CYCLING TIME TRIAL PERFORMANCE IMPROVED BY INGESTION OF A
CAFFEINE ENERGY DRINK

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Running Title: Energy drink improves exercise performance

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ABSTRACT

Not all athletic competitions lend themselves to supplementation during the actual event, underscoring the importance of pre-exercise 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 prior to exercise, on endurance performance. **Methods:** The study was a double-blind, randomized, crossover design. Following a 12h fast, 6 male and 6 female trained cyclists (mean age 27.3±1.7yr; mass 68.9±3.2kg; and VO$_2$ 54.9±2.3ml•kg$^{-1}$•min$^{-1}$) consumed either 500ml of flavored placebo or 500ml of Red Bull® Energy Drink (ED) (2.0g taurine, 1.2g glucuronolactone, 160mg caffeine, 54g carbohydrate, 40mg niacin, 10mg pantothenic acid, 10mg vitamin B6 and 10µg vitamin B12) 40min prior to a simulated cycling time trial. Performance was measured as time to complete a standardized amount of work equal to 1h of cycling at 70% W$_{MAX}$. **Results:** Performance improved with ED compared to placebo (3690±64sec vs. 3874±93sec, 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, was not different between treatments. **Conclusion:** These results demonstrate that consumption of a commercially available ED prior to exercise can improve endurance performance, and that this improvement might be due in part to increased effort without a concomitant increase in perceived exertion. **Key words:** aerobic endurance, β-endorphins, catecholamines, caffeine, glucose, taurine
INTRODUCTION

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 one-hour or less. Therefore, it becomes important to determine the most appropriate type of pre-exercise supplement to extend endurance and improve exercise performance.

The traditional sports drink composed of glucose and electrolytes may 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 et al., 1986; Ivy et al., 1983; Ivy et al., 2003; Sherman et al., 1989; Yaspelkis et al., 1993). However, the increased performance time-to-fatigue, or increased power output during the later stages of prolonged exercise occurs when supplementing during exercise. The effect of carbohydrate supplementation when provided prior to exercise, however, is equivocal with a few studies demonstrating a positive effect (Kirwan et al., 1998; Sherman et al., 1989), but most studies indicating a reduced (Foster et al., 1979) or no performance-enhancing effect (Hargreaves et al., 1987; Kuipers et al., 1999; Sherman et al., 1989).

Conversely, unlike carbohydrate supplementation, caffeine appears to have a very positive effect on exercise performance when taken prior to exercise as well as during exercise (Costill et al., 1978; Graham & Spriet, 1991; Ivy et al., 1979; Kovacs et al., 1998; Pierno et al., 1998; Spriet et al., 1992). Supplements of caffeine containing from 3 to 9 mg•kg\(^{-1}\) body weight provided 1 hour before exercise have been found to be effective in increasing exercise time-to-exhaustion at exercise intensities of 70 to 80% \(\text{VO}_{2\text{MAX}}\) (Graham & Spriet, 1995; Kovacs et al., 1998; Pasman et al., 1995).
Many of the new energy drinks currently being sold are marketed with the assertion of increasing endurance and improving 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 may also contribute to endurance performance by changing perception of effort. However, there has been little research to confirm the beneficial effects of such drinks, particularly when provided pre-exercise.

The purpose of the proposed study was to evaluate the exercise performance of trained cyclists after ingesting 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 prior to the onset of exercise.

**METHODS**

*Subjects.* The subjects were 6 male and 6 female competitive cyclists accustomed to cycling time trials. They averaged 27.3±1.7 yr of age (M = 27.5±3.0 yr; F = 27.0±2.0 yr), weighed an average of 68.9±3.2 kg (M = 75.1±3.7 kg; F = 62.6±3.8 kg) and had an average maximal aerobic power (VO$_{2\text{MAX}}$) of 54.9±2.3 ml•kg$^{-1}$•min$^{-1}$ (M: 60.5±1.8 ml•kg$^{-1}$•min$^{-1}$; F: 49.4±2.7 ml•kg$^{-1}$•min$^{-1}$). Before testing, all subjects 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 the start of the study.

**Experimental design.** A double blind, randomized, placebo-controlled, two-period, within-subjects crossover experimental design was used. Each subject completed two randomly-assigned treatments in which either 500mL of an artificially colored, flavored and sweetened water placebo (PL), or Red Bull® Energy Drink (ED) was provided 40 min prior to exercise since caffeine peaks in the blood within 30 to 60 minutes after consumption (Blanchard & Sawers, 1983; Cole et al., 1996; Essig et al., 1980; Graham & Spriet, 1995; Liguori et al., 1997). Red Bull GmbH, Fuschl am See, Austria provided the placebo and ED in 250ml units. Subjects consumed 500ml 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 500mL of ED consisted of 2.0g taurine, 1.2g glucuronolactone, 160mg caffeine, 54g carbohydrates, 40mg niacin, 10mg pantothenic acid, 10mg vitamin B6 and 10µg vitamin B12. At the beginning of exercise and every 20min thereafter, 300ml of water was provided. The subjects 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.** Subjects reported to the laboratory before the start of their experimental trials for determination of their VO$_{2\text{MAX}}$ and maximal workload (W$_{\text{MAX}}$). They also performed a practice trial. The 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 VO$_{2\text{MAX}}$/W$_{\text{MAX}}$ consisted of a 4 min warm up and then 2 min stages beginning at 100 watts (W) and increasing the workload by 50W every 2 min until 350W. After
350W the increase was 25W every minute until fatigue. Subjects breathed through a Daniel's valve, with expired gases directed to a mixing chamber for analysis of oxygen (O₂) and carbon dioxide (CO₂). 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, O₂ consumption (VO₂), CO₂ production, and respiratory exchange ratio (RER) every 30s. The criteria used to establish VO₂MAX was a plateau in VO₂ with increasing exercise intensity and RER > 1.10.

Two to three days after the VO₂MAX/WMAX test the subjects reported to the laboratory to perform a practice ride to familiarize them with the laboratory environment and the experimental protocol to be used. No blood samples were collected during the practice ride. The practice ride consisted of completion of a one-hour time trial. The ergometer was set in the linear, or pedal-rate dependent mode. After a short warm-up (5 min at 100W) the subjects performed a standardized amount of work equal to about one-hour of cycling as fast as possible. The measure of performance was the time to complete the target amount of work based on the subject’s WMAX and calculated according to Jeukendrup et al. (1996):

\[
\text{Target amount of work (J) = 0.75 \times W_{MAX} \times 3600 (s)}
\]

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 subject’s WMAX at 90 RPM. This corresponds to 75% WMAX at about 100 RPM, which is close to the preferential pedaling rate of most competitive cyclists. Subjects were informed that pedaling at 90 RPM or higher would complete the target amount of work in under 1h.

The subjects were instructed to maintain a training and dietary log for two days before the first experimental trial. The subjects were provided a copy of their training and dietary log and were
instructed to have the same dietary intake and activity the 48 h before their second trial. In addition, subjects 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 subjects reported to the lab in the morning after a 12 h fast during which time they were allowed to consume only water. On reporting 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 monitor (Polar Beat, Polar Electro Oy, Finland) was secured in place around the subject’s chest. A blood sample was drawn, and either the placebo or ED (500mL each) was provided in random order 40 min before the start of the time trial. Approximately 10 min before the start of the time trial, the subject was required to void after which resting heart rate (HR) was recorded. The subject then mounted the ergometer and a second blood sample drawn. Following the drawing of the blood sample, the time trial commenced as described for the practice trial.

Neither the subjects nor the investigators were aware as to whether the subjects were on the placebo or ED treatment, as 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 subjects during each trial. During each ride subjects were not aware of how long they had ridden, as all timing devices were removed from the sight of the subjects.

Sample collection and analyses. Ventilation, VO₂, CO₂ production, and RER were recorded with the respiratory gas analysis system described previously. Respiratory gas collections were measured between 5 to 10, 30 to 35, and 40 to 45 min of exercise. Only the last 3 min for each
collection were used to determine VO2 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 (ranging from 6 to 20) were obtained during exercise at the same time points as heart rate. Five milliliters of venous blood were drawn before ingestion of the treatment, while the subjects 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 0.3mL of EDTA (24mg/mL, pH 7.4) and reduced glutathione. A sample of this anticoagulated blood (0.5mL) was then transferred to another tube containing 1mL 10% perchloric acid. One drop of anticoagulated blood (150µL) was then used to measure blood glucose concentration in duplicate with a glucose meter (One Touch Basic, LifeScan, Inc., Milpitas, California, USA). Before using the glucose meter its validity and reliability were verified by comparing values obtained from the glucose meter 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, Inc.

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. Respectively, 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 (FFA). 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, Cat. No. 8E-S100-UGB). Following extraction, samples were dried under nitrogen using a SpedVac (Savant Instruments, Inc. Farmingdale, NY) and resuspended prior to 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-diolspecific affinity gel. Epinephrine and norepinephrine were then acylated to N-acylepinephrine and N-acylnorepinephrine, 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 et al. (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 means ± standard error of the mean (SEM).

RESULTS

Time to complete. Time to complete the time trial was significantly improved when subjects received ED prior to 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 subjects, 10 completed the time trial faster on ED than on placebo, whereas 2 subjects had very similar times for both drinks (Figure 1).

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

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

Glucose. Blood glucose during the ED trial was significantly higher than during the placebo trial immediately prior to exercise (Figure 3). However, by 10 minutes of exercise, blood glucose levels had returned to baseline during the ED trial and remained 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.

Lactate. There was a significant treatment and time effect for blood lactate. During exercise blood lactate rose significantly during both trials (Figure 4). However, blood lactate was significantly higher at pre-exercise, at 30 and 50 min of exercise and at completion of exercise for the ED treatment compared to placebo.

Insulin. ED significantly elevated plasma insulin levels immediately prior to 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 than placebo until completion of exercise. Plasma insulin levels were relatively stable during the placebo trial.

**Cortisol.** 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.** β-endorphin levels were similar between treatments pre-drink and immediately prior to exercise (Table 2). At the completion of exercise β-endorphin levels had risen slightly in both trials with the increase higher for ED. However, the difference between ED and placebo was not statistically significant (p=.10).

**Epinephrine.** Epinephrine levels were similar between treatments pre-dri nk and immediately prior to exercise. Epinephrine increased during exercise in both trials. However, the increase in epinephrine was significantly higher during the ED trial at 30 and 50 min during exercise and immediately post exercise (Table 2). Importantly, there was a very large increase in plasma epinephrine at the end of exercise during the ED treatment.

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

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

**FFA.** There was a significant treatment by time effect for plasma FFA (Figure 6). FFA decreased following consumption of ED and placebo. During exercise, FFA increased during
the placebo trial, but remained relatively stable during the ED trial. Plasma FFA were significantly higher during the placebo trial from 10 min to 50 min of exercise.

Substrate utilization. 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).

DISCUSSION
The major finding of this study was that ingestion of an ED, containing carbohydrates, taurine, glucuronolactone, caffeine and several B-vitamins, 40 min before exercise improved a one-hour cycling time trial. The improvement averaged 4.7%, with 83% of the subjects demonstrating a positive effect. Our performance improvements after ED are consistent with Alford et al. (2001), where performance was indirectly measured as the length of time subjects exercised and maintained heart rate within 65-75% HR_{MAX} and Geiß et al. (1994), where 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; Ivy et al., 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., 1986). However, research indicates that providing carbohydrate 30 to 60 min prior to exercise will increase the plasma insulin concentration prior to exercise, and that this causes a rapid decline in blood glucose due to the suppression of liver glucose output and increase in muscle glucose uptake. Elevated plasma
insulin also inhibits lipolysis and therefore increases reliance of muscle glycogen as a fuel source. Not surprisingly, providing carbohydrate 30 to 60 min prior to 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 subjects have been previously 12 h fasted. 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 may have blunted the normal increased reliance on carbohydrate as substrate when carbohydrate is provided prior to exercise. It also suggests that the caffeine may be primarily responsible for the improvement in exercise performance despite a blinded design (Foad et al., 2008; Geiß et al., 1994). Costill et al. (1978) found that providing 500mg of caffeine 1 h prior to exercise increased time-to-exhaustion while cycling at 80% VO\textsubscript{2MAX}. Ivy et al. (1979) reported that 500mg of caffeine provided in divided dosages prior to and during exercise could increase the amount of work that could be accomplishing during 2 h 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 present study, the ED did not increase, but actually decreased plasma FFA levels. While the 40mg of niacin in the ED may 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 500mg or more that are prescribed to alter blood lipid levels. Also, 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 et al., 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. However, epinephrine 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 et al., 2003) and inhibit muscle glucose uptake and transport (Aslesen & Jensen, 1998; Hunt & Ivy, 2002; Jensen et al., 1997; Kipnis & Cori, 1959). It is possible that the rise in epinephrine levels due to ED ingestion increased intramuscular triglyceride oxidation early in exercise and spared muscle glycogen as found by Essig et al. (1980) and Spriet et al. (1992). 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. Support for this hypothesis is a higher blood lactate level (p<.05) and rate of carbohydrate oxidation (p=.09) during the latter stage of the ED compared to the placebo time trial.

The effects of the caffeine on exercise performance may 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 subjects were able to exercise more intensely than with placebo, but with the same perception of effort. Similarly, Cole et al. (1996) observed that compared to placebo, a greater amount of work was performed at predetermined RPE levels after consuming 6mg caffeine•kg\(^{-1}\) body weight 1 h prior to 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. (2000) reported that following 2 h of cycling to exhaustion, plasma β-endorphin levels were significantly elevated when subjects consumed 6mg caffeine •kg⁻¹ body weight 90 minutes before exercise. In the present study, we found that at the end of exercise β-endorphin levels were higher for the ED trial compared with 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 will inhibit 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). Upon binding to its receptors, adenosine lowers brain dopamine levels (Feuerstein et al., 1985; Hauber & Münkle, 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., 2003).

It is also of interest to note that the amount of caffeine consumed averaged only 2.35mg•kg⁻¹ 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 see the improvement in exercise performance
observed. For example, taurine is known to modulate mood and to enhance the positive effects of caffeine on alertness (Mandel et al., 1985). Taurine may also help to 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 when caffeine was held constant, the drink containing taurine elicited better performance. Similarly, Zhang et al. (2004) observed increases in VO\textsubscript{2MAX}, time-to-exhaustion and maximum workload in subjects supplemented with taurine for one week. In addition to taurine, the B vitamins are known to play an important role in energy metabolism (Manore, 1994; Suboticanec et al., 1990). Additional research, however, will be required to determine the contributions of each ingredient on exercise performance.

In summary, it was found that consuming 500ml of the ED 40 min before a 1 h cycling time trial improved performance. Therefore, ED may be used effectively pre-exercise 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. However, the finding that rating of perceived exertion was similar for ED and placebo while work rate was higher for ED suggests that the positive effect of ED on performance was in part neuronal in nature.
REFERENCES


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FIGURE CAPTIONS

Figure 1. Time trial times for each subject following ingestion of the energy drink (ED) or Placebo. Means ± 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).

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

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

Figure 7. Average substrate utilization between 7-10, 32-35 and 42-45 min of cycling. †Difference between treatments approaching significance (p=.09).
Table 1. Heart rate (HR) and rating of perceived exertion (RPE) for energy drink (ED) and placebo (P).

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Data are means ± SEM. *Significant differences between treatments (p<.05). Abbreviations are:

PreD, pre-drink; PreEx, pre-exercise; ExC, exercise completion.
Table 2. Plasma hormone concentration for energy drink (ED) and placebo (P) before and during exercise.

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Data are means ± SEM. *Significantly different (p<.05); † p=.10.

Abbreviations are: PreD, pre-drink; PreEx, pre-exercise; ExC, exercise completion.
Figure 1
Figure 2

Work (% Completed) vs. TIME (minutes)

- ED
- Placebo

Figure 2
145x86mm (600 x 600 DPI)
Figure 3

Glucose (mM)

TIME (minutes)

PreD PreEx 10 20 30 40 50 ExC

ED

Placebo

*
Figure 5

Glycerol (mM) vs TIME (minutes)

- ED
- Placebo

*Significant difference
Figure 6

Comparison of FFA (mM) levels between ED and Placebo groups over time (in minutes). The graph shows a significant decrease in FFA levels for the ED group compared to the Placebo group, with significant differences marked by asterisks (*) at certain timepoints. The ED group maintains lower FFA levels throughout the exercise period (ExC).
Figure 7
201x94mm (600 x 600 DPI)