimate portion size. Nutritional information was

Table 2 Electrolyte of Fluids Consumed During "Two-a-Day" Practices

Electrolyte	Gatorade	GatorLYTES solution
$[Na^+]$	18.0	75.0
$[K^+]$	3.0	20.0
$[CL^-]$	11.0	90.0
$[Mg^{++}]$	—	2.8
$[Ca^{++}]$	—	2.9

Note. All units are mmol/L; GatorLYTES solution is a powder packet of electrolytes that is added to a standard 20-oz. Gatorade bottle to produce the electrolyte levels shown above.

obtained from all labels, and for cafeteria food, was derived from the nutritional breakdown of the menu plan provided by the university food service. Nutritionist Pro software (First Databank, San Bruno, CA) was used to quantify total sodium intake from meals and fluids.

Sample Analysis

Sweat electrolyte analysis followed extracting the fluid from sweat patches via centrifugation using a 0.45 micron porous filter tube (Microsep II MF, Pall Corp., East Hill, NY). From extracted fluid, sweat sodium and potassium concentrations were determined by flame photometry (IL943, Instrumentation Laboratory, Lexington, MA).

Calculations

Sweat loss was calculated from change in body weight pre- to post-practice after correcting for any urine loss and total fluid intake (assuming fluid volume of 1 L = 1 kg). Total sweat electrolyte loss was calculated by multiplying the volume (L) of water (sweat) that was lost by the concentration of each electrolyte (mmol/L, sodium or potassium) in the fluid. For fluids that were consumed during practice, volume (and concentration) were known values which allowed for calculation of sodium intake from fluids. No foods were eaten during practice. Over the entire day, sodium intake was recorded from the diet and sodium intake was calculated from diet and sports drinks combined. Daily urine sodium values were not available between practices so whole-body balance could not be estimated.

Statistical Analysis

Data were summarized using SPSS statistical software (version 11.0, SPSS, Inc., Chicago, IL) and are presented as mean ± standard deviation. For paired comparisons with small sample size and non-Gaussian distribution, we used a non-parametric procedure for paired comparisons (Wilcoxon Matched Pairs Signed Ranks test) to determine group differences. Statistical significance was accepted at an alpha level of $P < 0.05$. There was a ceiling effect, however, on the assessment of significance level due to small sample size where some data could not be used due to a player vomiting during practice. (The lowest significance level possible in these instances was $P = 0.063$). Thus, to further characterize the magnitude of the effect of the dependent measures between groups of small sample size, effect size estimates were calculated from mean and standard deviations and categorized by the continuum of effect size proposed by Cohen (8). For reference, “large” effect sizes are 0.8 or greater.

Results

For the football season prior to our study (2001-2002), each individual in the C group experienced at least one and no more than five episodes of heat cramps (mean 3.0 ± 1.9, $n = 5$) that required treatment. For the season following our study, C experienced in total ten episodes of heat cramping that required medical intervention.

NC did not experience any heat cramping for either the season preceding our study or the season following it. There were no differences in age, body weight, height, and college playing experience between C and NC players.

On-field environmental conditions were milder in the morning than evening. During the 2.5-h morning practice, ambient temperature ranged from 22.7 °C to 26.0 °C, relative humidity was 72 to 93% saturated, and heat stress (WBGT) rose from 22.8 °C to 27.2 °C. During the evening practice, air temperature dropped from 30.8 °C to 28.2 °C, humidity rose from 51% to 72% saturated, and heat stress (WBGT) ranged from 28.3 °C to 25.1 °C. This team purposefully avoided practicing during hotter periods of the day.

Urine samples before practices showed USG to be 1.025 ± 0.004 for C and 1.021 ± 0.002 for NC in the morning, and on returning for the evening practice USG was 1.015 ± 0.004 and 1.018 ± 0.004 for C and NC, respectively. Both groups drank a mix of water and sports drinks between practices. No difference in USG existed between groups at either time.

Sweat Losses and Fluid Intake by Practice Session

Sweat loss and fluid intake results specific to the practices are shown in Table 3. During the morning session, C appeared to lose more sweat (ES = 0.95) although using non-parametric statistics, this was not statistically different and C did not incur a significantly greater fluid deficit than NC (P = 0.313, ES = 0.98) due to large individual variation in fluid intake and small sample size. Intake of sodium in fluids consumed appeared greater for C than NC (2.6 ± 1.6 vs. 0.9 ± 1.3 g, P = 0.313, ES = 1.1) due predominantly to the consumption of sports drinks.

During the second practice session, one subject vomited several times, resulting in loss of fluid balance data for that subject in the C group. During the evening, C lost similar sweat (P = 1.000, ES = 0.30) and incurred a similar fluid deficit compared to NC (P = 0.875, ES = 0.32).

Sweat sodium concentration, sodium intake, and sweat sodium loss values are shown in Table 4. Intake of sodium from fluids appeared greater in C than NC (2.9 ±1.6 vs. 0.6 ± 0.6 g, P = 0.125, ES = 1.8) with very large effect sizes. C had a higher sweat sodium concentration compared to NC (55.6 ± 19.0 vs. 21.6 ± 7.6 mmol/L, P = 0.063, ES = 2.0) during the morning and this was consistent during the evening practices (C vs. NC, 53.6 ± 15.0 vs. 28.9 ± 11.7 mmol/L, P = 0.063, ES = 1.68); see Table 4. These values approached statistical significance with very large effect sizes. This resulted in total sweat sodium loss during each practice session appearing higher for C than NC with very large effect sizes. Total sweat potassium losses were small and were not different between C and NC.

Average Sweat Losses and Fluid Intake (Across the Day)

We combined data from morning and evening sessions to calculate “average” losses and intakes for each dependent variable. In this manner, gross sweat loss for a 2.5 h practice session was 4.0 ± 1.2 L in C and 3.5 ± 1.6 L in NC (P = 0.250, ES = 0.36). Fluid intake was similar between C and NC (2.6 ± 0.8 L vs. 2.8 ± 0.7 L;

Table 3 Fluid Intake and Sweat Loss Data in Crampers and Non-Crampers During "Two-a-Day" Practices

Variable	Crampers *N* = 5	Non-Crampers *N* = 5	*P*-value*	Effect size
Morning practice				
Fluid intake (L)	2.57 ± 1.03	2.32 ± 0.34	0.625	0.33
Sweat loss (L)	3.79 ± 1.54	2.54 ± 1.05	0.125	0.95
Sweat rate (L/h)	1.49 ± 0.60	0.99 ± 0.41	0.125	0.97
Fluid loss replaced (%)	70.0 ± 27.2	123.0 ± 96.8	0.313	0.75
Net fluid deficit (%)	1.07 ± 0.83	0.10 ± 1.12	0.313	0.98
Δ Body weight (kg)	–1.43 ± 0.84	–0.42 ± 0.84	0.313	1.20
Evening practice	*N* = 5 [a]	*N* = 5		
Fluid intake (L)	2.55 ± 0.64	3.18 ± 0.72	0.250	0.92
Sweat loss (L)	4.15 ± 0.67	4.49 ± 1.45	1.000	0.30
Sweat rate (L/h)	1.66 ± 0.27	1.80 ± 1.48	1.000	0.31
Fluid loss replaced (%)	63.0 ± 17.7	74.0 ± 15.4	0.625	0.66
Net fluid deficit (%)	1.52 ± 0.99	1.20 ± 1.03	0.875	0.32
Δ Body weight (kg)	–1.69 ± 0.93	–1.41 ± 1.05	0.875	0.28

Note. Values are means ± standard deviation. Column showing effect size compares Crampers and Non-crampers groups. * *P*-value represents exact significance (two-tailed) from Wilcoxon statistical test. [a] Means reflect missing data points for 1 subject. Effect size estimates are 0.2 (small), 0.5 (medium), and 0.8 (large) effects (8).

$P = 0.734$, ES = 0.25), as was fluid deficit (percent of body weight) by the end of practice (1.3 ± 0.9% vs. 0.7 ± 1.2%; $P = 0.250$, ES = 0.57), in part due to large variability in individuals tested.

Absolute forearm sweat sodium concentration in C was higher compared to NC (54.6 ± 16.2 vs. 25.3 ± 10.0 mmol/L; $P = 0.002$, ES = 2.05), and yielded an estimate of total sweat sodium loss more than twice that of NC (5.1 ± 2.3 g vs. 2.2 ± 1.7 g, $P = 0.039$, ES = 1.38). Sweat potassium was not different between C and NC (4.6 ± 0.9 vs. 4.9 ± 0.9 mmol/L; $P = 0.232$, ES = 0.33). Mean total sweat potassium loss was not different between C and NC (0.72 ± 0.22 g vs. 0.63 ± 0.24 g; $P = 0.426$, ES = 0.17)

The type of fluid consumed also differed between C and NC. Of the average total fluid during a 2.5-h practice, C consumed 17% water, 19% sports drink (~ 18 mmol/L Na^+), and 58% high-electrolyte sports drink (~ 75 mmol/L Na^+). NC consumed a fluid distribution emphasizing water: 66% water, 11% sports drink, and 23% high electrolyte sports drink. On average, the percent of daily sodium intake from consumed fluids was 39 ± 17% in C compared to 16 ± 20% in NC. Diet (meals) contributed the remaining sodium. Sodium intake from on-field fluids and daily meals, and sweat sodium loss are shown in Table 4. The differential between

Table 4 Sweat Sodium Concentration, Sodium Intake, and Sweat Sodium Loss in Crampers and Non-Crampers During "Two-a-Day" Practices

Variable	Crampers *N* = 5	Non-Crampers *N* = 5	*P*-value*	Effect size
Morning practice#				
$[Na^+]_{sweat}$ (g/L)	1.3 ± 0.4	0.5 ± 0.2	0.063	2.04
Na^+ $intake_{fluids}$ (g)	2.6 ± 1.6	0.9 ± 1.3	0.313	1.09
Na^+ $loss_{sweat}$ (g)	–5.0 ± 2.6	–1.5 ± 0.5	0.125	1.87
Evening practice#	*N* = 5	*N* = 5		
$[Na^+]_{sweat}$ (g/L)	1.2 ± 0.3	0.7 ± 0.3	0.063	1.67
Na^+ $intake_{fluids}$ (g)	2.9 ± 1.6	0.6 ± 0.6	0.125	1.80
Na^+ $loss_{sweat}$ (g)	–5.3 ± 2.2	–3.1 ± 2.0	0.375	1.05
Entire day (24 h)	*N* = 5	*N* = 5		
Na^+ $intake_{fluids}$ (g)	5.7 ± 3.2	1.5 ± 1.8	0.250	1.68
Na^+ $intake_{foods}$ (g)	10.3 ± 4.1	7.4 ± 3.3	0.125	0.78
Total Na^+ $loss_{sweat}$ (g)	–10.4 ± 4.1	–4.9 ± 2.3	0.250	1.66

Note. Values are means ± standard deviation. Column showing *P* and effect size compares Crampers and Non-Cramper groups. Effect size estimates are 0.2 (small), 0.5 (medium), and 0.8 (large) effects (8). # No urine loss during activity. Total loss of Na in sweat was estimated from gross sweat loss (L) multiplied by the concentration of sodium in sweat (g/L) for both practices, using 1 mmol/L Na^+ = 23 mg/L Na^+. * *P*- value represents exact significance (two-tailed) from Wilcoxon statistical test.

intake of sodium and that lost in sweat were similar between C and NC for morning ($P = 0.375$, ES = 0.58) and evening ($P = 0.875$, ES = 0.03) sessions. Over the entire day, sodium intake from fluids and meals appeared greater in C than NC, as was the loss of sodium in the sweat. This resulted in a similar sodium differential by the end of the day as intake and loss were proportionately larger in C compared to NC, with large effect sizes. Despite seemingly large fluid and sodium losses, no players in our study cramped on the day of data collection.

Discussion

The novel finding of this study, perhaps the first study of this nature using NCAA Division I football players on the field, is that cramp-prone players lost twice the sodium in sweat as did teammates who do not have a history of cramping. In contrast, sweat potassium loss was trivial and similar between groups. These results support our preliminary finding that cramp-prone professional football players have high sweat and sodium losses (21). Bergeron finds the same in top tennis players who have the same characteristics and a history of heat cramping (3, 4). Sodium depletion is a recurrent theme in heat cramping, and is reported to be one

of the factors contributing to cause, treatment, and prevention of exercise-associated muscle cramps (23).

The absolute amount of sodium lost in the sweat of the subjects in the present study could be debated. Our method of sweat collection by using a patch has been criticized for creating a microenvironment that might stimulate the sweat gland to secrete greater amounts of sodium than when the skin surface is open to the air. Shirreffs and Maughan (20) reported that values obtained using the patch methodology (on the back) were nearly 46% greater than when using the whole-body washdown method. Using that as a correction factor, the sodium losses during the practice might have been 2.4 g in the cramping group versus 1.1 g in the non-cramping group. Determination of the true amount of sodium lost is further complicated by the fact that the uniform worn by the players might also create an environment that promotes greater sodium loss on the skin surface, but this has yet to be investigated as a factor. In addition, our concentration estimates could be low because we collected samples early in the practice session. Unpublished work in our lab indicates that sweat sodium concentrations might increase by 20% as the exercise training progresses from the first to second hour of exercise. Data from Boisvert suggests this increase ranges from 25 to 32% (5). The amount of sodium lost in the present study is consistent with the results reported by the few studies that exist on fluid and electrolyte losses in football. Cade et al. studied 10 members of a Florida team in a 2-h practice (7). Average sweat loss exceeded 2 L and sweat sodium ranged from 37 to 114 mmol/L. Total sodium losses during training reportedly ranged from 0.8 to 8.5 g (mean 4.1 ± 2.2 g per 2 h).

In our study, cramp-prone players and non-cramp-prone players sweated heavily. Despite less environmental heat in the morning, C tended to lose more sweat volume than NC. The trend we observed is supported by Jacobson et al. who reported that those college football players with heat illness lost more fluid weight during practice than asymptomatic teammates (12). Heat illness was defined as any heat or dehydration-related event—including cramping—that took the player out of practice and required medical attention.

Perhaps subjects in C were less able to regulate sweat rate, had a lower sweating threshold, or were chronically hotter than counterparts in the NC group. Any of these factors could contribute to a higher sweat rate, but need further investigation as a possible explanation for greater sweat loss and fluid deficit by C during the morning practice. High sweat rates and high sweat sodium levels seem to be markers of players predisposed to heat cramps, but the mechanism behind this has yet to be determined.

Some argue that an acute sodium deficit induced by sweating and/or dehydration plays no role in heat cramping because studies of marathoners, those who cramp versus those who do not, show no difference in serum sodium or percent dehydration (13, 16). These field studies, however, focus on exercise-induced muscle cramps, an etiology that could differ from heat cramping. One study, however, showed a trend toward slightly lower serum sodium in C (16). We agree with those who argue that a deficit in total body exchangeable sodium often cannot be detected simply by measuring serum sodium (4). In separate observations (different subjects), we found that among six players with severe heat cramping, mean serum sodium was 139 mmol/L (unpublished data). Since serum or plasma sodium changes as if being distributed within total body water (15), a small reduction in serum sodium from 142 to 139 could remain within the limits for normal and yet translate into a substantial

loss in the total body exchangeable sodium. Fowkes-Godek et al. confirm this possibility with their observation that blood sodium levels decreased significantly (from 140 mmol/L to 136 mmol/L) in the face of a reduction in plasma volume (10). Changes in serum sodium levels—adjusting for urine sodium excretion—and sweat sodium loss in heat crampers requires further investigation.

The concept that heavy losses of sodium in sweat combined with a low dietary intake underlies heat cramping is widespread and longstanding, but has been more anecdotal than measured under controlled conditions. After learning that ocean-liner stokers mixed seawater with their drinking water, a London physician prevented heat cramps in industrial stokers via a saline drink (17). Similar remedies were suggested by the US Army (14) and by researchers in the Harvard Fatigue Lab, studying heat cramping in Hoover Dam construction workers (24). Deep in British coal mines, where in one shift a man could sweat out "20 grams of salt," cramps were reduced by "salt in water, about the composition of sweat" (6). Also supporting sodium depletion as a significant factor is that physicians and athletic trainers working with football, in medical tents of triathlons, and with top tennis players report that severe, full-body heat cramps are rapidly alleviated with intravenous saline. Experts continue to report this as of 2003 (23), just as they did 70 y earlier. The present study is the first, to our knowledge, to report under as well-controlled conditions as possible for a field setting, that high sweat sodium concentration (sodium loss) is associated with football players who have exhibited a history of heat cramps. That stated, more work is needed to confirm this relationship and ultimately explore cause and effect.

In our study, dietary and fluid sodium intakes were high in both groups, but cramp-prone players consumed more total sodium from fluids and diet combined compared to non-cramp-prone players (14.9 g vs. 9.0 g). This behavior was consequent to players learning from trainers and team physicians that high sodium intake might help prevent cramping. A portion of this behavior might also be related to development of sodium appetite, a naturally occurring phenomenon in humans with shifts in extracellular sodium concentration. A fair question is whether dietary sodium intake drives sweat sodium level. Some existing literature (1, 2, 11) hints at a relationship, although none of these studies were designed to test cause and effect. None of the studies compare to the present study in which both groups fell "near the high end" of total sodium intake; non-crampers at ~ 9 g versus crampers at ~ 15 g of sodium per day. This "difference at the high end" in sodium intake could account for some but not most of the two-fold higher sweat sodium in crampers. Others have demonstrated no association between dietary sodium and sweat sodium level assessed using the whole-body washdown method in endurance-trained athletes (18). Two pilot studies on the subjects in the present study—in the 2 months preceding our main study—showed consistently higher sweat sodium levels for the cramp-prone players when there was less emphasis to drink sodium-rich fluids. Finally, a report on acute sodium loading in our lab showed no short-term effect on sweat sodium levels, so sodium-rich fluids consumed in the morning were not proportionately associated with sweat sodium concentrations measured in the evening (22). Seemingly, large fluxes in sweat sodium are not proportionate to slight increases or decreases in the tightly regulated serum sodium. Nonetheless, the influence of dietary sodium on sweat sodium merits further study and a mechanistic explanation.

The sweat-induced body weight deficit in our study was minimal, but the groups studied did not appear well hydrated in agreement with the USG values obtained prior to practices. With euhydration, the average observed level of 1 to 1.5% fluid deficit might not be enough to critically impair physical performance, based on laboratory studies (25). However, a 1 to 1.5% fluid deficit in addition to preexisting hypohydration could impair performance with gross sweat losses, and thus gross salt losses. On the team studied here, good hydration (and salt use) was emphasized because large fluid and sodium losses during football practice in the heat must be replaced to keep athletes functional—maintaining the extracellular fluid space.

What remains to be determined is whether the large losses of sodium in the sweat of cramp-prone players lead to a negative sodium balance that precipitates the cramping episode. As noted, none of the players in the present study experienced a cramping episode while being studied during the early phase of two-a-day training when sodium intake appeared to be high, the players ate food provided at the training table, and the sports medicine staff was diligent in ensuring players were getting adequate sodium. Yet, during the course of the season following the study, the cramping group in total experienced 10 episodes of heat-related whole-body cramping even with an emphasis on dietary sodium intake. Consistently high sodium losses in the sweat combined with urinary and fecal losses and an inadequate cumulative sodium intake over a relatively short time (i.e., a couple of days) could culminate in a sodium deficit that contributes to the cramping. Carefully designed studies that account for intake and all routes of sodium loss will be needed before this can be concluded. Moreover, that the diet could have this impact relatively quickly could be a reason for the unpredictable nature of cramping.

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