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Effects of Conjugated Linoleic Acid Supplementation During Resistance Training on Body Composition, Bone Density, Strength, and Selected Hematological Markers

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ABSTRACT

Conjugated linoleic acids (CLA) are essential fatty acids that have been reported in animal studies to decrease catabolism, promote fat loss, increase bone density, enhance immunity, and serve as an antiatherogenic and anticarcinogenic agent. For this reason, CLA has been marketed as a supplement to promote weight loss and general health. CLA has also been heavily marketed to resistance-trained athletes as a supplement that may help lessen catabolism, decrease body fat, and promote greater gains in strength and muscle mass during training. Although basic research is promising, few studies have examined whether CLA supplementation during training enhances training adaptations and/or affects markers of health. This study evaluated whether CLA supplementation during resistance training affects body composition, strength, and/or general markers of catabolism and immunity. In a double-blind and randomized manner, 23 experienced, resistance-trained subjects were matched according to body mass and training volume and randomly assigned to supplement their diet with 9 $g \cdot d^{-1}$ of an olive oil placebo or 6 $g \cdot d^{-1}$ of CLA with 3 $g \cdot d^{-1}$ of fatty acids for 28 days. Prior to and following supplementation, fasting blood samples, total body mass, and dual-energy X-ray absorptiometry (DEXA) determined body composition, and isotonic bench press and leg press 1 repetition maximums (1RMs) were determined. Results revealed that although some statistical trends were observed with moderate to large effect sizes, CLA supplementation did not significantly affect ($p > 0.05$) changes in total body mass, fat-free mass, fat mass, percent body fat, bone mass, strength, serum substrates, or general markers of catabolism and immunity during training. These findings indicate that CLA does not appear to possess significant ergogenic value for experienced resistance-trained athletes.

Key Words: exercise, sport nutrition, ergogenic aids

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Introduction

Excessive consumption of dietary fat has generally been believed to increase the risk of various diseases. However, research over the last 25 years has indicated that certain components of fat may posses some health benefits (e.g., polyunsaturated fats, omega-3 fatty acids, etc). In the late 1970s, an extract of fat from beef was found to have anticancer properties for animals. The extract was identified as conjugated linoleic acid (CLA) by researchers at the University of Wisconsin in 1987. Since then, researchers have developed methods to purify CLA, and numerous studies have examined the potential health benefits of CLA.

For example, CLA supplementation has been reported to slow progression of heart disease in rabbits (25), mice (32), and hamsters (33). Conjugated linoleic acid has been reported to inhibit certain types of tumor growth in vitro (42), prostate cancer in dogs (11), and breast cancer in rats (19). The anticarcinogenic effects have recently been suggested to be because of an antioxidant effect of CLA (4). Conjugated linoleic acid has also been reported to improve glucose tolerance and thereby may have beneficial effects in controlling and/or preventing diabetes (17, 18). Moreover, CLA feedings in animals have been shown to lessen mark-

ers of catabolism (31); enhance the immune system (7, 46); increase bone content (26, 45); and decrease body fat accumulation in animals (3, 8, 12, 35–37). Consequently, research conducted on animals suggests that CLA may play an important role in fighting heart disease, obesity, cancer, diabetes, and osteoporosis while improving the immune system (28, 34, 38, 39).

To our knowledge, CLA was first marketed in the United States in 1996 as a potential ergogenic aid for resistance-trained athletes and bodybuilders. Marketing claims of the time suggested that CLA supplementation could benefit resistance-trained athletes by promoting fat loss, reducing catabolism, and enhancing immunity during training. More recently, there has also been some suggestion that CLA may help modulate insulin and/or serve as an antioxidant during exercise. Although basic animal research was promising, no studies had evaluated the effects of CLA supplementation on untrained or trained humans prior to CLA being marketed as an ergogenic for athletes.

Consequently, in 1996–1997 we conducted the first study to examine the safety and efficacy of CLA supplementation during resistance training on training adaptations in well-trained athletes. The study was designed in collaboration with our grant sponsors to evaluate the recommended dosage and length of supplementation for athletes on a number of dependent variables that animal studies had suggested may be affected by CLA supplementation (i.e., body composition, bone mass, markers of catabolism and immunity, and strength-training adaptations) as well as general clinical markers of health. Although subsequent studies have evaluated different dosages, lengths of supplementation, subject populations, and dependent variables, the preliminary results from this initial study that were presented at several scientific meetings helped form the basis for extending much of this work (13, 24). This paper presents the comprehensive results from this initial clinical trial evaluating the safety and efficacy of CLA supplementation during resistance training in well-trained athletes.

Methods

Experimental Approach to the Problem

This study was conducted as a double-blind, placebo-controlled, randomized clinical trial. The independent variable was CLA supplementation. Pre- and postintervention tests were performed to assess whether CLA supplementation promoted significantly different changes compared with a placebo in the selected dependent variables. The subject population studied, dependent variables assessed, dosage, and length of supplementation were determined in collaboration with researchers at Experimental & Applied Sciences (Golden, CO) and Pharmanutrients (Lake Bluff, IL), who served as sponsors to this study, as well as in consultation with researchers at the University of Wisconsin, who had pioneered CLA research. Since this was the first study evaluating the effects of CLA supplementation on training adaptations in humans, we adopted the null hypothesis for this experiment. However, given the available animal data at the time and marketing claims, we were interested in determining whether CLA supplementation would promote fat loss, increase fat-free and bone mass, reduce markers of catabolism and immune stress, and/or affect gains in strength during training.

Table 1. Subject characteristics.*

Variable	
Age (y)	23 ± 0.8
Weight (kg)	80.6 ± 2
Height (cm)	179 ± 1
Body fat (%)	15.5 ± 1
Resistance-training experience (y)	5.6 ± 0.8
Current training ($h \cdot wk^{-1}$)	7.0 ± 0.5
Bench press 1 RM ($kg \cdot kg^{-1}$ body weight)	1.3 ± 0.06
Hip sled/leg press 1 RM ($kg \cdot kg^{-1}$ body weight)	2.1 ± 0.6

* Data are means ± *SEM*.

Subjects

Twenty-three experienced resistance-trained men volunteered to participate in this study. Subjects were informed as to the experimental procedures and signed informed consent statements in adherence with the human subjects guidelines of The University of Memphis and the American College of Sports Medicine. Subject characteristics are presented in Table 1.

Entrance Criteria

In order to participate in the study, subjects had to (a) sign statements indicating they had no current or past history of anabolic steroid use; (b) be an experienced resistance-trained athlete (>1 year) who was currently training at least 3 $h \cdot wk^{-1}$ with a program that included bench press and leg press/squat exercises; (c) submit a detailed description of their current training program; (d) not have ingested creatine, CLA, or beta-agonists for an 8-week period prior to the start of supplementation; and (e) agree not to ingest any other nutritional supplements, proposed ergogenic aids, or nonprescription drugs during the course of the study.

Familiarization and Testing Sessions

Subjects participated in 2 familiarization sessions. In the first familiarization session, the procedures of the study were explained, the subjects were weighed, training and medical history forms were completed, and the subjects were familiarized to the strength-testing equipment and procedures. The subjects were in-

structed by a registered dietitian on how to report nutritional intake on nutritional log sheets. A certified strength and conditioning specialist instructed the subjects how to record training data (i.e., lifts performed, repetitions, amount of weight lifted, etc.) on training log forms. In the second session, subjects practiced using the bench press and hip sled/leg press strength testing equipment and were scheduled for presupplementation assessments. The investigators also clarified any questions the subjects had regarding methods of the study.

Presupplementation assessments included (a) a 4-day nutritional intake assessment (including 1 weekend day); (b) donation of an 8-hour fasting venous blood sample; (c) measurement of total body mass, total body water, and body composition; and (d) performance of 1 repetition maximum (1RM) strength tests on the isotonic bench press and hip sled/leg press. Following these assessments, subjects were match-paired according to total body mass, fat-free mass (FFM), years of training, hours per week of resistance training, and training program type/volume. In a double-blind and randomized manner subjects were then assigned to supplement their diet for 28 days with capsules containing either 9 $g \cdot d^{-1}$ of an olive oil placebo (P) or capsules containing 6 $g \cdot d^{-1}$ of CLA with 3 $g \cdot d^{-1}$ of fatty acids (Tonalin, Pharmanutrients). Previous gas chromatography analysis of this source of CLA revealed that it is comprised of 65% CLA isomers, of which 22.6% were *trans*-10, *cis*-12; 23.6% were *cis*-11, *trans*-13; 17.6% were *cis*-9, *trans*-11; 16.6% were *trans*-8, *cis*-10; 7.7% were *trans*-9, *trans*-11, and *trans*-10, *trans*-12; and 11.9% were other isomers (31, 48). This CLA was the highest purity of CLA available at the time and has served as the source of CLA in several subsequent studies (31, 48). Olive oil was selected as a placebo since it had similar caloric value, color, and consistency as CLA with no known ergogenic value. This dosage and length of supplementation represented the upper range of the amount of CLA recommended for athletes to take over a standard supplementation period (i.e., 1 month). Supplements were administered in opaque capsules and placed in coded generic bottles for double-blind administration. Subjects ingested 3 capsules with morning, midday, and evening meals. Subject compliance in taking the supplements was verified by collecting empty supplement bottles.

Subjects maintained their usual individualized training program and recorded all training on training log sheets during the supplementation period. Although resistance-training programs varied slightly among matched paired groups (i.e., types of lifts and the number of sets and repetitions performed for each lift), common lifts performed included bench press/incline bench press, chest flies, shoulder/military press, biceps curls, triceps extension, squats/leg press, leg extension, and leg curls. Subjects documented training volume into provided training logs throughout the study (i.e., lifts performed, number of sets, number of repetitions, and amount of weight lifted) in order to calculate total lifting volume. Subjects generally performed 2–4 sets of 6–10 repetitions on various lifts.

Following the 28-day supplementation period, subjects underwent postsupplementation assessments in a similar manner as the presupplementation tests. Specifically, diet was recorded for 4 days; subjects donated an 8-hour fasting venous blood sample; body mass, body water, and body composition were determined; and subjects performed 1RM strength tests on the isotonic bench press and hip sled/leg press.

Procedures

Subjects maintained their normal diet throughout the supplementation period. Nutritional intake was monitored for 4 days prior to the initiation of supplementation and during the final week of supplementation. Nutritional records were evaluated and analyzed by a registered dietitian using the Food Processor III nutritional analysis software (Nutritional Systems, Salem, OR).

Subjects did not exercise for 48 hours and observed an overnight 8-hour fast prior to reporting to the lab for baseline assessments. Subjects reported to the lab between 6:00 and 8:00 AM and donated fasting blood samples via venipuncture from an antecubital vein in the forearm using standard phlebotomy procedures. Blood samples were collected into two 10 ml serum separation tubes (SST) and a 5 ml anticoagulant tube containing K_3 (EDTA). The SSTs were centrifuged at 5,000 rpm for 10 minutes using a Biofuge 17R centrifuge (Heraeus Inc., Hanau, Germany). Serum from 1 SST was transferred into microcentrifuge tubes and frozen at $-80°$ C for subsequent analysis. Serum from the remaining SST was transferred into a 10-ml plain sterile tube. The plain and EDTA tubes were refrigerated and shipped overnight in cold containers to Ciba Corning Diagnostic Laboratories (St. Louis, MO) for clinical analysis. A complete clinical chemistry panel (31 items) was run on serum samples using the Technicon DAX model 96–0147 automated chemistry analyzer (Technicon Inc., Terry Town, NY) following standard clinical procedures. Cell blood counts with percent differentials were run on whole blood samples using a Coulter STKS automated analyzer using standard procedures (Coulter Inc., Hialeah, FL). These analyzers were calibrated daily to controls according to manufacturers' recommendations and federal guidelines for clinical diagnostic laboratories. Test to test reliability of these assays ranged from 2 to 6% for individual assays with an average variation of $\pm 3\%$. Samples were run in duplicate to verify results if the observed values were outside control values and/or

clinical norms according to standard lab procedures. Serum and whole blood analysis provided a basis to assess the clinical safety of CLA supplementation as well as determination of whether CLA supplementation affected markers of catabolism and/or immune stress.

Subjects were instructed not to exercise and to fast for 4 hours prior to body composition assessments. Total body mass was measured on a calibrated digital scale with a precision of ±0.02 kg (Sterling Scale Co., Southfield, MI). Total body water was estimated (41) using a Valhalla Bioelectrical Impedance Analyzer (Valhalla Scientific, San Diego, CA). Whole-body (excluding cranium) composition measurements were determined using a Hologic QDR-2000 DEXA with the Hologic version V 7, REV F software (Waltham, MA) using procedures previously described (21, 22). The DEXA scans regions of the body (right arm, left arm, trunk, right leg, and left leg) to determine the amount of bone mass, fat mass, and fat-free/soft tissue mass within each region. The scanned bone, fat, and fat-free/soft tissue mass for each region was then subtotaled to determine whole-body (excluding cranium) values. Percent body fat was calculated by dividing the amount of measured fat mass by total scanned mass (sum of bone mass, fat mass, and fat-free/soft tissue mass). DEXA has been shown to be a highly reliable and precise method for determining individual body composition segments (15, 16, 21, 22, 29).

DEXAs were performed under the supervision of a certified radiology technician. Quality control (QC) calibration procedures were performed on a spine phantom (Hologic X-CALIBER Model DPA/QDR-1 anthropometric spine phantom) prior to each testing session according to procedures previously described (22). Mean coefficients of variation in bone mineral content (BMC) and bone mineral density measurements on the spine phantom ranged between 0.41 and 0.55% throughout the life of the unit. Subjects were positioned on the DEXA table using standardized methods for each test. Test-retest reliability studies performed on male athletes with this DEXA machine yielded a mean deviation for total BMC and total fat free/soft tissue mass of 0.31% with a mean intra-class correlation of 0.985 (22).

On the same day that blood was collected and body composition was determined, subjects performed 1RM strength tests. A warm-up on the bench press was followed by 3–5 progressive 1RM attempts. Hand position on the bar was recorded and the weight plates were standardized between trials. Subjects were required to maintain good lifting form (i.e., feet maintaining contact with the floor, no arching of the back off of the bench, no bouncing of the weight off of the chest). Once 1RM was determined on the bench press, subjects rested for 10 minutes and began warming up for the hip sled/leg press test. The leg press 1RM test was performed on an AMF hip sled (AMF, Jefferson, IA). Subjects were positioned on their back in an adjustable back/shoulder support. The adjustable back/shoulder support was moved to allow each subject to be positioned so that his knees were bent past 90° with his thighs approximately 1 inch from his chest and his feet were comfortably positioned. Back/shoulder support position, foot placement position, athletic shoes worn, and the weight plates used were standardized between trials. Subjects were required to maintain standardized lifting form. Subjects typically made 4–6 lift attempts before achieving their 1RM on the leg press. Strength tests were performed in a competitive environment in order to motivate the subjects to perform to the best of their ability. All 1RM tests were performed under the supervision of certified strength and conditioning specialists using standardized lifting criteria (14, 23, 44). Test to test reliability of performing these strength tests on resistance-trained subjects in our laboratory have yielded low mean coefficients of variation and high reliability for the bench press (1.9%, intraclass $r = 0.94$) and leg press/hip sled (0.7%, intraclass $r = 0.91$).

Statistical Analyses

A priori power analysis revealed power values of 0.14, 0.71, and 0.99 for small (0.25), moderate (0.75), and large effect sizes (1.25), respectively, for the n size used in the study. These findings indicate that the n size used in the present study was sufficient to detect significant differences among groups. Dietary intake (energy, carbohydrate, protein, and fat) was evaluated by multivariate analysis of variance (MANOVA) using SPSS for Windows version 10.05 software (SPSS Inc., Chicago, IL) in order to assess overall differences between groups in dietary patterns. All remaining dependent variables were analyzed by univariate analysis of variance (ANOVA) with repeated measures. Data were considered significantly different when the probability of error was 0.05 or less. Delta scores (post–pre values) were calculated on selected variables and analyzed by 1-way ANOVA in order to assist in interpretation of data. Effect size was calculated on variables exhibiting statistical trends by dividing the difference between delta values for each group by the pooled *SD* (9). This was done to determine the strength of the effects observed in these trends. Data are presented as means ± *SE* of means.

Results

Side Effects

Analysis of poststudy questionnaires revealed that subjects tolerated the supplementation protocol well with no reports of medical problems or symptoms.

Table 2. Dietary intake data for the placebo and CLA groups.

Variable	Group		Day 0	Day 28
Energy intake ($kcal \cdot kg^{-1} \cdot d^{-1}$)	P	$\bar{x}$*	47.1	40.9
		±	4.4	2.9
	CLA	$\bar{x}$	42.1	35.5
		±	4.2	2.8
Carbohydrate intake ($g \cdot kg^{-1} \cdot d^{-1}$)	P	$\bar{x}$	6.2	4.9
		±	0.7	0.4
	CLA	$\bar{x}$	5.6	4.3
		±	0.6	0.4
Protein intake ($g \cdot kg^{-1} \cdot d^{-1}$)	P	$\bar{x}$	1.8	1.9
		±	0.2	0.2
	CLA	$\bar{x}$	1.9	1.5
		±	0.2	0.2
Fat intake ($g \cdot kg^{-1} \cdot d^{-1}$)	P	$\bar{x}$	1.7	2.8
		±	0.2	0.3
	CLA	$\bar{x}$	1.4	2.4
		±	0.2	0.2

* $\bar{x}$ = group mean; ± = *SE* of mean.

Training and Diet

No statistically significant differences were observed among groups in total lifting volume during the training period (P, 138,158 ± 21,549 kg; CLA, 128,248 ± 17,847 kg; $p = 0.35$). Table 2 presents dietary intake data for the P and CLA groups. MANOVA revealed that no significant differences were observed between groups among energy or macronutrient intake ($p = 0.36$).

Hematological Analysis

No significant differences were observed between groups in total protein, albumin, globulin, glucose, electrolytes, liver enzymes, lipid profiles, total bilirubin, hemoglobin, hematocrit, red blood cells, or white blood cells. These findings suggest that CLA supplementation does not promote clinically significant changes in general markers of health. Table 3 presents selected markers of catabolism and immunity observed for the P- and CLA-supplemented groups. No significant differences were observed in markers of catabolism (creatinine, blood urea nitrogen, creatine kinase, lactate dehydrogenase) or types of lymphocytes (neutrophils, lymphocytes, eosinophils, basophils, monocytes). However, there was some evidence that CLA supplementation tended to lessen changes in the ratio of blood urea nitrogen to creatinine (P, 2.3 ± 1.0; CLA, −0.41 ± 1.0, $p = 0.09$) and the ratio of neutrophils to lymphocytes (P, 8.0 ± 12; CLA, −25 ± 12%, $p = 0.07$). Effect size (ES) analysis revealed moderate to large effects for changes in the ratio of blood urea nitrogen to creatinine (ES = −0.74, $r = -0.35$) and the

Table 3. DEXA body composition and bone mass data for P and CLA groups.

Variable	Group		Day 0	Day 28		*p*
Body mass (kg)	P	$\bar{x}$*	79.5	79.4	Group	0.55
		±	3.1	3.3	Time	0.75
	CLA	$\bar{x}$	82.0	82.3	Group × time	0.43
		±	3.1	3.1		
Scanned mass (kg)	P	$\bar{x}$	73.2	73.3	Group	0.51
		±	3.1	3.2	Time	0.12
	CLA	$\bar{x}$	75.5	76.1	Group × time	0.26
		±	3.1	3.0		
Fat/bone-free mass (kg)	P	$\bar{x}$	59.0	59.0	Group	0.37
		±	1.9	1.9	Time	0.31
	CLA	$\bar{x}$	61.5	62.0	Group × time	0.49
		±	2.5	2.5		
Fat mass (kg)	P	$\bar{x}$	11.9	12.0	Group	0.92
		±	1.6	1.6	Time	0.20
	CLA	$\bar{x}$	11.5	12.0	Group × time	0.37
		±	2.0	2.0		
Bone mass (g)	P	$\bar{x}$	2,558	2,554	Group	0.74
		±	102	100	Time	0.21
	CLA	$\bar{x}$	2,492	2,516	Group × time	0.08
		±	121	117		
Body fat (%)	P	$\bar{x}$	15.9	15.9	Group	0.79
		±	1.6	1.5	Time	0.33
	CLA	$\bar{x}$	15.0	15.3	Group × time	0.44
		±	2.2	2.1		

* $\bar{x}$ = group mean; ± = *SE* of mean.

Table 4. Selected markers of catabolism and immunity for the P- and CLA-supplemented groups.

Variable	Group		Day 0	Day 28		*p*
Creatinine (μmol·L^{-1})	P	x̄*	99	97	Group	0.13
		±	3	1	Time	0.001
	CLA	x̄	107	101	Group × time	0.73
		±	3	2		
Blood urea nitrogen (mmol·L^{-1})	P	x̄	13.8	15.5	Group	0.82
		±	1.3	1.2	Time	0.83
	CLA	x̄	15.6	14.4	Group × time	0.11
		±	1.3	0.8		
Urea nitrogen/creatinine ratio	P	x̄	11.9	14.2	Group	0.81
		±	1.1	1.2	Time	0.23
	CLA	x̄	13.0	12.5	Group × time	0.09
		±	1.1	0.6		
CK (IU·L^{-1})	P	x̄	294	261	Group	0.23
		±	61	87	Time	0.71
	CLA	x̄	427	414	Group × time	0.87
		±	117	102		
LDH (IU·L^{-1})	P	x̄	166	139	Group	0.42
		±	9	9	Time	0.001
	CLA	x̄	178	147	Group × time	0.72
		±	9	10		
Neutrophil/lymphocyte ratio	P	x̄	1.6	1.6	Group	0.001
		±	0.3	0.2	Time	0.11
	CLA	x̄	1.4	1.0	Group × time	0.13
		±	0.2	0.2		

* x̄ = group mean; ± = *SE* of mean; CK = creatine kinase; LDH = lactate dehydrogenase.

neutrophil to lymphocyte ratio (ES = −0.66, r = −0.31).

Body Composition and Bone Density

Table 4 presents body composition and bone density results observed for the P and CLA groups. No significant differences were observed in changes in total body mass (P, −0.12 ± 0.3 kg; CLA, 0.29 ± 0.4 kg, p = 0.43), DEXA-scanned mass (P, 0.11 ± 0.3 kg; CLA, 0.60 ± 0.3 kg, p = 0.26), fat/bone-free mass (P, 0.05 ± 0.2 kg; CLA, 0.28 ± 0.2 kg, p = 0.49), fat mass (P, 0.06 ± 0.2 kg; CLA, 0.32 ± 0.2 kg, p = 0.37), or percent body fat (P, 0.03 ± 0.2%; CLA, 0.30 ± 0.3%, p = 0.44). However, there was some evidence that bone mass tended to increase in the CLA group (P, −4.0 ± 8.0 g; CLA, 25 ± 14 g, p = 0.08; P, −0.1 ± 0.3%; CLA, 1.1 ± 0.6%, p = 0.08). Effect size calculations revealed a moderate to large effect on changes in bone mass (ES = 0.75; r = 0.35).

Strength

Table 5 presents changes in strength observed for the P and CLA groups. No significant differences were observed between groups in changes in 1RM bench press (P, −2.4 ± 2.3 kg; CLA, 2.3 ± 2.2 kg, p = 0.18), leg press (P, 7.1 ± 3.0 kg; CLA, 15.5 ± 5 kg, p = 0.18), or overall gains in 1RM strength (P, 4.8 ± 4.0 kg; CLA, 17.6 ± 7 kg, p = 0.11).

Discussion

The major finding of this study was that although some interesting trends were observed, 28 days of CLA supplementation (6.2 g·d^{-1}) did not promote statistically significant changes in markers of catabolism, immunity, body composition, bone density, or strength in experienced resistance-trained athletes. These findings appear to contrast with studies conducted on animals as well as marketing claims that CLA supplementation may possess ergogenic value for resistance-trained athletes and bodybuilders. Initially, we felt that the lack of significance observed may simply be due to an inadequate length of supplementation and/or that well-trained athletes with relatively low body fat may be more resistive to CLA supplementation. However, as will be described below, the majority of studies conducted following this initial trial that evaluated longer supplementation periods in untrained, overweight, and/or trained populations have supported the results observed in this initial study. The following discussion examines the results of this initial study in light of more recent studies conducted on CLA supplementation in humans.

Previous research indicated that CLA reduced markers of catabolism and immunity in animals (7, 31, 46). Theoretically, if CLA supplementation produced similar effects in humans, CLA supplementation may

Table 5. One repetition maximum strength data for the P and CLA groups.

Variable	Group		Day 0	Day 28		*p*
Bench press (kg)	P	$\bar{x}$*	106.0	104.0	Group	0.64
		±	8.4	8.1	Time	0.92
	CLA	$\bar{x}$	109.0	111.0	Group × time	0.18
		±	7.4	7.4		
Bench press ($kg \cdot kg^{-1}$)	P	$\bar{x}$	1.3	1.3	Group	0.80
		±	0.9	0.8	Time	0.75
	CLA	$\bar{x}$	1.3	1.4	Group × time	0.24
		±	0.8	0.8		
Leg press (kg)	P	$\bar{x}$	166.0	173.0	Group	0.54
		±	12.8	14.8	Time	0.001
	CLA	$\bar{x}$	174.0	189.1	Group × time	0.18
		±	12.9	14.6		
Leg press ($kg \cdot kg^{-1}$)	P	$\bar{x}$	2.1	2.1	Group	0.57
		±	1.0	1.0	Time	0.001
	CLA	$\bar{x}$	2.1	2.3	Group × time	0.19
		±	1.0	1.0		

* $\bar{x}$ = group mean; ± = *SE* of mean.

allow an athlete to tolerate greater training loads and/or reduce incidence and/or severity of colds and infections during training. Results of the serum and whole blood analyses performed in the present study revealed 2 main findings: (a) CLA supplementation appears to be relatively safe and does not promote clinically significant changes in general markers of health; and (b) although some potentially beneficial trends were observed in the ratio of urea nitrogen/creatinine (a general marker of catabolism) and neutrophil/lymphocytes (a general marker of immune stress), CLA supplementation did not significantly reduce markers of catabolism or immune stress. Whether these findings were due to the length of supplementation, the training status of the subjects, and/or the type of training-employed stress is unclear. However, it is interesting to note that several studies conducted after this initial trial have also reported similar findings.

For example, Atkinson et al. (2) reported that CLA supplementation (2.7 $g \cdot d^{-1}$ for 6 months) did not significantly affect hormonal or hematological markers of health in 71 moderately overweight individuals engaged in an exercise/weight loss program. Likewise, Von Loeffelhotz et al. (43) reported that CLA supplementation (7 $g \cdot d^{-1}$ for 6 months) during training had no effects on selected blood markers among subjects assigned to a nontrained placebo group, a group of untrained subjects who initiated training during the study, or in an experienced group of resistance-trained subjects who trained during the study. Conversely, Beuker et al. (5) reported that CLA supplementation (12 $g \cdot d^{-1}$ for 7 weeks) decreased plasma cholesterol by approximately 15%. Additionally, Blankson et al. (6) reported that CLA supplementation (1.7, 3.4, 5.1, or 6.8 $g \cdot d^{-1}$ for 12 weeks) significantly decreased blood lipids in nontrained obese subjects. These researchers also reported that serum creatinine and bilirubin levels were significantly lower in subjects ingesting 5.1 $g \cdot d^{-1}$ of CLA and that creatine kinase levels were significantly lower in subjects ingesting 6.8 $g \cdot d^{-1}$ of CLA. These latter findings provide some limited support to the hypothesis that CLA may affect blood lipids and/or markers of catabolism. However, additional research is needed before definitive conclusions can be drawn.

Conjugated linoleic acid supplementation has been reported to increase total body mass from 36 to 57% (8, 9, 37) and reduce fat mass from 27 to 60% (10, 12, 36–38) in various animal populations. The reduction in body fat has been proposed to be due to an increased hormone-sensitive lipase activity and/or a norepinephrine-induced lipolysis (36, 37). Animal studies also indicate that CLA supplementation increases FFM (10), possibly by mediating anabolic hormone interactions (36, 37). Theoretically, CLA supplementation during training may therefore promote greater fat loss and increases in muscle mass.

Results from the present study, however, do not support this hypothesis. In this regard, results revealed that CLA supplementation (6.2 $g \cdot d^{-1}$ for 28 days) during training did not significantly affect DEXA-determined body composition alterations in experienced resistance-trained subjects. Initially, we thought that it was possible that trained subjects with average to low fat mass may be more resistant to body composition alterations with CLA and/or that the length of supplementation may have been too short to see an effect in trained subjects. However, the majority of studies that have been conducted following this ini-

tial trial have also reported that CLA supplementation (2.7–12 g·d^{-1} for 1.5–6 months) did not affect body composition alterations in overweight subjects (2, 30, 47), untrained subjects (40, 43, 47), or trained subjects (2, 5, 40, 43) as determined by skinfolds (5, 27, 40, 43), bioelectrical impedance (27, 47), hydrostatic weighing (2), or DEXA (47). Collectively, these findings suggest that CLA supplementation does not appear to promote fat loss or lean tissue accretion in untrained or trained populations.

We are aware of only 3 published studies that have reported that CLA supplementation affects body composition. In the first study, Lowery et al. (27) reported that CLA supplementation (7.2 g·d^{-1} for 6 weeks) during resistance training significantly increased arm mass (CLA +5.3%, P +2.8%) and body mass (CLA +2.4 kg, P 0.0 kg) with no differences observed in skinfold or bioelectrical impedance–determined body fat. The researchers suggested that the gains in weight were apparently due to an increase in fat-free mass. In the second study, Smedman and Vessby (40) reported that CLA supplementation (4.2 g·d^{-1} for 12 weeks) promoted significantly greater changes in age-adjusted body fat (CLA −1.0%, P −0.3%). These findings suggest that CLA may promote a modest reduction in fat mass. More recently, Blankson et al. (6) reported that overweight subjects supplementing their diet with CLA (3.4 and 6.8 g·d^{-1} for 12 weeks) experienced significantly greater losses in DEXA-determined fat mass (−1.7 and −1.3 kg, respectively) compared with the P group (+1.8 kg). These findings provide the strongest evidence to date that CLA supplementation may promote fat loss. However, since the significant differences observed were due in part to an increase in fat mass in the P group and no effects were observed when subjects were administered 1.7 or 5.1 g·d^{-1} of CLA, results of this study are somewhat difficult to interpret. Consequently, it is our view that additional research is needed to determine whether CLA supplementation may affect body composition alterations in untrained and trained populations before definitive conclusions can be drawn.

Previous research in animals has indicated that CLA feeding increases bone mass and/or bone ash, possibly by reducing the production of PGE_2 and upregulating insulin growth-like factor availability (26, 46). Consequently, there has been some interest in whether CLA supplementation may affect bone mass. In the present study, we used DEXA as the method of evaluating body composition, and therefore were able to assess whole-body bone mass alterations in response to CLA supplementation. Although it is generally believed that it is difficult to increase bone mass, training (14, 22) and nutritional interventions (1, 20, 22) have been reported to affect bone mass. Results of the present study indicate that CLA supplementation did not significantly affect bone mass. However, an interesting statistical trend was observed that deserves some comment. In this regard, subjects in the CLA-supplemented group experienced a 1.1% increase (p = 0.08) in total bone mass with a moderate to large effect size. In our view, this finding provides some support to the hypothesis that CLA may affect bone mass, which deserves additional study.

It has been hypothesized that CLA supplementation during resistance training may promote greater gains in strength, possibly in response to a decreased catabolism and/or enhanced muscle mass. In the present study, although some trends were observed in overall gains in strength, CLA supplementation did not significantly affect gains in bench press or leg press/hip sled 1RM. These findings suggest that CLA supplementation does not appear to possess significant ergogenic value. Since this initial study, several research groups have evaluated the effects of CLA supplementation on training adaptations. In support of the present findings, Von Loeffelhotz et al. (43) reported that CLA supplementation (7 g·d^{-1} for 6 months) did not affect gains in 1RM strength during training. However, Lowery et al. (27) reported that CLA supplementation (7.2 g·d^{-1} for 6 weeks) promoted greater gains in leg press strength during resistance training in novice bodybuilders (CLA +27%, P +13%). Additionally, Beuker et al. (5) reported that CLA supplementation (12 g·d^{-1} for 7 weeks) during training increased cycling power output efficacy. In our view, it is currently unclear whether CLA supplementation affects training adaptations, and more research is needed before definitive conclusions can be made.

Practical Applications

Conjugated linoleic acid has been marketed to resistance-trained athletes as a supplement that may promote fat loss while enhancing gains in strength and muscle mass during training. Results of this study indicate that although some interesting trends were observed that deserve additional research, 28 days of CLA supplementation (6 g·d^{-1}) does not appear to affect body composition alterations or gains in strength in experienced resistance-trained athletes. Therefore, CLA does not appear to be an effective ergogenic aid for this population.

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