Effects of Coenzyme Athletic Performance System as an Ergogenic Aid on Endurance Performance to Exhaustion

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This study examined the effects of the Coenzyme Athletic Performance System (CAPS) on endurance performance to exhaustion. CAPS contains 100 mg coenzyme Q10, 500 mg cytochrome C, 100 mg inosine, and 200 IU vitamin E. Eleven highly trained male triathletes were given three daily doses of either CAPS or placebo (dicalcium phosphate) for two 4-week periods using a double-blind crossover design. A 4-week washout period separated the two treatment periods. An exhaustive performance test, consisting of 90 minutes of running on a treadmill (70% VO₂max) followed by cycling (70% VO₂max) until exhaustion, was conducted after each treatment period. The mean (±SEM) time to exhaustion for the subjects using CAPS (223 ±17 min) was not significantly different (p=0.57) from the placebo trial (215 ±9 min). Blood glucose, lactate, and free fatty acid concentrations at exhaustion did not differ between treatments (p<0.05). CAPS had no apparent benefit on exercise to exhaustion.

This study was based on limited evidence that supplementation with vitamin E, coenzyme Q10, cytochrome C, or inosine may improve physical performance. Although some research has been conducted on the ergogenic effects of supplementation with the individual components of CAPS (i.e., vitamin E, coenzyme Q10, cytochrome C, or inosine), no research has been reported on the effects of supplementation with a combination of vitamin E, coenzyme Q10, cytochrome C, and inosine on endurance performance to exhaustion, alterations in blood

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Table 1
Summary of Metabolic Effects of Vitamin E, Inosine, Cytochrome C, and Coenzyme Q10

| Vitamin E: | -Catalyst in electron transport chain via cytochrome C reductase  
| | -Stabilizes cytochrome b and cytochrome c  
| | -Vitamin E deficiency associated with decreased oxidative phosphorylation (i.e., decreased ATP synthesis)  
| | -Decreased oxygen debt via maintaining ATP production |
| Inosine: | -Potentially enhances ATP and GTP synthesis  
| | -Enhanced 2, 3-diphosphoglycerate synthesis  
| | -May function as a vasodilator |
| Cytochrome C: | -Electron transport: maintain/enhance ATP synthesis |
| Coenzyme Q10: | -Constituent of electron transport chain  
| | -Regulation of mitochondrial succinate dehydrogenase, NADH dehydrogenase, and cytochrome b-cl complex |

parameters at exhaustion, or ratings of perceived exertion (RPE). The metabolic pathways in which these compounds participate are summarized in Table 1.

The results of several studies on the possible benefits of vitamin E on exercise have been equivocal (11, 14, 16, 18, 20, 23). Vitamin E may provide benefits to athletes at altitudes above 1,000 meters (16). Maximal oxygen consumption increased 9% at 1,667 meters and 14% at 5,000 meters when subjects were given vitamin E for 2 weeks (16).

We are not aware of any research on the effects of cytochrome C on exercise performance; however, increases in cytochrome C concentration following exercise training have been noted (2, 4, 26). Increased cytochrome C within the mitochondria with endurance training may increase oxidative capacity. Thus, cytochrome C supplementation may enhance exercise performance. Only Williams et al. (29) have examined the effects of inosine supplementation on exercise performance. No benefits were observed from oral inosine supplementation. Hypothetically, inosine may enhance oxygen transfer to hemoglobin molecules; however, this effect may not be present in vivo (29).

The effects of coenzyme Q10 on exercise performance remain largely uninvestigated. Coenzyme Q10 status may be related to the incidence of myocardial ischemia and reportedly may enhance exercise performance in healthy individuals (13). Van Fraechem and Folkers (27) reported that maximal oxygen uptake increased significantly following the administration of coenzyme Q10.

Theoretically, the combined intake of vitamin E, cytochrome C, inosine, and coenzyme Q10 may enhance oxidative phosphorylation and ATP resynthesis at the cellular level, but there is little convincing evidence of improved endurance performance as a result of using each of these supplements. Thus the purpose of this research was to determine whether these ergogenic aids, used in combination, would enhance endurance performance to exhaustion. The manufacturers of this ergogenic aid postulate "the maintenance of optimal energy production, reduces fatigue and shortens recovery time." The proposed
mechanism of action is a consequence of increased ATP synthesis, which will simultaneously increase "oxygen flow to muscles, increase oxygen efficiency and cellular respiration, increase glycolysis and decrease lactic acid production."

Methodology

Subjects

Twelve highly trained male triathletes 18 to 35 years of age were selected from a group of 21 volunteers to participate in this double-blind, placebo, repeated-measures crossover study. All signed consent forms approved by the human subjects review committee and completed a medical history. The following criteria were used to select subjects: (a) running a minimum of 30 miles a week and cycling at least 100 miles a week for at least 1 month prior to the study; (b) VO2 max of at least 50 ml·kg⁻¹·min⁻¹ for both running and cycling; (c) completion of a 3-hr trial endurance performance test; (d) resting blood pressure below 140/90 mm Hg; and (e) total cholesterol less than 200 mg%. 

VO2Max Determination

Running VO2 max was measured using an individualized graded exercise test protocol performed on a Quinton Q55 treadmill (Quinton Instrument Co., Seattle) which included a 3-min warm-up at a speed of 5 mph. Treadmill speed was increased every minute according to the subject’s capability (usually 1 or 2 mph) following the warm-up. After a maximum speed of 10 mph was reached, the grade was increased at 2-min intervals by 1% until the subject indicated he could not maintain the workload (volitional fatigue).

Cycling VO2 max was also determined using an individualized graded exercise test protocol performed on a modified BodyGuard 990 cycle ergometer (Jonas Ogaend A.S, Sandnes, Norway) with racing toeclips. After a 3-min warm-up at a resistance of 300 to 450 kpm, the workload was increased by 200 kpm over 2-min intervals until the subject could not maintain 80 rpm (volitional fatigue). Criteria for determining VO2 max included volitional fatigue, no change in oxygen consumption as the workload increased, and a respiratory quotient above 1.10.

Volumes of expired oxygen and CO2 were monitored continuously during each max test and samples were taken from a mixing chamber. The amount of expired air was measured using an Ametek turbine flow meter (Thermox Instruments Div., Pittsburgh, PA). Ametek’s stress software package and interface link (Ametek-71911KE) were used to calculate ventilation rate (L·min⁻¹ BTPS), VO2 (ml·kg⁻¹·min⁻¹), VCO2 (ml·kg⁻¹·min⁻¹), and the ventilatory exchange ratio (R) every 30 seconds.

Treatment

Subjects were randomly assigned to either the treatment or placebo group. CAPS and the placebo (dicalcium phosphate) were packaged in identical containers which were color coded. Each CAPS capsule contained 100 mg coenzyme Q10, 500 mg cytochrome C, 100 mg inosine, and 200 IU vitamin E. Each placebo
capsule contained 500 mg dicalcium phosphate. The funding agency selected dicalcium phosphate as the placebo and had reportedly used it as a placebo in other research.

The treatment and placebo packages contained either three capsules (one dose) or six capsules (two doses). Subjects were instructed to consume the package with six capsules 1 hour before a workout and to consume the package with three capsules within 30 minutes after a workout, for a total of three doses per workout. They were given enough packages for 10 workouts a week for each treatment period (4 weeks). All subjects completed the minimum of 10 workouts each week. Since each subject consumed all of his prescribed treatments, an average of 1,286 mg of coenzyme Q10, 1,286 mg inosine, 6,430 mg cytochrome C, and 2,572 IU vitamin E was ingested each day for the CAPS treatment period, or an average of 6,430 mg dicalcium phosphate was ingested each day for the placebo treatment period.

After 4 weeks of taking either CAPS or the placebo, subjects completed the exhaustive endurance test protocol (Figure 1) and then continued training as usual for 4 weeks while no treatment was administered. Performance Tests II and III were completed during the second and fourth weeks of the washout period. Test IV was administered after the second treatment period, that is, the end of Week 12.

**Endurance Performance Tests**

Subjects were instructed to treat each performance test as a race. They did not participate in any races within 5 days prior to endurance performance tests. Humidity was not controlled; however, a window air conditioning unit helped maintain a relatively constant environment. Room air temperature was approximately 23 °Celsius.

![Figure 1](image-url) — Time periods for V̇O_{2}max tests and performance tests.
After a 12-hr fast, subjects reported to the lab and their body weights were recorded using a Detecto™ balance scale. An 18-gauge venous catheter (Deseret Medical, Inc., Sandy, UT) was placed in a forearm vein and a baseline blood sample was obtained. The catheter was flushed every 15 minutes with 0.9% sterile saline to prevent clotting.

A 4-lead EKG was placed on each subject. After running at an individually selected warm-up pace for 3 to 5 minutes, subjects began the run portion of the performance test (Figure 2), which consisted of running on a motorized treadmill for 90 minutes at 70% of running VO$_2$max. Subjects consumed cold water ad libitum throughout each test. At the end of the run the subjects’ weights were recorded. The subjects then cycled for 90 minutes on the cycle ergometer at 70% of cycling VO$_2$max. At the end of 180 minutes the workload was increased by approximately 67 kpm for a 5-min interval, which was followed by a second increase in workload of approximately 67 kpm for 5 minutes. A third increase in the workload of approximately 67 kpm was maintained until exhaustion (i.e., failure to maintain a cadence above 70 rpm). Workloads increased to correspond to approximately 75, 80, and 85% of VO$_2$max. Subjects were encouraged to perform as long as possible.

Ratings of perceived exertion (RPE) using the Borg scale were measured every 15 minutes throughout the endurance performance test. Mean overall values were calculated for the CAPS and control groups. A physician was on call during the testing.

**Incentives.** Monetary rewards served as incentives to complete the study. Subjects were paid $450 for completing all performance tests. They were also eligible to win “primes” during each test for having the longest time to exhaustion. Individual times to exhaustion for each test were not revealed until all subjects had completed that performance test.

**Blood Analysis.** Blood samples were collected at the beginning of the endurance test (resting), 45 and 90 minutes into the run, 45 and 90 minutes into

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![Figure 2 — Performance test protocol.](image-url)
the ride, and at exhaustion. All samples were analyzed in duplicate for plasma glucose, lactate, FFA, hemoglobin, and hematocrit concentrations. Energy substrates were measured in order to document that the endpoint concentrations at exhaustion were consistent with expected values.

**Plasma Volume Shifts.** Changes in plasma volume concentrations can result in erroneous measurement of blood parameters (12). Plasma volume shifts were monitored by measuring hemoglobin and hematocrit. All energy substrate values were corrected for plasma volume shifts according to the procedure of Dill and Costill (12).

**Potentially Confounding Variables**

**Diet.** Nutritional adequacy for all nutrients listed in the 1989 Recommended Dietary Allowances was assessed using 4-day food records at the beginning of the study. Subjects were individually evaluated and educated as diet plans were made. They were instructed to maintain their usual food habits throughout the study.

**Anthropometry.** Percent body fat was determined using hydrostatic weighing according to the procedures of Sinning (25). The equation of Brozek et al. (5) was used to estimate body fat.

**Iron Status.** Since low serum iron levels can adversely affect physical performance, hemoglobin and serum ferritin were measured to assess iron status. Subjects with a serum ferritin below 40 ng/dl and/or a hemoglobin below 14 g/dl were instructed to increase their iron intake. Only one subject’s serum ferritin was less than 40 ng/dl. He was asked to increase his consumption of lean red meat by two 4-oz servings a week and to drink orange juice in order to enhance iron absorption.

**Training.** Increases in performance capacity often occur as a result of training. To minimize training effects, each subject kept a training log of daily mileage and the number of long-distance and interval workouts per week. Subjects were instructed to maintain a steady level of training (varying no more than 15% from the amount during Week 1), and to keep the numbers of long-distance and interval workouts similar for each week during the study. VO2max tests were performed at Weeks 0, 5, and 11 to monitor any possible training effects on the subjects’ performance times.

**Statistical Analysis**

All statistical analyses were performed using SAS analysis software (Cary, NC). All hypotheses were tested using the p<0.05 level of significance. Analysis of variance and Duncan post hoc analyses were used to test for significant differences in the blood parameters and RPE between treatments. Times to exhaustion during the performance tests and changes in VO2max values were analyzed using a paired-comparisons t test. In addition, times to exhaustion during Performance Tests I, II, and III were compared for subjects using the placebo, in order to monitor for possible training effects during the study.
Descriptive statistics for height, weight, age, percent body fat (hydrostatic), serum ferritin, total cholesterol, HDL-cholesterol, $VO_2$max (running and cycling), and trial performance time for the 11 subjects who completed the study are summarized in Table 2. The 12th subject dropped out of the study between Weeks 4 and 6 due to a back injury, thus none of his data were included in any analysis.

Figure 3 — Individual times to exhaustion during performance tests.

Results
Time to Exhaustion

The mean time to exhaustion (Figure 3) was 8 minutes longer for the CAPS treatment (223.5 ±17.0 min) than for the placebo treatment (215.5 ±9.6 min, p=0.56). Individual times ranged from 180.2 to 384.8 minutes. The variability (SEM) was nearly twice as large when subjects were tested while taking CAPS (17.0 min) compared to the placebo (9.6 min). The range was 200 minutes between subjects when tested while taking CAPS compared to 112 minutes when taking the placebo.

As noted in Figure 3, one subject’s times to exhaustion were considerably longer than for any of the other participants. We repeated the analysis without his data; however, mean times to exhaustion between the CAPS (210 ±6 min) and placebo treatments (207 ±6 min) were still not significantly different (p=0.50). Eliminating his data reduced the variability considerably and reduced the difference between treatments from 8 to 3 minutes.

Plasma Glucose, Lactate, and FFA

Mean glucose concentrations are summarized in Figure 4. Individual plasma glucose at exhaustion ranged from 2.47 mM to 5.87 mM and showed a moderate but significant correlation (r=−0.38, p=0.04) with times to exhaustion. Plasma glucose at exhaustion did not differ significantly between treatments (p=0.34; 4.05 ±0.19 mM for CAPS and 3.65 ±0.24 mM for placebo). Glucose decreased significantly (p=0.0001) from baseline (4.74 mM) to exhaustion (3.84 mM).

Mean plasma lactate concentrations are reported in Figure 5. Lactates at exhaustion were not significantly correlated (r=−0.06, p=0.70) with times to

![Figure 4](image-url) — Plasma glucose concentrations during performance tests (CAPS vs. placebo).
Plasma lactate concentrations during performance tests (CAPS vs. placebo).

Exhaustion. No significant difference was observed ($p=0.88$) for the mean lactate concentrations between CAPS (1.72 ±0.27 mM) and the placebo (1.77 ±0.29 mM). Plasma lactate concentration changes were analyzed for all four performance tests and were unaffected by treatment.

Free fatty acid (FFA) values are summarized in Figure 6. FFA at exhaustion were not significantly correlated ($r=0.03$, $p=0.84$) with the times to exhaustion. Mean FFA were not significantly different ($p=0.81$) at exhaustion between treatments (0.78 ±0.06 mM for CAPS and 0.78 ±0.06 mM for placebo). Plasma FFA increased significantly ($p=0.05$) from baseline (0.27 mM) to exhaustion (0.78 mM) (Figure 6).

**RPE, Diet, and Body Weight**

Mean values for ratings of perceived exertion at exhaustion did not differ significantly ($p=0.67$) between the CAPS (18.0 ±0.5) and placebo (18.3 ±0.3) treatments. Although mean RPE values tended to be slightly lower for the subjects using CAPS compared to the placebo, none of the means differed significantly from each other for any time period during the test.

Mean carbohydrate intake as a percentage of total calorie intake was 56.9 ±10.7%, ranging from 39 to 72%. Mean fat and protein intakes were 27.9 ±9.2% and 14.6 ±2.6%, respectively, and ranged from 16 to 41% for fat and from 11 to 19% for protein. Calorie intake was 4,146 ±1,504 (range: 2,629 to 6,400).

Body weights were recorded at the beginning of each performance test. The maximum weight change across the four performance tests for any subject was 3.4 kg while the minimum was 1.2 kg. An analysis of variance showed no significant differences among the mean preperformance test body weights across the four performance tests.
Training Effects

In order to check for training effects, the mean time to exhaustion for Performance Test III was compared to the mean times to exhaustion for Performance Tests I and II, separately. To eliminate any bias due to the possible effects of CAPS on the time to exhaustion, values for subjects using the placebo only were employed for this analysis. In addition, VO_{2\text{max}} values for running and cycling were measured at Weeks 0, 5, and 11 for all subjects. Mean times to exhaustion for subjects consuming the placebo were not significantly different (p=0.91) between any performance tests: Test I (231.0 ±17.6 min), Test II (193.8 ±9.3 min), and Test III (232.1 ±22.2 min). Mean VO_{2\text{max}} values for running and cycling did not differ significantly between any of the time periods (i.e., Weeks 0, 5, and 11) (Table 3).

<table>
<thead>
<tr>
<th>Week</th>
<th>Run M (SEM)</th>
<th>Bike M (SEM)</th>
</tr>
</thead>
<tbody>
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<td></td>
<td>67.7 (1.4)</td>
<td>66.7 (1.9)</td>
</tr>
<tr>
<td>5</td>
<td>68.8 (1.7)</td>
<td>67.7 (1.8)</td>
</tr>
<tr>
<td>11</td>
<td>70.2 (1.6)</td>
<td>68.2 (1.8)</td>
</tr>
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</table>

Table 3
VO_{2\text{max}} Values (ml·kg^{-1}·min^{-1}) for Run and Bike
Discussion

Time to Exhaustion

The mean times to exhaustion between subjects taking CAPS and those taking the placebo were not significantly different. To date, no studies have been reported that examined the effectiveness of CAPS on exercise performance. However, in studies examining time to exhaustion, the reported times to exhaustion vary greatly in magnitude. Coyle et al. (10) observed a time to exhaustion of 134 ±6 min in 10 trained cyclists while cycling at 74% VO₂max. In an investigation using a protocol similar to the one used in this study, the mean time to exhaustion was 186 min (no SEM given) (8). The overall mean time to exhaustion in the present study was 214 ±8 min. Differences in subjects’ training status, nutritional state, motivation, and psychological state may explain the longer mean time to exhaustion observed in the present study compared to previously reported data.

The mean difference in time to exhaustion between the CAPS and placebo treatments was 8 minutes. There was considerable variability in time to exhaustion among the subjects, which ranged from 180.2 to 384.8 minutes. The largest difference in times to exhaustion among an individual subject was 92.6 minutes; the smallest difference was 0.4 minute. Eliminating the data of one subject reduced variability 50 to 67% and reduced the difference between the mean times to exhaustion between the CAPS and placebo treatments from 8 to 3 minutes.

These data are consistent with findings reported in previous studies which show no improvement in exercise performance with the supplementation of vitamin E (1, 22) and inosine (29). Only two studies have reported an increase in exercise to exhaustion following supplementation with vitamin E (11, 16), and these improvements were only observed at high altitudes. Cureton and Pohndorf (11) did not report the exact amount of vitamin E given; however, wheat germ oil was reportedly administered. The benefits derived from wheat germ oil may have been due to substances other than the vitamin E in the wheat germ oil. Kobayashi (16) found increases in cycle ergometer performance after giving 12 subjects 1,200 IU vitamin E per day for 6 weeks. In the present investigation, it seems unlikely that the dosage of vitamin E in CAPS (i.e., 2,572 IU) was not high enough or administered long enough (4 vs. 6 weeks) to elicit positive changes. It is possible that vitamin E supplementation may not be beneficial at sea level, whereas decreased plasma vitamin E concentrations may be a limiting factor in exercise performance at high altitudes when oxygen demand is increased. Exercise was performed at high altitudes (1,667 and 5,000 meters) in the study by Kobayashi, as compared to 290 meters in the present study. Thus the differences between these studies may reflect the effects of altitude.

The placebo that we used (6,430 mg dicalcium phosphate per day) may have enhanced exercise performance to exhaustion, since Cortes et al. (9) and Schenck et al. (22) recently reported that phosphate loading enhanced exercise performance. However, these investigators used sodium phosphate, which may have produced a different effect than the dicalcium phosphate used in our study. Mean performance time to exhaustion was 207.5 ±9.4 minutes for the subjects during the washout period (Performance Tests II and III combined) compared to 222.4 ±8.7 minutes, and 216.7 ±17.6 minutes for Test Periods I and IV, respectively, when the subjects were receiving either the CAPS (223.5 ±17.0 min) or the placebo (215.5 ±9.6 min) treatment.
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Plasma Glucose, Lactate, and FFA

There was no significant difference between the mean plasma glucose concentration at exhaustion between treatments. No investigations on the combined effects of vitamin E, inosine, or coenzyme Q10 have examined changes in plasma glucose concentrations during a performance test; however, a series of studies were performed using a research design similar to our study (6, 7, 8, 10). The significant decrease in plasma glucose at exhaustion compared to baseline in the present study is consistent with the findings of Coggan and Coyle (8). The data support/validate that the subjects’ blood glucose had decreased to levels associated with exhaustion.

The subjects in this study were encouraged to consume a high carbohydrate diet. Bergstrom and Hultman (3) demonstrated that increased carbohydrate consumption adds to muscle and liver glycogen reserves. Differences in glucose concentrations at exhaustion between the present study and the one by Coggan and Coyle (8) could be due to lower muscle and/or liver glycogen reserves in their subjects, or possibly to differences in the protocols used (cycling only, vs. running and cycling). Additionally, differences in glycogen reserves might explain why our subjects were able to exercise longer to exhaustion.

Mean lactates at exhaustion did not differ significantly between treatments. The effects of vitamin E supplementation on plasma lactate concentrations following exercise have yet to be clearly identified. Results from studies that have examined the effects of vitamin E supplementation on plasma lactate concentration are conflicting. Lawrence et al. (17) observed no significant (p<0.05) changes in lactate concentration following intermittent swimming sprints when subjects were given 900 IU vitamin E per day for 6 months. However, Nagawa et al. (19) observed a significant decrease (no p value given) in plasma lactate concentration after an exhaustive cycle ergometer ride at altitudes of 2,700 and 2,900 meters after supplementation with 300 mg vitamin E per day for 44 days.

When the effects of inosine supplementation on plasma lactate concentrations were examined, no significant differences were observed (29). Blood lactates generally rise during endurance exercise (6, 7, 8, 10, 15, 24). Blood lactates increased significantly from 45 minutes into the run to exhaustion. Lactates at exhaustion were not significantly correlated with the times to exhaustion, perhaps because lactic acid in the blood is not a major limiting factor in aerobic exercise (28).

There were no significant differences in FFA at exhaustion between the CAPS and placebo treatments. Presently, no investigations evaluating the effects of vitamin E, coenzyme Q10, cytochrome C, or inosine on exercise performance have examined changes in plasma FFA. A steady rise in the plasma FFA across time was observed (from resting values of 0.28 ±0.02 mM to 0.85 ±0.04 mM at exhaustion). These data are consistent with the findings of several studies examining the FFA response during exercise (0.21 mM resting and 0.91 at exhaustion, no SEM reported) (6, 7, 8, 10).

Continuation of endurance performance may be dependent on FFA utilization as an alternative energy substrate compared to glucose. The regulation of alterations in glucose and FFA utilization have been described by Randle (21). There were no significant differences in mean glucose, lactate, or FFA levels
between the CAPS and placebo treatments, suggesting that differences in energy substrate metabolism were not confounding variables in evaluating the effects of CAPS.

**Ratings of Perceived Exertion**

Mean RPE did not differ significantly between the CAPS (18.0 ±0.5) and placebo (18.3 ±0.3) treatments at exhaustion. No other study has measured RPE at exhaustion between subjects using CAPS versus those using a placebo. The increased RPE at exhaustion in this research is consistent with the findings of several studies (6, 7, 8).

**Summary and Conclusions**

The mean times to exhaustion did not differ significantly between the CAPS and placebo treatments (p=0.56), although the mean difference in time to exhaustion was 8 minutes longer for the CAPS versus placebo (phosphate) treatment, and 17 minutes longer than the washout period. In addition, the observed mean plasma glucose, lactate, and FFA concentrations, as well as RPE, were not significantly different at exhaustion between treatments. Based on these data, the use of CAPS as an ergogenic aid in extending time to exhaustion cannot be recommended. Athletes should be advised that ergogenic aids are never a substitute for an appropriate training program.

It is recommended that future research be conducted on the effects of CAPS on exercise performance using a larger sample size, additional criteria in selecting subjects, and a protocol simulating distances associated with a U.S. Triathlon Series distance triathlon (1.5-Km swim, 40-Km bike, 10-Km run). Additional research is also suggested that would examine the postabsorptive fate of the individual components of CAPS (i.e., vitamin E, inosine, cytochrome C, and coenzyme Q10).

One subject’s ride to exhaustion (384.8 min) was more than 165 minutes longer than the mean ride to exhaustion (219.5 min), and more than 204 minutes longer than the shortest ride to exhaustion (180.2 min). The subject selection criteria and larger sample size could further minimize the effects of intersubject variability and strengthen the statistical power of the analyses performed. Eliminating this subject’s data did not alter our basic findings.

Times typically associated with a USTS triathlon range from 1.8 to 2.5 hours in duration. The use of an exhaustive endurance test represents one approach for evaluating the performance and metabolic effects of CAPS. When athletes reach exhaustion, numerous variables, both metabolic and nonmetabolic, may affect performance. Thus, other measures of performance such as the improvement of running or cycling speed (determined by completion of a measured run or bike course) provide alternative methods of evaluating the potential benefits of ingesting CAPS.

**References**


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