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Metabolic and exercise endurance effects of coffee and caffeine ingestion

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Graham, T. E., E. Hibbert, and P. Sathasivam. Metabolic and exercise endurance effects of coffee and caffeine ingestion. *J. Appl. Physiol.* 85(3): 883–889, 1998.—Caffeine (Caf) ingestion increases plasma epinephrine (Epi) and exercise endurance; these results are frequently transferred to coffee (Cof) consumption. We examined the impact of ingestion of the same dose of Caf in Cof or in water. Nine healthy, fit, young adults performed five trials after ingesting (double blind) either a capsule (Caf or placebo) with water or Cof (decaffeinated Cof, decaffeinated with Caf added, or regular Cof). In all three Caf trials, the Caf dose was 4.45 mg/kg body wt and the volume of liquid was 7.15 ml/kg. After 1 h of rest, the subject ran at 85% of maximal O₂ consumption until voluntary exhaustion (~32 min in the placebo and decaffeinated Cof tests). In the three Caf trials, the plasma Caf and paraxanthine concentrations were very similar. After 1 h of rest, the plasma Epi was increased ($P < 0.05$) by Caf ingestion, but the increase was greater ($P < 0.05$) with Caf capsules than with Cof. During the exercise there were no differences in Epi among the three Caf trials, and the Epi values were all greater ($P < 0.05$) than in the other tests. Endurance was only increased ($P < 0.05$) in the Caf capsule trial; there were no differences among the other four tests. One cannot extrapolate the effects of Caf to Cof; there must be a component(s) of Cof that moderates the actions of Caf.

norepinephrine; epinephrine; diuresis; free fatty acids; glucose; glycerol; lactate; performance; methylxanthines; doping

THERE HAVE BEEN NUMEROUS REPORTS that caffeine is an ergogenic aid; ingestion of the drug has been shown to increase endurance, particularly in prolonged exercise lasting 30–120 min (11–13, 23). Reviews of the physiological effects of caffeine commonly give an approximate equivalent of various food items such as coffee, tea, colas, etc. The implication would appear to be that ingesting the same amount of caffeine via a food source is as effective as ingesting pure caffeine.

This hypothesis has never been tested with regard to exercise metabolism. Coffee is certainly the most common mode of caffeine ingestion, and it also can provide caffeine in a more concentrated form than do other foodstuffs (1, 8). Caffeine and coffee ingestion have been shown to result in similar hemodynamic responses in resting subjects (5). However, it is important to note that coffee is a biological substance, and, when the beans are roasted, ground, and extracted in hot water, there are hundreds of compounds that are dissolved along with caffeine and the other methylxanthines (22, 28). Various compounds are present in much higher concentrations than is the caffeine; for example, lipids, carbohydrates, and proteins represent >60% of the compounds, whereas caffeine is ~2% (28). In some areas of metabolism it has been clearly demonstrated

that coffee can have metabolic actions independent of caffeine. For example, coffee is hypercholesterolemic, particularly if the brewing process does not include filtering. This effect has been attributed to a lipid fraction of the coffee and not to caffeine (1, 22, 24).

Additional compounds in coffee that could have metabolic importance are present in small concentrations, including nicotinic acid, opiate-receptor antagonists, and cholinomimetics (1, 22). Tse (26) isolated a cholinergic compound from both regular and decaffeinated coffees, purified it, and demonstrated that injecting it into rats resulted in decreases in heart rate and blood pressure. This illustrates that the effects of coffee should not automatically be attributed to caffeine.

Some investigators (3, 4, 9, 25, 31) have used regular coffee (or decaffeinated coffee with caffeine added) and decaffeinated coffee as the means to examine the metabolic actions of caffeine on exercising humans. In these situations they often, but not always (25), found a limited effect of the caffeinated beverage. This includes either modest or no improvement in performance (3, 31) and no increase in serum free fatty acids (FFAs) (9) or no decrease in respiratory exchange ratio (3, 4). In the original work by Costill and co-workers (9) decaffeinated coffee plus caffeine enhanced endurance ~21% compared with decaffeinated coffee. However, studies from our laboratory administering caffeine independent of coffee have demonstrated larger improvements in endurance (28–43%) for similar exercise intensities (11, 13, 20). Although our original studies (13, 20) used a larger caffeine dose (9 mg/kg) than that used by Costill et al., we have recently (14) shown that doses of 3–6 mg/kg are at least as effective. Thus the amount of caffeine administered does not appear to explain the discrepancy. One possibility to account for this difference is that one or more of the multitude of compounds in coffee beverages antagonize the actions of caffeine, resulting in a reduced response.

We examined the metabolic and endurance responses to the ingestion of coffee beverages (regular, decaffeinated, and decaffeinated plus caffeine) or caffeine in trained runners at rest and during strenuous, endurance exercise. Our hypothesis was that ingestion of caffeine in any form would result in an increase in plasma epinephrine levels, FFA concentration, and exercise endurance. We also hypothesized that the actions of caffeine would be greatest when the compound was consumed independent of coffee.

METHODS

Subjects. Nine, young adults who were actively training endurance runners [8 men, mean and range: age 27.8 yr (21–47 yr), wt 73.1 kg (59–81 kg), treadmill maximal O₂

consumption ($\dot{V}O_{2\max}$) 69.1 ml·kg⁻¹·min⁻¹ (65.7–71.0 ml·kg⁻¹·min⁻¹); and 1 woman: age 22 yr, wt 50 kg, and $\dot{V}O_{2\max}$ 52.5 ml·kg⁻¹·min⁻¹] volunteered to take part in the study after they were informed both verbally and in writing about the nature of the experiments. The protocol was approved by the Ethics Committee of the University of Guelph. The caffeine habits of the subjects varied from an abstainer and two very light users (<100 mg/day) to moderate users (<500 mg/day).

Preexperimental protocol. Each subject reported to the laboratory on two occasions before the actual experiments. On the first occasion they performed an incremental $\dot{V}O_{2\max}$ test on a treadmill. Subsequently, on a separate day, the subject performed a practice trial consisting of 20–30 min running on the treadmill at a workload predicted to require 85% of the $\dot{V}O_{2\max}$. The O₂ consumption was measured to confirm the selection was correct. In those cases in which it was not correct, the subject returned for another habituation run in which the workload was adjusted and confirmed to be 85% of $\dot{V}O_{2\max}$.

Experimental protocol. Each subject completed five trials in which they ran "to voluntary exhaustion" on the treadmill at a power output equivalent to ~85% of $\dot{V}O_{2\max}$. Exhaustion was defined as the point when they indicated that they could no longer run at the required speed and slope. The five trials were assigned in a random, double-blind fashion, and the subject was not told the duration of the exercise until after the last trial. The subjects were instructed to abstain from all caffeine-containing foods and beverages for 48 h and to prepare for the trials as they would for an athletic competition (i.e., well rested, consuming a high-carbohydrate diet) and to prepare for each trial in an identical fashion. To assist in this preparation, the subjects kept a food and activity diary for 48 h before each test. They were also told to record fluid intake the day of the trial and to keep this similar for each test. Each subject was tested at the same time of day, and there was at least 1 wk separating trials.

When the subjects reported to the laboratory they were asked to void their bladders as completely as possible. Subsequently, a catheter was placed into a medial antecubital vein, a normal saline drip (100–175 ml/h) was started to maintain catheter patency, and a resting sample was taken (referred to as *time -60*). Then the subject ingested one of the following: placebo (dextrose) capsules with water, caffeine capsules with water, regular coffee, decaffeinated coffee, or decaffeinated coffee plus caffeine. All of the "caffeine" (coffee or capsule) trials resulted in the ingestion of 4.45 mg caffeine/kg. In each experiment the quantity of fluid ingested by a given subject was the same (7.15 ml/kg), and it was consumed in 10 min.

To select a dose of coffee, we mixed a large quantity of ground coffee well to ensure uniformity. The coffee for all the trials was then prepared from this supply. In preliminary tests we prepared drip-filtered coffee as concentrated as the laboratory workers could tolerate with regard to taste and still consume approximately two "coffee mugs" (total of 500–600 ml) in 10 min. Subsequently, we analyzed the caffeine concentration (1.4% caffeine or 62.1 mg caffeine/100 ml coffee) and established that for a 70-kg individual two mugs of coffee would be 4.45 mg/kg and selected this as the test dose. For the decaffeinated coffee plus caffeine trial, the equivalent amount of caffeine was added to the beverage. This is in the midrange of an effective ergogenic caffeine dose (14). Thus the coffee was always prepared in the same fashion (40 g ground coffee and 1,000 ml of water), and the volume consumed was based on the weight of the subject to provide the required caffeine dose. When capsules were given the same volume of water

was consumed in the 10-min period. This was the only fluid ingestion permitted during the course of the experiment. As demonstrated in RESULTS, this procedure achieved conditions in which the circulating concentrations of methylxanthines were virtually identical.

After the fluid ingestion, the subject rested quietly for 1 h. To control for body posture influences on urine production, the subjects were allowed to either sit or lie down, but, whichever position was selected, they had to maintain it and use the same position in each trial. After the 1 h of rest, a second blood sample was taken (referred to as *time 0*), the subjects then voided their bladders and the urine volume was measured.

The subjects were allowed an active warm-up and/or stretching period. Whatever was selected on the first trial was noted, and they were instructed to reproduce the same preparation for subsequent trials. Then the subjects ran on the treadmill at a speed and slope calculated to require 85% of $\dot{V}O_{2\max}$ until voluntary exhaustion. After the exercise, the subjects again voided their bladders and the volume of urine was measured. There were no overt time clues, although blood and expired gas were collected every 15 min and an additional blood sample was taken 2–5 min before exhaustion. Retrospectively, many of the subjects were not able to identify which trials lasted the longest. The subjects were asked to complete a brief questionnaire after each trial to establish whether they believed that they could identify the treatment that they had received.

Analysis. The blood samples (7 ml) were immediately transferred to a sodium-heparinized tube. Hematocrit was measured in duplicate by high-speed centrifugation. A modest hemoconcentration occurred in the samples taken during exercise, but there was no difference among trials. A 100- μ l portion of the blood sample was added to 500 μ l of 0.6 M perchloric acid and centrifuged, and the supernatant was stored at -20°C for lactate, glucose, and glycerol analysis (2, 15, 18). Then, 120 μ l of a 0.24-M EGTA and reduced glutathione solution was added to the remaining heparinized blood. The plasma was derived from centrifugation and stored at -80°C. Later it was analyzed in duplicate for epinephrine and norepinephrine by HPLC (Waters) as described by Weicker et al. (29). In addition, plasma methylxanthines (caffeine, paraxanthine, theophylline, and theobromine) were analyzed by using a fully automated HPLC (Waters) system as follows: 150 μ l of plasma were added to ~40 mg ammonium sulfate and 50 μ l 0.05% acetic acid. After the addition of 25 μ l of internal standard solution (7- β -hydroxypropyl theophylline) and 3 ml chloroform-isopropyl alcohol (85:15, vol/vol) extracting solvent, the mixture was vortexed for 30 s and centrifuged for 10 min at 2,500 rpm. The organic phase was transferred and dried under O₂-free N₂ and was resuspended in HPLC mobile solvent (3% isopropanol, 0.05% acetic acid, and 0.5% methanol), and 100 μ l were injected onto a Beckman, Ultrasphere IP, C₁₈, 5- μ m column. Methylxanthines were measured at 282-nm wavelength.

Statistics. The data are presented as means \pm SE. The data were analyzed by a two-way ANOVA for repeated measures, and differences were accepted as significant if $P < 0.05$. Due to the fact that the total exercise time was different for a given subject in each trial and was also different among subjects, the data are only complete for the first 15 min of exercise and at exhaustion. Thus it is these data that were analyzed as described in *Analysis*.

RESULTS

The plasma concentrations of caffeine and paraxanthine are presented in Figs. 1 and 2, respectively. In

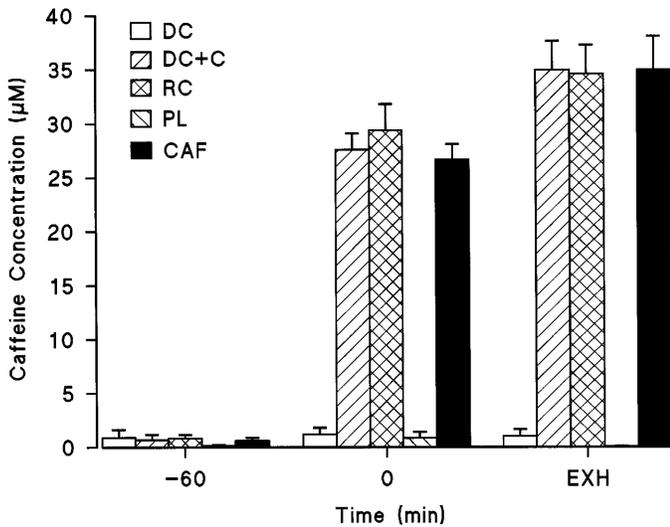


Fig. 1. Plasma caffeine concentration. Coffee beverages or capsules were ingested in a 10-min period after -60-min sample was taken, time 0 sample was taken with the subjects at rest 60 min after ingestion, and final sample was taken immediately before end of the exercise (EXH). DC, decaffeinated coffee; DC+C, decaffeinated coffee plus caffeine; RC, regular coffee; PL, placebo capsules; CAF, caffeine capsules. Dose of caffeine was the same in DC+C, RC, and CAF. Volume of fluid consumed was identical in each trial.

every trial subjects had only a very small, if any, plasma concentration of caffeine at -60 min and low values of paraxanthine (the major metabolite of caffeine), confirming that they had not ingested caffeine during the withdrawal period. On ingestion of either decaffeinated coffee or placebo capsules, there was no change in concentrations of these methylxanthines during the experiment. When the subjects ingested either of the caffeinated coffee solutions or the caffeine capsules, the responses were extremely similar. At 0 min the plasma caffeine concentration was ~28 µM in all three trials (Fig. 1), whereas paraxanthine, the main metabolite of caffeine, increased to ~3-5 µM (Fig. 2). Theophylline had a small but significant increase (data not shown), whereas the theobromine

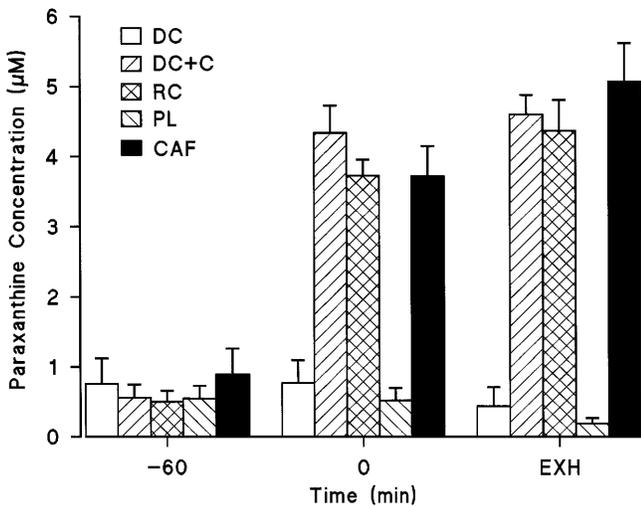


Fig. 2. Plasma paraxanthine concentration. Organization is the same for as Fig. 1.

concentration was low and did not change. By the time the subjects reached exhaustion, there was a modest, significant increase in plasma caffeine concentration from the onset of exercise; however, the responses among the three caffeine trials remained very similar. There was no time when the plasma concentrations of caffeine or paraxanthine were different among the three caffeine treatments.

The subjects varied in their ability to identify the treatment received; six of the nine correctly identified the decaffeinated coffee, and eight identified the placebo capsules. For the caffeine treatments, seven of nine identified the caffeine capsules correctly. In contrast, the subjects correctly identified the coffee beverages containing caffeine in only 13 of 18 trials. Because 2 of the 3 beverages contained caffeine, one would predict that 12 of 18 "guesses" would be correct by random chance. Only three subjects could correctly identify every trial. The reasons included "feeling jittery," feeling energetic, increased urine production (even though statistically there was no evidence of this; see below), and relief from a headache for the caffeine treatments. However, both in this respect and throughout the results there was no clear relationship between regular caffeine habits and the results.

The mean volume of fluid ingested was 7.15 ml/kg or 505 ml. There were no differences among trials in the volume of urine produced, but it was less after the exercise than before the exercise. The production during the 1 h preexercise was 349 ± 75, 423 ± 95, 428 ± 91, 354 ± 70, and 375 ± 125 ml for decaffeinated coffee, placebo capsules, decaffeinated coffee with caffeine, regular coffee, and caffeine capsules, respectively. The corresponding data for postexercise were 82 ± 16, 82 ± 19, 88 ± 24, 79 ± 25, and 79 ± 21 ml of urine.

Figure 3 summarizes the results for endurance; caffeine capsule ingestion resulted in a significant increase in endurance time of 7.5-10 min compared

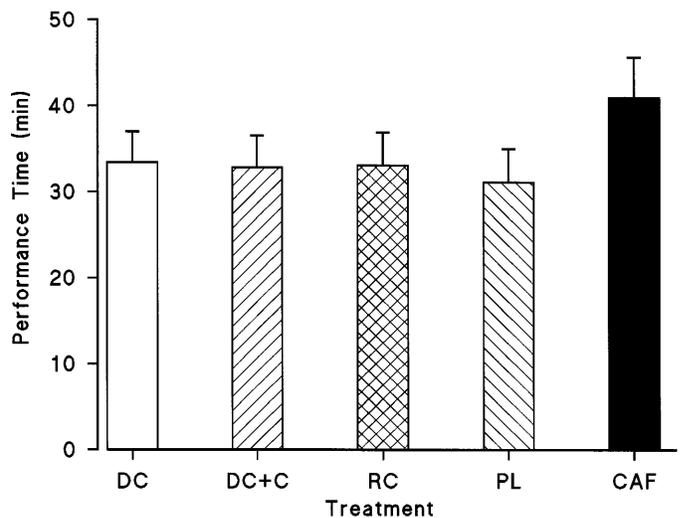


Fig. 3. Endurance time for the 5 trials. Caffeine capsule test resulted in endurance being increased ($P < 0.05$) beyond that of the other 4 trials. There were no differences among the other trials. Increased endurance time after caffeine capsules meant that subjects were able to run an additional 2-3 km at the prescribed speed and slope.

with the other four trials. Six subjects had their longest run of all five trials after ingestion of caffeine capsules, seven had longer runs after caffeine compared with placebo capsules, and eight ran longer after caffeine ingestion compared with after they drank decaffeinated coffee. In sharp contrast, there were no differences among the three trials in which coffee was ingested, and the results were not different from the placebo capsule trial. This is surprising in light of the remarkably similar data for plasma caffeine and paraxanthine concentrations for the three caffeine trials. Within the three trials with coffee beverages, there was no consistency in performance time and no apparent tendency for any one beverage to produce an ergogenic effect. For example, within these trials, three subjects had their longest run after decaffeinated coffee, whereas the other six subjects were equally divided between the other two caffeinated beverages.

The summary data for blood lactate and glucose and plasma glycerol are presented in Table 1. There were no treatment effects for blood glucose, but it increased progressively during exercise (the data at 15 min and exhaustion were significantly greater than at -60 min, and the data at exhaustion were also greater than at 15 min). Blood lactate increased progressively during exercise, and at both 15 min and at exhaustion the data for the regular coffee treatment were greater than those for placebo and for decaffeinated coffee. At 15 min it was also greater than the blood lactate for the caffeine trial. Plasma glycerol was not different among treatments, and it rose progressively during the exercise.

The data for plasma epinephrine and norepinephrine are presented in Figs. 4 and 5, respectively. At -60 min there were no differences among trials in plasma epinephrine; however, it increased during the next hour in the three caffeine trials (Fig. 4A). It is surprising that, despite virtually identical plasma methylxanthine data, the three caffeine treatments were not characterized by similar increases in epinephrine during the rest period. The value at 0 min for the caffeine trial was significantly greater than that of all the other trials, and that for the two caffeinated beverages was lower than that for caffeine capsules but greater than

that for placebo or decaffeinated coffee. During exercise, the plasma epinephrine increased in all conditions and the three caffeine treatments had greater concentrations than the placebo and decaffeinated trials (Fig. 4B). There were no treatment effects for plasma norepinephrine, and it increased progressively with the onset of exercise. In addition, from -60 to 0 min there was a modest, but significant, increase for all the trials except the decaffeinated coffee.

DISCUSSION

This study was designed to compare the metabolic and exercise endurance responses to the ingestion of the same amounts of caffeine as a coffee beverage and as pure caffeine with water. The caffeine was consumed in the same volume of coffee or water in the same period of time. It resulted in very similar plasma concentrations of plasma methylxanthines, but only when it was consumed independent of coffee was there an enhancement of endurance. In addition, in this trial the initial impact on circulating epinephrine concentration was greatest. Thus it appears that some component(s) in coffee interferes with the normal ergogenic response of caffeine.

In the present study the endurance after caffeine ingestion was improved 9.9 min or 31% over that for placebo capsules (and 7.6 min or 22.8% over that for decaffeinated coffee). The ingestion of caffeinated coffee did not enhance endurance in the present study. Of the two studies in which subjects were given caffeine with decaffeinated coffee before continuous, endurance exercise, one (3) did not demonstrate a significant effect on endurance (in agreement with the present data), and Costill and co-workers (9) found a 19.5% improvement. One reason for the inconsistencies in the literature may be due to the vehicle used to administer the caffeine.

Unfortunately, neither plasma methylxanthine nor catecholamine concentrations were determined in the previous investigations (3, 4, 9, 25, 31) that used coffee as the vehicle of administration. However, the doses of caffeine used in these previous studies were between 4 and 6 mg/kg, i.e., similar to that used in the present

Table 1. Summary of blood metabolites

Parameter	Time	Treatment				
		DC	DC+C	RC	Pl	Caf
Glucose, mM	-60 min	3.4 ± 0.2	3.6 ± 0.2	3.6 ± 0.2	3.7 ± 0.2	3.6 ± 0.4
	0 min	3.7 ± 0.3	4.2 ± 0.3	4.2 ± 0.2	3.7 ± 0.3	4.0 ± 0.2
	15 min	4.0 ± 0.4	4.3 ± 0.5	4.8 ± 0.4	4.3 ± 0.5	4.7 ± 0.5
	Exh	4.8 ± 0.6	5.6 ± 0.4	5.8 ± 0.5	5.4 ± 0.5	5.4 ± 0.5
Lactate, mM	-60 min	0.8 ± 0.1	0.7 ± 0.2	0.9 ± 0.1	0.7 ± 0.1	0.9 ± 0.3
	0 min	1.1 ± 0.2	1.0 ± 0.2	1.0 ± 0.0	0.7 ± 0.2	1.4 ± 0.3
	15 min	3.1 ± 0.6	2.8 ± 0.3	3.4 ± 0.8	2.4 ± 0.3	2.9 ± 0.6
	Exh	4.2 ± 0.8	4.1 ± 0.6	5.1 ± 1.2	3.4 ± 0.5	4.0 ± 1.0
Glycerol, μM	-60 min	59.64 ± 5.83	55.49 ± 6.61	55.67 ± 9.96	46.53 ± 5.18	59.39 ± 5.85
	0 min	50.24 ± 6.00	61.30 ± 6.41	53.33 ± 11.37	50.79 ± 6.31	57.35 ± 5.54
	15 min	90.74 ± 17.52	86.62 ± 11.11	99.86 ± 14.50	101.84 ± 11.78	86.05 ± 11.39
	Exh	136.15 ± 23.34	153.20 ± 31.25	133.10 ± 20.48	135.00 ± 19.76	136.41 ± 19.04

Values are means ± SE for 9 subjects. DC, decaffeinated coffee; DC+C, decaffeinated coffee plus caffeine; RC, regular coffee; Pl, placebo capsules; Caf, caffeine capsules; Exh, exhaustion.

study. The plasma methylxanthine data from the present study are in agreement with those that we reported previously (14). The data demonstrated that the differences among treatments are not due to plasma methylxanthine concentrations. It would appear that neither caffeine absorption nor metabolism was different among the caffeine trials and that the explanation for the differences is not due to the caffeine bioavailability.

Stavric (22) and Arnaud (1) pointed out that it should not be assumed that the only biologically active com-

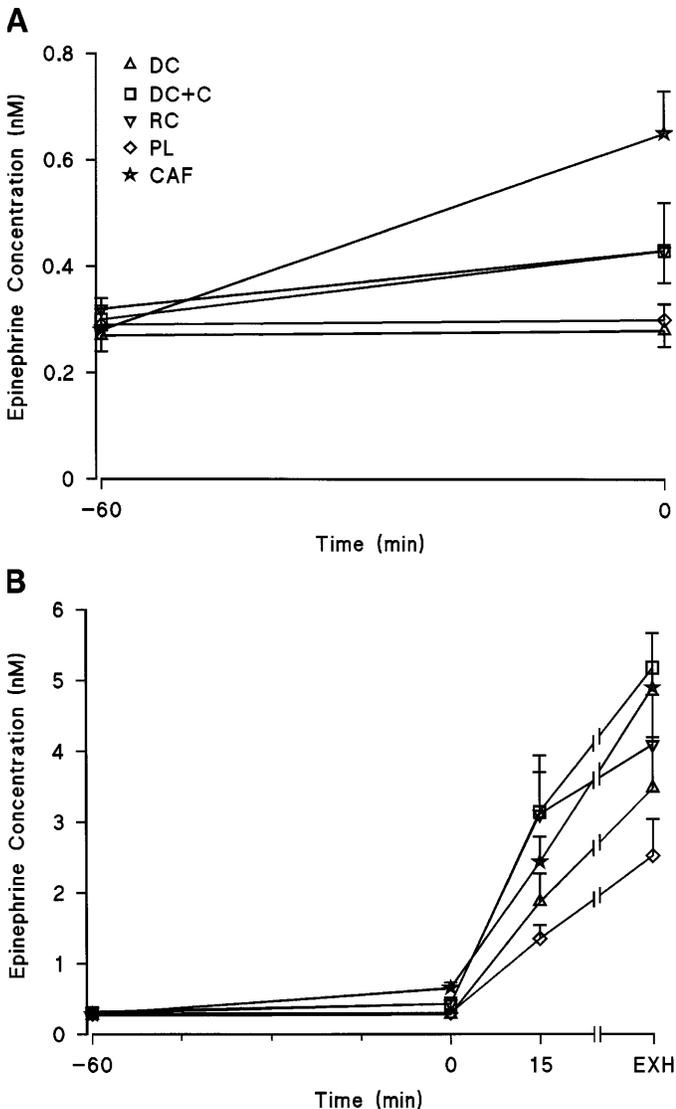


Fig. 4. Plasma epinephrine responses to caffeine, coffee, and exercise. Because of differences in absolute concentrations at rest and during exercise, resting data are presented in *A* with a more-sensitive concentration scale. Decaffeinated coffee and placebo were not different and did not change at rest. Two caffeinated beverages (regular coffee and decaffeinated coffee plus caffeine) resulted in an increase ($P < 0.05$) in epinephrine at *time 0*. Ingestion of caffeine independent of coffee resulted in a greater ($P < 0.05$) increase than in any other trial. *B* summarizes overall data and is organized the same as *A*, but some SE have been deleted for clarity. Exercise began at *time 0*, and the 3 caffeine tests all had increases in epinephrine that were similar and were greater ($P < 0.05$) than those of decaffeinated coffee and placebo.

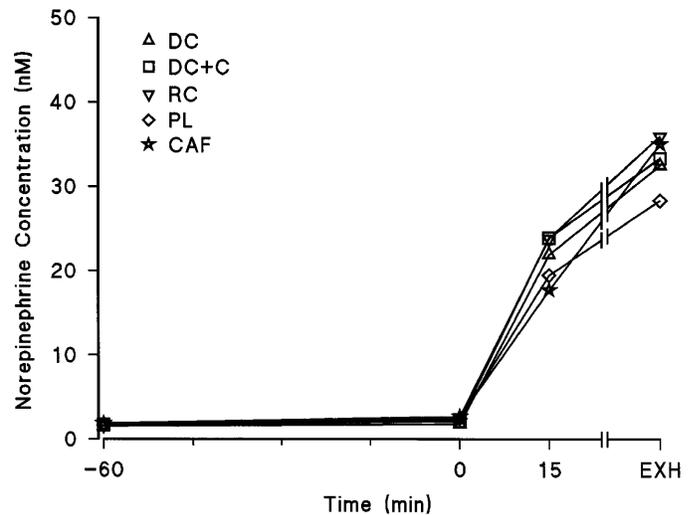


Fig. 5. Plasma norepinephrine responses to coffee, caffeine, and exercise. Organization is the same as for Fig. 4. There were no significant differences among the 5 treatments. SE bars have been deleted for clarity.

pound in coffee is caffeine. For example, coffee contains small amounts of nicotinic acid and opiate-receptor antagonists. In addition, Tse (26) demonstrated that both decaffeinated and regular coffee contain an unidentified cholinomimetic compound that produced abrupt depression in heart rate and blood pressure when injected into rats. It also caused a dose-dependent relaxation of vascular smooth muscle during *in vitro* tests. Its effects were blocked by atropine, but not by adrenergic, purinergic, or opiate antagonists (suggesting that it acted on muscarinic pathways), and it did not appear to act via the central nervous system. It may well be that this and/or other compounds have peripheral actions that antagonize the common responses to caffeine.

As noted earlier, the studies that used coffee as the caffeine vehicle have not measured plasma catecholamines. We have reported repeatedly (13, 14, 20) that caffeine ingestion results in an increase in plasma epinephrine within 1 h at rest and that this increase remains evident during exercise. The present data are consistent with this finding because the ingestion of caffeine capsules resulted in a doubling of plasma epinephrine during the 1 h of rest. Caffeine in the form of coffee resulted in the same plasma caffeine concentration, but the epinephrine response was only ~50% of that with the caffeine capsules. This suggests that the "cholinomimetic factor(s)" suppressed the sympathetic response either within the central nervous system or at the adrenal medulla itself. The present data cannot discriminate between these two possibilities, but a central nervous system action is more probable because the normal elevated epinephrine response to caffeine capsule ingestion is absent in tetraplegics (19, 27).

It is impossible in the present study to attribute a significant metabolic or endurance action to the differences in plasma epinephrine. It has been shown (6) that infusion of epinephrine to achieve concentrations similar to those induced by caffeine either at rest or during

exercise did not alter blood metabolites or intramuscular glycogen metabolism. In addition, in tetraplegics, who experienced no increase in plasma epinephrine after caffeine ingestion, there was an increase in plasma FFA concentrations (27) and increased endurance (19). Thus we do not feel that the differences in plasma epinephrine in the present study necessarily result in the differences in endurance. Our interpretation is that the rise in epinephrine at rest is a reflection of a caffeine-induced action and that the difference in plasma epinephrine is a clear demonstration that some aspect of the physiological responses to caffeine is modified by some component in both regular coffee and decaffeinated coffee.

One potentially detrimental aspect of caffeine ingestion could be diuresis and a reduction in plasma volume. Falk et al. (10) found that caffeine ingestion followed by prolonged exercise in the heat did not influence hemoconcentration or total body water loss and Wemple et al. (30) found no detectable diuresis due to caffeine ingestion during the first hour of rest and no changes in plasma volume or osmolality. The data from the present study support these findings. The subjects did not have more diuresis or hemoconcentration as a result of ingesting caffeine either with water or in coffee. The diuresis observed of ~500 ml appeared to be primarily due to the volume of fluid ingested independent of the caffeine content. Robertson et al. (20) found that caffeine caused a diuresis of ~100 ml during a 3-h rest, and this was associated with a 50% increase in renin activity. However, much of the renin increase occurred after the first hour; thus in the present study the exercise stress, beginning after 1 h of rest, could have interacted with the potential renin effect. In agreement with this interpretation, Wemple et al. (30) found a caffeine diuresis only from the second to the fourth hour of rest, and this did not occur if exercise was performed after the first hour.

The present data for the circulating concentrations of glucose, glycerol, and lactate generally failed to show any caffeine effects. Many studies have reported a lack of a difference in respiratory exchange ratio or in plasma FFA and glycerol concentrations after caffeine or coffee ingestion (11, 23). The literature is highly variable with respect to these measurements, with many of the reports being negative. It is common to have findings of a caffeine-associated increase in blood lactate (7, 13, 16); in the present study this was only associated with the regular coffee ingestion. We can offer no explanation for this finding. We have previously suggested (11, 27) that the actions of caffeine may not be associated with catecholamine responses or with the mobilization of fats. It is possible that caffeine affects excitation-contraction coupling mechanisms (23). In support of this hypothesis, studies in which human muscle has been electrically stimulated (17, 23) have shown that caffeine ingestion can enhance force production.

In summary, we investigated the effects of caffeine ingestion in association with coffee on endurance and metabolism during exercise. The results clearly demon-

strated that caffeine ingested in this form does not alter the bioavailability of caffeine but fails to enhance endurance. In addition, the increase in plasma epinephrine that is normally observed in the first hour after ingestion was moderated. The results suggest that other compounds in coffee act to antagonize the responses observed when caffeine is ingested independent of coffee.

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