Caffeinated Sports Drink: Ergogenic Effects and Possible Mechanisms


This double-blind experiment examined the effects of a caffeinated sports drink during prolonged cycling in a warm environment. Sixteen highly trained cyclists completed 3 trials: placebo, carbohydrate-electrolyte sports drink (CES), and caffeinated sports drink (CES+CAF). Subjects cycled for 135 min, alternating between 60% and 75% VO\textsubscript{2max} every 15 min for the first 120 min, followed by a 15-min performance ride. Maximal voluntary (MVC) and electrically evoked contractile properties of the knee extensors were measured before and after cycling. Work completed during the performance ride was 15–23% greater for CES+CAF than for the other beverages. Ratings of perceived exertion were lower with CES+CAF than with placebo and CES. After cycling, the MVC strength loss was two-thirds less for CES+CAF than for the other beverages (5% vs. 15%). Data from the interpolated-twitch technique indicated that attenuated strength loss with CES+CAF was explained by reduced intrinsic muscle fatigue.

Key Words: exertion, exercise, endurance, carbohydrate, muscle strength, performance

Sports drinks are designed to optimize performance during and in the recovery from prolonged exercise by providing carbohydrate (CHO) energy and replacing fluid and electrolytes. Commercial sports drinks consist of water, varying types of CHO in concentrations of ~4–8%, small amounts of electrolytes, and sometimes other ingredients designed or thought to increase physical performance or health. Caffeine has generally not been included in sports drinks because of its purported diuretic effect and assumed potential for increased loss of body fluids (17), especially in hot climatic conditions in which hydration is a priority. There is, however, a
mounting body of scientific evidence indicating that caffeine improves performance (12) and does not increase fluid loss during prolonged exercise (1, 37), which has increased interest in caffeinated sports drinks.

There is uncertainty about whether adding caffeine to a commercial sports drink has an ergogenic effect greater than that of the sports drink. CHO coingested with caffeine might counteract the ergogenic effect of caffeine (36), because CHO might reduce caffeine’s putative glycogen-sparing effect (35) or its effect on perception of effort (13). Therefore, studies in which caffeine was ingested without CHO might not be generalizable to the situation when caffeine is added to a sports drink containing CHO. The results of studies that have evaluated whether caffeine coingested with CHO has performance-enhancing effects greater than that of CHO alone have been equivocal. Jacobson et al. (20) found that the time required to complete a standard amount of work after 120 min of exercise at 70% of maximal oxygen uptake ($VO_2\text{max}$) was not improved by ingesting caffeine with a liquid CHO meal 60 min before exercise compared with a CHO meal without caffeine. Hunter et al. (18) found that 100-km cycling performance was not improved by the addition of caffeine to a 7% CHO-electrolyte solution ingested 60 min before and at 15-min intervals during their time trial. In contrast, Kovacs et al. (26) found that adding 225 or 320 mg/L of caffeine to a 7% CHO-electrolyte solution ingested during the 20 min before and during a ~1-h cycling time trial improved the time to complete a standard amount of work by 4%. Likewise, Cox et al. (10) found that caffeine added to a 6% CHO-electrolyte solution improved performance by 3% on a 7 kJ/kg cycling time trial after 2 h of exercise at 70% $VO_2\text{max}$, regardless of whether the caffeine was administered before or during exercise. They also reported that Coca-Cola®, with a higher CHO concentration (13%) and lower caffeine concentration, consumed late in exercise (at minute 100) had the same performance-enhancing effect as the 6% CHO-electrolyte solution with caffeine. In a second experiment, they confirmed the 3% performance-enhancing effect of decarbonated Coke relative to decaffeinated Coke and found that the extra CHO in Coke enhanced performance by 1%, whereas the caffeine without extra CHO enhanced performance by 2%.

The purpose of this study was to compare the effectiveness of a commercial caffeinated sports drink with the market-leading commercial noncaffeinated sports drink and a flavored-water placebo control. It was hypothesized that compared with the noncaffeinated sports drink, the caffeinated sports drink would be as effective in providing energy and more effective in maintaining muscle strength and improving prolonged cycling performance.

**Methods**

**Subjects**

**Study A.** Sixteen highly trained male cyclists were recruited and studied at 2 independent laboratories (10 subjects at the University of Georgia and 6 at the Georgia Institute of Technology). This sample size is greater than the minimum number of subjects needed to detect a moderate (0.5 SD) difference in cycling performance using a 2-tailed t-test for repeated measures (27) with an experiment-wise $\alpha$ level of 0.05 and power of 0.8, assuming a correlation between repeated trials of 0.9
The subjects’ mean (± SD) physical characteristics were age 27.5 ± 7.0 y, height 177.0 ± 5.9 cm, body mass 72.7 ± 6.4 kg, percentage body fat via skinfold thicknesses 12.2% ± 4.6%, VO_{max} 60.5 ± 7.2 mL·kg⁻¹·min⁻¹, and cycling training 264 ± 125 km/wk. All subjects habitually ingested caffeine. Caffeine consumption averaged 150 ± 113 mg/d but ranged from 9 to 482 mg/d. Approvals of the study design and consent forms were granted by the University of Georgia and Georgia Tech institutional review boards. All subjects provided written informed consent and were paid for their participation.

**Study B.** Five subjects, 4 of whom had participated in Study A at Georgia Tech, participated in this follow-up study. The subjects’ mean (± SD) physical characteristics were age 37 ± 4 y, height 180.9 ± 5.5 cm, body mass 68.7 ± 4.5 kg, and VO_{max} 66.4 ± 6.0 mL·kg⁻¹·min⁻¹. Approvals of the study design and consent forms were granted by the Georgia Tech institutional review board, and all subjects provided written informed consent.

**Beverage Treatments**

Three experimental beverages were used: an artificially sweetened, flavored-water control (placebo); a commercially available 6% CHO-electrolyte sports drink (CES; Gatorade®, Quaker Oats Co., Barrington, IL), and a commercially available 7% CHO-electrolyte sports drink containing 195 mg/L caffeine; 1.92 g/L taurine; 46 mg/L carnitine; vitamins B3, B6, and B12; and sucralose (CES+CAF; Powerade Advance®, The Coca-Cola Company, Atlanta GA). Beverages were administered in uniform containers and identifiable only by numeric code to both the investigators and the subjects. The fluid-ingestion schedule was patterned after recommendations of the American College of Sports Medicine (8). Subjects ingested half of a preexercise bolus of 6 mL/kg body weight 10 min before exercise and the other half immediately before exercise. They also received 3 mL/kg at 15-min intervals over the first 105 min of exercise. The total volume of fluid ingested was 2132 ± 219 for CES+CAF, 2128 ± 214 for CES, and 2100 ± 226 for placebo. Total CHO ingested averaged 149 ± 15 g for CES+CAF, 127 ± 12 g for CES, and none for placebo. Caffeine ingestion during the CES+CAF trial was 1.2 mg/kg before exercise, 2.9 mg/kg by 60 min, and 5.3 mg/kg for the entire protocol.

**Research Design**

**Study A.** A double-blind, placebo-controlled, repeated-measures experimental design was used, with all subjects serving as their own controls. After a preliminary test session in which subjects were familiarized with procedures and VO_{max} was measured, 3 experimental trials were completed. Beverage-treatment order for the individual subjects was randomly assigned. A period of at least 5 d separated experimental trials. For a given experimental trial, 1 of 3 beverage treatments was administered throughout a 2-h variable-intensity cycling bout followed by a 15-min performance ride. All experimental trials took place at approximately the same time of day in an environmental chamber at 28.5 °C and 60% relative humidity with fan airflow of ~2.5 m/s. Both before and after cycling, maximal voluntary and electrically evoked contractile properties were measured under normal laboratory conditions (21 °C, 45% relative humidity).
**Study B.** Study B was a follow-up study designed to probe the mechanisms for the improved cycling performance and lesser strength loss observed with the CES+CAF beverage. The research design was the same as for Study A except that only the CES and CES+CAF beverages were used and both before and after cycling, interpolated-twitch procedures were conducted to probe central and peripheral muscle-fatigue mechanisms.

**Protocol and Procedures (Study A)**

**Preliminary Test Session.** During the preliminary test session, a graded cycling exercise test was conducted to measure VO2max. The protocol consisted of cycling on an electronically braked ergometer (Lode Excalibur Sport, Lode B.V., Groningen, The Netherlands) with the power output starting at 200 W and increasing progressively by 25 W every 2 min until the subject could no longer continue. Oxygen uptake (VO2) and related gas-exchange measures were obtained by open-circuit spirometry using a PARVO Medics TrueOne 2400 metabolic measurement system (Parvo Medics, Inc., Salt Lake City, UT). After a recovery of ~20 min, parts of the experimental protocol were practiced, and the workloads necessary to elicit the desired metabolic intensities (60% and 75% VO2max) were determined. The cycling portion of the practice session concluded with 15 min of cycling in which subjects performed as much work as possible to practice the cycling performance ride. The subjects were also familiarized with the measurements of maximal voluntary and electrically evoked contractile properties of the right knee-extensor muscles.

**Experimental Trials.** In preparation for the experimental trials, subjects were instructed to perform a similar training volume for the 3 d before each test and to avoid vigorous exercise for 24 h before testing. In addition, they were instructed to report to the laboratory after maintaining a standard mixed diet for 2 d before each test session. Food records were kept before the first experimental test session, and this diet was replicated for the 2-d period before subsequent trials. Subjects were instructed not to consume alcohol, caffeine, or nonprescription drugs the day before and on the day of testing. On arrival at the laboratory, subjects completed a 24-h history form to assess compliance with pretest instructions. Subjects reported in a normally hydrated state. Euhydration was accomplished by instructing subjects to drink liberally the day before and to drink one 237-mL glass of water 1 h before testing. Urine specific gravity was measured with a refractometer to ensure that urine specimens provided by subjects before testing were <1.021 (2). In almost all cases, subjects had fasted overnight before each experimental session. For 4 subjects, however, the postprandial state was standardized to be a minimum of at least 3 h after the ingestion of a small meal or CHO drink.

Before cycling, maximal voluntary and electrically evoked isometric contractile properties of the right knee-extensor muscles were measured. After the baseline strength measures, a Teflon® catheter was inserted into an antecubital vein. Subjects drank the first aliquot of the experimental test beverage, and the initial blood sample was drawn. Ten minutes later, the second aliquot of the beverage was ingested and subjects began the cycling protocol.

The subjects cycled continuously for a total of 135 min, alternating the exercise intensity between 60% and 75% VO2max every 15 min for the first 120 min.
During the last 15 min of cycling, the subjects were instructed to ride as hard as possible, simulating an extended all-out effort at the end of a cycling race. Total work (in kilojoules) performed during the final 15 min was used as a measure of performance. During the ride, $\text{VO}_2$ and respiratory-exchange ratio were measured during the last 2 min of every 15-min interval during the first 120 min and then continuously during the performance trial using the ParvoMedics metabolic system. Total-body CHO oxidation was calculated from $\text{VO}_2$ and the respiratory-exchange ratio. Rating of perceived exertion (RPE) was assessed using the Borg 15-point category scale every 5 min during cycling. After cycling, the subjects exited the testing chamber into a cool laboratory to recover. Maximal voluntary and electrically evoked contractile properties were remeasured, beginning exactly 20 min after cessation of cycling.

**Blood Measures.** Ten-milliliter blood samples were obtained before, every 30 min during, and immediately after cycling via an indwelling catheter kept patent with heparin-lock solution. Blood lactate and glucose concentrations were measured using a YSI 2300 Stat Plus automated lactate/glucose analyzer (Yellow Springs Instruments, Inc., Yellow Springs, OH). Serum caffeine concentration was determined for 10 subjects (from the UGA test site) using a homogeneous enzyme immunoassay technique using commercially available reagents (Emit Caffeine Assay, Dade-Behring Syva, Cupertino, CA). Blood measures were not corrected for plasma-volume change.

**Maximal Voluntary and Electrically Evoked Isometric Contractile Properties.** Isometric torque production of the right knee-extensor muscles was assessed during both maximal voluntary contractions (MVC) and electrically evoked contractions. These measurements were made using a modified leg-extension/curl machine (model NT-1260, Nautilus Fitness Products, Louisville, CO). The subject was placed in a semireclined seated position on the machine with hip- and knee-flexion angles set at 80° and 70°, respectively; these angles were determined from pilot work to be optimal for isometric torque production. The leg-extension arm was attached to a force transducer (model SBO-300-T, Transducer Techniques, Temecula, CA), enabling the determination of isometric torque production about the knee. Using Velcro® straps, the subject’s right ankle was secured to the leg-extension arm. In addition, a seatbelt was strapped across the subject’s waist. To enable electrical stimulation of the knee-extensor muscles, two 8- by 10-cm adhesive electrodes (UniPatch 616SS, Wabasha, MN) were placed on the skin overlying the thigh, one each over the distal vastus medialis and proximal vastus lateralis muscles. Because the electrodes were removed for the cycling protocol, electrode position on the skin was marked with indelible ink to ensure that electrode positioning was the same after cycling as before.

MVC isometric torque production by the right knee-extensor muscles was determined before and after each cycling trial. Subjects performed three 3-s MVC trials at 1-min intervals; verbal encouragement was given to ensure maximal efforts. If the peak-torque values for the 3 trials were not similar (i.e., ±5%), 2 more trials were performed. The average of the 3 highest peak-torque values was used in the subsequent analyses. Electrically evoked isometric contractile properties of the knee-extensor muscles were then assessed using a procedure similar to that described previously (5, 33). Stimulations were produced with a
constant-current stimulator (model DS7AH, Digitimer, Hertfordshire, England) controlled by a 450-MHz Pentium computer using an A/D interface board (model DAS1802AO, Keithley Instruments, Cleveland, OH) and customized programs written with TestPoint software (version 5.0, Capital Equipment Corp., Billerica, MA). This system also sampled the torque data at 5 kHz from the force transducer on the leg-extension/curl machine. The initial electrical stimulations entailed 1-s trains of 0.2-ms pulses at 80 Hz. Stimulation current was initially set at 40 mA and was increased by 5–10 mA with each succeeding contraction; contractions were induced at 30-s intervals. Stimulation current was adjusted until peak isometric torque was 50–55% of MVC isometric torque. This level of stimulation current was used for subsequent measurements because we have found it to be about the highest tolerable by all subjects. Next, a series of 11 electrically evoked isometric contractions was induced. This entailed performing a 1-s contraction every 30 s with stimulation frequency progressing from low to high (i.e., 2, 4, 6, 10, 15, 20, 25, 40, 60, 80, and 100 Hz).

The strength-test session conducted after the cycling protocol commenced 20 min after cessation of cycling. The test protocol was identical to that conducted before cycling. It was found during pilot work that stimulating electrode impedance was reduced by ~50% after cycling, and thus at a given stimulation current, the torque elicited after cycling was much higher. This precluded pre–post comparisons of absolute strengths at the same stimulator settings. Therefore, strengths at the various stimulation frequencies were normalized to that at 100 Hz in order to enable pre–post comparisons. To assess the degree of intrinsic muscle fatigue after cycling, the degree of shift in the torque–frequency relationship was determined by comparing the stimulation frequencies eliciting a normalized torque equal to 50% (Freq50) for both pre- and postcycling trials. Freq50 was determined by fitting an asymmetric sigmoidal curve to a trial’s torque–frequency data using TableCurve software (version 5.01, Systat Software, Richmond, CA). Additional indices of intrinsic muscle fatigue that were calculated include the pre- to postcycling changes in the rates of torque production and relaxation observed during the 100-Hz electrically evoked contractions. The rate of torque production was calculated as the average rate (in Nm/s) in going from 20% to 80% of peak torque. The rate of torque relaxation was calculated as the average rate (in Nm/s) in going from the time of the last pulse in the stimulation train to the time at which torque had fallen to 50% of peak.

Protocol and Procedures (Study B)

The subjects performed the same cycling-test protocol (120-min variable-intensity ride followed by a 15-min performance ride) as described for Study A, except that no blood measures were made and the twitch-interpolation technique was used as part of the contractile-properties assessment. The twitch-interpolation technique enabled us to estimate how much of the difference between CES and CES+CAF in the MVC strength loss seen after cycling was the result of altered activation of muscle, and therefore an effect mediated by the nervous system, versus that resulting from a direct effect on muscle. A slightly modified version of the interpolated-twitch technique recommended by Shield and Zhou (32) was used.

Maximal Voluntary and Electrically Evoked Isometric Contractile Properties.
Subjects were positioned in the custom-built leg-extension chair and prepared for
electrical stimulation of the right knee-extensor muscles. Two electrical-stimulation procedures were used. The first procedure was conducted only before cycling and employed brief electrical stimulations (i.e., paired pulses, consisting of two 0.2-ms pulses with an interpulse interval of 10 ms) of the right knee-extensor muscles while they were relaxed. During this procedure, the stimulator current was progressively increased until isometric torque production of the knee extensors had plateaued. Current was initially set at 90 mA and was increased by 20 mA every contraction. There were 20 s between contractions. The current producing the greatest torque value within the plateau was determined to be the subject’s supramaximal stimulation current and was used in all subsequent stimulations that day.

The second electrical-stimulation procedure consisted of an interpolated-twitch electrical-stimulation procedure that enabled determination of the knee extensors’ MVC isometric torque, peak electrically evoked isometric torque, and the percentage muscle activation during MVC. For the interpolated-twitch procedure, subjects performed a 3-s isometric MVC of the knee-extensor muscles; at 2.5 s into the contraction the muscles received a paired-pulse stimulation, and the increase in torque over the MVC level (interpolated-twitch torque; ITT) was measured. At 2 and 4 s after the end of the MVC contraction, the paired-pulse stimulation was administered to the relaxed muscle to determine the peak electrically evoked torque (EET); the average value for the 2 stimulations was used in subsequent analyses. The percentage muscle activation was estimated as 100% \times (1 – ITT/EET). A sequence of 6 interpolated-twitch procedures was performed before cycling and also starting at 5, 15, and 25 min after cessation of cycling. Within a sequence, there was 1 min of rest between the interpolated-twitch procedures. For a given sequence, the 3 best trials were determined and their data averaged together.

**Statistical Analyses**

**Study A.** Data from the experimental trials were analyzed using 3-way (beverage × time × site) or 2-way (beverage × site) mixed-model ANOVA (beverage and time as repeated measures and site as a between-subjects factor) and post hoc paired t-tests with a Bonferroni correction to control the familywise error rate (25). There were no significant beverage × site interactions, indicating that the results related to differences among beverages were the same at the 2 test sites. Thus, data are presented as means with standard deviations (SD) representing all 16 subjects. All statistical testing was conducted using SPSS (version 13) and an overall \( \alpha \) level of 0.05.

**Study B.** The MVC isometric torque, peak electrically evoked isometric torque, and percentage muscle-activation data were analyzed using a 2-way (beverage × time) repeated-measures ANOVA. Post hoc tests included paired t-tests with a Bonferroni correction.

**Results**

**Study A**

In the 10 subjects assessed, the mean serum concentration of caffeine during the CES+CAF trial increased progressively from 3.2 \( \mu \)M just before beginning exercise
to 31.9 μM at the termination of cycling (Table 1). For the placebo and CES trials, the mean concentration remained less than 2.5 μM.

**Metabolic Responses.** The VO₂ response to the 3 experimental cycling trials, expressed as a percentage of maximum, is presented in Figure 1(A). As planned, after the first 15 min, VO₂ oscillated between about 60% VO₂max and 75% VO₂max every 15 min for the first 120 min. Values tended to increase slightly over time, but there were no differences among beverages except at Minute 75, when the VO₂ for CES+CAF was higher than for CES and placebo. Between 120 and 135 min, subjects performed as much work as possible (performance ride). During the CES+CAF performance ride, subjects were able to increase the work rate, and the VO₂ averaged 88–95% VO₂max. In the corresponding rides for the other 2 beverages, subjects first reduced their work rate so that the VO₂ decreased initially and

<table>
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<tr>
<th>Variable</th>
<th>Time (min)</th>
<th>Placebo</th>
<th>CES+CAF</th>
<th>CES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caffeine (μM)</td>
<td>Preexercise</td>
<td>2.3 ± 0.5</td>
<td>3.2 ± 0.3</td>
<td>2.5 ± 0.5</td>
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<td>30</td>
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<td>9.5 ± 0.4a</td>
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<td></td>
<td>60</td>
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<td>14.2 ± 0.9a</td>
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<td></td>
<td>90</td>
<td>—</td>
<td>23.0 ± 0.8a</td>
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<td></td>
<td>120</td>
<td>—</td>
<td>28.4 ± 1.1a</td>
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<td>Glucose (mM)</td>
<td>Preexercise</td>
<td>4.59 ± 0.70b</td>
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<td>4.54 ± 0.41</td>
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<td>4.93 ± 0.79</td>
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<td>Lactate (mM)</td>
<td>Preexercise</td>
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<td>0.61 ± 0.14</td>
<td>0.60 ± 0.12</td>
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<td>30</td>
<td>2.58 ± 0.93</td>
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<td>2.43 ± 0.93</td>
<td>2.29 ± 0.97</td>
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<tr>
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<td>120</td>
<td>2.05 ± 0.86</td>
<td>2.52 ± 0.96</td>
<td>2.25 ± 1.00</td>
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<tr>
<td>Postexercise</td>
<td>2.54 ± 1.27</td>
<td>5.04 ± 1.90f</td>
<td>3.25 ± 1.37</td>
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</tr>
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</table>

aSignificantly different from rest within same beverage treatment, P < 0.05. bPlacebo vs. CES, P < 0.05. cPlacebo vs. CES+CAF, P < 0.05. dPlacebo vs. others, P < 0.05. eCES+CAF vs. CES, P < 0.05. fCES+CAF vs. others, P < 0.05.

CES+CAF indicates caffeinated sports drink, and CES, carbohydrate-electrolyte sports drink.
then increased to 76–84% (CES) or 70–81% (placebo) of VO₂max. The %VO₂max for the CES+CAF trial was significantly higher than those for CES and placebo at Minutes 125, 130, and 135 ($P \leq 0.001$).

During the cycling bouts, blood lactate increased above the resting level but remained relatively low during the first 120 min (Table 1). Differences among the beverages over the first 120 min were small and not statistically significant. Blood lactate immediately after the ride was significantly higher in the CES+CAF trial than in the CES and placebo trials ($P < 0.0001$), reflecting the greater rate of work performed during Minutes 120–135 in the CES+CAF trial.
**Energy Provision.** The blood glucose level was significantly lower for placebo than for CES at rest (Table 1). It was also significantly lower for placebo than for CES+CAF at Minutes 60 and 90, and it was significantly lower for placebo than CES+CAF and CES at Minute 120. Differences were accentuated at the end of the performance ride because different rates of work were performed. Immediately after exercise, the blood glucose level was significantly lower for placebo than for the CES+CAF and CES trials, and it also was lower in the CES trial than in the CES+CAF trial.

Over the first 120 min of cycling, the rate of CHO oxidation increased and decreased with the oscillating exercise intensity and tended to decrease slowly over time for all 3 beverages as expected, reflecting increased use of fat for fuel (Figure 1[B]). There was a significant beverage × time interaction, reflecting higher values for CES+CAF than placebo at Minutes 45, 75, and 105 and thereafter. CES was higher than for placebo at Minutes 105 and 125 and was significantly lower than CES+CAF at Minutes 75, 125, and 130. Total CHO oxidation during the first 120 min of cycling was significantly greater \((P = 0.017)\) in CES+CAF \((291 \pm 57 \text{ g})\) than for placebo \((259 \pm 69 \text{ g})\) but not different from CES \((278 \pm 55 \text{ g})\). CES and placebo were not significantly different. Total CHO use during the 15-min performance trial was significantly greater in CES+CAF \((54 \pm 13 \text{ g})\) than in CES \((40 \pm 14 \text{ g}, P < 0.023)\) and placebo \((32 \pm 12 \text{ g}, P < 0.001)\). CES and placebo were not significantly different.

**Ergogenic Effects of the Beverages.** There was a significant main effect for beverage in the 2-way ANOVA of cycling performance during the 15-min performance ride. Post hoc tests indicated that the mean total work performed between Minutes 120 and 135 was significantly greater for CES+CAF \((218 \pm 31 \text{ kJ})\) than for placebo \((178 \pm 31 \text{ kJ})\), by 23% (effect size \(d = 1.31 \text{ SD}\)), and 15% (effect size \(d = 0.86 \text{ SD}\)) greater than CES \((190 \pm 36 \text{ kJ}; P \leq 0.009)\). There was no significant difference between CES and placebo. Evaluation of individual subject performances indicated that performance was better in CES+CAF than for placebo in 15 of 16 subjects and better in CES+CAF than in CES in 13 of 16 subjects (Figure 2).

Twenty minutes after cessation of cycling, MVC isometric strength of the knee-extensor muscles for the placebo and CES trials was 14.8% (± 7.4%) and 15.1% (± 8.5%) lower, respectively, than that measured before cycling, but the MVC strength reduction after cycling for the CES+CAF trial was about two-thirds less (i.e., 5.2% ± 7.1%, \(P \leq 0.001\)). Evaluation of individual subject performances indicated the MVC strength loss was less for CES+CAF than for placebo in 16 of 16 subjects and less for CES+CAF than for CES in 15 of 16 subjects (Figure 3). There was, surprisingly, no significant beverage effect on any electrically evoked contractile property. In fact, the data provided no evidence of fatigue intrinsic to the knee extensors 20 min after any experimental trial. There was no rightward shift in the torque–frequency relationship after cycling that one might expect with intrinsic muscle fatigue; the stimulation frequency eliciting 50% of maximal torque was not significantly altered (Table 2; \(P \geq 0.29\)). There was also no effect of cycling on the rate of torque production during the 100-Hz stimulation or on the rate of relaxation after stimulation (Table 2; \(P \geq 0.29\)).
Ratings of Perceived Exertion. RPE varied with exercise intensity and tended to increase over time during the first 120 min of exercise (Figure 4). There was a significant beverage × time interaction. Differences among beverage treatments for the first 35 min were not significantly different. Thereafter, ratings were significantly lower for CES+CAF than for placebo at Minutes 40, 55, 70, 85, and 95–120 and than for CES at Minutes 45, 70–85, and 95–120. At Minute 120, mean RPE for CES+CAF was 1.7 units lower than that for CES and 1.9 units lower than...
Table 2  Electrically Evoked Contractile Properties, Frequency at 50% max torque, and Rates of Torque Development and Relaxation, Measured for All 3 Beverages Both Before and After Cycling in Study A, Mean ± SD

<table>
<thead>
<tr>
<th>Variable</th>
<th>Time</th>
<th>Placebo</th>
<th>CES+CAF</th>
<th>CES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency at 50% max torque (Hz)</td>
<td>Preexercise</td>
<td>15.9 ± 2.0</td>
<td>15.9 ± 2.2</td>
<td>16.7 ± 2.6</td>
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<td>Postexercise</td>
<td>16.2 ± 3.6</td>
<td>16.9 ± 4.4</td>
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<td>Rate of torque development (Nm/s)</td>
<td>Preexercise</td>
<td>308.4 ± 64.3</td>
<td>314.1 ± 65.3</td>
<td>303.0 ± 47.0</td>
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<td>Postexercise</td>
<td>293.0 ± 48.3</td>
<td>311.6 ± 86.9</td>
<td>284.4 ± 72.4</td>
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<tr>
<td>Rate of torque relaxation (Nm/s)</td>
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<td>530.2 ± 82.4</td>
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<td>Postexercise</td>
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<td>541.9 ± 117.4</td>
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CES+CAF indicates caffeinated sports drink, and CES, carbohydrate-electrolyte sports drink.

Figure 4 — Ratings of perceived exertion during cycling with the 3 beverage treatments. CES+CAF indicates caffeinated sports drink, and CES, carbohydrate-electrolyte sports drink. *CES+CAF vs. placebo, P < 0.05; †CES+CAF vs. CES, P < 0.05.

that for placebo. There were no differences in RPE among beverages during the performance ride (Minutes 125–135).

Study B

To provide additional insight into the mechanisms underlying the significantly less MVC strength loss after cycling with CES+CAF than with CES, the interpolated-
twitch procedure was performed on 5 additional subjects both before and after the cycling protocol. As for the data presented for Study A, the MVC strength loss was significantly less with CES+CAF than with CES at all 3 time points after cycling (Figure 5[A]; main effect \( P = 0.049 \)). The MVC strength loss was always less during the CES+CAF trial than during the CES trial for each subject at all 3 time points. After the cycling bout, there was no change in the peak electrically evoked strength in the CES+CAF trial, but it was decreased \( \sim 10\% \), on average, in the CES trial (Figure 5[B]; main effect \( P = 0.039 \)). Again, for each subject at

![Graph showing changes in MVC strength and electrically evoked strength](image)

**Figure 5** — Changes in (A) maximal voluntary and (B) electrically evoked strengths after cycling in Study B. CES+CAF indicates caffeinated sports drink; CES, carbohydrate-electrolyte sports drink; and MVC, maximal voluntary contraction. *Change significant between beverages at \( P < 0.05 \).
all 3 time points, the electrically evoked strength loss was always less (or absent) during the CES+CAF trial. There was no difference between CES+CAF and CES in the change in percentage muscle activation after cycling ($P \geq 0.41$; Figure 6), indicating that the smaller MVC strength loss observed with CES+CAF than with CES was probably a direct effect of the beverage on muscle fatigue and not greater muscle activation.

Discussion

The overall purpose of these studies was to compare the effectiveness of a caffeinated sports drink with that of the market-leading noncaffeinated sports drink and a placebo control. Effectiveness was judged by the ability to provide CHO energy during prolonged exercise and to improve physical performance. The primary findings of the study are that the caffeinated sports drink is as effective in providing energy during prolonged exercise but is more effective in improving cycling performance and attenuating muscle fatigue than the noncaffeinated sports drink.

Cycling performance, as assessed by the total work performed during a 15-min performance ride after 2 h of moderate- to heavy-intensity cycling, was 23% greater in CES+CAF than in placebo and 15% greater in CES+CAF than in CES. Cycling performances for CES and placebo were not significantly different. The greater rate of work performed in CES+CAF than in placebo or CES was accompanied by a higher rate of VO$_2$ during the 15-min performance ride and a higher blood lactate level immediately afterward, indicating that subjects were able to maintain a higher rate of energy expenditure and tolerate a greater metabolic strain. A novel

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**Figure 6** — Changes in percentage muscle activation after cycling in Study B. There were no significant differences between beverages at any time point. CES+CAF indicates caffeinated sports drink, and CES, carbohydrate-electrolyte sports drink.
finding was that muscle strength was better maintained after prolonged exercise. These findings indicate that CES+CAF has a substantial ergogenic effect on work capacity in prolonged exercise compared with CES and placebo.

The design of this study does not permit isolation of the cause of the ergogenic effect. The effect is consistent with that previously reported for caffeine, but because CES+CAF contained ingredients in addition to caffeine not in CES or placebo (vitamins B3, B6, and B12; taurine; carnitine; and sucralose), it is possible that some or all of the ergogenic effect is caused by 1 or more of these other ingredients. The B vitamins (29), carnitine (6), and the artificial sweetener sucralose have no known ergogenic effect on work capacity in prolonged exercise, however. We are aware of only 1 study (15) in which an ergogenic effect was observed after acute administration of taurine that could not be attributed to caffeine that was included in the treatment along with taurine. Furthermore, the mechanism through which taurine might increase performance in prolonged exercise is unclear. In contrast, there is a voluminous literature establishing the ergogenic effect of caffeine on prolonged exercise performance. Although some studies have not found an ergogenic effect of caffeine during prolonged exercise, recent systematic qualitative (16, 34) and quantitative (12) reviews of the literature have concluded that caffeine enhances performance in prolonged exercise.

Inconsistent results among studies on the effects of caffeine on performance can potentially be explained by the many experimental-design factors that might influence the magnitude of a caffeine effect, including caffeine dose, the form in which caffeine is ingested (i.e., in pill form, in coffee, or in combination with other substances such as CHO), timing and pattern of administration, habitual dietary caffeine consumption by subjects, period of withdrawal of caffeine before testing, environment, and nature of the performance test (16).

The mechanisms underlying the ergogenic effect of CES+CAF cannot be established with certainty from the data collected in this study. If the ergogenicity is assumed to be primarily the result of caffeine, there are several possibilities. Caffeine has 3 biochemical mechanisms of action observed in vitro experiments: intracellular calcium release via direct interaction with ryanodine receptors (calcium-release channels) in the sarcoplasmic reticulum, phosphodiesterase inhibition, and adenosine-receptor antagonism (14, 23). The first 2 of these are generally thought to require millimolar caffeine concentrations that are toxic and are not found in humans. There are several studies, however, indicating that caffeine at much lower concentrations can have a force-potentiating effect on human muscle in vivo (28, 30, 31), suggesting that caffeine’s ergogenic effect might work via a direct effect on skeletal muscle. Nevertheless, the primary mechanism of action for orally ingested caffeine in humans is thought to be via adenosine-receptor antagonism (14). Of the 4 adenosine-receptor subtypes, the actions of caffeine in humans are thought to be mediated primarily by the A1 and A2a receptors located in most tissues throughout the body, including brain, spinal cord, peripheral nerves, adipocytes, heart, smooth muscle, and skeletal muscle.

In relation to performance enhancement during prolonged exercise, 3 possible mechanisms for caffeine have been considered important: increased mobilization of fat and possible sparing of muscle glycogen; effects on the central nervous system that might increase vigilance and motivation to continue, increase motor unit recruit-
ment and activity, and reduce sensations of effort, force, pain, and fatigue; and as mentioned previously, direct effects on skeletal muscle that might augment force production, increase endurance, and reduce fatigue. It is likely that more than one of these mechanisms might increase performance during prolonged exercise.

The mechanisms underlying the ergogenic effect of CES+CAF on cycling performance in our study do not appear to be related to altered substrate utilization or metabolism. Although differences in blood glucose concentration and in the rate of CHO oxidation at Minute 120, just before the performance trial, between CES+CAF versus placebo and CES versus placebo were substantial (19–28%), those between CES+CAF and CES were small (2–7%) and not statistically significant. Likewise, the difference between CES+CAF and CES in total CHO oxidized during the first 120 min of cycling was small (5%) and not statistically significant. The slightly higher values for CES+CAF than for CES might have reflected, in part, the difference in CHO concentration between the 2 beverages and difference in total CHO ingested in the 2 trials. Blood glucose concentration and CHO oxidation during the performance ride were greater in CES+CAF than in CES as would be expected because of the higher work rate during CES+CAF. Likewise, the VO$_2$ and concentration of blood lactate at Minute 120 were not different among trials. The absence of a difference in blood glucose concentration between CES+CAF and CES at the same submaximal work rate is similar to recent findings by Yeo et al. (38), but they found higher CHO oxidation with a caffeinated compared with a noncaffeinated CHO-containing beverages. The different findings might be related to their greater caffeine ingestion and lower exercise intensity. The deduction that the ergogenic effect of CES+CAF in our study is not related to altered substrate utilization or metabolism is consistent with conclusions of recent reviews that caffeine’s ergogenic effect during prolonged exercise is independent of altered substrate utilization or any associated metabolic changes known to enhance performance (12, 16, 34). The studies of Cox et al. (10) and Kovacs et al. (26), in which caffeine was coingested with CHO, as in our study, also found an ergogenic effect during prolonged exercise that was independent of altered substrate utilization.

If the ergogenicity of caffeine is not metabolic in origin, it is likely the result of an effect on the nervous system or a direct effect on skeletal muscle. A striking, but not unique, finding of this study was the reduced ratings of perceived exertion during the first 120 min of cycling for CES+CAF compared with both CES and placebo. The attenuated perceived effort for CES+CAF was evident at Minute 45 and increased in magnitude during the latter part of the 2-h ride when subjects rode at a set work rate. In contrast, during the maximal-effort performance ride after the initial 2 h of cycling, the difference in RPE was eliminated. Subjects increased their effort by performing at a higher rate of work and energy metabolism during CES+CAF, so their perception of effort was equivalent to that in the CES and placebo trials. These data indicate that enhanced performance in CES+CAF was not because the cyclists were more motivated to perform or tried harder under that condition. Rather, it suggests that perception of effort, and perhaps associated pain and discomfort, might have been altered by CES+CAF. If cycling during the performance ride was limited by feelings of discomfort, fatigue, and possibly pain, the altered perception could explain the improved performance. A lower RPE at the end of the 2-h ride could provide greater reserve for increasing power output up to the maximal level that can be tolerated.
Reduced RPE during constant-rate, submaximal exercise has been a common finding in studies reporting an ergogenic effect of caffeine during prolonged exercise (3, 4, 9, 10). The size of the effect in comparing CES+CAF with CES or placebo at Minute 120 (−1.7 to −1.9 units, −0.8 to −0.9 SD) was large and almost double that of the average effect (−0.5 SD) reported in a meta-analysis of studies on the effect of caffeine on RPE during constant-load exercise (13). In the meta-analysis, moderator variables including period of subject withdrawal before treatment, interval between caffeine ingestion and exercise test, and caffeine dose did not influence the effect magnitude. In the studies in that analysis, caffeine improved performance an average of 11% relative to placebo, whereas in our study, performance in the CES+CAF trials improved 15–23% relative to CES and placebo. Moreover, RPE during exercise accounted for ~29% of the variance in the improvement in exercise performance in the meta-analysis. The deduction that CES+CAF improved performance in part by reducing RPE during submaximal exercise and increasing the rate of work that can be tolerated for a sustained period of time is also supported by studies that have found caffeine to increase the rate of work performed at a given level of effort (7, 19).

In anticipation of observing possible ergogenic effects during cycling, we measured MVC and electrically evoked contractile properties in the right knee-extensor muscles both before and after cycling in order to provide insight into the mechanisms underlying any performance-enhancing effects of the beverages. We found in Study A that 20 min after cessation of cycling, the reduction in MVC isometric strength for CES+CAF (5%) was about two-thirds less than that for CES and placebo (15%). Surprisingly, there was no significant beverage effect on any electrically evoked contractile property measured in Study A nor evidence of intrinsic muscle fatigue being present 20 min after the cycling bout. There was no rightward shift in the torque–frequency relationship (data not shown), no change in the stimulation frequency eliciting 50% of maximal torque, and no alterations in the rate of torque production during a 100-Hz stimulation or in the rate of relaxation after stimulation. A rightward shift in the torque–frequency relation, an increased stimulation frequency required to elicit 50% maximal torque, and slowed rates of torque production and relaxation would be expected if there were fatigue-related changes in the excitation–contraction coupling process or in the contractile apparatus. These data suggest that the ability of CES+CAF to reduce the MVC strength loss was not a result of intrinsic changes in the active skeletal muscles.

Other explanations are possible, however, and the interpretation of the reduced MVC strength loss with CES+CAF compared with CES and placebo was unclear until Study B was conducted. Study B was designed to partition the MVC strength loss into 2 components, one being a fatigue intrinsic to the muscle tissue itself and the other being a fatigue resulting from an inability or lack of drive from the central nervous system to fully activate the muscle tissue. This latter fatigue, commonly called central fatigue, is thought to be attenuated by caffeine ingestion (24). Based on our results from Study A, we hypothesized that the lesser MVC strength loss observed in the CES+CAF trials was a result of less central fatigue occurring in those trials.

To directly test this hypothesis, the interpolated-twitch technique was employed to assess the percentage muscle activation both before and after cycling. In this technique, a brief supramaximal electrical stimulation is produced while the subject is performing an MVC. If torque goes up, then the muscle was not maximally
activated. The percentage muscle activation can be estimated by comparing the torque increase resulting from the stimulation imposed during the MVC with that elicited by the same stimulation while the muscles are at rest. We hypothesized that the percentage muscle activation during a maximal effort after cycling would be higher during the CES+CAF trials than during a trial done with CES. In these experiments, the interpolated-twitch technique was used both before and at 3 time points after the cycling bout. From these measurements, we were able to simultaneously measure the effect of prolonged cycling on the percentage muscle activation during a maximal effort and on strength assessed during a supramaximal electrical stimulation of relaxed muscle. This latter measurement when obtained pre- and postcycling provides a measure of fatigue intrinsic to the muscle tissue itself.

The results of Study B indicate that CES+CAF does not, as hypothesized, improve muscle activation during a maximal voluntary effort after prolonged cycling. The reduction in percentage muscle activation after cycling was not different between the CES+CAF and CES trials. After CES+CAF, however, there was markedly less fatigue intrinsic to the muscle tissue than that resulting from CES. These data appear to be in disagreement with those obtained from electrical-stimulation measures in Study A. In Study A, however, we did not use supramaximal electrical stimulation, and thus our measures of intrinsic muscle fatigue were indirect and less sensitive. To summarize Study B’s finding, the reduction in MVC strength loss after prolonged cycling when CES+CAF is ingested was replicated in 5 subjects and appears to be a result of less intrinsic muscle fatigue and not an improved ability of the central nervous system to activate the muscles as originally hypothesized. Presumably, this is caused by a direct effect of caffeine on the muscle tissue. Although there are a number of research studies indicating that caffeine can have a direct effect on human muscle (28, 30, 31), this possibility has generally been discounted in the literature because the effect was only thought to occur with nonphysiological (i.e., millimolar) levels of caffeine, not the more physiological micromolar levels (21).

Our data indicate that the ergogenic effect of CES+CAF compared with CES on cycling performance and loss of muscle strength after prolonged cycling is not explained by an ability to better activate the skeletal muscles involved. Rather, reduced intrinsic muscle fatigue is probably part of the explanation. Reduced intrinsic muscle fatigue during the CES+CAF trials might have contributed to reduced perception of effort toward the end of the 2 h of moderate- to heavy-intensity cycling and might have permitted subjects to increase their effort and cycle at a higher power output equivalent to the point of discomfort that subjects were willing or able to tolerate. Central and peripheral effects of caffeine on the nervous system that do not alter muscle activation might also have altered perception of effort and contributed to improved performance (11).

Conclusions

Based on the results of our 2 studies, we conclude that CES+CAF increases the provision of CHO energy by maintaining blood glucose concentration during prolonged exercise as well as CES does and enhances cycling performance and reduces strength loss relative to CES and placebo. The improved cycling performance
with CES+CAF is independent of any apparent alteration in substrate utilization or energy metabolism but is associated with a reduced perception of effort during submaximal cycling that apparently permits a higher rate of work to be performed at the maximum tolerable level of exertion and discomfort during an all-out effort. The ergogenicity of CES+CAF does not appear to be related to an increase in the ability to neurally activate muscles but, rather, to effects on the active musculature and nervous system that reduce fatigue and perceptions of effort, discomfort, and pain. These effects are consistent with the known actions of caffeine.

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