

Benefits of caffeine ingestion on sprint performance in trained and untrained swimmers

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Summary. The influence of specific training on benefits from caffeine (Caf) ingestion was examined during a sprint test in a group of highly trained swimmers (T) and compared with the response of a group of untrained occasional swimmers (UT). Seven T and seven UT subjects swam freestyle two randomly assigned 2 × 100 m distances, at maximal speed and separated by 20 min of passive recovery, once after Caf (250 mg) and once after placebo (Pla) ingestion. Anaerobic capacity was assessed by the mean velocity (meters per second) during each 100 m and blood was sampled from the fingertip just before and 1, 3, 5, 7, and 9 min after each 100 m for resting and maximal blood lactate concentration ($[la^-]_{b,max}$) determination. The $[la^-]_{b,max}$ was significantly enhanced by Caf in both T and UT subjects ($P < 0.01$). However, only T subjects exhibited significant improvement in their swimming velocity ($P < 0.01$) after Caf or any significant impairment during the second 100 m. In light of these results, it appears that specific training is necessary to benefit from the metabolic adaptations induced by Caf during supramaximal exercise requiring a high anaerobic capacity.

Key words: Caffeine – Specific training – Anaerobic performance – Swimming velocity – Lactate

Introduction

Although the physiological effects of caffeine ingestion prior to aerobic exercise of various intensities are well documented (Costill et al. 1978; Falk et al. 1989; Ivy et al. 1979; Powers et al. 1983; Sasaki et al. 1987), few studies of caffeine use during highly anaerobic activities have been made. In laboratory studies, it has been shown that a single intake of caffeine improves maximal anaerobic power in normally active subjects during a force-velocity test (Anselme et al. in press) but not

maximal anaerobic capacity during a Wingate test (Collomp et al. 1991). With both tests, there is an increase in peak blood lactate. Because blood lactate concentration only indirectly reflects muscle lactate production, the increase induced by caffeine may indicate an increase in muscle glycogenolysis, an increase in muscle lactate release, or both. If caffeine increases glycogenolysis, the lack of increase in maximal anaerobic capacity in untrained subjects could be explained by a parallel development of muscle acidosis. To verify indirectly this last hypothesis, we decided to test the effect of caffeine on maximal anaerobic capacity in anaerobically trained subjects. Indeed, anaerobic training has been shown to enhance the buffering capacity of muscle (Sharp et al. 1986) by an increase in intracellular bicarbonate and/or an efflux of hydrogen ions (Nevill et al. 1989). Thus it may be that only specifically trained athletes are able to optimize their maximal anaerobic capacity after caffeine ingestion.

The purpose of this study was to determine whether specific training is necessary to produce benefits from acute ingestion of caffeine by improving maximal anaerobic capacity during a spring swimming test (Keskinen et al. 1989).

Methods

Subjects. Fourteen healthy, nonsmoking volunteers (Table 1) gave written consent after being informed of the constraints of the experiment. An initial clinical examination verified the absence of hepatic impairment, gastric ulcers, cardiovascular or renal illness, and known allergies to xanthines. At the time of the study, the subjects were not under medical treatment. They kept to their normal dietary habits, although caffeine intake (coffee, tea, cola) was prohibited 1 week prior to and during the entire experiment to avoid caffeine tolerance (Robertson et al. 1981).

The subjects were divided into two groups: group T had been regional competitive swimmers (4 women and 3 men) for 5 years and they had been training (five to six times per week) for 4 months prior to the study. They were in a short-term sprint training period just before the test in preparation for a forthcoming meet. Group UT was composed of 7 untrained subjects (5 women and 2 men) who had been past members of a swimming club for

Table 1. Anthropometric characteristics of subjects and dose of caffeine administered

| <i>n</i> = 14 | Age (years) | Body mass (kg) | Caffeine (mg · kg ⁻¹) |
|---------------|-------------|----------------|-----------------------------------|
| Mean | 17 | 59.9 | 4.3 |
| SEM | 2.1 | 2.6 | 0.2 |

at least 3 years but who were swimming only occasionally at the time of the study. These UT subjects were normally active and practised between 2 to 4 h of varied sports each week.

Test. The sprint test consisted of 2 × 100 m swum freestyle at maximal speed separated by 20 min of passive recovery. Anaerobic capacity was assessed by the mean swimming velocity (S) during the first 100-m crawl (Keskinen et al. 1989). Differences in mean velocity between the first and second 100 m allowed us to evaluate indirectly the effects of caffeine on repeated periods of exercise.

Caffeine. Caffeine (Caf, 250 mg) and placebo (Pla, CaCO₃) gelatin capsules were prepared with the same packaging. The 250 mg of Caf represented approximately the same dose · kg⁻¹ body mass for all subjects (Table 1).

Analysis. The S was calculated from the performance time for each 100 m. Capillary blood was taken from the fingertip just before and 1, 3, 5, 7, and 9 min after each 100 m to determine resting blood lactate ([La⁻]_{b,resting,1} and [La⁻]_{b,resting,2}) and maximal blood lactate ([La⁻]_{b,max,1} and [La⁻]_{b,max,2}) concentrations. The sampling method was standardized in the following manner:

1. Prior to pricking, the finger was wiped dry with tissue
2. The first drop of blood was discarded and 20 µl of whole blood was then collected in a disposable calibrated nonheparinized capillary tube
3. The specimen was immediately put into 380 µl deproteinizing and conserving agent at 4°C for subsequent analysis by an electro-enzymatic method (Microzym, Setric Genie Industriel, Toulouse) later in the week.

Protocol. The experiment was conducted in a 25-m indoor swimming pool. The Caf and Pla trials were conducted in random double-blind order with a 3-day interval, during which time normal training continued. On the days of the experiment, Caf (250 mg) or Pla was administered at 11 a.m., 2 h after ingestion of a standard meal (300 kcal). Immediately after this the warm-up began. For T, this involved 1600 m of swimming as follows: 400-m medley, 4 × 100-m crawl, 8 × 50-m flutter kick and 16 × 25-m crawl sprints every 30 s. Warm-up in UT followed the same pattern but covered only a quarter of the total distance.

On completion, all the subjects remained at rest till 12 p.m. to allow for the development of maximal plasma concentrations of Caf and the return to basal values for the [La⁻]_b. The subjects then performed the first 100 m of the test. Passive recovery in a seated position was required for exactly 20 min before the second 100 m of the test.

Statistics. Comparisons of the Pla and Caf results ([La⁻]_b, S, and the differences in these parameters between the two 100 m tests) were conducted for each group using the Student's *t*-test for paired samples. As the results of T and UT subjects did not follow a Gaussian distribution, a logarithmic transformation was necessary to normalize the data (Bernardi et al. 1990; Csete et al. 1990) before using the Student's *t*-test for unpaired samples to compare the two groups. The level of significance was fixed at *P* < 0.05.

Results

Caffeine compared to Placebo

Performance. The S recorded for the subjects are presented in Fig. 1. The maximal nature of the test was verified for the T subjects. Under Pla, they attained 96.7% (SEM 0.7)% of their best 100-m performance times for the first 100 m and 94.3% (SEM 1.2)% for the second 100-m. In T subjects, Caf significantly improved S (Fig. 1) during the first and second 100 m (*P* < 0.01). The significant decrease in S during the second 100 m which occurred after Pla (*P* < 0.05) disappeared after Caf. In UT subjects, Caf did not improve performance during the first or the second 100 m when compared to Pla. The decrease in S during the second 100 m was significant (*P* < 0.05) after both Pla and Caf (*P* < 0.05).

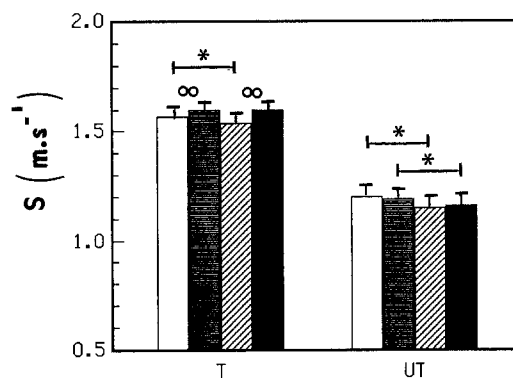


Fig. 1. Mean (SEM) swimming velocity (S) in a group of highly trained swimmers (T) and untrained occasional swimmers (UT) for the first (1) and the second (2) 100 m after placebo (Pla) and caffeine (Caf) ingestion. ° *P* < 0.05 and ∞ *P* < 0.01 significant difference between Caf and Pla; * *P* < 0.05 significant difference between the first and the second 100 m of the test. □ Caf1; ■ Pla1; ▨ Caf2; ■ Pla2

Table 2. Mean resting ([La⁻]_{b,resting}) and maximal blood lactate ([La⁻]_{b,max}) before and after the first (1) and the second (2) 100 m of the swimming test after placebo (Pla) and caffeine (Caf) ingestion in untrained occasional swimmers (UT) and highly trained swimmers (T) subjects

| Groups | [La ⁻] _{b,resting,1} | | [La ⁻] _{b,max,1} | | [La ⁻] _{b,resting,2} | | [La ⁻] _{b,max,2} | |
|------------------------|---|-----|---------------------------------------|--------------------|---|--------------------|---------------------------------------|--------------------|
| | mean | SEM | mean | SEM | mean | SEM | mean | SEM |
| mmol · l ⁻¹ | | | | | | | | |
| UT | | | | | | | | |
| Pla | 1.9 | 0.3 | 8.3 | 0.8 | 5.2 | 0.4 | 9.3 | 0.8 ^c |
| Caf | 2.2 | 0.4 | 9.1 | 0.6 ^a | 5.1 | 0.4 | 10.9 | 0.8 ^a |
| T | | | | | | | | |
| Pla | 2 | 0.2 | 11.9 | 0.2 ^d | 6.1 | 0.5 ^c | 13.1 | 0.2 ^{d,e} |
| Caf | 2.1 | 0.3 | 12.9 | 0.3 ^{b,d} | 8.2 | 0.4 ^{b,d} | 15.5 | 0.5 ^{b,d} |

^a *P* < 0.05 and ^b *P* < 0.01 significant difference between Caf and Pla; ^c *P* < 0.05 and ^d *P* < 0.01 significant difference between T and UT; ^e *P* < 0.05 significant difference Pla/Caf between the first and second 100 m ([La⁻]_{b,max})

Blood lactate. For all subjects, $[la^-]_{b,resting,1}$ just before the first 100 m did not appear significantly different after Caf and Pla (Table 2) but $[la^-]_{b,resting,2}$ just before the second 100 m was significantly higher after Caf ingestion. During the test, Caf significantly increased $[la^-]_{b,max}$ in T and UT subjects after both the first and the second 100 m ($P < 0.01$) compared to Pla. This Pla/Caf difference of $[la^-]_{b,max}$ was significantly greater after the second 100 m for all subjects ($P < 0.05$).

T compared to UT subjects

Performance. The S was always higher in T ($P < 0.01$).

Blood lactate. At rest, no significant difference was found between $[la^-]_{b,resting,1}$ in T and UT subjects. However, $[la^-]_{b,resting,2}$ after the 20 min of passive recovery was significantly higher in T ($P < 0.05$). During the test, with both Pla and Caf ingestion, $[la^-]_{b,max}$ was significantly higher in T subjects after both the first and the second 100 m ($P < 0.01$). However, the increase of $[la^-]_{b,max}$ induced by Caf after each 100 m was not significantly different for T and UT subjects.

Discussion

This study showed that although Caf (250 mg) increased $[la^-]_{b,max}$ in both T and UT swimmers after a sprint swimming test (2×100 m), it only improved performance in the highly trained athletes, with a greater relative improvement during the second part of the test.

Because of the relative insensitivity of the Wingate test to training adaptation (Jacobs et al. 1987), a field test was chosen to assess the effect of specific training on Caf benefits during supramaximal exercise involving maximal anaerobic capacity. The test, 2×100 m separated by 20 min of passive recovery, lasted too long to induce only anaerobic metabolism. However, Szögy (1988) has reliably estimated what he calls "anaerobic stamina" from S during a 100-m freestyle swim at maximal speed and recent research by Keskinen et al. (1989) has shown in a comparative study of $[la^-]_b$ tests that the repetition of maximal 100-m sprint swims allows the accurate evaluation of anaerobic capacity. We, therefore, decided to use this test to determine Caf effects in a double-blind study with two groups of subjects: highly trained swimmers (at least five times each week) and untrained but formerly regular swimmers, the latter to avoid an interaction of biomechanical factors.

With and without Caf, T subjects had significantly higher postexercise $[la^-]_b$. This greater $[la^-]_{b,max}$ with anaerobic training coupled with better performance was consistent with recent research (Chatard et al. 1988; Mebdo and Burgers 1990; Nevill et al. 1989) and may have reflected an increase in both muscle glycogenolysis and muscle buffer capacity. Indeed, short-term sprint or interval training has previously been shown to

result in an increase in adenosinetriphosphate (ATP) resynthesis from anaerobic glycolysis, which may be facilitated by an increased activity of phosphofructokinase or an increased efflux of H^+ from the muscle cell (Nevill et al. 1989) because of a decrease in membrane potential after the sprint training (Sjogaard 1983). Similarly, Sharp et al. (1986) found greater muscle lactate accumulation for the same level of muscle pH after 8 weeks of sprint training, which would indicate a significant increase in buffer capacity (37%). This finding was not confirmed by another study (Nevill et al. 1989) but it should be pointed out that the homogenate technique used in this latter work to determine buffer capacity did not take into account the transmembrane flux of ions.

According to earlier studies in this laboratory (Anselme et al. in press; Collomp et al. 1991), Caf ingestion has increased $[la^-]_{b,max}$, whatever the training status of the subjects. This phenomenon may have reflected a potentiating action of Caf on muscle glycogenolysis and/or an increase in lactate release.

The hypothesis of an increase in glycogenolysis by Caf appears, at first view, to be contrary to previous studies (Costill et al. 1978; Ivy et al. 1979) which have reported a glycogen sparing effect of Caf during submaximal exercise, explained by a greater free fatty acid consumption. This mechanism can hardly have been involved in short-term and much more intense exercise. Indeed, Caf has been shown to facilitate calcium liberation which activates both the enzymatic transformation of glycogen phosphorylase b to the more active form a and the glycogenolytic adrenaline secretion (Yamada et al. 1989; Collomp et al. 1991). Thus, it appears possible that one of the repercussions of these systems (Ca^{++} , catecholamines ...) activated by Caf was a greater lactate production. However, the increase of $[la^-]_{b,max}$ may also have resulted from a greater muscle lactate release. Unfortunately, with the indirect measurements of our study, we were unable to estimate the relative contribution of these two processes and the extent to which training status may have influenced these contributions.

The significant enhancement of S by Caf during the first 100 m which occurred only in T subjects confirmed the hypothesis of a necessary "specific training" factor to improve anaerobic capacity with Caf. As previously stated, how training interacts with Caf to improve sprint performance remains unclear. Despite the higher recovery $[la^-]_b$ in T subjects, there was no significant difference in Pla/Caf $[la^-]_b$ between T and UT subjects. Thus, it can be suggested that the improvement in performance of the T subjects after Caf ingestion may have resulted from the enhancement of buffering capacity by anaerobic training, which permitted a better release of muscle protons.

It is interesting to note that the Caf effect on $[la^-]_b$ was marked during the second part of the test for all subjects and was expressed in T swimmers by a greater improvement of S in comparison to Pla. The phenomenon involved may have been the same as after bicarbonate ingestion (Horswill et al. 1988). Indeed, the ef-

fect of bicarbonate ingestion on the improvement of sprint performance (2 min) was shown only after repeated periods of exercise, probably because a threshold pH gradient was required (Horswill et al. 1988) to induce bicarbonate action. It can be hypothesized that Caf activity became more effective when intramuscular H^+ accumulation exceeded the intracellular buffering capacity, thus resulting in a relatively greater S in the second part of the test. The maintenance of the initial S during the second 100 m may have indicated an accelerated rate of recovery. However, because of the same degree of decrease in S between the first and second 100 m after both Caf and Pla for UT subjects, the effective use of this process seemed to require specific training. Muscle biopsy would be necessary to confirm this hypothesis.

In summary, it is suggested that the intra and/or extracellular adaptations resulting from specific training are necessary to benefit from Caf during sprint performance. Further direct research evaluating Caf and specific training effects on muscle glycogenolysis and on acid-base status are needed, particularly concerning the influence of repeated periods of exercise, which are now frequent during competitive meets.

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