Effect of Conjugated Linoleic Acid Intake on Endurance Exercise Performance and Anti-fatigue in Student Athletes

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Abstract: Conjugated linoleic acids (CLA) intake has been reported to reduce body fat mass or increase lean body mass and to improve exercise outcome by modulating testosterone in humans. These reports have studied mostly overweight subjects; few were athletes. Therefore, in this study, the effect of CLA intake on endurance performance and anti-fatigue in student athletes was investigated. A double-blind, crossover study was conducted with 10 male student athletes. Each subject was administered with either CLA (net 0.9 g/day) or a placebo for 14 days. They were subjected to an exercise tolerance test (steady loading) using a cycle ergometer on days 0 and 14. Peak \( \dot{V}O_2 \) was determined for each subject using a graded loading test. The steady loading test was performed with a pedaling exercise load of 50% peak \( \dot{V}O_2 \) for 40 min and then with a load of 70% peak \( \dot{V}O_2 \) until exhaustion. Blood sampling and measurement of critical flicker frequency (CFF) were performed before and after exercise. The rate of perceived exertion (RPE) was measured serially during exercise. In the results, amount of body weight variation significantly increased and amount of body fat percentage variation tended to decrease by CLA intake, it might have an effect by increase in muscle mass. In addition, amount of exercise time variation significantly increased, amount of variation of CFF before and after exercise tended to increase, that of RPE during exercise tended to decrease, and that of creatine phosphokinase before and after exercise tended to decrease in the CLA group. These results suggested that CLA intake for 14 days might have an effect on endurance performance and anti-fatigue in student athletes.

Key words: conjugated linoleic acid, ergogenic, endurance performance, anti-fatigue

1 INTRODUCTION

Conjugated linoleic acid (CLA) is a group of positional and geometrical isomers of conjugated dienoic octadecadienoate fatty acids. There are at least 28 known isomers, but the two most common are cis-9, trans-11 (c9, t11) and trans-10, cis-12 (t10, c12). CLA is produced from linoleic acid by rumen bacteria, and is naturally occurring in foods from ruminant sources such as beef, lamb meats, and dairy products, mostly as a 9c, t11-isomer. In bovine milk, 92% of total CLA was c9, t11. CLA has been reported to show various functions such as anti-obesity, anti-carcinogenicity, anti-atherogenicity, anti-diabeticity, anti-mutagenicity, bone formation, etc. Amounts of CLA dietary intake were reported as 50.9–177.1 mg/day in Sweden, 97.5 mg/day in England, 430 and 350 mg/day for men and women in Germany, 212 and 151 mg/day for men and women in America, and 500–1500 mg/day in Australia. For Japanese individuals, the normal dietary intake of CLA is 37.5 mg/day. Although normal dietary intake and blood levels of CLA in Japanese individuals is lower than in the non-Japanese subjects reported in previous studies, long-term intake of 2.3 g CLA/day as a dietary supplement likely has a positive effect in Japanese individuals. 

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Recently, it was reported that CLA reduces cardiovascular diseases and cancer, modulates immune and inflammatory responses, and improves bone mass. It has been suggested that these CLA effects result from interactions between two major isomers, c9, t11 and t10, c12\(^2\). In particular, several human clinical trials have reported mixed results, with a few studies indicating that CLA reduces body fat mass or increases lean body mass (LBM)\(^1\), although others showed no effects on body composition\(^1\(-\)\(^3\)). CLA has also been reported to improve muscle hypertrophy, steroidogenesis, physical activity, and endurance capacity in mice\(^3\).

Additionally, the present study demonstrated that CLA supplementation of t10, c12, but not c9, t11, with exercise training enhances running endurance capacity of mice by increasing β-oxidation of muscle fat and reducing consumption of stored liver glycogen during prolonged exercise\(^4\). Several human intervention studies determining effects of CLA supplementation, 1.6–6.8 g/day for 3 weeks–6 months, on exercise outcome have been reported, possibly with CLA improving exercise outcome by modulating testosterone\(^5\)). These reports of CLA effects on reducing body fat mass or increasing LBM, endurance performance, and resistance performance have studied mostly overweight subjects, but few athletes. Therefore, this study’s purpose was to examine the effect of CLA intake on endurance performance and anti-fatigue in student athletes, by lower dosage and shorter duration of CLA supplementation than previous studies.

### 2 MATERIALS AND METHODS

#### 2.1 Subjects

Ten healthy male students at Kanazawa University who belong to university sports clubs (baseball, volleyball, and swimming) participated in this study. Informed consent was obtained from each subject after explanation of the experimental purpose and protocol. This study was conducted in accordance with the revised version of the Declaration of Helsinki 2013, and was approved by the Ethics Committee on Human Experimentation of Faculty of Human Science, Kanazawa University (approval number 2015-3).

#### 2.2 Pre-experimental protocol

Before the study’s experimental stage, each participant was asked to perform incremental cycling (Bicycle ergometer 232C model 50, Combi, Tokyo, Japan) until volitional fatigue, to determine peak \(\dot{V}O_2\) and was also asked to measure their body weight and body fat percentage (Karada Scan HBF-362, Omron, Kyoto, Japan). Moreover, they answered a questionnaire about food frequency (Excel Eiyo-kun Food frequency questionnaire based on food groups, ver. 4.0, Kenpakusha, Tokyo, Japan) to check nutrient intake.

#### 2.3 Experimental design

This research was conducted as a double-blind, crossover study. Participants were given two treatments separated by a washout period of 14 days. Participants were asked to ingest test foods (Table 1), 1.8 g/day of CLAce Powder (CLA group; CLA net 0.9 g/day, The Nisshin OilliO Group, Ltd., Tokyo, Japan) or placebo (Pla group; Magical Ace Powder, Miyoshi Oil & Fat Co., Ltd., Tokyo, Japan) after dinner for 14 days. Participants were also instructed

<table>
<thead>
<tr>
<th>Table 1 Composition of test foods.</th>
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<tr>
<td><strong>CLAce powder</strong></td>
</tr>
<tr>
<td>Energy (kcal/100 g)</td>
</tr>
<tr>
<td>Lipid (%)</td>
</tr>
<tr>
<td>Fatty acid composition in lipid (%)</td>
</tr>
<tr>
<td>CLA</td>
</tr>
<tr>
<td>c9, t11(^1)</td>
</tr>
<tr>
<td>t10, c12</td>
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<td>c9, c11 / c10, c12</td>
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<tr>
<td>t9, t11 / t10, t12</td>
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<tr>
<td>Oleic acid</td>
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<td>Palmitic acid</td>
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<tr>
<td>Stearic acid</td>
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<td>Linoleic acid</td>
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<tr>
<td>Other</td>
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<td>Linolenic acid</td>
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<td>Erucic acid</td>
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<td>Behenic acid</td>
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<td>Palmitoleic acid</td>
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</tbody>
</table>

\(^1\) c, cis; t, trans

Effect of CLA intake on endurance exercise performance


2.4 Exercise regimen

The exercise regimen is shown in Fig. 1. On days 0 and 14, after ingesting test food, participants were asked report to the laboratory 45 min before the start of exercise. They were queried about their condition according to an interview sheet. They rested quietly in a sitting posture for 30 min, and at 10 min before beginning warm-up, their body weights and body fat percentages were measured. Next, critical flicker frequency (CFF; T.K.K. flicker meter, type I, Takei Scientific Instruments, Niigata, Japan), heart rate (HR), and systolic and diastolic blood pressures (HEM-7200, Omron, Kyoto, Japan) in the sitting posture were measured. After that, participants were asked drink 285 mL of water.

After a 5-min warm-up at a fixed workload of 40 W of the bicycle ergometer, they began exercising at a pedaling frequency of 50–60 rpm and at a workload corresponding to 50% peak \( \dot{V}O_2 \) for 40 min. Then, the workload was increased to a level corresponding to 70% peak \( \dot{V}O_2 \), and they continued pedaling until exhaustion. Their exhaustion point was established at the inability to maintain 50 rpm of pedaling. After exercise, their CFF, HR, and systolic and diastolic blood pressures in the sitting posture were measured. During exercise (0, 15, 30, and 40 min after the start of exercise) and at exhaustion, the rate of perceived exertion (RPE) was examined using Borg’s scale.

2.5 Blood analysis

At 10 min before the start of warm-up and after exercise, blood sampling was conducted. Each blood sample, 2 mL in an EDTA tube and 7 mL in a plain tube, were collected from the antecubital vein. The following were determined: hematological value and serum level of creatine phosphokinase (CK), glucose, triglyceride (TG), non-esterified fatty acid (NEFA), and isozymes of lactate dehydrogenase (LD), LD \(_{1,5} \). These analyzes were conducted by LSI Medience Corporation (Tokyo, Japan).

2.6 Statistical analysis

The data were expressed as mean ± SD. Differences in values between the CLA group and the Pla group were tested by a paired Student’s t-test. Repeated two-way analysis of variance (ANOVA) was used to examine RPE during exercise in the two groups (group × measurement time). The level of significance was set at \( p < 0.05 \).

3 RESULTS

3.1 Characteristics and nutrient intake before the experimental trial of subjects

Participants’ physical characteristics and nutrient intake before the experimental trial are shown in Table 2. Participants were divided into two groups, with no significant differences in age, height, body weight, body fat percentage, peak \( \dot{V}O_2 \), and intake of energy, protein, and dairy products. The intake rate of test foods for 14 days also did not show any significant difference between the two groups (Pla group: 90.0 ± 8.0%, CLA group: 92.2 ± 5.0%).

3.2 Exercise

Amount of exercise time variation (subtracting exercise time to exhaustion of day 0 from that of day 14) in the CLA group substantially increased (149 ± 102 sec), whereas that in the Pla group decreased (−30 ± 172 sec), a significant difference \( (p<0.05) \). The laboratory temperature and humidity during exercise were at 25.4 ± 1.0 °C and 51.7 ± 6.3%, respectively.

<table>
<thead>
<tr>
<th>Table 2 Characteristics and nutrient intake of subjects.</th>
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<tbody>
<tr>
<td>n=10</td>
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<tr>
<td>Age (year)</td>
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<tr>
<td>Height (cm)</td>
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<tr>
<td>Body weight (kg)</td>
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<tr>
<td>Peak ( \dot{V}O_2 ) (mL/kg/min)</td>
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<tr>
<td>Energy intake (kcal/day)</td>
</tr>
<tr>
<td>Protein intake (g/day)</td>
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<tr>
<td>Dairy product intake (g/day)</td>
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Values are expressed as the mean ± SD.

Fig. 1 Exercise regimen.

↓ Measurement of body mass, ▽ blood sampling, measurement of CFF, HR, and blood pressure, ● measurement of RPE, ○ drink 285 mL of water.
3.3 Body mass, CFF, HR, and blood pressure

Changes in body mass, CFF, HR, and blood pressure of the two groups are shown in Table 3. Row data indicate no significant differences between the CLA and the Pla group. As for body weight, the amount of body weight variation (subtracting the value of day 0 from that of day 14) in the CLA group, 0.650 ± 1.04 kg, significantly increased compared with the Pla group value of −0.430 ± 0.809 kg (p < 0.05). On the other hand, the amount of body fat percentage variation decreased in both groups, −0.220 ± 1.05% in the CLA group and −3.50 ± 0.916% in the Pla group, showing no significant difference between the two groups. The amount of variation of CFF in the CLA group increased both before and after exercise, respectively, 1.85 ± 4.55 Hz and 2.35 ± 3.54 Hz, whereas there was no significant difference between the two groups. The amount of variance of CFF in the CLA group significantly decreased with the value of −0.900 ± 10.6 beat/min in the Pla group (p < 0.05). Blood pressure before and after exercise did not show significant difference between the CLA and the Pla group.

3.4 Blood analysis

Changes in hematological and serum biochemical parameters are shown in Table 4. Although all parameters showed no significant difference between the CLA and the Pla group, the amount of CK variation in the CLA group and that of TG variation in the Pla group were substantially lower than that of the other group before and after exercise. The amount of CK variation before and after exercise in the CLA group was −49.8 ± 111 U/L and −64.1 ± 131 U/L, respectively, and that in the Pla group was 9.10 ± 147 U/L and −4.90 ± 177 U/L, respectively. The amount of TG

| Table 3 Changes in body mass, critical flicker frequency, heart rate, and blood pressure of subjects. |
|--------------------------------------------------|-----------------|-----------------|-----------------|-----------------|
| Pla                                              | 0 day           | 14 day          | △ day 14-0      | CLA             | 0 day           | 14 day          | △ day 14-0      |
| Body mass                                        |                 |                 |                 |                 |
| Body weight (kg)                                 | 67.6±5.35       | 67.1±5.28       | −0.430±0.809    | 66.7±5.19       | 67.4±5.25       | 0.650±1.04*     |
| Body fat percentage (%)                          | 11.8±2.02       | 11.5±1.67       | −0.350±0.916    | 11.7±1.67       | 11.5±2.23       | −0.220±1.05     |
| Critical flicker frequency (Hz)                  |                 |                 |                 |                 |
| Before ex.                                       | 43.8±4.04       | 42.1±2.61       | −1.70±3.10      | 42.3±3.36       | 44.2±4.85       | 1.85±4.55       |
| After ex.                                        | 43.7±4.79       | 42.6±1.82       | −1.15±4.15      | 42.8±3.70       | 45.1±4.05       | 2.35±3.54       |
| Heart rate (beat/min)                            |                 |                 |                 |                 |
| Before ex.                                       | 67.8±8.51       | 66.9±6.82*      | −0.900±10.6b    | 74.0±9.31*      | 64.5±8.65*      | −9.50±6.17b     |
| After ex.                                        | 98.1±11.0       | 91.9±12.9*      | −6.20±6.05      | 100±9.74        | 93.8±8.08       | −6.20±9.70      |
| Blood pressure (mmHg)                            |                 |                 |                 |                 |
| Systolic phase                                   |                 |                 |                 |                 |
| Before ex.                                       | 121±9.39*       | 117±7.79        | −3.90±6.69      | 124±11.6        | 120±8.10        | −4.30±10.2      |
| After ex.                                        | 112±8.73        | 114±12.1        | 2.40±12.9       | 111±12.9        | 110±3.98        | −1.70±11.6      |
| Diastolic phase                                  |                 |                 |                 |                 |
| Before ex.                                       | 68.9±5.41*      | 66.0±6.05       | −2.90±5.99      | 67.6±5.66       | 66.1±6.22      | −1.50±7.53      |
| After ex.                                        | 65.4±5.99       | 66.4±8.86       | 1.00±7.66       | 65.6±6.34       | 63.1±4.16       | −2.50±4.01      |

Values are expressed as the mean ± SD.
*p < 0.05, values with the same letters are significantly different at p < 0.05.
variation before and after exercise in the CLA group was −6.50 ± 11.5 mg/100 mL and −6.00 ± 19.9 mg/100 mL, respectively, and that in the Pla group was −28.3 ± 40.5 mg/100 mL and −32.6 ± 41.7 mg/100 mL, respectively.

Changes in LD isozymes are shown in Table 5, and row data indicate no significant difference between the CLA and the Pla group. Amount of LD1 variation after exercise in the Pla group significantly increased compared with the value of −0.310 ± 1.12 mg/100 mL, whereas there was no significant difference in the LD1/LD2 ratio between the two groups and between before and after exercise. The amount of LD5 variation after exercise in the Pla group significantly decreased compared with the value of 0.100 ± 1.03 mg/100 mL.

3.5 Fatigue evaluation

Amount of variations of RPE during exercise in the CLA and Pla groups are shown in Fig. 3. Values are expressed as data that subtract the value of day 0 from that of day 14, and that of start time of exercise from that of each measurement time. The variation of RPE during exercise in the CLA group indicated a minus value during exercise and remained at a low level compared with that of the Pla group, whereas it did not show a significant interaction effect between groups and measurement times by repeated two-way ANOVA.

4 DISCUSSION

In this study, we investigated the effect of CLA intake on endurance exercise performance and anti-fatigue in student athletes. Compared to the previous studies, the
Results show that the CLA intake caused significant increased body weight and insignificant decreased body fat percentage. It indicated that the increase of body weight was caused by increased muscle mass.

Several previous studies have reported that dietary CLA reduces body fat mass (BMF) or increases LBM in overweight and/or obese humans. Blankson et al. reported that BMF decreased by 1.7 g/day and LBM increased by 6.8 g/day, compared with placebo and baseline, respectively, for 12 weeks of CLA intake. Watras et al. reported that body weight and BMF decreased by 3.2 g/day for 6 months of CLA intake compared with placebo. And Chen et al. reported that body weight and BMF decreased by 1.7 g/day for 12 weeks of CLA intake compared with baseline.

As for normal weight subjects, several studies have reported that CLA supplementation has no effect on body mass or BMF. Kreider et al. reported that BMF decreased by 1.7 g/day and LM increased by 6.8 g/day, compared with placebo and baseline, respectively, for 12 weeks of CLA intake. Sneddon et al. reported that CLA plus n-3 long-chain polyunsaturated fatty acids (n-3 LC-PUFA) supplementation, 3 g/day CLA plus 3 g/day n-3 LC-PUFA for 12 weeks, has no effect on BMF in young lean male individuals (age 30.5 ± 4.9 years, BMI 23.6 ± 1.5 kg/m², body fat 16.1 ± 5.4%). And Macaluso et al. reported that there were no significant differences in total body mass, BMF, and LBM by CLA supplementation, 6 g/day for dosage and duration of CLA used in this study, 0.9 g/day for 14 days, was low and short.

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As for normal weight subjects, several studies have reported that CLA supplementation has no effect on body mass or BMF. Kreider et al. reported that CLA supplementation, 6 g/day for 4 weeks, did not significantly affect changes in total body mass, fat-free mass, BMF, and percent body fat in resistance-trained male subjects (age 23 ± 0 years, body fat 15.5 ± 1%). Sneddon et al. reported that CLA plus n-3 long-chain polyunsaturated fatty acids (n-3 LC-PUFA) supplementation, 3 g/day CLA plus 3 g/day n-3 LC-PUFA for 12 weeks, has no effect on BMF in young lean male individuals (age 30.5 ± 4.9 years, BMI 23.6 ± 1.5 kg/m², body fat 16.1 ± 5.4%). And Macaluso et al. reported that there were no significant differences in total body mass, BMF, and LBM by CLA supplementation, 6 g/day for dosage and duration of CLA used in this study, 0.9 g/day for 14 days, was low and short.

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3 weeks, in resistance-trained male subjects (age 27.4 ± 3.7 years, BMI 25.9 ± 2.6 kg/m², body fat 15.1 ± 3.1%). On the other hand, some studies have reported that CLA reduces BMI or increases LBM in normal weight subjects. Thom et al. reported that body fat was significantly reduced and no effects on body weight were observed by CLA supplementation, 1.8 g/day for 12 weeks, in female plus male subjects (age 27.5 ± 3.0 years, BMI 23.2 ± 2.4 kg/m²), and Colakoglu et al. reported that fat-free mass was induced and body weight was reduced by CLA supplementation, 3.6 g/day for 6 weeks, in female subjects (age 20.4 ± 1.7 – 21.9 ± 2 years, BMI 20.8 ± 1.6 – 23.3 ± 1.2 kg/m²).

Reduced body fat mass by CLA intake suggested that CLA causes reduction in lipid uptake by adipose cells because of an effect on lipoprotein lipase and stearoyl-CoA desaturase and carnitine palmitoyltransferase activity in muscle cells, which is the rate-limiting enzyme in β-oxidation, is increased. On the other hand, increased muscle mass by CLA intake was suggested by increased serum testosterone level. Testosterone can induce increase of energy expenditure by elevating mitochondrial biogenesis in skeletal muscle in mice. As previously mentioned, CLA intake has been reported to improve stereoidogenesis, muscle hypertrophy, physical activity, and endurance capacity in mice.

Barone et al. reported that trained mice showed an increase in free plasma testosterone and an upregulation of 17α-hydroxylase/17, 20-lyase (CYP17A1) mRNA and protein. The effect of training on CYP17A1 expression and testosterone biosynthesis was significantly higher in the trained mice supplemented with CLA compared with placebo. Barone et al. also reported that CLA supplementation induced hypertrophy of types I and IIb muscle fibers in plantaris and gastrocnemius muscles of mice by endurance exercise and induced only a specific hypertrophy of type IIX muscle fibers in the plantaris muscle.

This study’s participants were student athletes. In this study, the amount of exercise time variation in the CLA group significantly increased compared with the Pla group. CLA supplementation with training increased free testosterone and exercise performance. Hemoglobin and hematocrit were increased by higher testosterone, and that was associated with increased erythropoietin levels. In this study, there was no significant difference in the amount of hemoglobin and hematocrit variation between the two groups.

On the other hand, Mizunoya et al. reported that the maximum swimming time until fatigue and the muscle lipoprotein lipase activity of mice were significantly higher in the CLA intake group than in the control group. These results suggest that CLA ingestion increases endurance exercise capacity by promoting fat oxidation during exercise. Kim et al. reported that the maximum running time on a treadmill in CLA intake mice was significantly longer than that of the control. This result likely due to increased fat utilization and reduced consumption of stored liver glycogen as substrates for energy metabolism by CLA intake. Kim and Park reported that CLA treatment was shown to activate peroxisome proliferator-activated receptor γ co-activator 1α (PGC-1α) in C2C12 mouse myoblasts; this was linked with upregulation of mitochondrial biogenesis. PGC-1α is a transcriptional coactivator that controls the expression of genes involved in regulation of fatty acid oxidation, glucose metabolism, and antioxidants. It has been reported that PGC-1α may induce many of the changes associated with endurance training, including mitochondrial biogenesis, fiber-type switching, stimulation of fatty acid oxidation, angiogenesis, and resistance to muscle atrophy.

On the other hand, Parra et al. reported that CLA treatment did not show any effect for PGC-1α increase and gene expression of myosin heavy-chain isoforms in mice and that it did not support an enhancement of β-oxidation in skeletal muscle contributing to the observed antiobesity effect of CLA.

In contrast, the concentration of transforming growth factor-β (TGFB-β) in cerebrospinal fluid increased with increased intensity of exercise used to cause fatigue. And the TGFB-β acted on the brain and modulated activities of neurons; this changed whole-body metabolism to utilize more fat and enhanced the oxidation of fatty acid. In this study, the amount of TG variation before and after exercise in the Pla group was substantially lower than in the CLA group, whereas there was no significant difference in the amount of TG and NEFA variation between the two groups.

Although there was no significant difference in the amount of RPE and CFF variation between the CLA and Pla groups, the amount of RPE variation in the CLA group during exercise was lower than that of the Pla group, and the amount of CFF variation of the CLA group increased both before and after exercise in this study. These results indicate that possibility of exercise fatigue was reduced by CLA intake.

Iwaki and Harada reported that a frequency threshold with “flickering” can be perceived when the frequency of the intermittent point-light stimulus gradually decreases. The flicker perception threshold changes according to the excitability of the central nervous system, including the brain cortex or the change in arousal level due to accumulation of fatigue, and thereby CFF declines with accumulation of fatigue. During dynamic exhaustive exercise, cerebral oxygenation decreased, suggesting that decreased cerebral activity occurs with decreased muscular functions, that is, severe exercise-induced decreases of cerebral function. These reports suggest that, in this study, the brain cortex fatigue of the CLA group may be lower than that of the Pla group. But there was no significant difference in amounts of RPE and CFF variation between the
two groups; this result may indicate no significant difference in serum NEFA between the two groups.

Some reports suggested that CLA intake enhanced endurance capacity of mice by increasing fat utilization and reducing consumption of stored liver glycogen\(^{30}, 31\). Moreover, Kim et al.\(^{15}\) investigated the isomer-specific effects of CLA and reported that dietary t10, c12 CLA, not c9, t11, induced significant increase in maximum running time and distance until exhaustion, with a dramatic reduction of total adipose depots in mice compared to a control group. In this study, there was no significant difference in serum NEFA between the two groups. Further study needs to investigate fat oxidation by analysis of expired gas.

LD isozymses, LD\(_{1,5}\), in serum before and after exercise were measured in this study. Many previous studies measured total LD after exercise\(^{38-41}\). They reported that LD increased significantly, and the degree of increase depended on the exercise’s intensity and duration\(^{40}\), LD activity is commonly used as a marker indirectly indicating damage to skeletal muscles\(^{45}\), whereas few studies have measured LD isozymses after exercise\(^{44-46}\). LD\(_1\) and LD\(_2\) are cardiac-predominant isozymses. Increase of these isozymses in serum is considered generally supportive of myocardial injury\(^{47}\). Huang et al.\(^{48}\) reported that endurance training accelerates exhaustive exercise-induced apoptosis in left ventricles of rats, as a result of exhaustive running tests on a treadmill. In this study, the amount of LD\(_1\) variation after exercise in the CLA group was significantly lower than that of the Pla group. CLA intake may reduce apoptosis in ventricles in exhaustive exercise. On the other hand, Wolf et al.\(^{49}\) reported that total LD increased after repetitive treadmill testing in male distance runners, but the fractions represented by LD\(_{1,5}\) were unaltered, with no change in the LD\(_1)/LD\(_2\) ratio. This result indicated that total LD elevations did not derive from the heart. There was also no significant difference in the LD\(_1)/LD\(_2\) ratio between the two groups in this study. For most, LD\(_1\) predominates in skeletal muscle and the liver\(^{47}\), and it increased in serum in patients with non-traumatic acute rhabdomyolysis\(^{49}\). In this study, there was no significant difference in LD\(_1\) between the two groups, and there was no indication that CLA intake inhibited leaking of LD\(_1\) from skeletal muscle cells.

Serum CK was also measured as a marker of muscle damage after exercise\(^{45}, 50-52\). The CK level after exercise relates to duration and intensity of exercise, type of exercise, and the individual’s physical characteristics and training background. Baird et al.\(^{52}\) reported that higher intensity exercise elicited greater serum CK, glutamic oxaloacetic transaminase, and serum LD levels than lower-intensity exercise, and magnitude of exercise intensity may have greater influence on cellular response to exercise-induced muscle damage than the duration. Pantoja et al.\(^{56}\) reported that significant increase in serum CK was observed at 48 hours post-exercise on land, and significant differences were found between land and water. Hackney et al.\(^{57}\) reported that CK level was significantly increased at 24 hours post for both resistance trained and untrained (UT) participants and at 48 and 72 hours post for UT compared only with baseline, by an acute bout of high-volume, full-body resistance training. Lippi et al.\(^{58}\) reported that, compared to before the run, the level of CK directly after a half-marathon run increased 1.4-fold. A significant difference was observed in samples between CK levels before and after the run; then, it increased 24 hours after the run. In this study, although there were no significant differences between the two groups, the CK level before and after exercise in the CLA group was substantially lower than that of the Pla group. This result suggests that exercise-induced muscle damage was reduced by CLA intake, and it may have contributed to increased endurance performance in this study.

5 CONCLUSIONS

In this study, the amount of body weight variation significantly increased through CLA intake, and such intake might have an effect by increasing muscle mass. In addition, the amount of exercise time variation significantly increased; the amount of variation of CFF before and after exercise tended to increase; that of RPE during exercise tended to decrease; and that of CK before and after exercise tended to decrease in the CLA group. These results suggested that CLA intake for 14 days might have an effect on endurance performance and anti-fatigue in student athletes. Although the number of subjects in this study was relatively small and may be a limiting factor in reaching general conclusions, the results above provide an intriguing fact for the effect of CLA intake. Further studies need to investigate fat oxidation by analysis of expired gas with more subjects.

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References


Effect of CLA intake on endurance exercise performance


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Effect of CLA intake on endurance exercise performance


