Caffeine as a Lipolytic Food Component Increases Endurance Performance in Rats and Athletes

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(Received October 6, 2000)

Summary Caffeine is one of the famous ergogenic aids in the athletic field. Caffeine has been known to stimulate lipolysis that spares stored glycogen utilization during moderate intensity exercise. Therefore, we investigated the effects of caffeine ingestion on exercise performance in rats and athletes. Rats were administered the caffeine (6 mg/kg) 1 h prior to the exercise then were run on a treadmill at a speed of 20 m/min. They were decapitated at 0 min, 30 min, 60 min of exercise, and exhausted time point. Human subjects ingested the caffeine (5 mg/kg) 1 h prior to the exercise. They exercised on a cycle ergometer at 60% of their VO2max for 45 min, and then the exercise intensity was increased to 80% of their VO2max until exhaustion. Blood and breathing gas samples were collected and calculated every 10 min during exercise. Respiratory exchange ratio of the caffeine trial was significantly lower than that of the placebo trial in the athletes’ study (p<0.05). Blood free fatty acid (FFA) levels in studies of both rats and athletes were increased by caffeine ingestion during exercise (p<0.05). Blood lactate levels were also increased during exercise in both rats and athletes (p<0.05). Increased FFA and glycerol concentrations reduced glycogen utilization during exercise compared with placebo group in rats. In addition, endurance time to exhaustion was significantly increased by the caffeine ingestion in both rats and athletes (p<0.05). These results suggest that the caffeine ingestion enhanced endurance performance resulting from spare stored glycogen with increasing lipolysis from adipose tissues and fat oxidation during exercise both in rats and in athletes.

Key Words caffeine, exercise, fat oxidation, rat, athletes

It is well known that caffeine ingestion enhances fat metabolism by altering the lipolytic hormones and fat oxidative capacity in skeletal muscle resulting in glycogen sparing effect. In other words, caffeine ingestion prior to exercise enhances fatty acids mobilization that is used as the major energy source during endurance exercise under 60% of maximal oxygen consumption (VO2max) (1). Caffeine inhibits phosphodiesterase activity in cells by catecholamines release, and the increase of cAMP accumulation in skeletal muscle and adipose tissues. Caffeine is also known to increase hormone sensitive lipase (HSL) activity and to enhance fat mobilization from the skeletal muscles and adipose tissues, which results in increased free fatty acid (FFA) in the blood stream (2). Endogenous epinephrine secretion is increased by exercise and release of this hormone during exercise may be augmented by many factors including caffeine ingestion (3). Therefore, caffeine ingestion before endurance exercise has been investigated as an ergogenic aid by many researchers (3–7). They reported consistently that caffeine increases endurance exercise performance time and delays fatigue.

To enhance endurance performance, effective use of stored glycogen in muscle and liver has been researched. Unfortunately, glycogen storage capacity in body is limited. It is well known that amount of stored glycogen is about 400 g in 80 kg of healthy male. Therefore, the chief causes of fatigue during prolonged endurance exercise are hypoglycemia and stored glycogen depletion (8).

The increase of fatty acids utilization during such an exercise reduces the glycogen depletion rate and improves endurance exercise performance (9, 10). Therefore, the increase of fatty acids utilization before exercise is thought to be important for endurance performance. As reported by researchers above, caffeine is a meaningful source because increased plasma FFA concentrations reduce glycogen depletion (11–13).

In the sports science field, many research groups have investigated the effects of food supplements on endurance performance. They used to investigate the effect of supplements we previously suggested as lipolytic foods, such as carnitine (14), capsaicin in red hot pepper (15) and caffeine (16, 17) with human muscle biopsy and/or isotope techniques. The indirect energy consumption method from expired gas analysis is also used in this field. However, endurance exercise time to the exhaustion stage is an actual parameter for endurance exercise performance. From this point of view,
in the present study, we investigated the effects of caffeine ingestion prior to endurance exercise on endurance performance in rats and athletes.

**MATERIALS AND METHODS**

*Experiment 1 (Rats)*

**Animal care and experimental design.** Forty-eight Sprague-Dawley male rats 3 wk of age were obtained from Korea Institute of Chemical Technology (Daejeon, Korea). During the experimental period, including 3 wk of environmental adaptation, rats were fed with a commercial chow diet (Samyang Oil & Feed Co., Seoul, Korea) ad libitum, had free access to water and were kept in rooms maintained on a 12-h light (2000-0800)/12-h dark (0800-2000) cycle at 23°C. All rats were subjected to running exercise on a motor-driven treadmill designed for the rats during the darkness cycle.

The intensity and duration of the exercise was gradually increased during the first week of the exercise program (including pre-exercise training for adaptation: 5 min/d and 5 m/min for three days, and 10 min/d and 10 m/min for two days), with the final speed and duration of 20 m/min and 30 min/d, respectively. The rats were exercised five days per week throughout the experimental period.

On the final day of the experiment, 6 mg/kg of caffeine was offered by oral intubation using a round-ended needle to the caffeine (CA) group and 1.0 mL of water was administered to the placebo (PL) group in the same way 1 h prior to the exercise. They were sacrificed by decapitation before the exercise (0 min), at 30 min of exercise (30 min), 60 min of exercise (60 min), and the exhaustion time point, respectively. Blood was collected after decapitation for the measurement of blood glucose, lactate, FFA and plasma glycerol concentrations. Liver and muscles were clamped then frozen in the liquid nitrogen to hold activation. All samples were stored at -70°C in a deep-freezer for future analysis. The experimental procedures are shown in Fig. 1.

**Measurements and analysis.** Blood glucose and lactate were analyzed using an auto-analysis system (YSI 2300 Plus, Yellow Springs Institute, USA) (15). Plasma FFA and glycerol concentrations were analyzed using a NEFAzyme-Kit (Eiken, Tokyo, Japan) and a Glycerol-Kit (Boehringer Mannheim GmbH, Mannheim, Germany) (18). Glycogen contents in liver and muscle were assayed by the method reported previously (18, 19).

*Statistics.* Data are described as mean and standard error (M±SE). To compare the differences between groups, Student’s unpaired t-test method was used. The *p*<0.05 was obtained if statistically significantly difference was found between the groups.

*Experiment 2 (Athletes)*

**Subjects.** Five healthy rugby players participated in this experiment. They understood and agreed with the purpose of the study and gave written consent. They did not have any kind of disease or use of drug for at least 1 year. Even though they were not habitual caffeine consumers, we educated them never to drink caffeine-containing drinks. The subjects were trained at least 5 years and had an average maximal oxygen uptake (VO₂max) of 53.2 mL/kg/min. VO₂max was measured using indirect calorimetric technique during an incremental exercise bout (15). Subject characteristics are shown in Table 1. The experiment was approved by Institute of Medical Science of Kyungpook National University in accordance with the Helsinki Declaration of 1975.

Subjects arrived 150 min before endurance exercise at the laboratory. They consumed 640 kcal of a formal diet (bread, eggs, and orange juice) at 120 min before exercise, then drank caffeine or water 1 h after the meal. After the meal, rest-expired gas was measured for 5 min. Caffeine ingestion group ingested 5 mg/kg of caffeine, dissolved in 250 mL of water. International Olympic Committee (IOC) decided caffeine ingestion is illegal if urinary caffeine is over 12 mg/L. Jackman et al. (20) reported that 6 mg/kg of caffeine was offered by oral intubation using a round-ended needle to the caffeine (CA) group and 1.0 mL of water was administered to the placebo (PL) group in the same way 1 h prior to the exercise. They were sacrificed by decapitation before the exercise (0 min), at 30 min of exercise (30 min), 60 min of exercise (60 min), and the exhaustion time point, respectively. Blood was collected after decapitation for the measurement of blood glucose, lactate, FFA and plasma glycerol concentrations. Liver and muscles were clamped then frozen in the liquid nitrogen to hold activation. All samples were stored at -70°C in a deep-freezer for future analysis. The experimental procedures are shown in Fig. 1.

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**Table 1. Physical characteristics of the subjects (Experiment 2).**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Value (M±SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>19.4±0.36</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>178.9±1.74</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>74.3±1.59</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>19.2±0.36</td>
</tr>
<tr>
<td>VO₂max (mL/kg/min)</td>
<td>53.2±1.59</td>
</tr>
</tbody>
</table>

Values are mean±SE. *Body fat was analyzed using a body fat analyzer (TBF-105, Tanita, Japan).*

**Training**

<table>
<thead>
<tr>
<th>Age (wk)</th>
<th>Treadmill, 20 m/min 0% incline, 30 min/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

**Final day**

- Remove meal
- Caffeine
- Sacrifice
- Treadmill exercise

-120 min -60 min 0 30 min 60 min Exhaustion

Fig. 1. Experimental procedures of Experiment 1 (rats).
caffeine ingestion before exercise is not over the IOC's limitation. From this point of view, we hypothesized that 6 mg/kg of caffeine ingestion had maximum effect in human. Therefore, 5 mg/kg of caffeine was selected in this experiment.

The subjects did stretching exercises for 2–3 min after 5 min of resting metabolic rate (RMR) measurement before 10 min of exercise. A 3-way catheter was inserted in the anticubital vein prior to exercise for blood collection every 10 min of exercise for 45 min. They exercised on a cycle ergometer at 60% VO$_{2}$max for 45 min, then exercise intensity was increased to 80% VO$_{2}$max until exhaustion. Exhaustion was defined when a subject could not maintain 50 rpm speed of pedaling. Expired gas was collected in a Douglas bag (Fukuda Sangyo, Tokyo, Japan) every 2 min at the first period of exercise at 60% VO$_{2}$max and 2 min at 80% of VO$_{2}$max.

Blood glucose, lactate, and FFA assays were same as in Experiment 1. The experimental procedures are shown in Fig. 2.

Statistics. Data are described as mean and standard error (M$\pm$SE). To compare the differences during exercise and between groups, one-way ANOVA repeated measure method was used. In addition, Student's t-test method was used to compare exercise time to exhaustion between groups. The p<0.05 was obtained if statistically significantly difference was found between groups.

RESULTS

Experiment 1 (Rats)

In the present experiment, Sprague-Dawley male rats were used as the subjects. They ingested caffeine (6 mg/kg) in CA group or water (1.0 mL) in PL group 1 h prior to exercise on the treadmill. We investigated the effects of caffeine on the energy substrates and glycogen utilization in Experiment 1.

Blood measurements

Plasma glucose levels in CA group were significantly lower at 60 min and exhaustion compared with those of PL group (p<0.05) (Fig. 3; upper). Blood lactate concentrations were not significantly different at pre-exercise between the two groups, but significantly higher in CA group at 30 min, 60 min, and exhaustion (p<0.05) (Fig. 3; lower). Plasma FFA concentration was not different at pre-exercise, 30 min, and exhaustion between groups, but higher in CA group than in PL group at 60 min (p<0.05) (Fig. 4; upper). Glycerol concentrations were significantly higher in CA group at 0 min, 30 min, 60 min, and exhaustion compared to PL group (p<0.05) (Fig 4; lower).

Glycogen contents

Glycogen contents in soleus muscle were not different at 0 min, 30 min, 60 min, and exhaustion between the two groups (Fig. 5; top). Gastrocnemius white muscle glycogen contents were not significantly different at 0 min, 30 min, and exhaustion between the two groups, but higher in CA group than in PL group at 60 min (p<0.05) (Fig 5; middle). Liver glycogen contents were not different at 0 min, 30 min, and 60 min between groups, but significantly higher in CA group at exhaustion compared to PL group (p<0.05) (Fig. 5;
Endurance exercise time

Exercise time to exhaustion was significantly longer in CA group compared to PL group \((p<0.05)\) (Fig. 6).

Experiment 2 (Athletes)

In this Experiment 2, five healthy male rugby players participated as the subjects. They ingested caffeine (5 mg/kg dissolved in 250 mL water) in CA trial or water (250 mL) in PL trial 1 h prior to cycling exercise performance. We investigated the effects of caffeine on calorimetry and energy substrates utilization in Experiment 2.

Expired gas measurements

Oxygen uptake was not significantly different at 0 min nor during exercise between the two trials (Table 2). Respiratory exchange ratio (RER) was slightly lower in CA trial though not significantly different at 0 min but it was significantly higher in CA trial than PL trial throughout the periods of exercise \((p<0.05)\) (Table 2).

Fat and carbohydrate oxidation

Fat oxidation was higher in CA trial than in PL trial during exercise for 45 min \((p<0.05)\) (Fig. 7: upper). However, carbohydrate oxidation was lower in CA trial during exercise \((p<0.05)\) (Fig. 7: lower).

Blood measurements

Blood glucose levels were not different at pre-exercise and during exercise between the two trials (Table 3). Blood lactate levels during the exercise were higher in
Table 2. The oxygen consumption and respiratory exchange ratio during exercise at 60% VO₂max (Experiment 2).

<table>
<thead>
<tr>
<th>Group</th>
<th>Exercise time (min)</th>
<th>Oxygen consumption (mL/min/kg)</th>
<th>RER</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td>Placebo</td>
<td>3.63±0.05</td>
<td>28.95±1.22</td>
<td>33.50±1.25</td>
</tr>
<tr>
<td>Caffeine</td>
<td>3.68±0.05</td>
<td>27.33±1.54</td>
<td>32.90±1.03</td>
</tr>
<tr>
<td>Placebo</td>
<td>0.82±0.10</td>
<td>0.90±0.01</td>
<td>0.88±0.01</td>
</tr>
<tr>
<td>Caffeine</td>
<td>0.80±0.02</td>
<td>0.86±0.02*</td>
<td>0.85±0.01*</td>
</tr>
</tbody>
</table>

Values are M±SE. * Statistically different from placebo of the same time point (p<0.05).

Table 3. Blood glucose, lactate and free fatty acid levels during exercise at 60% VO₂max (Experiment 2).

<table>
<thead>
<tr>
<th>Group</th>
<th>Exercise time (min)</th>
<th>Blood glucose (mg/100 mL)</th>
<th>Blood lactate (mmol/L)</th>
<th>Plasma FFA (μEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>10</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>Placebo</td>
<td>81.6±3.1</td>
<td>80.0±3.8</td>
<td>79.8±4.0</td>
<td>78.6±4.3</td>
</tr>
<tr>
<td>Caffeine</td>
<td>80.4±3.0</td>
<td>82.8±2.9</td>
<td>83.2±2.9</td>
<td>79.8±2.7</td>
</tr>
<tr>
<td>Placebo</td>
<td>1.32±0.13</td>
<td>1.92±0.15</td>
<td>1.70±0.16</td>
<td>1.60±0.24</td>
</tr>
<tr>
<td>Caffeine</td>
<td>1.32±0.13</td>
<td>1.92±0.15</td>
<td>1.70±0.16</td>
<td>1.60±0.24</td>
</tr>
<tr>
<td>Placebo</td>
<td>495±51</td>
<td>462±87</td>
<td>550±62</td>
<td>686±57</td>
</tr>
<tr>
<td>Caffeine</td>
<td>630±123</td>
<td>540±60</td>
<td>705±34</td>
<td>876±104</td>
</tr>
</tbody>
</table>

Values are M±SE. * Statistically different from placebo of the same time point (p<0.05).

Fig. 7. Changes of fat oxidation (upper) and carbohydrate oxidation (lower) during cycle ergometer exercise at 60% VO₂max in Experiment 2. Values are M±SE. * Significant difference between the two groups (p<0.05).

CA trial compared with PL trial, but not significantly. Plasma FFA levels were higher at 40 and 45 min of the exercise in CA trial as compared with PL trial (p<0.05).

Endurance exercise time
Exercise time to exhaustion was significantly longer in CA trial compared to PL trial (p<0.05) (Fig. 8).

DISCUSSION

This study provides more information about "caffeine effect", as a lipolytic food component, on endurance performance in rats and athletes. A lipolytic food is one that stimulates lipolysis and/or fat oxidation at rest or during exercise in humans. We have studied and sur-
Caffeine ingestion 60 min prior to exercise resulted in an increase of FFA concentration (26), which has been shown to increase lipid metabolism and decrease muscle glycogen utilization. Fat oxidation is enhanced with increased FFA concentration. Lipolysis was determined by blood glycerol concentration that dissolved with 3 mol of fatty acids from stored triglyceride, because FFA was reesterified in the adipose tissue. In support of this contention, glycerol concentration was significantly elevated following exercise (Fig. 4, lower). This result suggested that caffeine enhances lipolysis activity in adipose tissue. In addition, as reported by Klein et al. (27), about half of the FFA mobilized from adipose tissue was utilized in contractive muscles during moderate intensity endurance exercise. It seems from their report that caffeine increases FFA mobilization (Fig. 4, upper) that becomes the main energy source during endurance exercise. Moreover, in Experiment 2, RER was significantly lower in the caffeine ingestion trial than the placebo trial from 0 min to 45 min of exercise (Table 2). These results also suggest that fatty acids increased by caffeine ingestion were utilized as a major energy source during moderate endurance exercise. Paraxanthine, known to have the lipolytic effect of caffeine, is significantly correlated (r=0.93) with FFA concentration (28). In addition, caffeine reduces phosphodiesterase activity that is stimulated by catecholamines. Increased catecholamine level in blood enhances cyclic adenosine monophosphate (cAMP) and HSL activity in skeletal muscle and adipose tissue, respectively. Therefore, augmented hydrolysis of the stored TG releases more FFA in the blood (2).

Falk et al. (29) reported that FFA concentration was significantly increased by caffeine ingestion during exercise. However, plasma lactate accumulation was also increased at the latter stage of exercise because liver and skeletal muscle glycolysis increased (30). Therefore, it is possible that caffeine attenuates fatty acid oxidation in the skeletal muscle during exercise. Poehlman et al. (31) reported that 300 mg of caffeine ingestion at rest increases RMR in untrained subjects compared to trained subjects. At the cellular levels, caffeine affects muscle, adipose and central nervous tissue by indirectly mediating the level of cAMP, thus promoting lipolysis by activation of HSL and its related calcium release from the intracellular storage sites (32). More recent work using caffeine, which causes the release of calcium from the sarcoplasmic reticulum (33), indicates that an increase in cytosolic calcium concentration leads to increased muscle glucose transport even at cytosolic calcium concentrations too low to elicit muscle contraction (34). Thus, the increase in cytosolic calcium concentration might be an important initiator for the enhanced glucose transport during contractions. In the present study, however, both experiments in rats and athletes were performed with trained subjects. Therefore, even though caffeine stimulates glycogenolysis, lipolysis suppression effects were less in the present studies. However, lactate levels in CA groups were highly maintained compared to PL groups in both experiments. We cautiously suggest from these results that caffeine might attenuate the process by which lactate converts into pyruvate to maintain high lactate concentrations.

Spriet et al. (35) reported that ingestion of caffeine 1 h before exercise decreases muscle glycogen utilization about 55% during 15 min of exercise compared to that of subjects receiving placebo. The glycogen thus saved becomes an available energy source for the following phases of exercise, which delays onset of fatigue. As shown in Experiment 1, glycogen concentrations in muscle and liver were reduced more slowly during exercise in CA group than PL group. Dyck et al. (36) suggested that the elevation of plasma FFA during intense cycling exercise spares muscle glycogen by post-transformational regulation of phosphorylase. Their result may be due to blunted increases in the contents of adenosine monophosphate (AMP). These results suggest that FFA mobilized by caffeine ingestion is used as a main energy source during moderate intensity exercise in rats, which enhances exercise time to exhaustion.

In most studies which have investigated the effects of caffeine on exercise performance, exercise duration was about 1 h long. Of these, Costill et al. (37) reported increased time to exhaustion with caffeine ingestion. Ivy et al. (38), who also reported enhanced exercise performance and found a significant difference in lipid oxidation. In addition, time to exhaustion was significantly increased by 16.25 min after caffeine treatment (39). In the present study, endurance time to exhaustion in both experiments was significantly higher in CA groups compared with PL groups. In Experiment 1, although both groups were run on the treadmill at the same running speed and incline until exhaustion, exercise time to exhaustion of CA groups was prolonged. Moreover, in Experiment 2, subjects were exercised on the cycle ergometer at 60% VO2max for 45 min, then exercise intensity was increased at 80% VO2max until exhaustion. This experimental design means that FFA was utilized at the former stage of exercise, resulting in spared glycojen content, which produced more power during the latter stage of exercise. We could propose that increased fat oxidation by caffeine at the former stage enhanced endurance performance at the last spurt. Several recent studies have examined the performance and metabolic effects of caffeine in well-trained subjects. The studies examined the effects of caffeine ingestion of 9 mg/kg body weight in running and cycling performance at 80–85% VO2max (35, 39), and the effects of moderate...
caffeine ingestion (5 mg/kg) on performance of repeated 30 min bouts of cycling at 85–90% VO2max (40). These studies reported that endurance performance was improved by 20–50% compared to placebo trial in elite and recreationally trained subjects.

In summary, caffeine ingestion 1 h prior to exercise enhances endurance performance. We proposed caffeine as a lipolytic food for its significant effects in producing glycerol sparing. There are two reasons as follows: first, caffeine enhances lipolysis activity that maintains higher FFA concentration during moderate intensity exercise, which becomes the main energy source in the skeletal muscle; second, glycogen spared by caffeine ingestion can be used as an important energy source during the latter stage of exercise, especially if the exercise intensity is increased at that point. Therefore, caffeine is a useful ergogenic aid 1 h prior to endurance exercise in rats and humans at moderate intensity exercise.

Acknowledgements

We thank the laboratory staff of Exercise Nutrition at Kyungpook National University for their animal care and the rugby team of Kyung-Hee University for their participation as subjects.

REFERENCES


