

Plasma Potassium Concentration and Content Changes After Banana Ingestion in Exercised Men

Kevin C. Miller, PhD, LAT, ATC

Department of Health, Nutrition, and Exercise Sciences, North Dakota State University, Fargo

Context: Individuals prone to exercise-associated muscle cramps (EAMCs) are instructed to eat bananas because of their high potassium (K^+) concentration and carbohydrate content and the perception that K^+ imbalances and fatigue contribute to the genesis of EAMCs. No data exist about the effect of bananas on plasma K^+ concentration ($[K^+]_p$) or plasma glucose concentration ($[glucose]_p$) after exercise in the heat.

Objective: To determine whether ingesting 0, 1, or 2 servings of bananas after 60 minutes of moderate to vigorous exercise in the heat alters $[K^+]_p$ or $[glucose]_p$ and whether changes in $[K^+]_p$ result from hypotonic fluid effluxes or K^+ ion changes.

Design: Crossover study.

Setting: Laboratory.

Patients or Other Participants: Nine euhydrated men (age = 27 ± 4 years, height = 180.3 ± 8.4 cm, mass = 84.9 ± 26.1 kg, urine specific gravity ≤ 1.006) without EAMCs volunteered.

Intervention(s): On 3 separate days, participants completed 60 minutes of moderate to vigorous cycling (temperature = $36.4^\circ\text{C} \pm 1.1^\circ\text{C}$, relative humidity = $19.4\% \pm 2.5\%$) and then ate 0 g (0 servings), 150 g (1 serving), or 300 g (2 servings) of bananas. Blood samples were collected at –3, 5, 15, 30, and 60 minutes postingestion.

Main Outcome Measure(s): The $[K^+]_p$, changes in plasma K^+ content, plasma volume changes, and $[glucose]_p$.

Results: The $[K^+]_p$ differed between conditions at 60 minutes; 2 servings (4.6 ± 0.3 mmol/L [conventional unit = 4.6 ± 0.3 mEq/L]) was greater than 1 serving (4.5 ± 0.2 mmol/L [conventional unit = 4.5 ± 0.2 mEq/L]) and 0 servings (4.4 ± 0.3 mmol/L [conventional unit = 4.4 ± 0.3 mEq/L]) ($P < .05$). The $[K^+]_p$ was greater at 60 minutes than at –3 and 5 minutes in the 1-serving condition and was greater at 30 and 60 minutes than at –3 and 5 minutes in the 2-servings condition ($P < .05$). Percentage change in K^+ content was greater only at 30 and 60 minutes postingestion than at baseline in the 2-servings condition ($4.4\% \pm 3.7\%$ and $5.8\% \pm 2.3\%$ increase, respectively) ($P < .05$). The plasma volume changes among conditions were unremarkable. The $[glucose]_p$ was greater in the 2-servings condition than in all other conditions at 15, 30, and 60 minutes ($P < .05$).

Conclusions: The effect of banana ingestion on EAMCs is unknown; however, these data suggested bananas are unlikely to relieve EAMCs by increasing extracellular $[K^+]_p$ or $[glucose]_p$. The increases in $[K^+]_p$ were marginal and within normal clinical values. The changes in $[K^+]_p$, plasma K^+ content, and $[glucose]_p$ do not occur quickly enough to treat acute EAMCs, especially if they develop near the end of competition.

Key Words: electrolytes, fruit, glucose, muscle cramps

Key Points

- Eating up to 2 servings of bananas caused marginal increases in plasma potassium concentration.
- The small increases in plasma potassium concentration occurred 30 to 60 minutes postingestion of bananas.
- Eating bananas is unlikely to be an effective treatment for exercise-associated muscle cramping.

Despite estimates by some researchers that exercise-associated muscle cramps (EAMCs) affect up to 95% of the general population,¹ their cause remains unknown. The dehydration/electrolyte imbalance theory is a popular explanation for the cause of EAMCs² and postulates that fluid and ion shifts from the extracellular space result in EAMCs.³ Whereas proponents of this theory traditionally have focused on sodium (Na^+) losses and EAMCs,^{4,5} potassium (K^+) imbalances (eg, hypokalemia, hyperkalemia) also have been listed as possible contributors to the genesis of EAMCs.^{6,7}

In several quasi-experimental studies, investigators have shown no differences in plasma K^+ concentration ($[K^+]_p$) between athletes with and without EAMCs.^{8–10} Given that these researchers^{8–10} often compared hematologic characteristics between crampers and noncrampers postexercise, a potential limitation is how quickly postexercise blood sampling was performed, because $[K^+]_p$ can return to

normal levels within 5 minutes postexercise.^{11,12} The electrolyte concentrations in the extracellular and intracellular compartments during an EAMC are unknown. Whereas K^+ involvement in the genesis of EAMCs is unclear, my clinical experience has been that some coaches, health care professionals, and lay community members believe that eating bananas is an effective treatment for EAMCs because of their high K^+ content and because they believe that electrolyte losses contribute to the genesis of EAMCs. No researchers have examined $[K^+]_p$ or plasma K^+ content after the ingestion of varying quantities of bananas after exercise in the heat.

Not knowing how much bananas alter $[K^+]_p$ may have important clinical implications. Eating large quantities of bananas while the extracellular fluid compartment is hypertonic may result in modest hyperkalemia. Based on animal studies and in situ heart preparations, modest hyperkalemia ($6\text{--}7$ mmol/L [conventional unit = $6\text{--}7$

mEq/L)) may cause cardiac abnormalities, such as QT-wave shortening,¹³ but little or no clinical evidence of hyperkalemia preceding cardiac arrhythmias has been demonstrated in humans.¹⁴ High extracellular $[K^+]_p$ also is thought to contribute to premature muscle fatigue.¹⁵ If EAMCs result from fatigue-induced changes in muscle afferent activity as speculated,¹⁶ eating bananas may expedite fatigue and increase the risk of EAMCs. Therefore, elucidating the effects of eating varying quantities of bananas on $[K^+]_p$ and plasma glucose concentration ($[glucose]_p$) postexercise is necessary.

The purpose of my study was to determine whether eating 0, 1, or 2 servings of bananas after 60 minutes of moderate to vigorous exercise in the heat altered $[K^+]_p$ or $[glucose]_p$ and whether changes in $[K^+]_p$ resulted from hypotonic fluid effluxes or K^+ ion changes. Understanding when and if these blood variables change after banana ingestion has important clinical implications for whether bananas could be used to restore K^+ or $[glucose]_p$ during exercise or postexercise. I hypothesized that $[K^+]_p$ would not increase after banana ingestion. Moreover, I expected that $[glucose]_p$ would increase after banana ingestion, but 30 minutes would lapse before appreciable changes would be observed.

METHODS

Participants

I recruited a convenience sample of 9 healthy men to participate in this study (age = 27 ± 4 years, height = 180.3 ± 8.4 cm, mass = 84.9 ± 26.1 kg). Volunteers were excluded from participating if they (1) had experienced any upper extremity injury or undergone surgery within the 6 months before the study; (2) self-reported any neurologic, cardiovascular, or blood-borne diseases; (3) had a known food allergy to bananas; or (4) had a history of heat syncope, heat exhaustion, or heat stroke during exercise or postexercise in their lifetimes. Participants provided written consent, and the study was approved by the North Dakota State University Institutional Review Board.

Experimental Design

A 3×5 factorial, crossover design with repeated measures on time guided data collection for blood dependent variables. The independent variables were servings of bananas (Dole Food Co, West Lake Village, CA) ingested (0, 1 [150 g], or 2 servings [300 g] of sliced, ripened bananas) and time (–3, 5, 15, 30, and 60 minutes postingestion). From a practical standpoint, the amounts of bananas ingested in the 1-serving and 2-servings conditions were approximately 1.5 and 3 medium-sized (22.2 cm in length) bananas, respectively. These amounts were chosen based on my clinical experience that athletes typically eat 1 to 2 bananas during competitions if they develop EAMCs (high external validity) and to minimize the amount of time required to ingest the bananas. The blood dependent variables were $[K^+]_p$ in millimoles per liter, hematocrit in percentage of red cell volume, hemoglobin concentration in grams per deciliter, and $[glucose]_p$ in millimoles per liter.

Testing Procedures

Participants were instructed to drink water consistently during the day before testing, avoid strenuous exercise for 24 hours before testing, and fast for 12 hours before testing. Although they did not keep a diet log, participants were instructed to maintain a consistent dietary regimen over the course of the study. Compliance with these instructions was determined by self-report before each testing session.

Participants reported to the laboratory on 3 different days. On each testing day, participants voided their bladders completely, and urine specific gravity was assessed via refractometry (model SUR-Ne; Atago USA Inc, Bellevue, WA) to determine if they were euhydrated (urine specific gravity ≤ 1.02).¹⁷ If euhydrated, participants were weighed in socks, shorts, and undergarments. If hypohydrated, participants ingested 3 mL/kg of body mass of tap water, and specific gravity was reassessed 30 minutes later. After weight measurement, participants donned a heart rate monitor (Polar Electro, Inc, Lake Success, NY).

They entered an environmental chamber (temperature = $36.4^\circ\text{C} \pm 1.1^\circ\text{C}$, relative humidity = $19.4\% \pm 2.5\%$) and began a 60-minute bout of exercise on a semirecumbent cycle ergometer (model 846i; Precor, Woodinville, MA). Participants exercised at a heart rate from 145 to 155 beats per minute. Posttesting analysis using the participants' ages and heart rate ranges during exercise indicated the intensity of exercise was moderate to vigorous for 7 participants and vigorous for the other 2 participants.¹⁸ I monitored exercise intensity at 10-minute intervals.

Upon completion of the 60 minutes of exercise, participants biked at a self-selected lower intensity for 5 minutes to cool down. They exited the environmental chamber, towel dried themselves, removed the heart rate monitor, and were weighed. Next, they sat upright in a chair in a climate-controlled room (temperature = 23°C , relative humidity = 16%) with one of their upper extremities resting on a padded treatment table for 30 minutes to allow their body fluid compartments to equilibrate. Participants stayed in this position for the remainder of the study and were instructed to minimize movement for the rest of the testing session. During this equilibration period, the cubital fossa of the upper extremity resting on the table was cleaned with isopropyl alcohol, and a sterile, 20-gauge venous catheter was inserted into a superficial forearm vein. After the 30-minute equilibration period, a 5-mL blood sample was collected (–3-minute sample).

Bananas were sliced and weighed (BL3831; Taylor Precision Products, Oak Brook, IL) before ingestion. The nutritional content of raw, sliced bananas was determined from a food composition table.¹⁹ According to this table, a single serving (150 g) of raw, sliced bananas contains 594 mg (15.2 mmol) of K^+ , 111 g (74%) of water, 138 calories, 2 g of protein, 35 g (23%) of carbohydrates, 4 g of dietary fiber, 1 g of fat, and 1 mg (0.043 mmol) of Na^+ .

Participants were given 3 minutes to eat 0, 1, or 2 servings of bananas. The amount of bananas ingested was counterbalanced with a Latin square a priori, and participants randomly chose a testing order on the first day of testing. No fluids were given to help with banana ingestion or after exercise had been initiated until completion of testing. At 5, 15, 30, and 60 minutes postingestion, 5-mL blood samples were collected. After the last blood sample was collected, the catheter assembly

was removed, and participants were excused. They were instructed to report at least 48 hours later at the same time of day for the next testing session.

Blood Analysis Procedures

Whole blood was analyzed for hematocrit and hemoglobin concentration. Blood for hematocrit analysis was drawn into heparinized microcapillary tubes, centrifuged at 3000 rpm (model IEC Micro-MB; International Equipment Co, Needham Heights, MA) for 5 minutes, and read using a microcapillary reader (model IEC 2201; Damon/IEC, Needham Heights, MA). Hemoglobin concentration was measured by mixing 20 μ L of whole blood with 5 mL of cyanmethemoglobin reagent, and the absorbance was read at 540 nm on a standard spectrophotometer (iMark Spectrophotometer; Biorad, Hercules, CA). Hematocrit and hemoglobin concentration were measured in triplicate immediately after sampling and averaged for each blood sample for statistical analysis and calculations. Any remaining blood not used for hematocrit and hemoglobin concentration was stored on ice until centrifugation.

After collection of the 60-minute blood sample, the remaining blood was centrifuged at 3000 rpm for 15 minutes at 3°C (model 5804R; Eppendorf North America, Inc, New York, NY). Plasma was removed and frozen in an ultralow (−80°C) freezer and later thawed by placing the cuvettes into a 37°C water bath for 5 minutes. The cuvettes were centrifuged for 5 minutes and analyzed in duplicate for $[K^+]_p$ and $[glucose]_p$ with an ion-selective electrode analyzer (NOVA 16; Nova Biomedical, Waltham, MA). Laboratory equipment restrictions precluded our ability to measure total body K^+ content. Thus, K^+ content was estimated using plasma $[K^+]$, hematocrit, and hemoglobin concentration data and the equation of Greenleaf et al²⁰; data are reported as percentage change in K^+ content. Hematocrit and hemoglobin concentration data were inserted into the equation of Dill and Costill²¹ to estimate percentage change in plasma volume.

Statistical Analysis

Separate 2-way repeated-measures analyses of variance (ANOVAs) were used to determine differences in plasma variables among banana conditions over time. A 1-way repeated-measures ANOVA was used to analyze urine specific gravity before testing and percentage of hypohydration postexercise. Tukey-Kramer multiple comparison tests were used when I found F values that were different. The α level was set at .05. I used NCSS (version 2007; NCSS, Kaysville, UT) to analyze the data.

RESULTS

Data are reported as means $\pm$ SDs. Participants self-reported compliance with all pretesting instructions on each testing day and were euhydrated similarly before testing (0 servings = 1.006 ± 0.005 , 1 serving = 1.005 ± 0.005 , 2 servings = 1.005 ± 0.003) ($F_{2,16} = 0.1$, $P = .94$). Participants were hypohydrated similarly postexercise each day (0 servings = $1.29\% \pm 0.49\%$, 1 serving = $1.30\% \pm 0.54\%$, 2 servings = $1.33\% \pm 0.59\%$) ($F_{2,16} = 0.2$, $P = .86$). Participants ingested 0 ± 0 g, 150.6 ± 2.4 g, and $300.8 \pm$

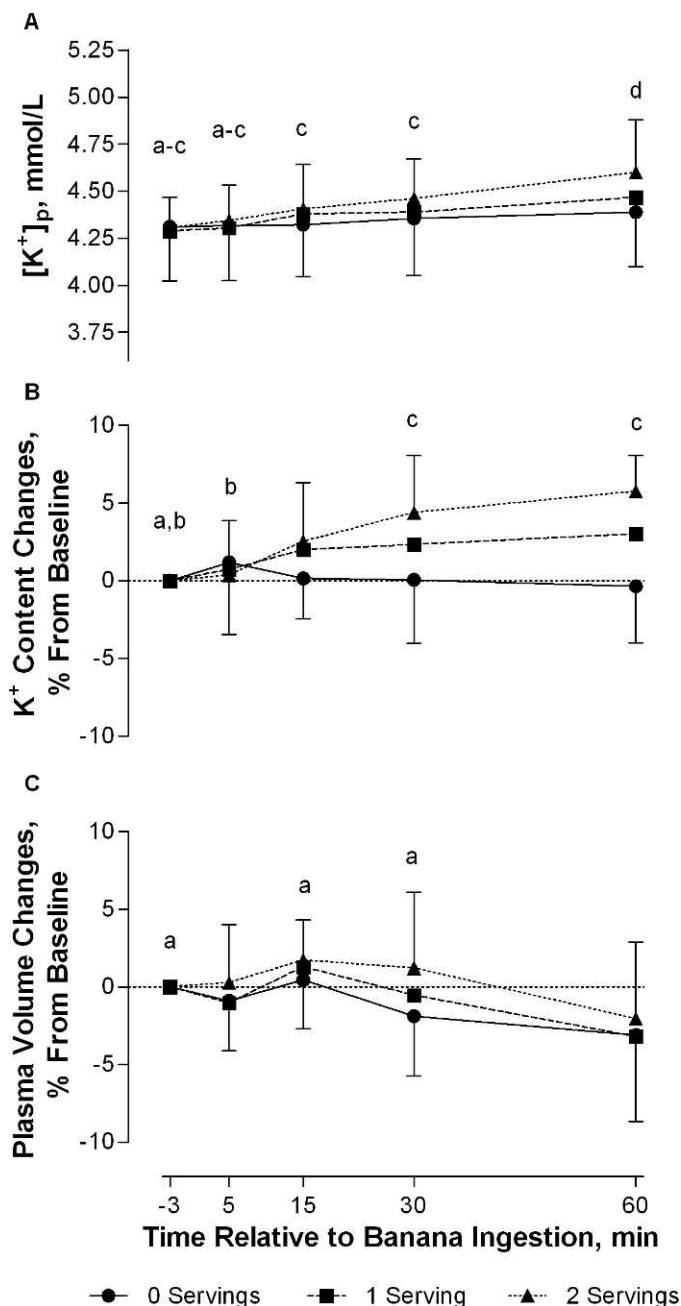


Figure 1. A, Plasma potassium concentration ($[K^+]_p$) over 60 minutes postingestion of 0, 1, or 2 servings of bananas (mean $\pm$ SD). ^a Indicates 1 serving < 1 serving at 60 minutes. ^b Indicates 2 servings < 2 servings at 30 minutes. ^c Indicates 2 servings < 2 servings at 60 minutes. ^d Indicates 0 servings and 1 serving < 2 servings. B, Percentage change in plasma potassium (K^+) content from baseline over 60 minutes postingestion of 0, 1, or 2 servings of bananas (mean $\pm$ SD). ^a Indicates 2 servings < 2 servings at 30 minutes. ^b Indicates 2 servings < 2 servings at 60 minutes. ^c Indicates 2 servings > 0 servings. C, Plasma volume changes over 60 minutes postingestion of 0, 1, or 2 servings of bananas (mean $\pm$ SD). ^a Indicates greater than 60 minutes. One and 2 servings of bananas are equivalent to 150 and 300 g of bananas, respectively.

1.1 g of bananas in the 0, 1, and 2 servings of banana conditions, respectively.

Plasma K^+ concentration differed among conditions over time ($F_{8,64} = 3.6$, $P = .002$) (Figure 1). The only difference in $[K^+]_p$ among conditions occurred at 60 minutes post-ingestion; $[K^+]_p$ was greater in the 2-servings condition

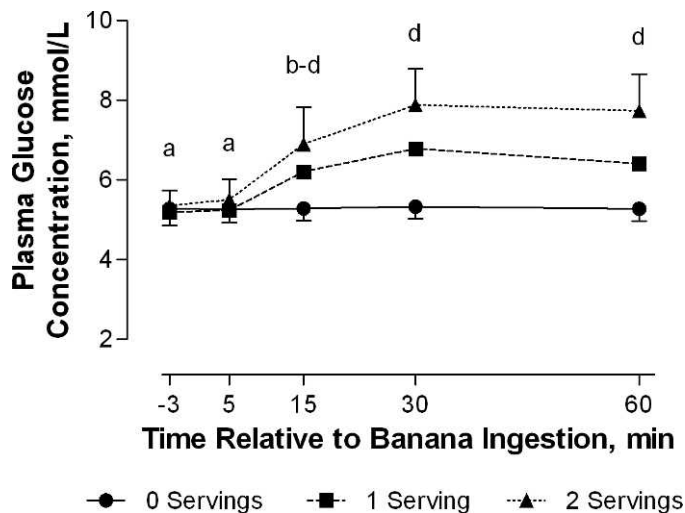


Figure 2. Plasma glucose concentration over 60 minutes post-ingestion of 0, 1, or 2 servings of bananas (mean $\pm$ SD). ^a Indicates < 15, 30, and 60 minutes within 1 and 2 servings. ^b Indicates 1 serving < 1 serving at 30 minutes. ^c Indicates 2 servings < 2 servings at 30 and 60 minutes. ^d Indicates 0 servings < 1 serving < 2 servings. One and 2 servings of bananas are equivalent to 150 g and 300 g of bananas, respectively.

than in the 1-serving and 0-servings conditions ($P < .05$). Although no changes occurred to $[K^+]_p$ in the 0-servings condition over 60 minutes ($P > .05$), $[K^+]_p$ increased in the 1-serving and 2-servings conditions. In the 1-serving condition, $[K^+]_p$ was lower at -3 and 5 minutes than at 60 minutes post-ingestion ($P < .05$). In the 2-servings condition, $[K^+]_p$ was lower at -3 and 5 minutes than at 30 and 60 minutes post-ingestion ($P < .05$). In addition, $[K^+]_p$ was greater at 60 minutes post-ingestion than at 15 and 30 minutes post-ingestion in the 2-servings condition ($P < .05$).

Percentage change in plasma K^+ content differed among conditions over time ($F_{8,64} = 3.2$, $P = .004$) (Figure 1). Percentage change in plasma K^+ content remained unaltered for the duration of testing in the 0-servings and 1-serving conditions, and no differences were observed between these conditions at any time ($P > .05$). However, percentage change in plasma K^+ content at 30 and 60 minutes was greater in the 2-servings condition than in the 0-servings condition ($P < .05$). Within the 2-servings condition, percentage change in plasma K^+ content was greater at 30 and 60 minutes than at -3 minutes ($P < .05$). In addition, within the 2-servings condition, percentage change in plasma K^+ content was lower at 5 minutes than at 60 minutes ($P < .05$).

For percentage change in plasma volume, I observed no interaction between servings of bananas and time ($F_{8,64} = 0.4$, $P = .94$) or among servings of bananas ($F_{2,16} = 1.2$, $P = .33$). However, plasma volume changed over time ($F_{4,32} = 6.4$, $P < .001$) (Figure 1). Plasma volume was higher at -3, 15, and 30 minutes post-ingestion than at 60 minutes post-ingestion ($P < .05$).

Plasma glucose concentrations differed between serving conditions and time ($F_{8,64} = 29.7$, $P < .001$) (Figure 2). Plasma glucose concentration was greater in the 1-serving and 2-servings condition than in the 0-servings condition at 15, 30, and 60 minutes ($P < .05$). Similarly, $[glucose]_p$ was greater in the 2-servings condition than in the 1-serving condition at 15, 30, and 60 minutes ($P < .05$). No changes

in $[glucose]_p$ occurred in the 0-servings condition over 60 minutes ($P > .05$). The $[glucose]_p$ in the 1-serving and 2-servings conditions was greater at 15, 30, and 60 minutes than at -3 and 5 minutes. It also increased from 15 to 30 minutes in the 1-serving condition ($P < .05$), whereas in the 2-servings condition, $[glucose]_p$ was lower at 15 minutes than at 30 and 60 minutes post-ingestion ($P < .05$).

DISCUSSION

The most important observation was that eating 1 or 2 servings of bananas increased $[K^+]_p$. Although differences were observed, the increases in $[K^+]_p$ are unlikely to be clinically important. For example, the highest $[K^+]_p$ occurred in the 2-servings condition, yet these increases were marginal: 0.16 mmol/L (conventional unit = 0.16 mEq/L) increase at 30 minutes and 0.3 mmol/L (conventional unit = 0.3 mEq/L) increase at 60 minutes post-ingestion. Moreover, the $[K^+]_p$ values were well within the normal clinical range (ie, 3.8–5 mmol/L [conventional unit = 3.8–5 mEq/L])²² and substantially lower than those $[K^+]_p$ values thought to cause cardiac abnormalities or performance deficits (ie, 6–10 mmol/L [conventional unit = 6–10 mEq/L]). These small increases in $[K^+]_p$ occurred despite 15 to 30 mmol of K^+ being delivered to the gut in the 1-serving and 2-servings conditions, respectively. Thus, most of the ingested K^+ appears to have been excreted in either the urine or feces. If EAMCs are due to K^+ imbalances, the marginal increases in $[K^+]_p$ that I observed provide strong evidence against the anecdote that eating bananas could relieve EAMCs by affecting an athlete's intravascular K^+ content.

It is known that $[K^+]_p$ increases substantially after moderate-intensity to high-intensity exercise because of K^+ release from contracting muscle.¹² At exercise intensities similar to those I used, Vollestad et al¹² observed that $[K^+]_p$ increased from 4.4 ± 0.2 mmol/L (conventional unit = 4.4 ± 0.2 mEq/L) to 6.4 ± 0.2 mmol/L (conventional unit = 6.4 ± 0.2 mEq/L) shortly after exercise began (1.5 minutes) and remained elevated until exercise ceased. However, the increases in $[K^+]_p$ in my study cannot be explained by muscle-contraction-induced K^+ effluxes, because arterial and venous $[K^+]_p$ quickly return to or fall below resting levels within 5 minutes of exercise cessation.^{11,12} This rapid return to resting $[K^+]_p$ can be attributed to the high activity of $[Na^+]-[K^+]-ATPase$ pumps and to erythrocyte and noncontractile tissue uptake of K^+ .^{14,23} In my study, participants rested for 30 minutes postexercise, thereby allowing $[K^+]_p$ to return to basal levels before eating bananas. The $[K^+]_p$ at -3 minutes in each condition confirmed that $[K^+]_p$ had returned to normal resting values (approximately 4.3 mmol/L [conventional unit = 4.3 mEq/L]). Thus, the changes in $[K^+]_p$ are best explained by banana ingestion. However, the increases in $[K^+]_p$ between the 1-serving and 2-servings conditions are due to different causes. The small increase in $[K^+]_p$ that occurred in the 1-serving condition at 60 minutes post-ingestion can be attributed primarily to an efflux of hypotonic fluid out of the intravascular space (3.6% reduction in plasma volume), because percentage change in K^+ content did not increase over 60 minutes. Mitchell et al²⁴ observed similar decreases (approximately 2.5%) in plasma volume at 15, 30, and 45 minutes post-ingestion of

bananas in rested participants. In contrast, the increase in $[K^+]_p$ that occurred in the 2-servings condition was due primarily to an increase in K^+ ions in the intravascular space rather than changes in plasma volume. At 30 and 60 minutes postingestion, percentage change in plasma K^+ content increased 4.4% and 5.8% from baseline, respectively. At these same time intervals, plasma volume increased 1.2% at 30 minutes and decreased 2% from baseline at 60 minutes. Therefore, if athletes want to increase $[K^+]_p$, they must eat at least 2 servings of bananas.

The timing of the alterations in $[K^+]_p$ and plasma K^+ content after banana ingestion also has not been described. I observed that $[K^+]_p$ did not increase until 60 minutes postingestion when 1 serving of bananas was ingested and 30 minutes postingestion when 2 servings were ingested. Thus, athletes wanting to increase $[K^+]_p$ as quickly as possible need to ingest at least 2 servings of bananas and wait at least 30 minutes before any substantive increase is observed. Delayed gastric emptying likely explains the time course of changes in $[K^+]_p$ and K^+ content. Whereas no researchers explicitly have examined the gastric emptying of bananas, several authors have confirmed delays in gastric emptying because the ingested bolus was solid rather than liquid,²⁵ contained dietary fiber,²⁶ or had a large [glucose].²⁷ Bananas fit all of these criteria.

The delay in changes to $[K^+]_p$ has clinical implications for health care professionals attempting to treat athletes experiencing EAMCs by having them eat bananas. Because many athletes develop EAMCs near the end of competitions (eg, beginning of fourth quarter of an American football game, end of marathon),²⁸ a competition may be over or the athlete would lose considerable time if he or she waited 30 minutes for $[K^+]_p$ to increase. Moreover, our participants rested for 30 minutes before eating the bananas; thus, blood flow to the gut, and therefore digestion, presumably had enough time to return to normal. Because exercise at moderate intensities (65%–85% of maximal oxygen consumption) delays gastric emptying,^{29–32} the increases in $[K^+]_p$ may be delayed more if the athlete resumes competition shortly after eating bananas. These facts make it even more unlikely that bananas are an effective strategy for relieving acute EAMCs by altering $[K^+]_p$.

Whereas eating bananas is unlikely to relieve EAMCs by altering extracellular $[K^+]_p$, bananas may help prevent their recurrence by increasing [glucose]_p. Some scientists have hypothesized that EAMCs are caused by fatigue-induced changes in the muscle.¹⁶ It has long been established that ingesting carbohydrates or glucose prolongs exercise duration and performance.^{33,34} Murdoch et al³⁵ observed similar results after banana ingestion. In their study, participants cycled or ran at 70% maximal oxygen consumption for 180 minutes. Next, they ingested a placebo or 3 slurried or solid bananas. Finally, they cycled at 70% of maximal oxygen consumption until volitional exhaustion. The authors noted that eating bananas improved time to exhaustion by 16 and 18 minutes for the slurried and solid banana conditions, respectively. Moreover, they observed that eating solid bananas increased [glucose]_p by 1.1 mmol/L (conventional unit = 19.82 mg/dL) after 15 minutes and that [glucose]_p was maintained in the exhaustion bout of exercise (postingestion of banana = 4.7 mmol/L [conventional unit = 84.68 mg/dL], post-exhaustive exercise = 4.6 mmol/L [conventional unit =

82.88 mg/dL]) compared with the placebo (postingestion of placebo = 4.3 mmol/L [conventional unit = 77.48 mg/dL], postexhaustive exercise = 3.5 mmol/L [conventional unit = 63.06 mg/dL]).³⁵ I observed similar increases in [glucose]_p at 15 minutes. In the 1-serving and 2-servings conditions, [glucose]_p was 1.0 mmol/L (conventional unit = 18.02 mg/dL) and 1.5 mmol/L (conventional unit = 27.03 mg/dL) greater than baseline, respectively. Moreover, [glucose]_p was maintained for the duration of testing. As with $[K^+]_p$, the increases in [glucose]_p occurred too slowly to immediately affect acute EAMCs. However, [glucose]_p increased 15 minutes postingestion in the 1-serving and 2-servings conditions. If an athlete with EAMCs ingested bananas when he or she had a longer period before re-entering competition (eg, halftime in American football), he or she could expect a modest increase in [glucose]_p.

Other strategies for increasing [glucose]_p include ingesting a carbohydrate-electrolyte beverage (ie, sports drink), which has been shown to increase [glucose]_p within 30 minutes of ingestion during cycling in the heat.³⁶ Thus, eating bananas may help prevent the recurrence of EAMCs caused by fatigue. Whereas this hypothesis is compelling, authors of 2 cohort studies in triathletes¹⁰ and distance runners⁹ observed no differences in [glucose]_p between crampers and noncrampers. However, other authors³⁷ have observed that the onset of EAMCs can be delayed from 14.6 ± 5 minutes to 36.8 ± 17.3 minutes by consuming a carbohydrate-electrolyte beverage rather than no fluid. Given their findings, I infer that the exogenous carbohydrate ingested was likely the cause of the delay in EAMC onset, because the sweat rates of participants were 1.8 L/h and EAMCs occurred after 14 minutes (420 mL of total fluid lost). Thus, large fluid, and presumably electrolyte, losses were not incurred. Indeed, participants only lost approximately 1% of body mass because of testing.³⁷ In the future, researchers should examine whether bananas can delay the onset of EAMCs or prevent their occurrence and should examine other potential mechanisms by which bananas may prevent or treat EAMCs.

tions of my study. Most K^+ ions are located intracellularly rather than extracellularly. Thus, the higher $[K^+]_p$ observed after banana ingestion possibly was due to decreased K^+ uptake by the $[Na^+]-[K^+]$ -ATPase pumps into the cells after the body had reached an equilibrium. However, this scenario is unlikely. For K^+ reuptake into the cell to occur, the equilibrium potential of K^+ must become less negative than the muscle's resting membrane potential (eg, -85 mV).²² If the baseline $[K^+]_p$ data from the 1-serving and 2-servings conditions are inserted into the Nernst equation and an intracellular $[K^+]$ of 160 mmol/L (conventional unit = 160 mEq/L) is assumed,³⁸ the equilibrium potential of K^+ would have been approximately -96.7 mV before banana ingestion in both conditions. Inserting the highest $[K^+]_p$ values from the 1-serving and 2-servings conditions indicates that the equilibrium potential of K^+ would have increased by 1.0 mV and 1.7 mV, respectively. These negligible changes in the equilibrium potential of K^+ indicate that the predominant driving force for K^+ would have remained toward the outside of the cell, because the equilibrium potential still would be lower than the resting membrane potential of the muscle. Therefore, it may be assumed that passive diffusion continued to be the predominant force guiding K^+ movement inside and outside of the cell and that the increases in

extracellular K^+ content reflect new K^+ ions from the breakdown of the ingested bananas.

The first potential limitation of my study was that the amount of bananas given was not tailored for each participant by body mass or resting metabolic rate. I did this to maximize the external validity of the study. Health care professionals are not likely to know the metabolic rates of the individuals to whom they give bananas after EAMC occurrence. Rather, in my clinical experience, health care professionals provide bananas according to serving size or without any conscious predetermination. These observations guided the design of my experiment. Therefore, some internal validity was sacrificed in this study, and the reader should interpret these data in light of this fact. The second limitation was that $[K^+]_p$ was studied in men who had not experienced acute EAMCs. In several studies, researchers^{8–10} have observed that $[K^+]_p$ does not differ between athletes who do and do not develop EAMC before or after competition. Therefore, studying participants who had not experienced EAMCs is unlikely to have negatively affected the results or conclusions of my investigation. However, future study on the effects of banana ingestion after EAMCs is needed.

CONCLUSIONS

Eating 2 servings of bananas increased $[K^+]_p$ at 30 and 60 minutes postingestion. Dangerous levels of hyperkalemia did not occur, and because all $[K^+]_p$ were within normal limits, banana ingestion postexercise likely did not affect performance or health. The increase in $[K^+]_p$ after ingestion of 2 servings of bananas is caused primarily by changes in extracellular K^+ content. In contrast, the increase in $[K^+]_p$ occurring at 60 minutes postingestion in the 1-serving condition primarily is due to plasma volume effluxes. If bananas relieve EAMCs, it is unlikely because of changes in K^+ for 2 reasons: (1) although different, the increases in $[K^+]_p$ were marginal and well within normal clinical values; and (2) the changes in $[K^+]_p$ and plasma K^+ content do not occur quickly enough to help athletes in many sports of short to moderate duration, especially if athletes develop EAMCs toward the end of competition. However, if the athlete has 15 minutes before resuming competition and fatigue contributes to the genesis of EAMCs, the increase in $[glucose]_p$ that occurs after ingestion of 1 or 2 servings of banana may prevent the occurrence of EAMCs. In the future, researchers should examine this hypothesis and confirm these data in individuals experiencing acute EAMCs.

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Address correspondence to Kevin C. Miller, PhD, LAT, ATC, Department of Health, Nutrition, and Exercise Sciences, North Dakota State University, PO Box 6050, Department 2620, Fargo, ND 58108. Address e-mail to Kevin.C.Miller@ndsu.edu.