# Caffeine Ingestion Does Not Alter Performance During a 100-km Cycling Time-Trial Performance

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This study analyzed the effect of caffeine ingestion on performance during a repeated-measures, 100-km, laboratory cycling time trial that included bouts of 1- and 4-km high intensity epochs (HIE). Eight highly trained cyclists participated in 3 separate trials—placebo ingestion before exercise with a placebo carbohydrate solution and placebo tablets during exercise (Pl), or placebo ingestion before exercise with a 7% carbohydrate drink and placebo tablets during exercise (Cho), or caffeine tablet ingestion before and during exercise with 7% carbohydrate (Caf). Placebo (twice) or 6 mg · kg$^{-1}$ caffeine was ingested 60 min prior to starting 1 of the 3 cycling trials, during which subjects ingested either additional placebos or a caffeine maintenance dose of 0.33 mg · kg$^{-1}$ every 15 min to trial completion. The 100-km time trial consisted of five 1-km HIE after 10, 32, 52, 72, and 99 km, as well as four 4-km HIE after 20, 40, 60, and 80 km. Subjects were instructed to complete the time trial and all HIE as fast as possible. Plasma (caffeine) was significantly higher during Caf (0.43 ± 0.56 and 1.11 ± 1.78 mM pre vs. post Pl; and 47.32 ± 12.01 and 72.43 ± 29.08 mM pre vs. post Caf). Average power, HIE time to completion, and 100-km time to completion were not different between trials. Mean heart rates during both the 1-km HIE (184.0 ± 9.8 Caf; 177.0 ± 5.8 Pl; 177.4 ± 8.9 Cho) and 4-km HIE (181.7 ± 5.7 Caf; 174.3 ± 7.2 Pl; 175.6 ± 7.6 Cho; $p < .05$) was higher in Caf than in the other groups. No significant differences were found between groups for either EMG amplitude (IEMG) or mean power frequency spectrum (MPFS). IEMG activity and performance were not different between groups but were both higher in the 1-km HIE, indicating the absence of peripheral fatigue and the presence of a centrally-regulated pacing strategy that is not altered by caffeine ingestion. Caffeine may be without ergogenic benefit during endurance exercise in which the athlete begins exercise with a defined, predetermined goal measured as speed or distance.

*Key Words:* caffeine, cycle time trial, performance, EMG

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## Introduction

The modern interest in the possible performance-enhancing effects of caffeine were initially raised by the finding of Costill and co-workers (8, 12, 21) that caffeine ingestion prior to exercise increased time to exhaustion or the amount of work that could be performed in a known time.

An early theory was that caffeine effects substrate metabolism during exercise. The glucose–fatty acid cycle theory (35) postulated that caffeine would enhance performance by increasing fat oxidation during exercise, thereby reducing the rate of muscle glycogen use. Since muscle glycogen depletion is believed to be the principal determinant of exhaustion during prolonged exercise, this effect of caffeine would delay the onset of terminal glycogen depletion, thereby enhancing performance (12).

More recently, there has been a considerable increase in evidence (48) supporting a substantial ergogenic effect of caffeine on endurance but not sprint activities (10, 15, 18, 34). The vast majority of those studies showing an ergogenic effect of caffeine on endurance exercise have evaluated performance during open-ended cycling (1, 2, 17, 22, 33) or running (10, 14) trials in which subjects maintain the same exercise intensity for as long as possible.

It is difficult to extrapolate findings from time-to-exhaustion trials to human sporting performance, since most human competitive sport requires either that a specified distance must be completed as rapidly as possible or, much less commonly, as much distance as possible must be covered in a given time, also fixed and known before the exercise begins.

To our knowledge, very few studies have evaluated the effects of caffeine ingestion on performance during a prolonged laboratory trial in which the endpoint is predetermined, not open-ended as in the majority of the reported studies. It is known that inter-test variability is substantially reduced in this form of closed trials compared to open-ended trials (37). However the very marked ergogenic effect of caffeine in the open-ended trials that have been reported (2, 10, 14, 17, 22, 33), precluded the possibility that a false positive finding might have resulted from the relative insensitivity of that experimental method.

Although an ergogenic effect of caffeine has been reported in runners (46), cyclists (25), and rowers (5) who completed closed performance time trials in which a specified distance was completed in a faster time after caffeine ingestion, the duration of these trials were relatively brief, lasting from 4 min (46) to 60 min (25).

There are good reasons why the effects of caffeine-ingestion should also be investigated during exercise in which the endpoint is predetermined and the duration of exercise is more prolonged—for example, hours rather than minutes. First, the majority of recreational athletes for whom advice about caffeine ingestion is provided by exercise scientists, probably compete in long-distance running and cycling events, including marathons and triathlons, that last hours rather than minutes. Second, the effects of caffeine could conceivably differ depending not only on the type of exercise undertaken (closed vs. open trials) but also on its duration. Third, the method of action of caffeine could conceivably be different under different circumstances. For example, it would be unlikely that a glycogen sparing effect would explain any ergogenic effect of caffeine during exercise of short duration, whereas that effect could be important during more prolonged exercise, lasting some hours.

Accordingly in this study, we evaluated the effects of caffeine ingestion on performance during a 100-km cycling time trial, which included high intensity epochs (HIE). This protocol was previously used to study the effects of carbohydrate loading (6).

## Methods

### Subjects

Fifteen competitive, endurance-trained male cyclists volunteered for this study, with 8 completing the full trial. Seven of the original 15 subjects were excluded—2 because they were unable to achieve the required cycling speed and 5 because they found the trial "too arduous". At the time of the study, the trialists were cycling between 200–500 km · wk$^{-1}$ and had completed at least one local, specified, annual 104-km road race under 3 hours during the previous 2 years. Subjects were well-trained and accustomed to exercising for prolonged periods (3–4 hours). The mean age, weight, body fat, $\dot{V}O_{2max}$, and peak power output (PPO) of the 8 subjects who completed the trial were 23.5 ± 6.7 years, 66.8 ± 6.2 kg, 10.6 ± 3.5%, 64.6 ± 7.9 ml · kg$^{-1}$ · min$^{-1}$ and 385 ± 61 W, respectively. All subjects were fully informed of the nature of the investigation, which was to evaluate the effects of caffeine or carbohydrate drink ingestion on cycling performance, after which they gave written informed consent. The study was approved by the Research and Ethics Committee of the Faculty of Health Sciences within the University of Cape Town.

### Kingcycle Ergometry System

All testing was conducted on a Kingcycle ergometry system (Kingcycle Ltd., High Wycombe, UK), which allows cyclists to ride on their own racing bicycles in the laboratory. After the front wheel was removed, the subject's bicycle was attached to the ergometry system by the front fork and supported by an adjustable pillar under the bottom bracket. The bottom bracket support was used to position the rolling resistance of the rear wheel correctly on an air-braked flywheel. A photo-optic sensor monitored the velocity of the flywheel in revolutions per second (RPS), from which an IBM-compatible computer calculated the power output (W) that would be generated by a cyclist riding at that speed on a level terrain, using the following equation:

$$W = 0.000136 \, RPS^3 + 1.09 \, RPS$$

The Kingcycle was calibrated before both the incremental tests to exhaustion and time trials. For the calibration, subjects were asked to accelerate to a workload of ~200 W and instructed to immediately stop pedaling as soon as they reached the desired workload, while remaining seated in their riding position. The bottom bracket support was then adjusted until the computer display indicated that the slowing of the flywheel matched a predetermined reference power decay curve. The time taken for a laboratory simulated 20-km and 40-km time trials on the Kingcycle ergometer system has previously been shown to be highly reproducible (CV 1.1 ± 0.9% and 1.0 ± 0.5%, respectively; 20, 32).

## Preliminary Testing

To determine PPO, the modified protocol as described by Hawley and Noakes (19) was used. Subjects performed a 10-min warm up on the Kingcycle. The starting power output was determined by multiplying the subject's body weight by 3.3 W. The load was subsequently increased at a rate of $1 \text{ W} \cdot \text{kg}^{-1}$ of body weight $\cdot 150 \text{ s}^{-1}$. The subject was required to match a continuously increasing power output displayed in analogue form on the computer monitor. The test was terminated when the subject failed to match the target power. The highest mean power output achieved during any 150-s period was recorded as the subject's PPO. The PPO was subsequently converted into a predicted $\dot{V}O_{2max}$ value using the following equation as described by Keen et al. (24):

$$\dot{V}O_{2max} \text{ (L} \cdot \text{min}^{-1}) = 0.011 \text{ PPO (W)} + 0.081$$

## Experimental Procedure

After measurement of the PPO, each subject reported to the laboratory on four separate occasions. During the first visit, subjects familiarized themselves with the equipment and laboratory conditions; thereafter, they completed a familiarization 100-km cycling time trial (TT), during which they ingested their own chosen fluid replacement solutions (e.g., cordial, water, and sports drinks) ad libitum. All other conditions were identical except that subjects were also not given tablets, nor were they forced to drink at specific, regular intervals during the familiarization trial. Subjects completed a TT during each of the next three visits. Each visit was separated by a minimum of 6 and a maximum of 8 days.

For each subject, the 3 TTs were conducted at the same time of day in the environmental chamber (Scientific Technology Corporation, Cape Town, South Africa) at an ambient temperature of 27 °C, a relative humidity (Rh) of $50 \pm 0.9\%$, and a wind velocity (v) of $15 \pm 0.4 \text{ km} \cdot \text{h}^{-1}$. The trials were randomized, and subjects were blinded to the nature of each trial. The subjects were requested to perform the same type of training for the duration of the trial and to refrain from heavy physical exercise on the day before a TT. Subjects completed a nutritional information sheet on which they recorded their food and fluid intake for 3 days preceding the TT. Subjects were instructed to avoid any caffeine containing products for 48 hours before each time trial. They were specifically asked to abstain from all the obvious sources of caffeine including coffee, tea, cola drinks, chocolate, and over-the-counter caffeine containing pharmaceuticals. They were requested to report any deviations from these instructions. The day prior to the trial, the subjects followed a prescribed diet, which consisted of $5 \text{ g} \cdot \text{kg}^{-1}$ of carbohydrate and $1.3 \text{ g} \cdot \text{kg}^{-1}$ protein (60% and 17%, respectively). They were instructed to repeat the same dietary regimen before each subsequent trial.

In addition, before each experiment, subjects were issued a standardized breakfast consisting of 30 g of cornflakes and 150 ml of 2% fat milk, which was consumed 3 hours prior to commencing the TT. Only subjects who followed the standardized dietary and training protocol were allowed to continue with the study.

## Isometric Testing

To normalize EMG recordings during cycling, it was first necessary to perform isometric testing. All subjects were tested on a Kin-Com isokinetic dynamometer (Chattanooga Group Inc., U.S.). The knee extensors were tested isometrically at stop and start angles of 60º and 65º, respectively. Each subject performed four submaximal familiarization trials on the isokinetic dynamometer. Following the warm-up, subjects performed three MVCs for each test. The highest average force in these tests was used for subsequent analysis. The subjects were verbally encouraged during the test to exert maximal effort.

## Electromyographic (EMG) Testing

Prior to maximal isometric strength testing on the Kin-Com isokinetic dynamometer, EMG electrodes were attached to the subject's lower limb midway between the superior surface of the patella and the anterior superior iliac crest of the "belly" of the rectus femoris as previously described (17). The electrodes were heavily taped down and surrounded with cotton swabs to minimize sweat-induced interference. The overlying skin on the muscles was carefully prepared. Hair was shaved off, the outer layer of epidermal cells abraded, and oil and dirt were removed from the skin with an alcohol swab. Triode electrodes were placed on the muscle sites as described above and linked via a fiber-optic cable to the Flexcomp/DSP EMG apparatus (Thought Technology, U.S.) and host computer. A 50-Hz line filter was applied to the EMG data to prevent electrical interference from electrical sources. Each EMG measurement was sampled at a 1984-Hz capture rate for both MVC and the cycle TT. EMG was captured at 5-s bouts during MVC, whereas during the TT, four successive bursts of EMG activity representing the cycle contractions were selected to standardize measurement because subjects selected their own cadence. Four bursts of consecutive raw EMG data from the cycle contractions were collected at the midpoint of each 1-km HIE (10.5 km, 32.5 km, 52.5 km, 72.5 km, and 99.5 km) and each 4-km HIE (22 km, 42 km, 62 km, and 82 km) during each TT. The raw EMG data was automatically anti-aliased by the hardware (Thought Technology, U.S.). Then the EMG signals were full-wave rectified, movement artifact removed using a high-pass second-order Butterworth filter with a cutoff frequency of 15 Hz, then smoothed with a low-pass second-order filter with a cutoff frequency of 5 Hz. This was performed using MATLAB™ gait analyses software. This integrated data (IEMG) was used for subsequent analyses.

　　Following isometric testing, the subjects ingested gelatin capsules containing placebo (white flour) with 150 ml of either a sports electrolyte solution containing a sweetener (1.7% carbohydrate; Pl) or the same solution with 7% carbohydrate without sweetener (Cho), or gelatin capsules containing caffeine (6 mg $\cdot$ kg$^{-1}$) with the 7% carbohydrate solution (Caf). This caffeine dose was selected because it is believed to produce an ergogenic effect without exceeding the International Olympic Committee's (IOC) legal limits (18). Moreover, caffeine doses of 5–6 mg $\cdot$ kg$^{-1}$ bodyweight (BW) saturate the cytochrome P-450 system in the liver (17). Furthermore, because a considerable amount of caffeine is excreted through sweat during endurance exercise (25) and as exercise increases the expression of cytochrome P450 1A2 (45), decreasing peak plasma caffeine levels and half-life (7), it was considered necessary that subjects also ingest a maintenance dose of 0.33 mg $\cdot$ kg$^{-1}$

BW of caffeine every 15 min during the Caf trial. This maintenance dose was calculated to ensure that the overall caffeine dosage did not exceed $9 \text{ mg} \cdot \text{kg}^{-1}$ BW. This dose maintains mean urinary caffeine levels below the IOC limit of $12 \text{ mg}^{-1}\text{ml}$ (11, 16, 33, 43, 44). Hence a bolus amount of $6 \text{ mg} \cdot \text{kg}^{-1}$ BW caffeine allowed a maintenance dose of $3 \text{ mg} \cdot \text{kg}^{-1}$ BW over the TT of approximately 2.5 hours. This allowed 9 3 15-min dosages, each containing $0.33 \text{ mg} \cdot \text{kg}^{-1}$ BW. During the two other trials (Pl and Cho), flour-containing gelatin tablets were ingested every 15 min. One hour after ingesting the initial solution and capsules, a resting blood sample was taken and urine sample collected, and body weight was measured on a precision scale (Soehnle, Germany).

After a standardized 5-min warm up of easy cycling, subjects commenced with the 100-km time trial. Every 15 min the subjects were given either the caffeine or placebo capsules together with 150 ml of either the carbohydrate or placebo drinks. To mimic the stochastic nature of cycle road races, the time trial included a series of HIE, during which subjects were requested to ride "as fast as possible" according to the methods previously described (6). There were five 1-km HIE after 10, 32, 52, 72, and 99 km, as well as four 4-km HIE after 20, 40, 60, and 80 km. Subjects were instructed to complete the total distance in "the fastest time possible", taking into consideration the HIE that were included. Just before commencement of a sprint, the investigator gave a distance countdown and instructed the cyclist to complete the HIE in the fastest possible time as soon as he reached the specific distance at which the sprint started. Subjects viewed a diagram of the "course profile", which graphically illustrated where the 1-km and 4-km HIE occurred, before and during each ride. Otherwise subjects received no external clues to influence their performance other than their elapsed distance and heart rate. Subjects were not informed of the elapsed time or the times for the HIE until completion of all three trials.

Throughout each trial, power output and elapsed time were monitored continuously and stored on computer. Heart rate was recorded using a Sport Tester monitor (Polar Electro, Kempele, Finland). Instantaneous power output and heart rate was recorded at each 500-m split of both the 1-km and 4-km HIE to provide an estimate of the average power output for the HIE. Rating of perceived exertion (RPE; 4) was recorded in the middle of each HIE.

Upon completion of the TT, subjects had their bodyweight recorded, urine sample collected, and a final blood sample drawn. Finally, subjects were asked whether they could determine which solution, caffeine or placebo, they had received and to describe the basis on which they made that choice.

## Analysis

### Blood Samples

Fifteen milliliter venous blood samples were drawn by venepuncture into EDTA, sodium fluoride and potassium oxalate, and SST® gel clot activator vacutainer tubes for determinations of caffeine, lactate, and free fatty acid (FFA) concentrations, respectively. Immediately after the completion of the trial, the tubes were centrifuged at $3000 \times g$ for 10 min at $4 \text{ °C}$, and the supernatants were stored at $-20 \text{ °C}$ for later analyses. Plasma lactate concentrations were measured with spectrophotomeric (Beckman Model 35, Beckman Instruments Inc., Fullerton, CA) enzymatic assay

[Lactate PAP, BioM (Rieux, Lyon, France) NEFA half-micro test (Boehringer Manheim, Germany)]. Serum FFA concentrations (FFA one and a half–micro test, Boehringer Manheim, Germany) were also determined by spectrophotomeric enzymatic assay using commercial kits. Plasma caffeine, theophylline, paraxanthine, and theobromine concentrations were measured using high-performance liquid chromatography (HPLC; Gilson Inc., Middleton, WI). Plasma samples were processed as described by Tang-Liu et al. (42) with modifications. Briefly, plasma proteins were precipitated by the gradual addition of 2 ml of acetonitrile to 0.5 ml of plasma containing 20 ml of 1 mg$^{-1}$ ml paracetomol as an internal standard. The samples were mechanically agitated and centrifuged at 3000 $\times$ g for 5 min, and the supernatants were transferred to a fresh tube, frozen in liquid nitrogen, and freeze dried for at least 24 hours. The dried samples were resuspended in 200 $\mu$l of the mobile phase (90 mM phosphate, pH 3.6; 2.3 mM tetrabutyl ammonium hydrogen sulphate; 6% acetonitrile) and stored at 4 °C until HPLC analysis. Twenty $\mu$l of each sample was injected and resolved on a 150 $\times$ 4.6 mm Ultracarb 5 ODS (20) analytical column protected by 30 $\times$ 4.6 mm ultracarb 5 ODS (20) guard column (Phenomenex, St. Torrance, CA). The methylxanthine samples were measured at 280 nm.

## EMG

The spectrum of the frequency for each epoch of data collected during the TT HIE was assessed using the raw EMG data by using a fast Fourier transformation algorithm. The analyses for frequency spectrum were restricted to frequencies of the 5–500 Hz range, due to the EMG signal content consisting mostly of noise when it is outside of this bandwidth. The frequency spectrum from each epoch of data was compared with that derived from the MVC, and the amount of spectral compression was estimated. This technique was performed as described by Lowery et al. (27), which is a modification of the work of Lo Conte and Merletti (26) and Merletti and Lo Conte (30). The spectrum of the raw signal of each epoch was obtained and the normalized cumulative power at each frequency was calculated for each epoch. The shift in percentile frequency was then examined (i.e., at 0%, . . . 50%, . . . 100% of the total cumulative). The percentile shift was then estimated by calculating the mean shift in all percentile frequencies throughout the mid-frequency range (i.e., 5–500 Hz). This method has been suggested as a more accurate estimate of spectral compression than median frequency analyses, which uses single value of (50th) percentile frequency (27). This change in mean percentile frequency (MPFS) data was used for subsequent analyses.

All EMG data were normalized by dividing the value obtained midway during each HIE in the TT by the EMG value obtained during the MVC performed before the start of each TT. IEMG and MPFS data were therefore expressed as a percentage of this MVC data.

## Statistical Analyses

Data are presented as means and standard deviations. A two-way ANOVA for repeated measures was used to evaluate statistical significance of all the variables measured. A Scheffe's post hoc test was used to reduce the possibility of incurring a type I error and automated checks for spericity were computed by the Statistica program. The data was analyzed by a 3 (condition) $\times$ 5 (time) and 3 (condition) $\times$ 4

(time) ANOVA for the 1-km and the 4-km HIE, respectively. Significance was accepted at $p \le .05$.

## Results

### Plasma and Urinary Caffeine Concentrations

Subjects had low plasma caffeine concentrations in both non-caffeine trials, confirming their abstinence from caffeine-containing products before the trial (Table 1). Plasma caffeine and its metabolite paraxanthine concentrations were significantly higher during, and over time, in Caf compared to both Pl or Cho trials ($p < .01$; Table 1). Plasma theophylline and theobromine concentrations were also higher ($p < .05$) in Caf versus Cho and over time between Caf versus Cho (Table 1).

### Performance

Due to technical difficulties with the Kingcycle instrumentation, average power was not recorded during the familiarization ride. TT performance measured as average power during the TT or as time to complete the TT was not different in the three trials (Figure 1). The fastest average finishing time, although not significant, occurred during the familiarization trial when subjects ingested their own fluid solutions ad libitum. There was no significant differences in any of the HIE times

**Table 1   Plasma Concentrations of Caffeine and Its Metabolites Before and After the 100-km Cycle Time Trials for Both Carbohydrate and Caffeine, When Subjects Ingested Either Placebos (Pl), Carbohydrate and Placebo (Cho), or Carbohydrate and Caffeine (Caf) During Exercise**

| Variable | Pl | | Cho | | Caf | |
|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Pre | Post |
| Caffeine (µM) | 4.6 ± 7.2 | 2.5 ± 4.9 | 0.4 ± 0.6 | 1.1 ± 1.8 | 47.3 ± 12** | 73 ± 27.8** |
| Theophylline (µM) | 0.7 ± 0.6 | 0.5 ± 0.5 | 0.3 ± 0.4 | 0.5 ± 0.6 | 1.9 ± 2.1† | 4.2 ± 4.7† |
| Paraxanthine (µM) | 1.4 ± 1.2 | 1.3 ± 1.4 | 0.7 ± 0.4 | 0.6 ± 0.8 | 3.9 ± 2.8** | 9.3 ± 7.2** |
| Theobromine (µM) | 1.6 ± 2.3 | 1.4 ± 2 | 0.6 ± 0.7 | 0.5 ± 0.5 | 1.6 ± 1.1* | 2.7 ± 1.3* |

*Note.* Values are means ± *SD*. *Significant differences ($p < .05$) were found in the interaction of Caf versus Cho and changes over time; **highly significant differences ($p < .01$) Caf versus Cho and Pl; †significant differences ($p < .05$) were found in the interaction of Caf versus Pl and differences between groups.

between Pl, Cho, and Caf for both 1 km and 4 km (Figure 2). The values ranged from1.1 ± 1.2 min to 1.36 ± 1.3 min for 1-km HIE and from 5.22 ± 5.4 min to 6.05 ± 5.8 min for 4-km HIE.

## Heart Rate

There was a trend for mean heart rates to be higher during the 1-km HIE in Caf compared to Pl or Cho; this trend became significant during the 4-km HIE ($p < .05$). Heart rate in all groups responded similarly over time. Mean heart rates during both the 1-km (184.0 ± 9.8 Caf; 177.0 ± 5.8 Pl; 177.4 ± 8.9 Cho) and 4-km (181.7 ± 5.7 Caf; 174.3 ± 7.2 Pl; 175.6 ± 7.6 Cho) HIE were similar under the different trial conditions and tended to fall with successive HIE. Mean heart rate for the 100 km was significantly higher ($p < .01$) in the Caf group compared to the Cho group (181.6 ± 1.9 Caf; 175.5 ± 2.7 Cho).

## EMG

Due to technical difficulties with the EMG instrumentation, complete EMG data could only be collected for Cho and Caf. IEMG is expressed as a percentage of the
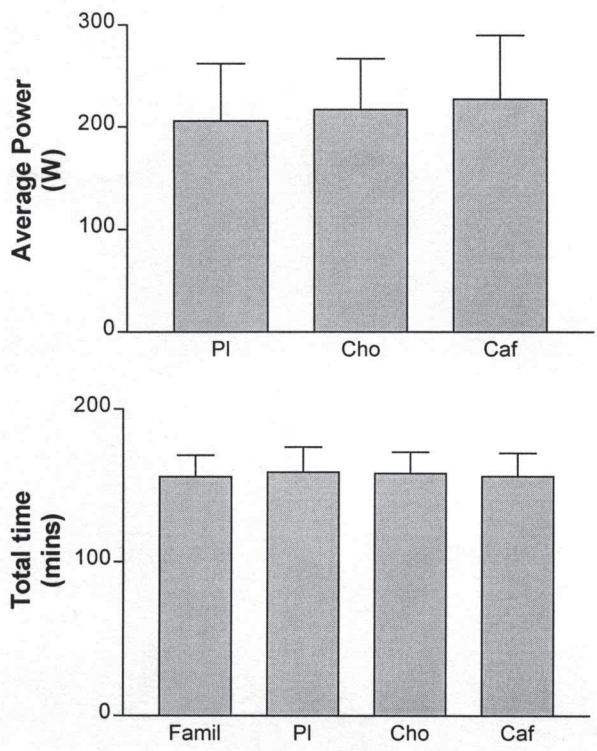


**Figure 1 — Average power (W; top) and total time (mins; bottom) to complete familiarization (Famil; top only), placebo (Pl), carbohydrate (Cho), and caffeine 100-km cycle time trials.**

IEMG recorded during the MVC. There were no significant differences in IEMG values in any of the HIE for Caf or Pl. The values ranged between $15.2 \pm 1.0\%$ and $41.3 \pm 17.3\%$ in the 1-km HIE and between $19.9 \pm 4.4\%$ and $26.3 \pm 7.7\%$ in the 4-km HIE. There were no differences between groups or over time. The mean IEMG was $25.4 \pm 18.3\%$ for 1-km and $22.0 \pm 13.2$ for 4-km HIE. The MPFS, expressed as a percentage of the MPFS measured during the MVC, also showed no differences between group or time. The mean MPFS was $1.08 \pm 0.2\%$ for 1 km and $1.10 \pm 0.2\%$ for 4 km.

### Serum FFA and Lactate Concentrations

Serum FFA and lactate concentrations increased significantly during exercise in all TT ($p < .01$). There were no differences in serum FFA and lactate concentrations between groups. Mean pre FFA and lactate values for all three groups were $0.22 \pm 0.1$ mmol $\cdot$ L$^{-1}$ and $1.33 \pm 0.2$ mmol $\cdot$ L$^{-1}$, respectively, compared to post values of $1.7 \pm 0.2$ mmol $\cdot$ L$^{-1}$ for FFA and $4.5 \pm 0.4$ mmol $\cdot$ L$^{-1}$ for lactate.

### RPE

RPE values rose progressively ($p < .01$) in successive 1-km and 4-km HIE. Mean RPEs were also similar in the consecutive 1-km ($19.8 \pm 0.4$ Caf; $19.6 \pm 0.5$ Pl; $18.7 \pm 1.5$ Cho) and 4-km ($18.4 \pm 1.9$ Caf; $18.2 \pm 1.8$ Pl; $18.5 \pm 1.5$ Cho) HIE, but there was no difference between Pl, Cho, and Caf.

### Weight Loss

The percent weight loss of subjects during the three trials ($3.5 \pm 1.8\%$ Caf; $3.0 \pm 1.7\%$ Pl; $2.7 \pm 1.5\%$ Cho) were similar.

### Drug Identification

Seven of the 8 subjects correctly identified when they had ingested caffeine—citing symptoms of reduced concentration and elevated heart rate—before, during, and after the trial and difficulty in sleeping the following night.

## Discussion

Caffeine is listed as a stimulatory drug by the International Olympic Committee (IOC), and its use during those sporting competitions sanctioned by the IOC is subject to certain restrictions. It is inherent in this assumption that any stimulatory effect of caffeine will be performance enhancing.

Indeed the bulk of current evidence suggests that caffeine ingestion enhances performance to quite a marked extent during more prolonged endurance exercise (40), although there appears to be little if any effect during high intensity exercise of short duration (10, 14, 18, 34), a fact which seems to have escaped the attention of the international doping control authorities.

However, most of the evidence for this large effect comes from studies in which subjects begin exercise unaware of any fixed endpoint other than to continue exercising for "as long as possible" (16). One result is that subjects do not have a fixed goal around which to plan their pacing strategy. We (38) and others (29) have

shown that the coefficient of variance for such open-end testing is large. In contrast, performance during closed trials, in which subjects begin exercise knowing the endpoint in terms of distance to be covered or total exercise time, is far more reproducible (37). In addition, relatively few studies have evaluated the effects of caffeine on the performance of better performing athletes of the quality studied in this trial and who, even then, were clearly not elite athletes of Olympic quality.

Accordingly, the most important finding of this study was that, in contrast to the large ergogenic effect measured in open-ended trials, caffeine did not significantly enhance overall performance during the 100-km cycling time trial that included bouts of high intensity exercise. This is consistent with other trials which show that any effect of caffeine is much greater in open-ended than closed trials. For example, the magnitude of the ergogenic effect of caffeine in open-ended trials is of the order of 20–50% (17), whereas the average performance enhancement in closed trials was 1.8% (range, 1.7–2.5%; 3, 5, 25, 28, 46). In this study, subjects were 1.3% faster when using caffeine compared to placebo, but 0.1% slower when ingesting caffeine than in the familiarization trial when they ingested their chosen drinks ad libitum.

Plasma caffeine concentrations were low in the Pl and Cho trials but were markedly elevated in the Caf trial; hence, illicit use of caffeine before the Pl or Cho trials could not explain our failure to detect an ergogenic effect of caffeine. The significantly higher heart rates with caffeine ingestion, especially during the 4-km HIE, confirm that the ingested caffeine was physiologically active. In contrast, HIE
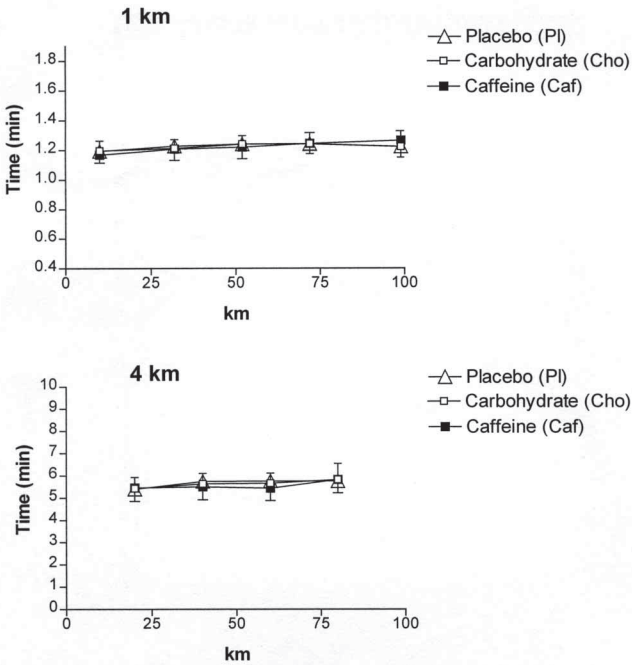


Figure 2 — Time taken to complete 1-km (top) and 4-km high intensity epochs (HIE) (bottom) within the100-km cycle time trials for subjects ingesting placebo (Pl), carbohydrate (Cho), or caffeine (Caf).

performances when ingesting placebo or carbohydrate were reproduced exactly (Figure 2).

Thus, despite only receiving heart rate as a external cue, subjects employed essentially the same pacing strategies when ingesting either carbohydrate or placebo, whereas caffeine ingestion may have modified imperceptibly the pacing strategy. Interestingly, in the studies of Bruce et al. (5), caffeine enhanced the performance of rowers during a simulated 2000-m trial specifically by increasing their speed only over the first 500 m of the race. This suggests that, in that trial, caffeine may have acted directly on the subconscious brain centers that direct the early pacing strategies during exercise.

Indeed, the noteworthy feature of this and other studies (33, 36, 37, 39) is the reproducibility of the pacing strategies used by subjects during closed trials of the type used in this study. This invites the question of the nature of the internal physiological cues that determine such a response, since the subjects in all our trials receive no external cues other than the distance covered.

In this study, we found that, whereas performance time (Figure 1) remained unchanged, RPE rose progressively with consecutive 1-km and 4-km HIE, peak heart rate was essentially the same, in both 1-km and 4-km HIE and, if anything, decreased during the course of the exercise. Peak EMG activity was more variable, in contrast to our previous study in which EMG activity was significantly lower in the final 1-km and the last two 4-km HIE in subjects who modified their diets in the 3 days before exercise (9). However, sprinting performance in that trial also fell significantly in the final 1-km and 4-km HIE, whereas in this study, performance was unchanged in the successive 1-km and 4-km HIE (Figure 2).

Significantly, IEMG activity was about 12% higher during the 1-km than during the 4-km HIE, which is to be expected since subjects cycled faster during the 1-km than the 4-km HIE. However, as also reported in our previous study (41), subjects recruited only ~25% of their maximal neuromuscular activation during the 1-km sprints and only ~22% during the 4-km HIE.

Skeletal muscle activation and performance was greater in the 1-km than the 4-km HIE. However, if peripheral metabolite accumulation or muscle glycogen depletion determines performance during the 4-km HIE or indeed the pacing strategy during the sustained exercise between the HIE, then even higher performance should not have been possible during the 1-km HIE. Rather, the higher metabolic rate during the 1-km HIE should have increased the concentration of fatigue-inducing metabolites, thereby progressively impairing performance. But this was not the case (Figure 2).

Indeed in a previous study employing shorter 40-km time trials, which also included repeated HIE, we (39) found that sprinting performance fell progressively as did blood lactate concentrations, whereas pH rose, the opposite of the predicted effect if fatigue is regulated by peripheral metabolites.

Furthermore, only 22–25% of the total neuromuscular activation was recruited, even from the outset of the 1- and 4-km HIE. Hence, something "constrains" neuromuscular activation so that less than 25% of the available muscle mass is ever recruited, even from the start of exercise when muscle metabolite concentrations are likely to be the least perturbed. We have proposed that this constraint exists centrally in the brain (4, 23).

According to this theory, the consistency of performance in all these trials results from a centrally-determined pacing strategy that is uninfluenced by caffeine

ingestion. Since the neuromuscular activation does not rise during the repetitive sprints, the absence of a "peripheral" fatigue is confirmed, since the definition of peripheral fatigue requires a greater neuromuscular activation to maintain the same, or a lesser, power output (31).

In summary, this study establishes that a substance considered ergogenic because of its stimulatory effects on the central nervous system and shown to have this effect during prolonged open-ended exercise (8, 12, 16, 17, 21, 33), fails to improve performance when the trial has a defined endpoint.

If this is true, the IOC ruling on stimulatory drugs needs to be reviewed. Indeed, we have previously shown that another substance banned by the IOC, pseudoephedrine, did not improve 40-km time-trial performance (13). Now we show the same result, but at a longer distance, for caffeine.

## References

1. Alves, M.N., W.M. Ferrari-Auarek, K.M. Pinto, K.R. Sa, J.P. Viveiros, H.A. Pereira, A.M. Ribeiro, and L.O. Rodrigues. Effects of caffeine and tryptophan on rectal temperature, metabolism, total exercise time, rate of perceived exertion and heart rate. *Braz. J. Med. Biol. Res.* 28:705-709, 1995.
2. Bell, D.G., I. Jacobs, and J. Zamecnik. Effects of caffeine, ephedrine and their combination on time to exhaustion during high-intensity exercise. *Eur. J. Appl. Physiol.* 77:427-433, 1998.
3. Berglund, B., and P. Hemmingsson. Effects of caffeine ingestion on exercise performance at low and high altitudes in cross-country skiers. *Int. J. Sports Med.* 3:234-236, 1982.
4. Borg, G.A. Perceived exertion: a note on "history" and methods. *Med. Sci. Sports* 5:90-93, 1973.
5. Bruce, C.R., M.E. Anderson, S.F. Fraser, N.K. Stepto, R. Klein, W.G. Hopkins, and J.A. Hawley. Enhancement of 2000-m rowing performance after caffeine ingestion. *Med. Sci. Sports Exerc.* 32:1958-1963, 2000.
6. Burke, L.M., J.A. Hawley, E.J. Schabort, G.A. St Clair, I. Mujika, and T.D. Noakes. Carbohydrate loading failed to improve 100-km cycling performance in a placebo-controlled trial. *J. Appl. Physiol.* 88:1284-1290, 2000.
7. Collomp, K., F. Anselme, M. Audran, J.P. Gay, J.L. Chanal, and C. Prefaut. Effects of moderate exercise on the pharmokinetics of caffeine. *Eur. J. Clin. Pharmacol.* 40:279-282, 1996.
8. Costill, D.L., G.P. Dalsky, and W.J. Fink. Effects of caffeine ingestion on metabolism and exercise performance. *Med. Sci. Sports* 10:155-158, 1978.
9. Dodd, S.L., E. Brooks, S.K. Powers, and R. Tulley. The effects of caffeine on graded exercise performance in caffeine naive versus habituated subjects. *Eur. J. Appl. Physiol.* 62:424-429, 1991.
10. Doherty, M. The effects of caffeine on the maximal accumulated oxygen deficit and short-term running performance. *Int. J. Sport Nutr.* 8:95-104, 1998.
11. Duthel, J.M., J.J. Vallon, G. Martin, J.M. Ferret, R. Mathieu, and R. Videman. Caffeine and sport: role of physical exercise upon elimination. *Med. Sci. Sports Exerc.* 23:980-985, 1991.
12. Essig, D., D.L. Costill, and P.J. Van Handel. Effects of caffeine ingestion on utilization of muscle glycogen and lipid during leg ergometer cycling. *Int. J. Sports Med.* 1:86-90, 1980.

13. Gillies, H., W.E. Derman, T.D. Noakes, P. Smith, A. Evans, and G. Gabriels. Pseudoephedrine is without ergogenic effects during prolonged exercise. *J. Appl. Physiol.* 81:2611-2617, 1996.

14. Graham, T.E., E. Hibbert, and P. Sathasivam. Metabolic and exercise endurance effects of coffee and caffeine ingestion. *J. Appl. Physiol.* 85:883-889, 1998.

15. Graham, T.E., J.W. Rush, and M.H. Van Soeren. Caffeine and exercise: metabolism and performance. *Can. J. Appl. Physiol.* 19:111-138, 1994.

16. Graham, T.E., and L.L. Spriet. Performance and metabolic responses to a high caffeine dose during prolonged exercise. *J. Appl. Physiol.* 71:2292-2298, 1991.

17. Graham, T.E., and L.L. Spriet. Metabolic, catecholamine, and exercise performance responses to various doses of caffeine. *J. Appl. Physiol.* 78:867-874, 1995.

18. Greer, F., C. McLean, and T.E. Graham. Caffeine, performance, and metabolism during repeated Wingate exercise tests. *J. Appl. Physiol.* 85:1502-1508, 1998.

19. Hawley, J.A., and T.D. Noakes. Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. *Eur. J. Appl. Physiol.* 65:79-83, 1992.

20. Hickey, M.S., D.L. Costill, G.K. McConell, J.J. Widrick, and H. Tanaka. Day to day variation in time trial cycling performance. *Int. J. Sports Med.* 13:467-470, 1992.

21. Ivy, J.L., D.L. Costill, W.J. Fink, and R.W. Lower. Influence of caffeine and carbohydrate feedings on endurance performance. *Med. Sci. Sports* 11:6-11, 1979.

22. Jackman, M., P. Wendling, D. Friars, and T.E. Graham. Metabolic catecholamine, and endurance responses to caffeine during intense exercise. *J. Appl. Physiol.* 81:1658-1663, 1996.

23. Kay, D., F.E. Marino, J. Cannon, A.G. St Clair, M.I. Lambert, and T.D. Noakes. Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *Eur. J. Appl. Physiol.* 84:115-121, 2001.

24. Keen, P., L. Passfield, and T. Hale. *Indirect determination of $\dot{V}O_{2max}$ using a sports specific (cycling) ergometry system.* Unpublished doctoral dissertation, West Sussex Institute of Higher Education, Human Performance Laboratory, Chichester, 2000.

25. Kovacs, E.M., J.H.C.H. Stegen, and F. Brouns. Effect of caffeinated drinks on substrate metabolism, caffeine excretion, and performance. *J. Appl. Physiol.* 85:709-715, 1998.

26. Lo Conte, L.R., and R. Merletti. Estimating EMG spectral compression: comparison of four indices. *18th Ann. Int. Con. IEEE Eng. Med. Biol. Sci.* 5:2-5, 1996.

27. Lowery, M., M. O'Malley, C. Vaughan, and A. St Clair Gibson. A physiologically based stimulation of the electromyographic signal. In: *Proceedings of the 12th International Society and Kinesiology,* A.B. Arsenault, P. McKinley, and B. McFadyen (Eds.), June 12–13, 1988.

28. MacIntosh, B.R., and B.M. Wright. Caffeine ingestion and performance of a 1,500-metre swim. *Can. J. Appl. Physiol.* 20:168-177, 1995.

29. McLellan, T.M., S.S. Cheung, and I. Jacobs. Variability of time to exhaustion during submaximal exercise. *Can. J. Appl. Physiol.* 20:39-51, 1995.

30. Merletti, R., and L.R. Lo Conte. Advances in processing of surface myoelectric signals: Part 1. *Med. Biol. Eng. Comput.* 33:362-372, 1995.

31. Moritani, T., M. Muro, and A. Nagata. Intramuscular and surface electromyogram changes during muscle fatigue. *J. Appl. Physiol.* 60:1179-1185, 1986.

32. Palmer, G.S., J.A. Hawley, S.C. Dennis, and T.D. Noakes. Heart rate responses during a 4-d cycle stage race. *Med. Sci. Sports Exerc.* 26:1278-1283, 1994.

33. Pasman, W.J., M.A. van Baak, A.E. Jeukendrup, and A. de Haan. The effect of different dosages of caffeine on endurance performance time. *Int. J. Sports Med.* 16:225-230, 1995.

34. Paton, C.D., W.G. Hopkins, and L. Vollebregt. Little effect of caffeine ingestion on repeated sprints in team-sport athletes. *Med. Sci. Sports Exerc.* 33:822-825, 2001.

35. Randle, P.J., P.B. Garland, C.N. Hales, and E.A. Newsholme. The glucose-fatty acid cycle: its role in insulin sensitivity and the metabolic disturbances of diabetes mellitus. *Lancet* 1:785-789, 1963.

36. Schabort, E.J., J.A. Hawley, W.G. Hopkins, and H. Blum. High reliability of performance of well-trained rowers on a rowing ergometer. *J. Sports Sci.* 17:627-632, 1999.

37. Schabort, E.J., J.A. Hawley, W.G. Hopkins, I. Mujika, and T.D. Noakes. A new reliable laboratory test of endurance performance for road cyclists. *Med. Sci. Sports Exerc.* 30:1744-1750, 1998.

38. Schabort, E.J., W.G. Hopkins, and J.A. Hawley. Reproducibility of self-paced treadmill performance of trained endurance runners. *Int. J. Sports Med.* 19:48-51, 1998.

39. Schabort, E.J., G. Wilson, and T.D. Noakes. Dose-related elevations in venous pH with citrate ingestion do not alter 40-km cycling time-trial performance. *Eur. J. Appl. Physiol.* 83:320-327, 2000.

40. Sinclair, C.J., and J.D. Geiger. Caffeine use in sports. A pharmacological review. *J. Sports Med. Phys. Fitness* 40:71-79, 2000.

41. St Clair, A.G., E.J. Schabort, and T.D. Noakes. Reduced neuromuscular activity and force generation during prolonged cycling. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 281:R187-R196, 2001.

42. Tang-Liu, D.D., and S. Riegelman. An automated HPLC assay for simultaneous quantitation of methylated xanthines and uric acids in urine. *J. Chromatogr. Sci.* 20:155-159, 1982.

43. Van der Merwe, P.J., H.G. Luus, and J.G. Barnard. Caffeine in sport. Influence of endurance exercise on the urinary caffeine concentration. *Int. J. Sports Med.* 13:74-76, 1992.

44. Van Soeren, M.H., P. Sathasivam, L.L. Spriet, and T.E. Graham. Caffeine metabolism and epinephrine responses during exercise in users and nonusers. *J. Appl. Physiol.* 75:805-812, 1993.

45. Vistisen, K., H.E. Poulsen, and S. Loft. Foreign compound metabolism capacity in man measured from metabolites of dietary caffeine. *Carcinogenesis* 13:1561-1568, 1992.

46. Wiles, J.D., S.R. Bird, J. Hopkins, and M. Riley. Effect of caffeinated coffee on running speed, respiratory factors, blood lactate and perceived exertion during 1500-m treadmill running. *Br. J. Sports Med.* 26:116-120, 1992.

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