

Improved Cycling Time-Trial Performance After Ingestion of a Caffeine Energy Drink

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Context: Not all athletic competitions lend themselves to supplementation during the actual event, underscoring the importance of preexercise supplementation to extend endurance and improve exercise performance. Energy drinks are composed of ingredients that have been found to increase endurance and improve physical performance.

Purpose: The purpose of the study was to investigate the effects of a commercially available energy drink, ingested before exercise, on endurance performance. **Methods:** The study was a double-blind, randomized, crossover design. After a 12-hr fast, 6 male and 6 female trained cyclists (mean age 27.3 ± 1.7 yr, mass 68.9 ± 3.2 kg, and VO_2 54.9 ± 2.3 ml \cdot kg⁻¹ \cdot min⁻¹) consumed 500 ml of either flavored placebo or Red Bull Energy Drink (ED; 2.0 g taurine, 1.2 g glucuronolactone, 160 mg caffeine, 54 g carbohydrate, 40 mg niacin, 10 mg pantothenic acid, 10 mg vitamin B6, and 10 μ g vitamin B12) 40 min before a simulated cycling time trial. Performance was measured as time to complete a standardized amount of work equal to 1 hr of cycling at 70% W_{max} . **Results:** Performance improved with ED compared with placebo ($3,690 \pm 64$ s vs. $3,874 \pm 93$ s, $p < .01$), but there was no difference in rating of perceived exertion between treatments. β -Endorphin levels increased during exercise, with the increase for ED approaching significance over placebo ($p = .10$). Substrate utilization, as measured by open-circuit spirometry, did not differ between treatments. **Conclusion:** These results demonstrate that consuming a commercially available ED before exercise can improve endurance performance and that this improvement might be in part the result of increased effort without a concomitant increase in perceived exertion.

Keywords: aerobic endurance, β -endorphins, catecholamines, glucose, taurine

Not all sporting events lend themselves to supplementation during the actual event. For example, it is very difficult to consume appropriate amounts of supplements during soccer matches or cycling time trials that last 1 hr or less. Therefore, it becomes important to determine the most appropriate type of preexercise supplement to extend endurance and improve exercise performance.

Traditional sports drinks composed of glucose and electrolytes might not be of much benefit in this regard. Research has consistently documented improved endurance performance in athletes using carbohydrate beverages, compared with water or other placebo beverages (Coyle, Coggan, Hemmert, & Ivy, 1986; Ivy et al., 1983; Ivy, Res, Sprague, & Widzer, 2003; Sherman et al., 1989; Yaspelkis, Patterson, Anderla, Ding, & Ivy, 1993). The increased performance time to fatigue, however, or increased power output during the later stages of prolonged exercise occurs when supplementing during exercise. The effect of carbohydrate supplementation when provided before exercise, however, is equivocal, with a few studies demonstrating a positive effect (Kirwan, O’Gorman, & Evans, 1998; Sherman et al.) but most studies indicating a reduced (Foster, Costill, & Fink, 1979) or no performance-enhancing effect (Hargreaves, Costill, Fink, King, & Fielding, 1987; Kuipers, Franssen, & Keizer, 1999; Sherman et al.).

Conversely, unlike carbohydrate supplementation, caffeine appears to have a very positive effect on exercise performance when taken before exercise, as well as during exercise (Costill, Dalsky, & Fink, 1978; Graham & Spriet, 1991; Ivy, Costill, Fink, & Lower, 1979; Kovacs, Stegen, & Brouns, 1998; Pierno, De Luca, Camerino, Huxtable, & Camerino, 1998; Spriet et al., 1992). Supplements of caffeine containing 3–9 mg/kg body weight provided 1 hr before exercise have been found to be effective in increasing exercise time to exhaustion at exercise intensities of 70–80% $\text{VO}_{2\text{max}}$ (Graham & Spriet, 1995; Kovacs et al.; Pasmán, van Baak, Jeukendrup, & de Haan, 1995).

Many of the new energy drinks currently being sold are marketed with the assertion that they will increase endurance and improve physical performance. The main ingredients of energy drinks are carbohydrates, taurine, glucuronolactone, caffeine, and B vitamins. Energy drinks contain caffeine and carbohydrate and therefore have the potential to improve endurance performance. These drinks also contain ingredients such as taurine that have been found to elevate mood, alertness, and concentration (Mandel et al., 1985) and therefore might also contribute to endurance performance by changing perception of effort. There has been little research, however, to confirm the beneficial effects of such drinks, particularly when provided preexercise.

The purpose of the proposed study was to evaluate the exercise performance of trained cyclists after they ingested a commercially available energy drink containing glucose, caffeine, and taurine, as well as other ingredients that might benefit performance. We hypothesized that the time to complete a standardized workload on a cycle ergometer would be enhanced by consuming the energy drink 40 min before the onset of exercise.

Methods

Participants

The participants were 6 male and 6 female competitive cyclists accustomed to cycling time trials. They averaged 27.3 ± 1.7 years of age (men 27.5 ± 3.0 , women 27.0 ± 2.0), weighed an average of 68.9 ± 3.2 kg (men 75.1 ± 3.7 , women 62.6 ± 3.8), and had an average maximal aerobic power ($\text{VO}_{2\text{max}}$) of 54.9 ± 2.3 ml · kg⁻¹

· min⁻¹ (men 60.5 ± 1.8, women 49.4 ± 2.7). Before testing, all participants were given a detailed explanation of the procedures to be used and the potential risks of the study. They had to successfully complete a health-history questionnaire and not be taking any prescription drugs such as blood-pressure, cardiac, or glucose-control medications that might affect the outcome of the study or participant safety. They then completed the consent-to-participate form according to the protocol described in the University of Texas at Austin's *Institutional Review Board Procedures Manual for Faculty, Staff, and Student Researchers With Human Participants*. The study was approved by the University of Texas at Austin Institutional Review Board before its commencement.

Experimental Design

A double-blind, randomized, placebo-controlled, two-period, within-participants crossover experimental design was used. Each participant completed two randomly assigned treatments in which 500 ml of either an artificially colored, flavored, and sweetened-water placebo or Red Bull Energy Drink (ED) was provided 40 min before exercise because caffeine peaks in the blood within 30–60 min after consumption (Blanchard & Sawers, 1983; Cole et al., 1996; Essig, Costill, & Van Handel, 1980; Graham & Spriet, 1995; Liguori, Hughes, & Grass, 1997). Red Bull GmbH, Fuschl am See, Austria, provided the placebo and ED in 250-ml units. Participants consumed 500 ml of ED, the equivalent of two cans of Red Bull Energy Drink, which contains the minimum average amount of caffeine found to be effective (Graham & Spriet, 1995). The concentration of ingredients in 500 mL of ED consisted of 2.0 g taurine, 1.2 g glucuronolactone, 160 mg caffeine, 54 g carbohydrates, 40 mg niacin, 10 mg pantothenic acid, 10 mg vitamin B6, and 10 µg vitamin B12. At the beginning of exercise and every 20 min thereafter, 300 ml of water was provided. The participants performed each trial in a room maintained at 19–21 °C, at the same time of day and the same day of the week 1 week apart.

Preliminary Testing

Participants reported to the laboratory before the start of their experimental trials for determination of their $\text{VO}_{2\text{max}}$ and maximal workload (W_{max}). They also performed a practice trial. The $\text{VO}_{2\text{max}}/W_{\text{max}}$ test was performed on the same ergometer used in the practice and experimental trials (Lode Excalibur Sport Cycle Ergometer, Groningen, The Netherlands). The protocol for establishing $\text{VO}_{2\text{max}}/W_{\text{max}}$ consisted of a 4-min warm-up and then 2-min stages beginning at a workload of 100 W and increasing by 50 W every 2 min until 350 W. After 350 W the increase was 25 W every minute until fatigue. Participants breathed through a Daniel's valve, with expired gases directed to a mixing chamber for analysis of oxygen (O_2) and carbon dioxide (CO_2). Inspired volumes were measured using a dry gas meter (Max-1, Physio-Dyne Instruments Corp., Quogue, NY). Outputs from these instruments were directed to a laboratory computer for calculation of ventilation, O_2 consumption (VO_2), CO_2 production, and respiratory-exchange ratio (RER) every 30 s. The criteria used to establish $\text{VO}_{2\text{max}}$ were a plateau in VO_2 with increasing exercise intensity and RER >1.10.

Two to three days after the $\text{VO}_{2\text{max}}/\text{W}_{\text{max}}$ test the participants reported to the laboratory to perform a practice ride to familiarize themselves with the laboratory environment and the experimental protocol to be used. No blood samples were collected during the practice ride, which consisted of a 1-hr time trial. The ergometer was set in the linear, or pedal-rate-dependent, mode. After a short warm-up (5 min at 100 W) the participants performed a standardized amount of work equal to about 1 hr of cycling as fast as possible. The measure of performance was the time to complete the target amount of work based on the participant's W_{max} and calculated according to Jeukendrup, Saris, Brouns, and Kester (1996):

$$\text{Target amount of work (J)} = 0.75 \times \text{W}_{\text{max}} \times 3,600 \text{ (s)}$$

The ergometer was set in the linear mode according to the formula $W = L \times \text{rpm}^2$, where L was selected to achieve 70% of the participant's W_{max} at 90 rpm. This corresponds to 75% W_{max} at about 100 rpm, which is close to the preferred pedaling rate of most competitive cyclists. Participants were informed that pedaling at 90 rpm or more would complete the target amount of work in under 1 hr.

The participants were instructed to maintain a training and dietary log for 2 days before the first experimental trial. They were provided a copy of their log and instructed to have the same dietary intake and activity during the 48 hr before their second trial. In addition, participants were instructed to keep their caffeine consumption stable throughout the period of study participation. The experimental trials were separated by a minimum of 7 days.

Experimental Protocol

The participants reported to the laboratory in the morning after a 12-hr fast, during which time they were allowed to consume only water. When they reported to the laboratory a catheter fitted with a three-way stopcock and catheter-extension tubing was inserted into a forearm vein and tapped in place. Next, a heart-rate (HR) monitor (Polar Beat, Polar Electro Oy, Finland) was secured in place around the participant's chest. A blood sample was drawn, and either the placebo or ED (500 mL each) was provided 40 min before the start of the time trial. Approximately 10 min before the start of the time trial, the participant was required to void, after which resting HR was recorded. The participant then mounted the ergometer and a second blood sample was drawn, and then the time trial commenced as described for the practice trial.

Neither the participants nor the investigators were aware of which participants were on the placebo or ED treatment; the two treatments were similar in color, taste, and texture and were randomly distributed by a laboratory technician not involved in the data collection. Constant verbal encouragement was given to the participants during each trial. During each ride participants were not aware of how long they had ridden, because all timing devices were removed from their sight.

Sample Collection and Analyses

Ventilation, VO_2 , CO_2 production, and RER were recorded with the respiratory-gas-analysis system described previously. Respiratory-gas collections were mea-

sured between 5–10, 30–35, and 40–45 min of exercise. Only the last 3 min for each collection were used to determine VO_2 and RER. These data were then used to estimate the rate of carbohydrate and fat oxidation. HR was recorded at the beginning of exercise and at 10-min intervals during exercise. Subjective ratings of perceived exertion on a Borg scale (of 6–20) were obtained during exercise at the same time points as HR. Five milliliters of venous blood were drawn before ingestion of the treatment while the participants were seated on the cycle ergometer immediately before the start of exercise, at every 10 min of exercise, and immediately after exercise. Each blood sample was mixed with 0.3 ml of EDTA (24 mg/ml, pH 7.4) and reduced glutathione. A sample of this anticoagulated blood (0.5 ml) was then transferred to another tube containing 1 ml 10% perchloric acid. One drop of anticoagulated blood (150 μl) was then used to measure blood glucose concentration in duplicate with a glucose meter (One Touch Basic, LifeScan, Inc., Milpitas, CA). Before using the glucose meter we verified its validity and reliability by comparing values obtained from it with those from a YSI 23A glucose analyzer (Yellow Springs, OH). The glucose meter was calibrated at the beginning of each experimental trial using standards provided by LifeScan.

All blood samples were maintained on ice until the completion of the exercise trial and were then centrifuged for 15 min at 3,000 rpm with a JS-7.5 rotor in a Beckman J2-21 centrifuge. Plasma and perchloric acid extracts were transferred and stored at -80°C for further analysis. Plasma samples were analyzed for insulin, cortisol, human β -endorphins, epinephrine, norepinephrine, and free fatty acids (FFAs). Insulin and cortisol were analyzed by commercially available double-antibody RIA kits (insulin: Diagnostics Systems Laboratories, Inc., Webster, TX, intra-assay CV 6.0%; cortisol: Diagnostic Products, Los Angeles, CA, intra-assay CV 6.8%). Human β -endorphin was analyzed by ELISA (MD Biosciences, Inc., St. Paul, MN) after column extraction using a Strata well plate (Phenomenex, Torrance, CA). After extraction, samples were dried under nitrogen using a SpedVac (Savant Instruments, Inc., Farmingdale, NY) and resuspended before assay. The plasma catecholamine concentrations were determined using ELISA (Labor Diagnostika Nord GmbH & Co., Nordhorn, Germany; intra-assay CV: epinephrine 11.0%, norepinephrine 13.0%) after extraction using a cis-diol-specific affinity gel. Epinephrine and norepinephrine were then acylated to N-acyl epinephrine and N-acyl norepinephrine, followed by their enzymatic conversion into N-acylmetanephrine and N-acylnormetanephrine, respectively, during the detection phase. Plasma (intra-assay CV 1.2%) FFAs were measured using the colorimetric assay procedure of Duncombe (1964) but modified by using the extraction reagent of Noma, Okahe, and Kita (1973). Blood lactate (intra-assay CV 9.8%) and glycerol (intra-assay CV 8.0%) were determined from the perchloric acid extract by enzymatic analysis according to Hohorst (1963) and Weiland (1974), respectively.

Statistical Analysis

Exercise-performance data were analyzed using a paired Student's *t* test. The blood data were analyzed using a general linear model for repeated measures. Ratings of perceived exertion were analyzed using a nonparametric Kruskal–Wallis test. Post

hoc analyses were performed using a Bonferroni test. Differences were considered significant at $p < .05$. Data are expressed as $M \pm SEM$.

Results

Time to complete the time trial was significantly improved when participants ingested ED before exercise (Figure 1). This occurred as a result of a higher work rate from 30 to 40 min of exercise during the ED treatment, with this difference in work then maintained throughout the remainder of the exercise (Figure 2). The average improvement in performance was 4.7%. Of the 12 participants, 10 completed the time trial faster on ED than on placebo; 2 participants had very similar times for both drinks (Figure 1).

Resting HR was similar for ED and placebo treatments (Table 1). HR increased with exercise but remained similar between treatments until the 50-min exercise period. At 50 min of exercise HR was slightly but significantly higher during the ED trial and remained significantly higher until work completion.

Rating of perceived exertion increased significantly with exercise duration during both trials. Despite the increased rate of work during the ED trial, rating of perceived exertion was not different between treatments (Table 1).

Blood glucose during the ED trial was significantly higher than during the placebo trial immediately before exercise (Figure 3). By 10 min of exercise, however, blood glucose levels had returned to baseline during the ED trial and remained

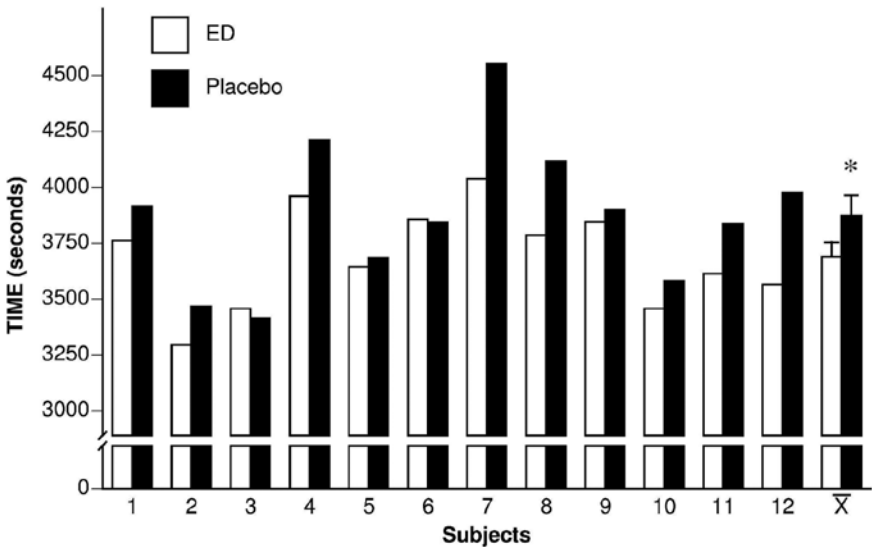


Figure 1 — Time-trial times for each participant after ingestion of the energy drink (ED) or placebo. $M \pm SEM$ are also presented for each treatment. *Significant difference between treatments ($p < .05$).

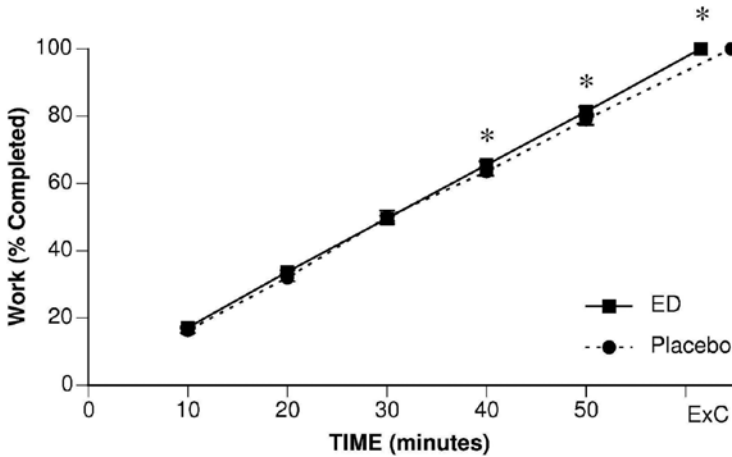


Figure 2 — Percentage of work completed every 10 min of cycling until completion of the time trial (ExC) for the energy drink (ED) and the placebo trial. *Significant difference between treatments ($p < .05$).

stable for the rest of the exercise. They also remained stable during exercise in the placebo trial. Thus, there was no difference in blood glucose levels between trials during exercise.

There was a significant treatment-by-time effect for blood lactate. During exercise blood lactate rose significantly during both trials (Figure 4). It was significantly higher, however, preexercise, at 30 and 50 min of exercise, and at completion of exercise with the ED treatment than with placebo.

ED significantly elevated plasma insulin levels immediately before exercise and during the first 10 min of exercise (Table 2). By 30 min of exercise, plasma insulin levels had returned to baseline during the ED trial and were not significantly different from placebo until completion of exercise. Plasma insulin levels were relatively stable during the placebo trial.

There was no significant treatment, time, or treatment-by-time effect for cortisol (Table 2). Thus, there were no differences in the cortisol responses for the ED and placebo trials.

β -Endorphin levels were similar between treatments predrink and immediately before exercise (Table 2). At the completion of exercise β -endorphin levels had risen slightly in both trials, with the increase higher for ED. The difference between ED and placebo was not statistically significant ($p = .10$), however.

Epinephrine levels were similar between treatments predrink and immediately before exercise. Epinephrine increased during exercise in both trials. The increase in epinephrine was significantly higher, however, during the ED trial at 30 and 50 min during exercise and immediately postexercise (Table 2). There was a very large increase in plasma epinephrine at the end of exercise during the ED treatment.

Table 1 Heart Rate (HR) and Rating of Perceived Exertion (RPE) for Energy Drink and Placebo, $M \pm SEM$

Treatment	PreD	PreEx	Min					ExC
			10	20	30	40	50	
HR, beats/min	57.6 \pm 3.0	63.6 \pm 3.9	164.0 \pm 3.2	168.8 \pm 3.4	170.3 \pm 3.2	169.4 \pm 3.8	173.0* \pm 4.0	182.2* \pm 4.3
RPE, Borg scale	60.9 \pm 3.4	56.5 \pm 2.9	161.1 \pm 4.2	164.0 \pm 4.2	165.8 \pm 4.6	165.3 \pm 5.2	166.5 \pm 5.8	170.5 \pm 5.7
energy drink			11.9 \pm 1.6	13.2 \pm 2.0	13.8 \pm 1.4	14.2 \pm 1.4	14.6 \pm 1.5	
placebo			12.2 \pm 1.9	13.0 \pm 2.0	13.7 \pm 2.1	14.3 \pm 1.5	15.0 \pm 1.6	

Abbreviations: PreD = predrink; PreEx = preexercise; ExC = exercise completion.

*Significant differences between treatments ($p < .05$).

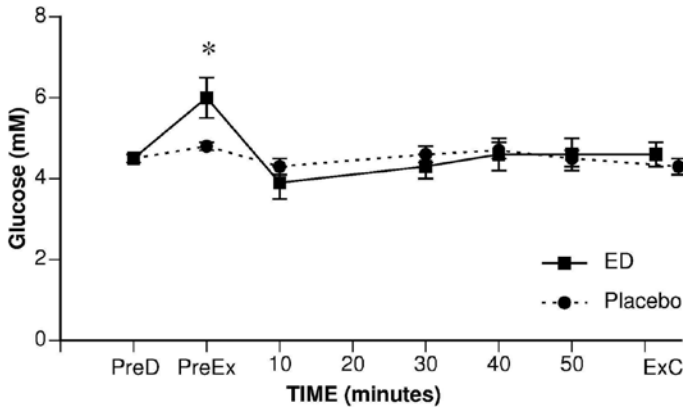


Figure 3 — Blood glucose before ingestion of energy drink (ED) or placebo (PreD), immediately before the time trial (PreEx), during the time trial, and on completion of the time trial (ExC). *Significant difference between treatments ($p < .05$).

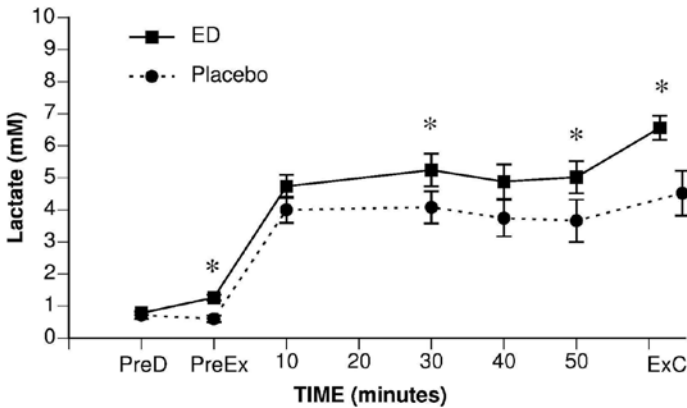


Figure 4 — Blood lactate before ingestion of energy drink (ED) or placebo (PreD), immediately before the time trial (PreEx), during the time trial, and on completion of the time trial (ExC). *Significant difference between treatments ($p < .05$).

Overall plasma norepinephrine levels increased with exercise and peaked immediately postexercise in both trials (Table 2). The increases in norepinephrine were similar for both treatments.

Blood glycerol rose steadily during exercise for both treatments (Figure 5). Glycerol levels were slightly higher during the placebo trial than the ED trial. Significant differences only occurred, however, during the first 10 min of exercise.

There was a significant treatment-by-time effect for plasma FFA (Figure 6). FFA decreased after consumption of ED and placebo. During exercise, FFA

Table 2 Plasma Hormone Concentrations for Energy Drink and Placebo Before and During Exercise, $M \pm SEM$

Hormone	Treatment	PreD	PreEx	Min					ExC
				10	30	40	50		
Insulin, nmol/ml	energy drink	78.9 \pm 5.7	213.8* \pm 27.3	111.9* \pm 15.0	68.1 \pm 4.3	63.1 \pm 5.7	62.4 \pm 6.4	69.5 \pm 9.3	
	placebo	78.9 \pm 4.3	73.1 \pm 3.5	60.3 \pm 5.0	58.1 \pm 4.3	60.3 \pm 2.9	54.5 \pm 2.9	51.0 \pm 4.3	
Cortisol, ng/ml	energy drink	263 \pm 34	272 \pm 33	275 \pm 34	277 \pm 22	286 \pm 28	284 \pm 26	293 \pm 26	
	placebo	266 \pm 34	235 \pm 22	235 \pm 26	250 \pm 24	250 \pm 22	264 \pm 26	262 \pm 24	
Epinephrine, pg/ml	energy drink	83.6 \pm 10.8	72.7 \pm 10.0		461.0* \pm 122.5		470.8* \pm 96.9	1,011.8* \pm 295.1	
	placebo	75.5 \pm 12.0	68.4 \pm 12.6		195.6 \pm 43.8		219.5 \pm 55.4	287.1 \pm 69.7	
Norepinephrine, pg/ml	energy drink	283.1 \pm 68.2	287.4 \pm 43.5		1,971.3 \pm 287.0		1,943.9 \pm 293.9	2,476.3 \pm 297.2	
	placebo	225.7 \pm 44.5	265.4 \pm 31.8		1,584.5 \pm 216.6		1,492.2 \pm 244.0	2,211.8 \pm 442.0	
β -Endorphin, ng/ml	energy drink	0.47 \pm 0.04	0.45 \pm 0.05					0.72 \dagger \pm 0.9	
	placebo	0.48 \pm 0.06	0.44 \pm 0.06					0.59 \pm 0.04	

Abbreviations: PreD, predrink; PreEx, preexercise; ExC, exercise completion.

* $p < .05$. $\dagger p = .10$.

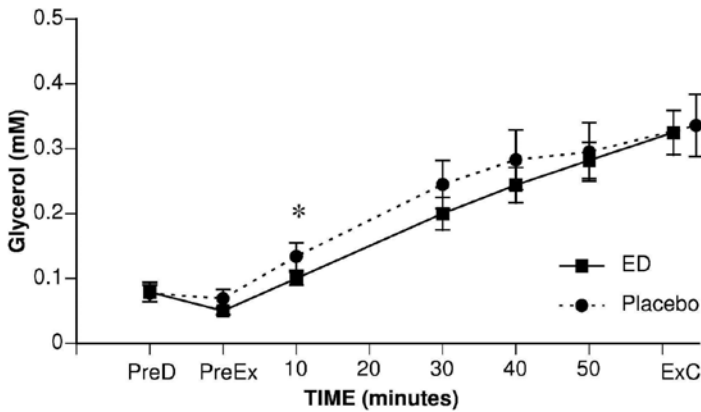


Figure 5 — Blood glycerol before ingestion of energy drink (ED) or placebo (PreD), immediately before the time trial (PreEx), during the time trial, and on completion of the time trial (ExC). *Significant difference between treatments ($p < .05$).

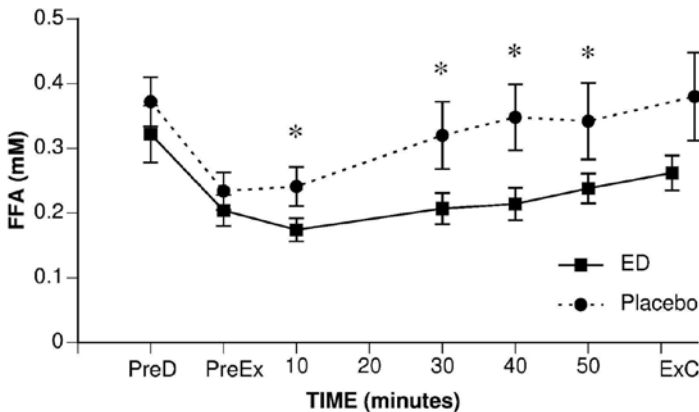


Figure 6 — Blood free fatty acids (FFA) before ingestion of energy drink (ED) or placebo (PreD), immediately before the time trial (PreEx), during the time trial, and on completion of the time trial (ExC). *Significant difference between treatments ($p < .05$).

increased during the placebo trial but remained relatively stable during the ED trial. Plasma FFAs were significantly higher during the placebo trial from 10 min to 50 min of exercise.

With exercise duration, carbohydrate oxidation decreased and fat oxidation increased in both trials (Figure 7). Although there was a trend for carbohydrate oxidation to be higher during the ED trial after 45 min of exercise, carbohydrate oxidation did not differ significantly between treatments ($p = .09$).

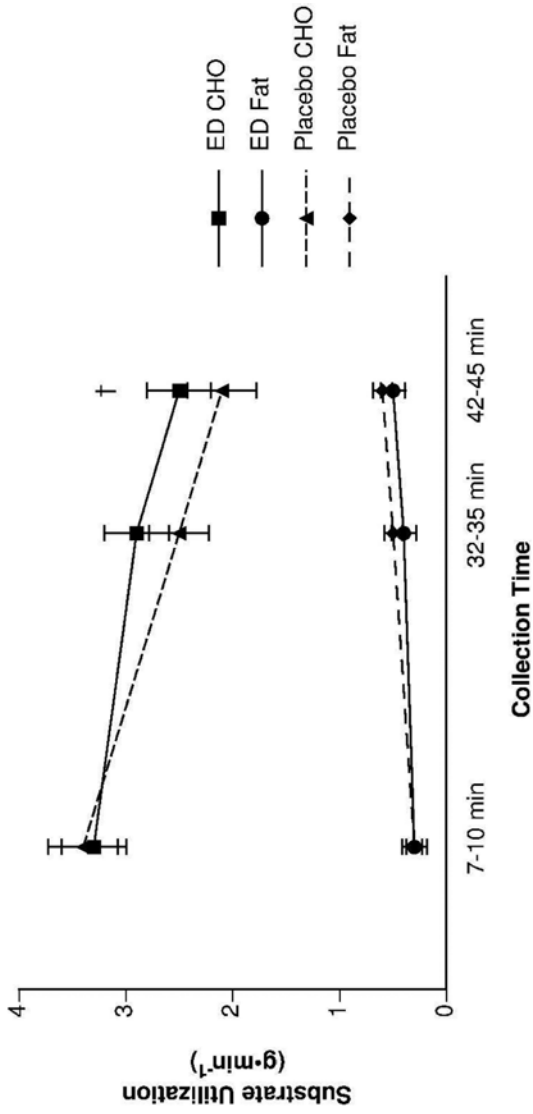


Figure 7 — Average substrate utilization at 7–10, 32–35, and 42–45 min of cycling. †Difference between treatments approaching significance ($p = .09$).

Discussion

The major finding of this study was that ingesting an ED containing carbohydrates, taurine, glucuronolactone, caffeine, and several B vitamins 40 min before exercise improved performance on a 1-hr cycling time trial. The improvement averaged 4.7%, with 83% of participants demonstrating a positive effect. Our performance improvements after ED are consistent with those found by Alford, Cox, and Wescott (2001), in whose study performance was indirectly measured as the length of time participants exercised and maintained HR within 65–75% of HR_{max} , and Geiß, Jester, Falke, Hamme, and Waag (1994), in whose study performance was directly measured as time to exhaustion.

The mechanism by which ED improved performance is not immediately clear. It is well established that providing carbohydrate during prolonged aerobic exercise increases endurance and exercise performance (Coyle et al., 1986; Ivy et al., 1983, 2003; Yaspelkis et al., 1993). This improvement in exercise performance is believed to be related to maintaining an adequate glucose supply to the active muscles (Coyle et al.). Research indicates, however, that providing carbohydrate 30–60 min before exercise will increase the plasma insulin concentration before exercise and that this causes a rapid decline in blood glucose because of the suppression of liver glucose output and increase in muscle glucose uptake. Elevated plasma insulin also inhibits lipolysis and therefore increases reliance on muscle glycogen as a fuel source. Not surprisingly, providing carbohydrate 30–60 min before exercise generally has not been found to enhance endurance performance (Foster et al., 1979; Hargreaves et al., 1987; Kuipers et al., 1999; Sherman et al., 1989) even when participants have previously fasted for 12 hr. Therefore, it is unlikely that the ED increased exercise performance by simply providing additional calories or maintaining blood glucose availability. This is supported by the finding that blood glucose during exercise did not decline during either treatment and did not differ between treatments.

The finding that substrate utilization was not different between treatments, particularly early in exercise, suggests that the caffeine in the ED might have blunted the normal increased reliance on carbohydrate as substrate when carbohydrate is provided before exercise. It also suggests that caffeine might be primarily responsible for the improvement in exercise performance despite a blinded design (Foad, Beedle, & Coleman, 2008; Geiß et al., 1994). Costill et al. (1978) found that providing 500 mg of caffeine 1 hr before exercise increased time to exhaustion during cycling at 80% VO_{2max} . Ivy et al. (1979) reported that 500 mg of caffeine provided in divided doses before and during exercise increased the amount of work that could be accomplished during 2 hr of cycling. Similar to Cole et al. (1996), these studies found that caffeine raised plasma FFA levels and increased reliance on fat as substrate during exercise. In the current study, the ED did not increase but actually decreased plasma FFA levels. Although the 40 mg of niacin in the ED might have contributed, along with an elevated insulin response, to inhibition of lipolysis and subsequent release of FFAs from adipocytes, this niacin dose is lower than typical pharmacologic doses of 500 mg or more that are prescribed to alter blood lipid levels. In addition, if niacin were the primary inhibitor of plasma FFAs, rebounding of FFA levels during the ride would be expected, which did not occur (Carlson, 1963; Kamanna, Vo, & Kashyap, 2008). It is

possible, however, that caffeine was able to offset elevated carbohydrate utilization by increasing reliance on intramuscular triglyceride stores during the early phase of the time trial (Essig et al., 1980).

Cortisol was similar between treatments, as was norepinephrine. Epinephrine, however, was significantly elevated during the ED trial. Caffeine has been found to elevate epinephrine levels during exercise (Spriet et al., 1992). Although epinephrine is generally associated with an increase in muscle glycogen utilization, studies have found that it can increase intramuscular triglyceride oxidation (Langfort et al., 1999; Watt, Stellingwerff, Heigenhauser, & Spriet, 2003) and inhibit muscle glucose uptake and transport (Aslesen & Jensen, 1998; Hunt & Ivy, 2002; Jensen, Aslesen, Ivy, & Brørs, 1997; Kipnis & Cori, 1959). It is possible that the rise in epinephrine levels resulting from ED ingestion increased intramuscular triglyceride oxidation early in exercise and spared muscle glycogen, as found by Essig et al. (1980) and Spriet et al. This would have increased its potential as substrate during the second half of the time trial, when performance during the ED treatment was superior to that of placebo. A higher blood lactate level ($p < .05$) and rate of carbohydrate oxidation ($p = .09$) during the latter stage of the ED than the placebo time trial supports this hypothesis.

The effects of the caffeine on exercise performance might not have been limited to possible shifts in substrate utilization. Ivy et al. (1979) reported that caffeine increased self-selected exercise intensity early in exercise and that, relative to exercise intensity, rating of perceived exertion was lowered by caffeine. That is, with caffeine participants were able to exercise more intensely than with placebo but with the same perception of effort. Similarly, Cole et al. (1996) observed that compared with placebo, a greater amount of work was performed at predetermined levels of perceived exertion after participants consumed 6 mg caffeine/kg body weight 1 hr before exercise. This ability to influence the psychological state and alter pain perception can significantly affect exercise performance. During high levels of physical activity, an increase in the release of β -endorphins has been proposed to limit discomfort and pain, invoke euphoria, and reduce sensation of effort (Gambert et al., 1981; Laurent et al., 2000). In this regard, Laurent et al. reported that after 2 hr of cycling to exhaustion, plasma β -endorphin levels were significantly elevated when participants consumed 6 mg caffeine/kg body weight 90 min before exercise. In the current study, we found that at the end of exercise β -endorphin levels were higher for the ED trial than for the placebo trial. This difference, however, only approached significance ($p = .10$).

It is also possible that caffeine improved exercise response during the ED trial by increasing or maintaining a high central nervous system drive. It has been found that caffeine inhibits the brain's adenosine receptors (Davis et al., 2003). During exercise adenosine levels increase in skeletal muscle and blood in proportion to the rate of ATP hydrolysis. Although it has not been determined how adenosine levels change in the brain during exercise, adenosine can cross the blood-brain barrier (Latini & Pedata, 2001). On binding to its receptors, adenosine lowers brain dopamine levels (Feuerstein, Hertting, & Jackisch, 1985; Hauber & Munkle, 1997). This lowers the serotonin:dopamine ratio, resulting in sensations of weariness and central nervous system fatigue (Garrett & Griffiths, 1997; Myers & Pugsley, 1986). By blocking the adenosine receptors, it is believed that caffeine

is able to maintain alertness and vigor and delay sensation of fatigue (Davis et al.).

It is also interesting that the amount of caffeine consumed averaged only 2.35 mg/kg body weight, which is at the lower end of caffeine dosage found to be effective. Thus, it is possible that the caffeine was working in combination with the other functional ingredients in the ED and that this combination is required to obtain the improvement in exercise performance observed. For example, taurine is known to modulate mood and enhance the positive effects of caffeine on alertness (Mandel et al., 1985). It might also help improve performance by enhancing skeletal-muscle contractility (Pierno et al., 1998; Warskulat et al., 2004). Geiß et al. (1994) compared ED with and without taurine and found that when caffeine was held constant, the drink containing taurine elicited better performance. Similarly, Zhang et al. (2004) observed increases in $\text{VO}_{2\text{max}}$, time to exhaustion, and maximum workload in participants supplemented with taurine for 1 week. In addition to taurine, the B vitamins are known to play an important role in energy metabolism (Manore, 1994; Suboticanec, Stavljenic, Schalch, & Buzina, 1990). Additional research, however, will be required to determine the contributions of each ingredient to exercise performance.

In summary, it was found that consuming 500 ml of the ED 40 min before a 1-hr cycling time trial improved performance. Therefore, ED might be used effectively preexercise when supplementation during exercise is not possible. There are several possible mechanisms that could account for this improvement, including increased caloric availability and sparing of muscle glycogen. The finding that rating of perceived exertion was similar for ED and placebo while work rate was higher for ED, however, suggests that the positive effect of ED on performance was in part neuronal in nature.

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