(-)-Hydroxycitric Acid Ingestion Increases Fat Utilization During Exercise in Untrained Women

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Summary (-)-Hydroxycitric acid (HCA) is a competitive inhibitor of the enzyme ATP-citrate-lyase, which inhibits lipogenesis in the body. Moreover, HCA increases endurance exercise performance in trained mice and athletes. However, had not been investigated in untrained animals and humans. Therefore, we investigated the effects of short-term HCA ingestion on endurance exercise performance and fat metabolism in untrained women. In two experiments designed as a double-blind crossover test, six subjects ingested 250 mg of HCA or placebo (same amount of dextrin) via capsule for 5 days and then participated in cycle ergometer exercise. They cycled at 40% VO2max for 1 h and then participated in cycle ergometer exercise. They cycled at 40% VO2max for 1 h and then the exercise intensity was increased to 60% VO2max until exhaustion on day 5 of each experiment. HCA tended to decrease the respiratory exchange ratio (RER) and carbohydrate oxidation during 1 h of exercise. In addition, exercise time to exhaustion was significantly enhanced (p<0.05). These results suggest that HCA increases fat metabolism, which may be associated with a decrease in glycogen utilization during the same intensity exercise and enhanced exercise performance.

Key Words hydroxycitrate, RER, fat oxidation, exercise

METHODS

Subjects. Six females agreed to participate in the study after being informed of the nature of the experiments. Subjects were nonsmokers and drink alcoholic beverages socially 2–3 times a month. They did not take any kind of drug or pharmaceutical and had not undertaken any type of exercise training for at least the past 6 mo. We did not control their food consumption or private lifestyle. However, lipolytic food consumption like caffeine in coffee, green tea and black tea was inhibited during the experimental period except for cap-
saicin resultant from the intake of Kimuchi, a traditional Korean food, considering the dietary habits of Koreans. Their physical characteristics are presented in Table 1.

Each subject signed a written consent form that outlined possible risks of the procedures. The nature of the study, approved by the institutional ethics committee in Kyung-Hee University and the Helsinki Declaration of 1975, was explained to all subjects, who thereafter gave their written informed consent to participate.

**Experimental procedures.** Subjects reported to the laboratory 1 wk before the start of the experiment for an incremental maximum oxygen consumption (\(\dot{V}_O_2\)max) test on a cycle ergometer as reported by Lim et al. (14). Their mean \(\dot{V}_O_2\)max was 26.07±2.79 ml/kg⁻¹/min⁻¹. We calculated the exact 40% and 60% of each \(\dot{V}_O_2\)max exercise bout based on their \(\dot{V}_O_2\)max. HCA or a placebo was ingested for 5 d in a double-blind crossover manner. At least 2 d between trials was established to minimize any possible effects of HCA.

**Experimental design.** The protocol for each trial was designed in the same manner. Subjects reported to the laboratory 2 h before the start of the experiment (07:00). They ingested a 640 kcal meal (breads, eggs, and orange juice) and 250 mg of HCA (water-soluble type) or placebo 2 h before exercising as reported previously (5). Exercise was conducted at the time of peak HCA concentration in the blood (13). After the meal, subjects were allowed to rest in a seated position. After resting for 100 min, they warmed up with 5 min of stretching exercises 10 min prior to beginning exercise. During the resting periods, a venous 3-way catheter was inserted into the antecubital vein and was kept silent with a saline infusion. A resting blood sample and expired gas were collected and analyzed.

The subjects exercised using a bicycle ergometer (Combi Aerobike, 75TXL-2, Japan) at a pedaling frequency of 50 rpm and an intensity of 40% of \(\dot{V}_O_2\)max for 60 min and then the intensity was elevated to 60% until exhaustion. The protocol of two different exercise intensities (40% for 60 min and 60% \(\dot{V}_O_2\)max exercise until exhaustion) was selected to investigate endurance performance reported by other researchers (5, 13, 14).

**Analysis.** Expired gas samples were analyzed using an auto-analyzer (Sensor Medic, Vmax 229, USA) previously calibrated for \(O_2\) and \(CO_2\). Fat and carbohydrate oxidation during exercise were calculated as previously described (16).

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\text{Carbohydrate oxidation} = 4.585\ VCO_2 - 3.226\ VO_2 \\
\text{Fat oxidation} = 1.695\ VO_2 - 1.701\ VCO_2
\]

Blood samples were collected with an EDTA-coated tube. Whole blood (500 \(\mu\)L) was immediately separated by microcentrifugation for blood glucose and lactate analysis using an auto-analysis system (YSI 2300 Plus, Yellow Springs Institute, USA). Another blood sample was centrifuged and plasma was collected and stored in a −70°C freezer for future free fatty acid (FFA) analysis.

**Statistical analysis.** The results are described as mean±SE. The data were analyzed using Student’s t-test in the same time point. The level of significance was set at \(p<0.05\).

**RESULTS**

**Oxygen consumption and respiratory exchange ratio**

The oxygen consumption increased at the initiation of exercise and reached a plateau at 15 min during exercise (Fig. 2A). The oxygen consumption at rest and during exercise was not different between the placebo and HCA trials, indicating that the ingestion of HCA did not affect \(VO_2\) at rest or during exercise for 60 min. The RER tended to be lower in the HCA trial than in the placebo trial during 1 h exercise, and was significantly different between the trials at 30 min (\(p<0.05\); Fig. 2B).
Carbohydrate and fat oxidation

Carbohydrate oxidation (Fig. 3A) increased at the initiation of exercise and slightly decreased during 1 h exercise, but fat oxidation (Fig. 3B) increased in the late period of exercise. Carbohydrate oxidation during the exercise period was not different between the HCA and placebo trials. Fat oxidation in HCA trial tended to increase as compared to that in the placebo trial, but the difference was not significant.

Blood measurements

The blood glucose levels were increased by consuming the meal (−120 min) and slightly decreased during exercise in the HCA trial as compared to the placebo trial (Fig. 4A). The lactate levels during exercise were increased in placebo trial, but not to a significant degree (Fig. 4B). The FFA concentrations were decreased by consuming the meal and increased at the initiation of exercise (Fig. 4C). The levels during exercise were slightly lower in the HCA trial than in the placebo trial.

Endurance exercise capacity

Exercise time to exhaustion at 60% \( \text{VO}_2\text{max} \) after 40% \( \text{VO}_2\text{max} \) for 60 min was significantly longer in the HCA trial (\( p<0.05 \); Fig. 5).

DISCUSSION

The aim of the present study was to investigate the short-term ingestion of HCA on endurance exercise performance in untrained women. We tried to show in this study that whether or not the ingestion of 250 mg HCA for 5 d would increase fat oxidation and improve endurance performance.

HCA is an active ingredient that is extracted from the rind of the fruit *Garcinia cambogia*, a native species of India. HCA is a potent competitive inhibitor of the extra-mitochondrial enzyme ATP citrate-lyase (10), and also increases the rate of hepatic glycogen synthesis and decreases body weight gain (17).

Focused on body weight control, HCA ingestion for a period of months failed to result in the loss of body weight and body fat (7, 18). This is unlikely, since the conversion of citrate into acetyl-CoA by ATP citrate-lyase only occurs when energy intake exceeds the energy requirements of the body (19). It is well known that during low-to-moderate intensity exercise, more FFA are released from adipocytes into the blood than in resting condition. These FFA are re-esterified or used as an energy source during exercise. Therefore, when energy intake exceeds the energy needs in the active muscle, the conversion of citrate into acetyl-CoA occurs during exercise. Consequently, HCA would be an effective inhibitor of carbohydrate oxidation, especially during moderately intense exercise. Therefore, the effects of HCA ingestion might be emphasized with physical activity.

However, acute ingestion of HCA did not change fat oxidation during exercise or at rest (20). On the contrary, Ishihara et al. (11) reported that short-term ingestion of water-soluble type HCA, which we used in the present study, increased fat oxidation in trained mice. The physical conditions as to trained or untrained showed different energy metabolisms during exercise. Endurance exercise training reduces malonyl-CoA concentration (1), increases mitochondrial number (2) and
mitochondrial uptake of long-chain fatty acids (3), and so on. Therefore, fatty acid oxidation increased and carbohydrate oxidation decreased during exercise of the same relative intensity for trained subjects.

Acute ingestion of HCA did not affect energy substrate utilization, especially in fat oxidation (7, 21). However, the short-term administration of HCA lowers RER and increases fat oxidation in trained subjects (13). In untrained subjects, HCA ingestion significantly decreased RER after 30 min of exercise as shown in the present study (Fig. 2B). However, the effects were lower than those of trained subjects (12, 13), because training might enhance the fat oxidation rate. Our experiment was designed to test two different exercise intensities, 40% and 60% VO2max. These two intensities showed the effects of glycogen sparing during prolonged endurance exercise performance. The fat oxidation rate during the latter stage of exercise increased in trained subjects (13) and increased slightly in untrained subjects in the present study (data not shown).

In the present study, blood glucose and lactate concentrations during the former stage of cycle ergometer exercise were not significantly affected by HCA ingestion (Fig. 4). However, Van Loon et al. (20) reported that the lactate concentration was decreased by acute ingestion of HCA during exercise, although not significantly, as in the present study. These findings suggest that short-term ingestion of HCA might delay the lactate threshold since HCA has an inhibiting effect on carbohydrate oxidation (11, 13). Plasma FFA concentrations were not significantly affected by HCA ingestion. Considering the characteristics of HCA, it does not have a lipolysis effect, which increases FFA concentration as Ishihara et al. (11) reported. They suggested that the acute ingestion of HCA increases serum FFA concentrations in resting mice.

The enhancement of fat oxidation during exercise could lead to increased endurance exercise performance. In the present study, the cycle ergometer exercise time to exhaustion after 1 h of 40% VO2max exercise was significantly enhanced by HCA ingestion. The increased fat oxidation with HCA ingestion may be attributed to the modification of CPT-I (carnitine palmitoyltransferase-I) activity (19); malonyl-CoA inhibits CPT-I activity in the cytosol, but its effect is suppressed by HCA as described above even in untrained state.

In summary, the short-term ingestion of HCA enhanced fatty acid utilization during moderately intense exercise in untrained women. Depressed energy utilization of carbohydrates by HCA during the former stage of exercise became important for keeping the energy source during the latter stage of high-intensity exercise in untrained subjects as observed in the trained subjects. Therefore, HCA ingestion (250 mg per day) at least 5 d before exercise can be considered an effective nutritional ergogenic aid for untrained people before participating in some types of exercise activities.

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REFERENCES


