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The Effect of Conjugated Linoleic Acid Supplementation on Lipid-Related Cardiovascular Biomarkers in Obese Adults

Fatemeh Esmaeili Shahmirzadi; MSc[1, 2], Saeid Ghavamzadeh; PhD*[1- 3] & Arash Rashidi; PhD[4]

[1] Food and Beverage Safety Research Center, Urmia University of Medical Sciences, Urmia, Iran; [2] Department of Nutrition, Medicine Faculty, Urmia University of Medical Sciences, Urmia, Iran; [3] Maternal and Child Obesity Research Center, Urmia University of Medical Sciences, Urmia, Iran; [4] Nutrition Research Department, National Nutrition and Food Technology Research Institute, Faculty of Nutrition Sciences and Food Technology, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

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Corresponding author:
ghavamzadeh@hotmail.com
Medicine Faculty, Urmia University of Medical Sciences, Nazloo Highway, Pardis-e-Daneshgah, Urmia, Iran.

Postal code: 5714783734
Tel: +98 914 341 3616

ABSTRACT

Background: Studies have shown incompatible findings regarding the effects of conjugated linoleic acid (CLA) supplementation on cardiovascular diseases (CVDs) risk factors. The aim of this study was to evaluate the effect of daily CLA supplementation on serum insulin and lipid- related CV biomarkers in obese adults. **Methods:** This randomized double-blind clinical trial was conducted on 54 adults categorized as class I obesity. The participants were randomly assigned into two groups (n=27) receiving a total of 3,000 mg/d of a 50:50 mixture of CLA isomers for three months in intervention group (IG) and 500 mg/d paraffin in placebo group (PG). Moreover, fasting serum levels of insulin, lipid profile, non-HDL-Cholesterol (non-HDL-C), atherogenic index of plasma (AIP), total triglyceride (TG)/HDL-C, and cholesterol/HDL-C ratio were measured. The main statistical analysis method was independent t-test for changes. **Results:** Changes between the groups showed a significant decrease in total cholesterol (P=0.03), LDL-C (P=0.04), and non-HDL-C (P=0.03), and also a significant increase in AIP (P=0.04) in IG compared to the PG. A remarkable decrease was found in HDL-C and cholesterol/HDL-C ratio. In addition, a remarkable increase was observed in TG in this context. Serum insulin, VLDL-C, and LDL-C/HDL-C ratio showed no significant changes during the intervention period. The use of CLA supplementation could help reduce some adverse fractions of serum lipid profile, particularly TC, non-HDL-C and LDL-C. **Conclusions:** Regarding the augmenting effects of CLA intake on AIP as a strong predictive marker for CVDs, it is difficult to confirm the beneficial effects of CLA supplementation in preventing CVDs.

Keywords: *Linoleic acid; Conjugated linoleic acid; Cardiovascular risk; Obesity.*

Introduction

Obesity refers to excessive accumulation of fat in body, which is known not only as a fitness problem, but also a multi-causative disease (Finucane *et al.*, 2011). The danger of growing prevalence of obesity as an epidemic has long been highlighted in Iran, and its prevalence has been

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rising in recent decades in this country (Bakhshi *et al.*, 2016, Musaiger, 2011).

Evidences has suggested that obesity and excess mortality are linked to each other through increased risk of essential hypertension, type 2 diabetes mellitus (T2DM), and cardiovascular diseases (CVDs) among obese individuals (Chaudhary *et al.*, 2019, Dal Canto *et al.*, 2020, Wang *et al.*, 2021). Insulin could stimulate glucose disposal in non-diabetic healthy population by various degrees. As a result, a large number of these individuals may be known as insulin resistant subjects (Park *et al.*, 2021, Reaven *et al.*, 1993).

Insulin resistance and compensatory hyperinsulinemia were strongly correlated with CVDs, independent from the other risk factors (Ormazabal *et al.*, 2018).

The disturbance of the lipolysis in adipose tissue can lead to increased plasma free fatty acid levels. It seems that this mechanism is responsible for insulin resistance in obesity and T2DM. Hormone sensitive lipase in adipose probably results in the production of most of the free fatty acids. However, there is a separate process, known as spillover, in which endothelial lipase, especially lipoprotein lipase, acts on triglyceride-rich lipoproteins (Eckel *et al.*, 2005, Miles and Nelson, 2007). Both lipolysis inhibitory effect and the stimulation of lipoprotein lipase depend on insulin. It seems that the substantial substrate for spillover would be chylomicrons derived from dietary fat.

In insulin resistance status, the adipose tissue produces more fatty acids due to increased amount of lipolysis of stored triacylglycerol molecules. These fatty acids play an inhibitory role in antilipolytic effect of insulin, developing additional lipolysis (Eckel *et al.*, 2005, Ormazabal *et al.*, 2018). It seems that chronic hyperinsulinemia could stimulate very low-density lipoprotein (VLDL) production, too (Julius, 2003).

Biological active isomers of conjugated linoleic acid (CLAs) are one of the supplements used in weight loss. Since the discovery of CLA, many studies focused on the traits of CLA in animal models and cell culture environments (humans and animals); therefore, in one study, it was concluded that CLA supplementation could reduce body fat and serum leptin (Esmaeili Shahmirzadi *et al.*, 2019). It has also been shown CLA could have beneficial effects on health including antiadipogenic (den Hartigh, 2019, Pariza *et al.*, 2001) anti-carcinogenic (den Hartigh, 2019, McGowan *et al.*, 2013), anti-inflammatory properties (Bassaganya-Riera *et al.*, 2003, Fleck *et al.*, 2021, Lordan *et al.*, 2018), anti-diabetic (Moloney *et al.*, 2007), and also antiatherogenic properties (Koba *et al.*, 2002, Kritchevsky *et al.*, 2004). According to the current documents, CLA's effect on CVDs biomarkers remains indecisive. It has been found that CLA could result in hypocholesterolemic, hypotriglyceridemic, and anti-atherosclerotic properties in animals (McLeod *et al.*, 2004). CLA supplementation for 8 weeks reduced plasma total triglycerides (TG) and very low-density lipoprotein cholesterol (VLDL-C) among normolipidemic men and women (Noone *et al.*, 2002). However, other studies reported no effects on different doses of CLA supplements regarding blood lipids (Eftekhari *et al.*, 2014, Joseph *et al.*, 2011). Because of the importance of CVDs, as one of the most common problems in Iranian society, and due to the above-mentioned contradictory findings regarding the effect of CLA on CVDs risk indexes; the authors decided to design this study to evaluate the effect of CLA supplementation on serum lipid-related CVDs biomarkers in obese adults. The secondary objective was to study the effect of CLA supplementation on insulin sensitivity as a link between obesity and CVDs in the studied population.

Materials and Methods

Participants and study design

In line with Nikan Hospital in Tehran, 120 obese people who went to Weight Loss Clinic for weight reduction were considered as candidates for this study, and afterwards, 64 people were assigned to this study in terms of the inclusion criteria. All the eligible participants were allocated into two groups using random allocation rule method as follows: the intervention group (IG, n=32) and the placebo

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group (PG, n=32).

According to the inclusion criteria, these individuals were included in the study if they were between 18 and 65, and were at grade 1 obesity (BMI of 30-34.9 kg/m^2). The exclusion criteria were having any illnesses e.g., diabetes and CVDs, a history of cigarette smoking, taking any medications, taking CLA supplement, and having a weight loss diet in the past 3 months. As a result, none of the participants were addicted to cigarettes, alcohol, and narcotics. They suffered from no other diseases such as endocrine, liver, diabetes, renal, cardiac or respiratory illnesses. The studied subjects have also followed no therapeutic diet since the last three months. All these mentioned characteristics were considered as exclusion criteria in this study. In total, 120 participants were included with grade 1 obesity; however, 56 subjects were excluded in the eligibility phase due to not meeting inclusion criteria. 5subjects in each group were dropped out due to some reasons reflected in **Figure 1**. Therefore, the rate of compliance was 85% for both groups.

Random allocation was performed by selection of a total study size of 64. Afterwards, two groups of cards, named A and B, were placed in a hat and were randomly drawn without replacement. The sequence generation would randomly order 32 subjects in IG and 32 participants in the PG.

From a statistical viewpoint, using the formula suggested for clinical trials, having 15 participants in each group was adequate; however, Considering a type 1 error (α) of 0.05 and type 2 error (β) of 0.05 (power = 95%) according to Blankson *et al.'s* study (Blankson *et al.*, 2000), the mean±SD of changes in body fat mass (BFM) in IG (3.4 g) and PG were 1.73±1.9 and 1.47±2.43 during 12 weeks of intervention, respectively. However, with regard to the participants who dropped out of the study and enhanced its validity, the authors decided to increase the sample size as far as possible (n=32). Finally, there were 5 dropouts for each group (**Figure 1**) and we completed the study with 27 people in each of the studied groups.

Data collection

In this randomized double-blind clinical trial, the participants were divided into two groups as follows: IG received a daily dose of 3000 mg of CLA supplements (1000 mg t.d.s containing 50:50 mixture of cis-9, trans-11 and trans-*10*, and cis-12 CLA isomers), and the PG received the same number of placebos daily (500 mg t.d.s paraffin oil) for 12 weeks. CLA capsules used in this study were prepared by Nutricentury®/Canada, and placebo capsules were from Zahravi Co. Pharmaceutical Company in Iran. Blinding was applied at two levels of the participants and data gatherer co-worker. Both CLA and placebo capsules were completely similar in size and color. The encoded boxes of both capsules were presented to the participants by an assistant who was aware of the boxes' contents. They were instructed to take 3 capsules before eating each meal. Individuals who reported that they had taken less than 80% of the supplements based on self-reports or counting the number of capsules in the package delivered at the end of the study, were excluded from the final analysis of the data. Participants were urged not to change their physical activity, diet, or lifestyle during the study and quickly report any abnormal feelings. Furthermore, at the beginning and at the end of the study, general information questionnaires, dietary intake, and international physical activity questionnaire (IPAQ) were completed and collected. In order to measure physical activity, a Persian translated IPAQ was used. The validation and reliability of the questionnaire had been confirmed in a published document (Vasheghani-Farahani *et al.*, 2011).

The questionnaire was completed by self-administering the samples themselves at the beginning and end of the study. They were asked to not change their activities until the end of the study.

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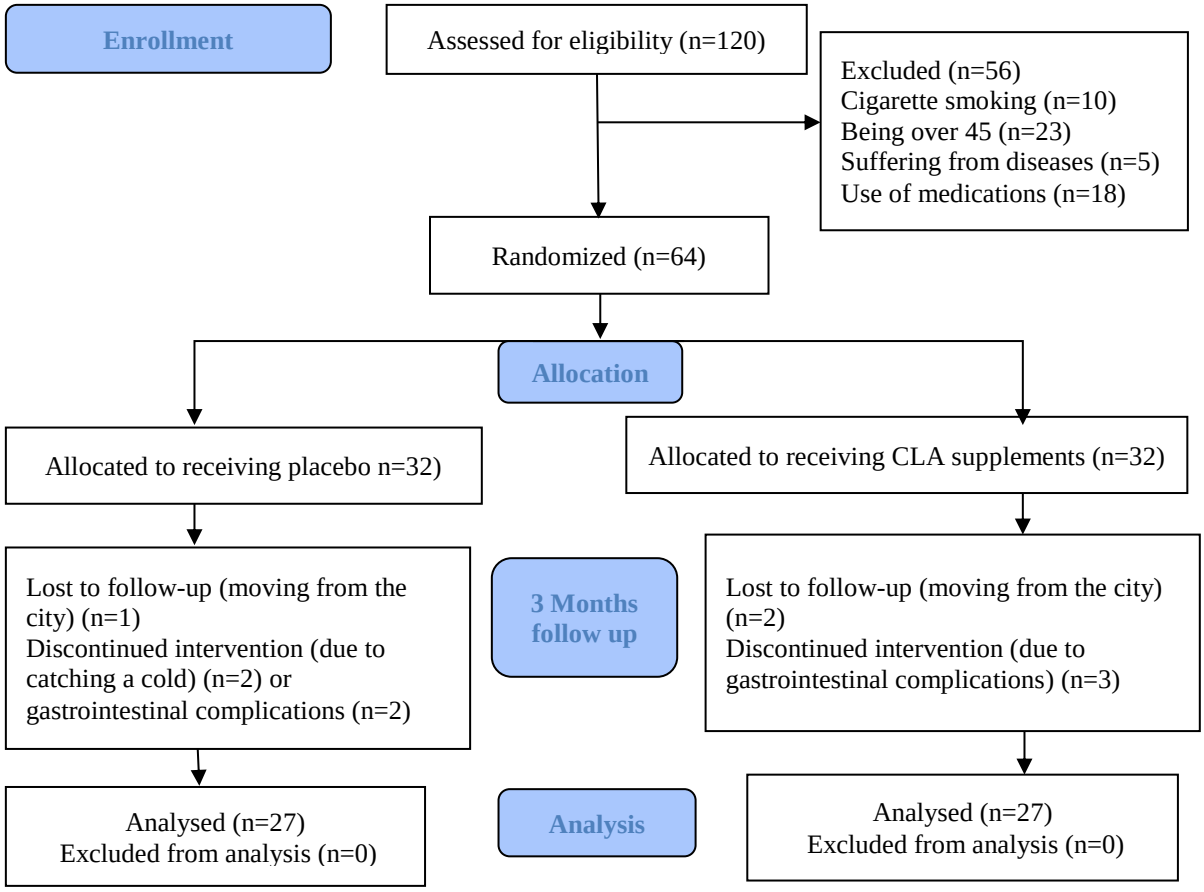

Figure 1. The flowchart of sample selection in two groups according to the criteria for entering, leaving and dropping the samples.

Dietary assessment: Dietary assessment was fulfilled by the Three-Day Written Diet Record form. For this purpose, the participants were trained in two public sessions by an experienced fieldworker. Accordingly, energy and macronutrients intakes were estimated in 3 consecutive days (one day off and two working days). A modified version of Nutritionist IV software for Iranian community was used to analyze nutritional data. The energy and macronutrients intake as well as vitamin D and calcium intake were reported based on kilocalories and grams per day, respectively.

Laboratory tests

Fasting venous blood samples (5 ml) were collected from the participants by a trained laboratory technician. Blood samples were moved to anticoagulant tubes of EDTA and non-anticoagulant tubes, and were then transferred to Nikan hospital's clinical laboratory. EDTA-containing tubes were closed by parafilm and slowly mixed with anticoagulants to prevent clotting. To prepare serum, the tubes were centrifuged for 10 minutes at 1500 rpm. Serum samples were then stored in encoded microtubes for each patient and stored at -80° C to retain, if needed. Biochemical measures included the measurement of serum insulin, fasting plasma glucose, total cholesterol (TC), TG, LDL-C, and High-density lipoprotein cholesterol (HDL-C). In order to obtain greater reliability, biochemical tests, food intake, and anthropometric measurements were performed by a certain person and/or device.

The concentrations of TC, TG, HDL-C, and LDL-C were measured using an automatic biochemistry

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analyzer (BT 1500, Italy). Also, TC, TG, HDL-C, and LDL-C levels were measured using an enzymatic assay (Biosystem, Spain). VLDL-C was calculated based on Friedewald estimation: VLDL-C = TG/5 (Friedewald *et al.*, 1972) .

AIP was derived as the logarithmically transformed ratio of molar concentrations of TG to HDL-C (Dobiásová and Frohlich, 2001, Wu *et al.*, 2018). Non-HDL Cholesterol was obtained by subtracting HDL-C level from TC (Jiang *et al.*, 2004). LDL-C/HDL-C ratio was calculated to estimate CVDs risk (Khazaál, 2014, Wu *et al.*, 2018), and cholesterol/HDL-C ratio was calculated via dividing TC by HDL-C number (Lemieux *et al.*, 2001). Seated blood pressure (BP) was measured by mercury sphygmomanometer (Riester Co., Germany) after a 10-min rest in waiting room.

Ethical considerations

All the participants were requested to complete the informed consent form approved by the Ethics Committee of Urmia University of Medical Sciences. All the information collected from the subjects remained confidential at all the stages of the investigation. In addition, as the research protocol states, the participant was urged to quit the research in case of any problem. All the stages of study were conducted by a trained expert. This research was approved by the Ethics Committee of Urmia University of Medical Sciences under the number umsu.rec.202. It was also registered at Iran Clinical Trials Center (www.irct.ir) under the code IRCT2014052413678N2.

Data analysis

Data were reported as mean±SD. The statistical significance level in all the analyses was considered less than or equal to 0.05. Chi-square test and/or Fisher's exact test were used to examine statistical relationships between qualitative variables. Normality of data was examined by Kolmogorov-Smirnov test, and if data distribution was normal, paired t-test was used to compare the values before and after the intervention in each of the IG and PG. If not, Wilcoxon Signed Rank test was used. Mann-Whitney U test was also used to compare the mean of data in IG, and for PG, given they were normal, independent *t-test* was used. Analysis between groups regarding differences in changes was also done using independent *t-test*. Data were analyzed using SPSS software version 20.0.

Results

General characteristics are shown in **Table 1**. The age of all the participants was between 29 and 64 with the mean±SD of 36.72±5.78 year for PG and 38.22±7.74 year for IG (*P*=0.23). No significant difference in literacy levels, occupational, or marital status was found among the groups at the beginning of the study (*P*>0.05).

Table 1. General characteristics of the participants at baseline (n=54).

Variables	PG (n=27)	IG (n=27)	P-value[b]
Gender			
Male	13 (48.2)[a]	12 (44.4)	0.78
Female	14 (51.8)	15 (55.6)	
Literacy levels			
Illiterate	0 (0.0)	1 (3.7)	0.23
Below high school diploma	8 (29.7)	10 (37.0)	
High school diploma	9 (33.3)	7 (26.0)	
Academic literacy	10 (37.0)	9 (33.3)	
Occupational status			
Business	9 (33.3)	10 (37.0)	0.46
Governmental employee	6 (22.3)	9 (33.3)	
Housekeeper	12 (44.4)	8 (29.7)	
Marital status			
Married	21 (77.7)	23 (85.2)	0.88
Divorced	1 (3.7)	0 (0.0)	
Single	5 (18.6)	4 (14.8)	

[a]: n (%); [b]: Chi square test. **PG**: Placebo group ; **IG:** Intervention group

Table 2 shows the findings of the study on laboratory indicators of the participants. None of the laboratory indices pointed out any significant differences among the two studied groups, neither at beforee nor after intervention stage. However, group changes showed a significant decrease in TC, LDL-C, and Non-HDL-C and a significant increase in AIP in IG in comparison with the PG. Furthermore,

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a remarkable decrease was found (but not significant) in HDL-C and cholesterol//HDL-C ratio and a remarkable increase was also observed in TG in this regard. The researchers also compared the data of systolic BP and diastolic BP between the groups**.** According to **Table 2**, of systolic BP and diastolic BP were not altered during the intervention.

Table 2 illustrates dietary intake of the subjects. As shown in the tables, among the variables studied, the intake of no studied nutrients significantly changed during the study period. Analysis of physical activity data revealed that most of the subjects were at the minimally inactive category in both groups and in all of the studied phases within a range of 38% to 48% (P<0.05).

Table 2. Comparison of the laboratory indicators, blood pressure and dietary intake at the two studied stages in studied groups.

Variables	Placebo group (n=27)	Intervention group (n=27)	P-value[a]
Total cholesterol (mg/dl)			
Before	179.63±46.3[c]	193.11±35.76	0.11
After	173.13±36.57	177.07±35.77	0.02
P-value[b]	0.22	0.08	
Changes	-6.50±9.32	-16.04±10.24	0.03
Triglyceride (mg/dl)			
Before	145.81±93.27	148.51±92.44	0.16
After	147.40±85.30	151.59±84.58	0.09
P-value	0.54	0.24	
Changes	1.59±22.63	3.08±31.34	0.08
Low density lipoprotein (mg/dl)			
Before	105.36±25.21	113.37±22.69	0.11
After	104.13±22.75	108.14±21.97	0.16
P-value	0.31	0.26	
Changes	-1.23±6.26	-5.23±10.12	0.04
High density lipoprotein (mg/dl)			
Before	42.22±35.76	43.69±9.61	0.14
After	42.63±9.96	40.36±10.30	0.09
P-value	0.61	0.12	
Changes	0.41±3.69	-3.33±4.62	0.07
Very low density lipoprotein (mg/dl)			
Before	28.89±8.72	28.67±6.93	0.44
After	29.48±7.72	30.49±4.46	0.14
P-value	0.35	0.29	
Changes	0.59±3.64	1.82±4.96	0.19
Atherogenic index of plasma			
Before	0.17±0.06	0.18±0.10	0.44
After	0.18±0.03	0.21±0.08	0.09
P-value	0.35	0.29	
Changes	0.01±0.60	0.04±0.87	0.04
Non-High density lipoprotein (mg/dl)			
Before	116.25±23.45	123.89±25.67	0.44
After	113.56±29.21	115.43±26.17	0.30
P-value	0.35	0.29	
Changes	-2.69±14.57	-8.46±26.24	0.03
Low density lipoprotein /High density lipoprotein			
Before	2.78±1.12	2.34±1.03	0.19
After	2.56±1.11	2.86±1.42	0.09
P-value	0.35	0.29	
Changes	-0.22±1.25	0.52±0.96	0.10

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Cholesterol//HDL-C ratio			
Before	4.56±2.12	4.84±2.32	0.33
After	4.33±1.96	4.22±2.01	0.64
P-value	0.77	0.43	
Changes	-0.23±1.22	-0.62±1.95	0.07
Insulin (mIU/l)			
Before	14.81±7.27	14.82±8.04	0.98
After	15.07±8.61	14.22±7.64	0.22
P-value	0.32	0.18	
Changes	0.26±4.26	-0.60±5.24	0.27
Diastolic blood pressure (mmHg)			
Before	7.45±1.67	7.19±1.62	0.32
After	7.34±1.21	7.42±1.32	0.43
P-value	0.29	0.21	
Changes	-0.11±2.07	0.23±1.75	0.15
Systolic blood pressure (mmHg)			
Before	12.35±2.12	12.89±1.12	0.64
After	11.86±2.32	12.23±1.96	0.28
P-value	0.32	0.31	
Changes	-0.49±1.94	-0.66±1.69	0.35
Energy (kcal/d)			
Before	1853±279	1890±350	0.39
After	1813±318	1820±379	0.34
P-value	0.25	0.16	
Changes	-40±259	-70±316	0.23
Protein (g/d)			
Before	64±17	66±22	0.17
After	62±13	64±18	0.17
P-value	0.34	0.29	
Changes	-2±14	-2±18	0.33
Carbohydrate (g/d)			
Before	234±59	241±58	0.49
After	228±57	236±53	0.45
P-value	0.39	0.44	
Changes	-6±49	-5±46	0.50
Fat (g/d)			
Before	66±19	67±24	0.68
After	66±17	65±20	0.81
P-value	0.79	0.43	
Changes	0±14	-2±17	0.12
Calcium (mg/d)			
Before	689±62	636±78	0.37
After	629±73	654±59	0.50
P-value	0.10	0.29	
Changes	-60±64	18±52	0.49
Vitamin D (µg/d)			
Before	3.32±0.52	2.96±0.68	0.22
After	3.46±0.79	3.12±0.69	0.23
P-value	0.54	0.65	
Changes	0.14±0.24	0.16±032	0.16
Soluble fiber (g/d)			
Before	1.73±0.27	1.77±0.28	0.25
After	1.59±0.32	1.71±0.29	0.33
P-value	0.24	0.41	
Changes	-0.14±0.28	-0.06±0.08	0.20

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Insoluble fiber (g/d)			
Before	12.24±1.18	12.64±1.48	0.27
After	11.63±1.22	12.12±1.63	0.22
P-value	0.43	0.49	
Changes	-0.61±0.99	-0.52±1.09	0.44
Sodium (mg/d)			
Before	3234±451	3281±489	0.09
After	3356±522	3517±422	0.12
P-value	0.29	0.17	
Changes	122±375	236±462	0.08

[a]: Independent sample t-test; [b]: Paired t-test; [c]: Mean±SD.

Discussion

While studies are still at their preliminary phase, the desirable potential effects of dairy fats including CLA on CV biomarkers have attracted remarkable research interests (Lordan *et al.*, 2018). Despite the fact that several investigations have revealed no significant effect on serum lipid profile, some trials reported both beneficial and adverse effects on TC, LDL-c, HDL-c, and TGs (Agueda *et al.*, 2009, Eftekhari *et al.*, 2014, Joseph *et al.*, 2011, Steck *et al.*, 2007).

The study by Steck *et al.* was similar to this study in terms of dose and complementary CLA and duration of the intervention. This clinical trial was performed on obese people with a BMI of 30-35 kg/m2 for 12 weeks with CLA supplementation (50:50 ratios of cis-9, trans-11 and trans-10, cis-12 isomers). Also, the supplements were given in two doses of 3.2 and 6.4 g/d CLA. The results showed that CLA supplementation may lead to a significant decrease (P<0.05) in serum HDL-C among the participants receiving 6.4 g/d CLA. Furthermore, a significant fall could be observed in HDL-C in placebo group. Also, no changes were observed in serum TG, TC, and LDL-C in studied groups (Steck *et al.*, 2007). Based on the findings, a remarkable decrease could be detected in HDL-C levels; however, it was not a statistically significant decrease. These effects were shown at a dose of 3 g/d while in Steck *et al's* study, it was achieved only at a dose of 6.4 g/d. The results of the changes regarding differences between the groups were not seen in Steck *et al's* study, whereas a significant decrease was detected between the groups regarding HDL-C in this study. The study population of the survey, unlike the sample population of this study, belonged to different racial groups. Moreover, there were differences in sex ratio between the two studies. Besides, dietary intake of sodium and soluble and insoluble fibers were not reported. It was possible that the participants could not achieve a stable dietary intake of effective nutrients in serum lipid profile during the study period. Accordingly, the findings of this study showed that the studied groups achieved no significant difference in terms of the above-mentioned nutrients (P>0.05). Based on the findings reported by Josef et al., CLA's 8-week supplementation could not achieve a significant decline in serum lipid profile including TC, TG, VLDL-C, LDL-C, and HDL-C. The participants this study were overweight hyperlipidemic men, and the dietary intake had not been controlled in that study (Joseph *et al.*, 2011).

In their report, Mougios *et al.* showed that serum HDL-C significantly decreased (P <0.001) and TG as well as TC tended to be reduced in CLA group receiving 0.7 g of CLA for four weeks. These data indicated that daily supplementation of 0.7–1.4 g CLA daily for 4–8 weeks may modulate serum lipids and increase CLA content of serum lipids in humans (Mougios *et al.*, 2001). Participants were not obese (BMI<30) and dietary control had been fulfilled. Although such changes in serum lipids have not been documented by this low dose of CLA in other sources, the results were approximately similar to the findings except for TG figures. Also, dietary intakes of three nutritional factors affecting serum lipid profile including fibers, calcium, and vitamin D were not analyzed in this paper.

According to our results, serum insulin was not altered during the intervention period. Although

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the associations between serum insulin and some components of serum lipids were reported by some studies conducted on patients with diabetes (Moussa *et al.*, 1998, Yan *et al.*, 2016); Based on some recent studies, no clear correlations were found between them in participants such as non-diabetic people (Bedogni *et al.*, 2019). Accordingly, that may explain the significant changes in serum lipid components in spite of no statistical changes in serum insulin in this study.

Regarding serum lipid-related CVDs predicting indexes, the findings of this study were more confusing. In IG, while Non-HDL-C were significantly decreased (P=0.03) and cholesterol//HDL-C ratio tended to decline (P=0.07), AIP was significantly risen (P=0.04). The mentioned variables were rarely reflected in findings of the available studies. Eftekhari *et al.* reported no changes in LDL-C/HDL-C among CLA supplemented group in comparison with placebo group. Serum LDL-C was not directly measured, and also it was obtained using Friedewald equation (Eftekhari *et al.*, 2014). It seems that AIP as a novel predictive indicator for CVDs is a stronger predictor for the disease compared to others (Cai *et al.*, 2017). Nevertheless, the researchers could reveal no changes in systolic BP, Diastolic BP, dietary intakes of macronutrients, calcium, sodium, soluble and insoluble dietary fibers, and physical activity among the two studied groups during the intervention. Additionally, no changes were observed in serum insulin as a major hormone affecting the lipid metabolism. In a meta-analysis, Yang *et al.* included 8 studies with 9 trials, which involved 638 participants with CLA supplementation ranging from 2.0 g/day to 6.8 g/day (Yang *et al.*, 2015). The findings of this meta-analysis did not support the overall favorable effect of CLA supplementation on BP regulation.

It should be noted that in this research, the authors measured no inflammation markers affecting the lipid levels such as hs-CRP and TNF-α. They also could not measure CLA dietary intake. To the best of the author's knowledge, the effect of CLA on some lipid-related biomarkers including AIP, Non-HDL-C, LDL-C/HDL-C, and Cholesterol//HDL-C ratio in obese adults was studied for the first time.

Conclusion

The authors concluded that the use of CLA supplementation could help reduce some adverse fractions of serum lipid profile, particularly TC, Non-HDL-C, and LDL-C. Regarding the augmenting effects of CLA intake on AIP as a strong predictive marker for CVDs, it is difficult to confirm the beneficial effects of CLA supplementation on preventing CVDs. The authors suggest that epidemiological and animal studies be continued in this field.

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Authors' contributions

All the authors were involved in designing, data gathering, statistical analysis and writing the original draft of the manuscript, and they approved of the final manuscript.

Conflicting interests

The authors declared no conflict of interests.

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