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ORIGINAL COMMUNICATION

Effect of conjugated linoleic acid supplementation after weight loss on appetite and food intake in overweight subjects

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Objective: To study the effects of 13 weeks conjugated linoleic acid (CLA) supplementation in overweight subjects on body-weight maintenance, parameters of appetite and energy intake (EI) at breakfast after weight loss.
Design: This study had a double-blind, placebo-controlled randomized design.
Subjects: A total of 26 men and 28 women (age 37.8±7.7 y; body mass index 27.8±1.5 kg/m^2).
Interventions: Subjects were first submitted to a very-low-calorie diet (VLCD; 2.1 MJ/day) for 3 weeks after which they started with the 13-week intervention period. They either received 1.8 g CLA or placebo per day or 3.6 g CLA or placebo per day. Additionally, subjects of the high dosage intervention replaced their habitual lunch by one meal of a protein-rich, low-energy supplement. EI was measured at breakfast and appetite profile after an overnight fast.
Results: The mean body weight loss was 6.9±1.7% of their original body weight. Multiple regression analysis showed that at the end of the 13-week intervention, CLA did not have an effect on body weight regain. Feelings of fullness and satiety were increased and feelings of hunger were decreased after 13 weeks intervention by CLA compared to placebo, independent of %body weight regain. However, EI measured at breakfast was not affected by CLA.
Conclusion: Appetite (hunger, satiety and fullness) was favorably, dose-independently affected by a 13-week consumption of 1.8 or 3.6 g CLA/day. This did not result in a lower EI at breakfast or an improved body-weight maintenance after weight loss.
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Keywords: CLA; satiety; appetite; body-weight regulation; energy intake

Introduction

Conjugated linoleic acid (CLA) refers to a group of positional and geometrical isomers of linoleic acid containing conjugated double bonds. It is naturally found in beef, milk and milk products since it is produced by rumen bacteria from linoleic acid (Kepler *et al*, 1970, 1971).

Numerous physiological effects in relation to body weight regulation have been attributed to CLA ingestion in animals. In different animal models, consumption of CLA has been shown to increase lean body mass (Park *et al*, 1997, Park *et al*, 1999a, b; DeLany *et al*, 1999) and to reduce body fat mass (Park *et al* 1997; Park *et al*, 1999a, b; West *et al*, 1998, 2000; DeLany *et al*, 1999; Azain *et al*, 2000). However, different effects in lean and obese rats have been observed (Sisk *et al*, 2001), that is, CLA ingestion decreased fat mass in lean rats, whereas it caused an increase of fat mass in obese rats. The results of studies on the effects of CLA on body weight are inconsistent. Some investigators found reduced body weight after a CLA diet (West *et al*, 1998; DeLany *et al*, 1999; Park *et al*, 1999a), whereas others found no effect (Park *et al*, 1997, Park *et al*, 1999b; West *et al*, 2000; Miner *et al*, 2001; Sisk *et al*, 2001) or an increase in body weight (Miner *et al*, 2001). Furthermore, CLA supplementation is associated with an increased energy expenditure (West *et al*, 1998, 2000; Ohnuki *et al*, 2001). The results of studies on the effects of CLA on energy intakes are inconsistent. Some studies found decreased energy intakes by CLA (West *et al*, 1998; Park *et al*, 1999b; Miner *et al*, 2001), whereas others observed no effect on food intake (DeLany *et al*, 1999; Azain *et al*, 2000; West *et al*, 2000; Sisk *et al*, 2001).

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Only a few human studies have been conducted to study the effect of CLA ingestion on body weight, BMI and/or fat mass. Even though fat mass (Blankson *et al*, 2000) and sagital abdominal diameter (Riserus *et al*, 2001) were lowered by CLA, it did not result in body weight loss (Berven *et al*, 2000; Blankson *et al*, 2000; Zambell *et al*, 2000; Riserus *et al*, 2001). In these studies, weight loss or fat mass loss was assessed. However, the effects of CLA supplementation might appear more clearly while subjects are in a state of weight (re)gain, since CLA reduces fat uptake into adipocytes (Park *et al*, 1997, 1999a; Choi *et al*, 2000), but does not enhance lipolysis (Pariza *et al*, 2001) and therefore it could block body fat gain instead of reducing body fat level. Blocking body fat gain during weight regain may lead to relatively more regain of fat-free mass. Since gain of fat-free mass is more costly than gain of fat mass (Stock, 1999), this may lead to a relatively smaller weight regain. Only two studies have been investigating the effect of CLA supplementation on appetite (Blankson *et al*, 2000; Medina *et al*, 2000) and observed no effect. Moreover, no human study has investigated the effect of CLA ingestion on energy intake. It is known that intake of CLA decreases the uptake of fatty acids in adipocytes and enhances β-oxidation in muscle cells. So, there might be an increased flux of fatty acids towards muscle cells and therefore a shift to fat oxidation. So, glycogen will be spared, which in turn may serve as a satiety signal as has been proposed by different researchers (Flatt, 1996; Melanson *et al*, 1999; Westerterp-Plantenga & Kovacs, 2002).

The aim of this study was to determine the effect of two dosages of CLA after weight loss on body-weight maintenance, appetite profile and energy intake at breakfast. We hypothesized that CLA supplementation might affect appetite profile, that is, reduced feeling of hunger and increased feelings of satiety and fullness, which in turn might lower the energy intake (EI) and therefore improve weight maintenance.

Research methods and procedures

Subjects

A total of 60 overweight men and women (BMI between 25 and 30 kg/m^2) aged between 20 and 50 y were recruited by advertisements in local newspapers and participated in this study. Selection was based upon being healthy and at least 3 months weight stable prior to the study, no use of any medication known to affect body weight and/or appetite, being nonsmoking, and at most moderate alcohol-users (max 10 glasses/week). Subjects had to be unrestrained eaters. The degree of dietary restraint was determined by the Three-Factor Eating Questionnaire (TFEQ, score F1$\leq$9) (Stunkard & Messick, 1985; Westerterp-Plantenga & Verwegen, 1999) and by the Herman/Polivy restraint questionnaire (HP, score$\leq$15) (Herman & Polivy, 1980). Height was measured using a wall-mounted stadiometer (Seca, model 220, Hamburg, Germany). Body weight (in underwear) was measured on a digital balance (Seca, model 707, Hamburg, Germany; weighing accuracy of 0.1 kg) in fasted state and after voiding the bladder. BMI was calculated as weight/(height^2). In all, 54 subjects completed the study. Six subjects dropped out for several reasons: one subject for illness not related to the treatment, one subject because of use of medication and four subjects because of motivation reasons. Of these, 27 subjects (15 women and 12 men) completed the low-dosage study and 27 (13 women and 14 men) subjects completed the high-dosage study. Table 1 shows the baseline characteristics of the subjects. There were no significant differences in the characteristics between CLA and placebo groups at baseline.

All subjects gave their written informed consent. The study was approved by the Medical Ethics Committee of Maastricht University.

Intervention protocol

The study had a randomized placebo-controlled and double-blind design.

Before the intervention period, all subjects were submitted to a 3-week very-low-calorie diet (VLCD, 2.1 MJ; Modifast, Novartis). Subjects replaced their habitual breakfast, lunch and dinner by three meals of Modifast. No snacks, except fruit or salad, (no dressing) were allowed. Drinks consisted of coffee, tea (without milk and sugar) and water were allowed *ad libitum*. After the 3 weeks on a VLCD, subjects started the intervention period. At that moment, subjects were randomly assigned to the low-dosage study or to the high-dosage study. In low-dosage study, subjects were randomized to 1.8 g CLA (Tonalin™ CLA 75% TG, Tonalin™, Hovdebygda, Norway) (three capsules/day with 600 mg CLA/capsule, $n=14$) or 1.8 g placebo (oleic acid, three capsules/day with 600 mg oleic acid/capsule, $n=13$) to be taken before breakfast, lunch and dinner. In the high-dosage study, subjects were randomized to 3.6 g CLA (six capsules/day with 600 mg CLA/capsule, $n=13$) or 3.6 g placebo (oleic acid, six capsules/day with 600 mg oleic acid/capsule, $n=14$) to be taken before breakfast, lunch and dinner. Additionally, all subjects of the high-dosage study were required to replace their habitual lunch by one meal of a protein-rich, low-energy supplement (0.7 MJ, 17.3 g protein) to prevent possible

Table 1 Baseline characteristics of the subjects in the conjugated linoleic acid (CLA) and placebo (oleic acid) supplementation group

	CLA (n=27)	*Placebo (n=27)*
Age (y)	39±7	37±9
Weight (kg)	85.1±8.0	82.7±9.0
BMI (kg/m^2)	27.8±1.6	27.8±1.4
Body fat (%)	31.3±7.5	33.0±7.0
F1[a]	5±3	5±2

[a]Factor 1 (cognitive restraint) of the TFEQ (Stunkard & Messick, 1985)

decrease of protein intake in case of decreased food intake. Since CLA appears to enhance fat-free mass (Park *et al*, 1997; DeLany *et al*, 1999; Park *et al*, 1999a, b; Blankson *et al*, 2000), an optimal supply of amino acids is desirable.

The duration of the intervention period in both studies was 13 weeks.

Test protocol

Body weight (in underwear, after voiding the bladder) was measured after an overnight fast before the VLCD (week −3), before (week 0), during (weeks 3 and 8) and at the end of the treatment period (week 13). During three of the visits (week −3, 0 and 13), a TFEQ (Stunkard & Messick, 1985) and a questionnaire for parameters of appetite were completed. The appetite profile was measured with the following questions using an anchored 100 mm visual analogue scale: 'How full are you?' 'How hungry are you?', and How satiated are you?'. Those questions were anchored with 'not at all' – 'extremely'.

EI during breakfast after an overnight fast, after ingesting the usual dosage CLA or placebo, was measured using the Universal Eating Monitor (Westerterp-Plantenga, 2000) on week −3, 0 and 13. At week −3, subjects could choose between fruit yogurt (3.2 kJ/g) and yogurt with cereals (5.35 kJ/g). The breakfast they had chosen had to be consumed also at week 0 and 13.

Tolerance

Tolerance of the capsules was determined at the end of the intervention period using a questionnaire on the occurrence of gastrointestinal and other complaints, and scored on a 5-point scale (0 = not at all, 1 = little, 2 = sometimes, 3 = relatively much, 4 = often).

Statistics

Differences between subjects of the CLA and placebo intervention groups for baseline characteristics were analyzed with an unpaired *t*-test (Statview SE Graphics™).

Changes in body weight, feelings of hunger, satiety and fullness, EI at breakfast, and Factors 1–3 of the TFEQ from week −3 to week 0 were tested with paired *t*-test (Statview SE Graphics™) for all subjects together.

The effect of CLA supplementation at week 13 for the dependent variables %body weight regain, feelings of hunger, satiety and fullness, EI at breakfast, and Factors 1–3 of the TFEQ were analyzed by linear multiple regression model with treatment (0 = placebo; 1 = CLA), gender (0 = men; 1 = women) and dosage (0 = LD, 1 = HD) as independent variables. Furthermore, for the dependent variables EI at breakfast and the factors of TFEQ, the values at week −3 and 0 of those parameters were also included in the model as independent variables. For the dependent variables satiety, fullness and hunger, the values at week −3 and 0 of those parameters and %regain of body weight were included in the model as independent variables (SPSS Inc. Chicago, IL, USA).

A possible relation between any of the parameters of appetite and EI at breakfast was tested with a simple linear regression (Statview SE Graphics™).

The regression coefficient (RC) with 95% confidence interval (CI) of the CLA intervention was calculated for each dependent variable. The level of significance is set at $P<0.05$. Data are presented as means and standard deviations.

Results

Tolerance

The occurrence of adverse events remained low and was not different between CLA and placebo intervention.

Body weight

As a consequence of the VLCD, body weight at week 0 was significantly lowered (Figure 1). The mean weight loss was 6.9 ± 1.7% from the original body weight.

After 13 weeks of intervention, the subjects of the CLA group had a regain of 40.2 ± 69.3%, while the placebo group

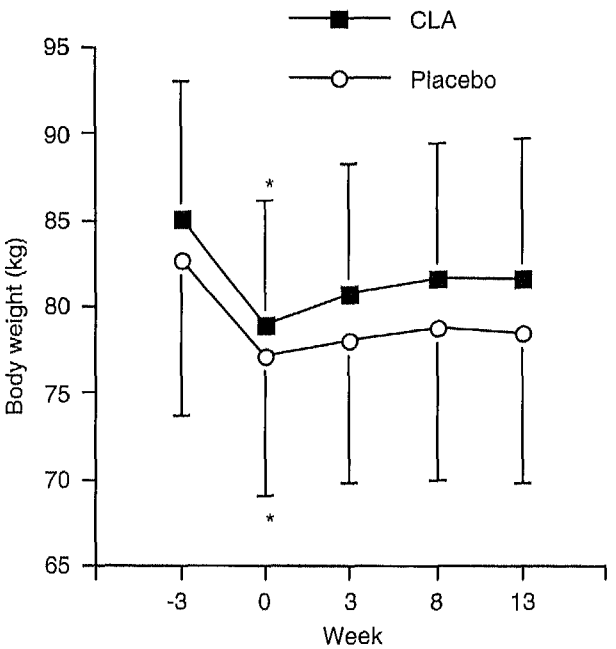

Figure 1 Body weight (kg) of subjects before VLCD (−3), after VLCD and before intervention (0), and at 3, 8 and 13 weeks of intervention with 1.8 or 3.6 g conjugated linoleic acid/day (CLA, $n=27$), and 1.8 and 3.6 g placebo/day (oleic acid, $n=27$). The results are presented as CLA and placebo, with the low and high dosage combined. * Repeated measures ANOVA for all subjects together showed a significant decrease in body weight from week −3 to week 0 ($P<0.0001$). Multiple regression showed that the body-weight regain was not affected by CLA supplementation (RC: 13.9; CI: −16.1 to 44.0, NS).

had a regain of 24.8±33.6% (NS). Thus, body weight regain during the intervention (week 13) was not influenced by CLA (RC 13.9; CI −16.1 to 44.0, NS). Moreover, both independent variables dosage (RC −11.5; CI −41.5 to 18.6, NS) and gender (RC −14.4; CI −44.5 to 15.7, NS) did not affect body weight regain.

Appetite profile

The feeling of fullness (Figure 2) and satiety (Figure 3) remained unchanged after the VLCD compared to before. CLA intake increased the feelings of fullness (RC 14.9; CI 3.46–26.4, $P<0.05$) as well as satiety (RC 12.2; CI 0.9–25.3, $P<0.05$) during the intervention. The rise in feelings of fullness was independent of %body weight regain (RC 0.01; CI −0.13 to 0.13, NS), dosage (RC −1.7; CI −13.2 to 9.8, NS) and gender (RC 0.2; CI −11.2 to 11.6, NS). Also, the increase in feelings of satiety was independent of % body-weight regain (RC −0.01; CI −0.14 to 0.13, NS), dosage (RC −6.5; CI −17.8 to 4.9, NS) and gender (RC −3.6; CI −14.6 to 7.3, NS).

The hunger level (Figure 4) was increased after the VLCD compared to before ($P<0.001$). The feeling of hunger was significantly decreased by CLA (RC −14.0; −25.0 to −3, $P<0.05$) during the intervention independent of % body weight regain (RC −0.05; CI −0.2 to 0.08, NS), dosage (RC 7.7; −3.3 to 18.7, NS) or gender (RC −5.1; −16.1 to 5.8, NS).

Energy intake

The EI at breakfast after VLCD was similar to before the diet (1.2±0.6 and 1.1±0.6 MJ, respectively; NS). CLA used for 13 weeks, as well as just before the test-breakfast, did not affect EI at breakfast at week 13 (CLA: 1.2±0.6 and placebo 1.1±0.6 MJ; RC: −0.07; CI: −0.3 to 0.1, NS). Moreover, dosage (RC 0.1; CI −0.1 to 0.3, NS) did not affect the EI at breakfast although a gender effect was observed (RC −0.2; CI −0.4 to −0.0, $P<0.05$).

There was no relation between any of the appetite scores and EI at breakfast at week −3 and 0. For the CLA as well as for the placebo groups, no relation between any parameters of appetite and EI at breakfast at week 13 was observed. Furthermore, there was no relation between change in any of the appetite scores from week 0 to 13 and change in EIs from week 0 to 13 for the CLA groups and placebo groups.

Three-factor eating questionnaire

All subjects were unrestrained eaters measured with Factor 1 of the TFEQ (cognitive restraint) at the start of the study

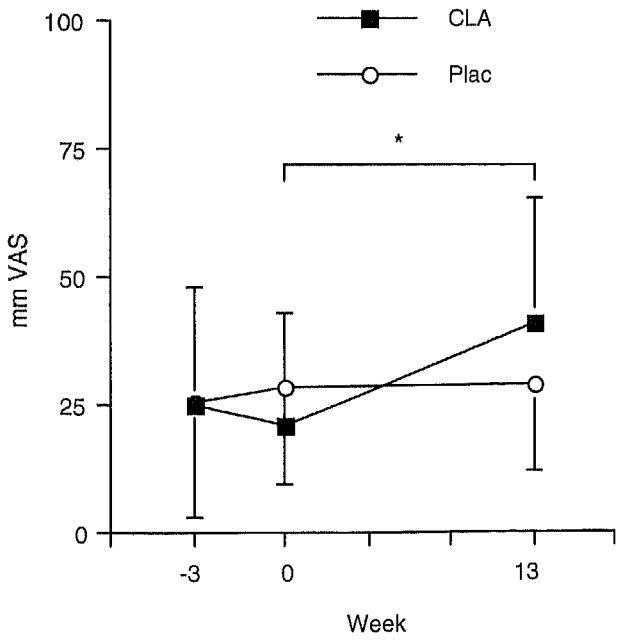

Figure 2 Feelings of fullness measured with an anchored 100 mm visual analogue scale (VAS) before VLCD (−3), after VLCD and before intervention (0), and at 13 weeks of intervention with 1.8 or 3.6 g conjugated linoleic acid/day (CLA, $n=27$) and 1.8 or 3.6 g placebo/day (oleic acid, $n=27$). The results are presented as CLA and placebo, with the low and high dosage combined. * Multiple regression showed that the feeling of fullness during intervention was increased by CLA compared to placebo (RC: 14.9 (3.46–26.4), $P<0.05$).

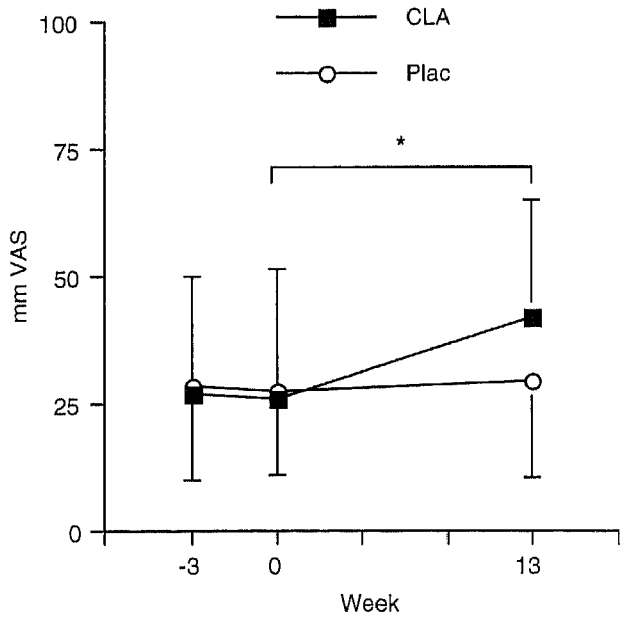

Figure 3 Feelings of satiety measured with an anchored 100 mm visual analogue scale (VAS) before VLCD (−3), after VLCD and before intervention (0), and at 13 weeks of intervention with 1.8 or 3.6 g conjugated linoleic acid/day (CLA, $n=27$) and 1.8 or 3.6 g placebo/day (oleic acid, $n=27$). The results are presented as CLA and placebo, with the low and high dosage combined. * Multiple regression showed that the feeling of satiety during intervention was increased by CLA compared to placebo (RC: 12.2 (0.9–23.5), $P<0.05$).

(Table 1). Cognitive restraint was significantly increased by VLCD ($P<0.01$), disinhibition (Factor 2 of TFEQ) significantly decreased disinhibition ($P<0.01$), while general hunger (Factor 3 of TFEQ) remained unchanged after the VLCD compared to before. Cognitive restraint, disinhibition and general hunger were not affected by the CLA intervention or by dosage or gender (Table 2).

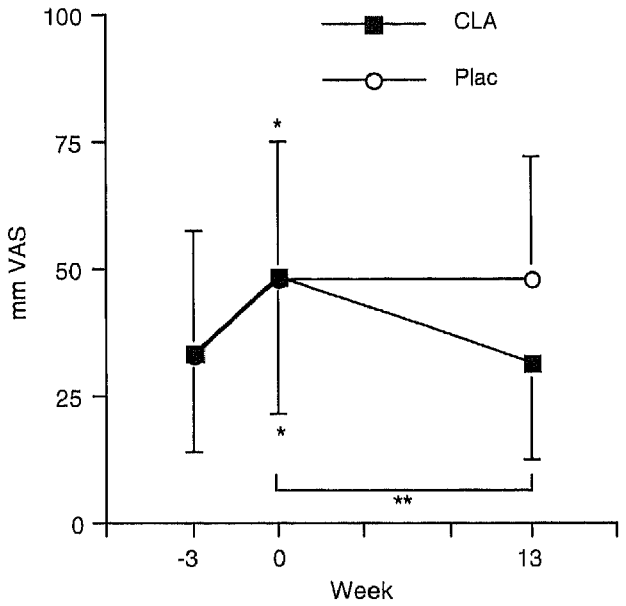

Figure 4 Feelings of hunger measured with an anchored 100 mm visual analogue scale (VAS) before VLCD (−3), after VLCD and before intervention (0), and at 13 weeks of intervention with 1.8 or 3.6 g conjugated linoleic acid/day (CLA, $n=27$) and 1.8 or 3.6 g placebo/day (oleic acid, $n=27$). The results are presented as CLA and placebo, with the low and high dosage combined. * Repeated measures ANOVA for all subjects together showed a significant increase in feelings of hunger from week −3 to week 0 ($P<0.001$). ** Multiple regression showed that the feeling of hunger during intervention was decreased by CLA compared to placebo (RC: −14.0 (−25.0 to −3), $P<0.05$).

Discussion

In the present study, the effect of CLA or placebo (oleic acid) after a 3-week VLCD on body-weight maintenance, parameters of appetite and EI was investigated. It was shown that a 13-week supplementation with CLA after body weight loss did not affect body-weight maintenance, but favorably altered the parameters of appetite compared to placebo independent of %body weight regain. We observed that feelings of fullness and satiety were increased by CLA ingestion compared to the placebo, while the feeling of hunger was decreased during the weight maintenance period. However, this did not result in a lower EI measured at breakfast as well as overall weight maintenance.

Previously, in two studies the effects of CLA on appetite were investigated, although both did not observe an effect compared to placebo. The mechanisms by which CLA might affect appetite as reported here are speculative. It is possible that the post-ingestive effects of CLA could have modulated satiety since it is known from *in vitro* studies that CLA reduces lipid uptake by adipose cells because of an effect on lipoprotein lipase (Park *et al*, 1997; Park *et al*, 1999a) and stearoyl-CoA desaturase (Choi *et al*, 2000; Pariza *et al*, 2001). As a result of a decreased uptake of fatty acids by adipocytes, there might be an increased flux of fatty acids to muscle cells. When the use of fatty acids is increased, less glucose is needed for combustion. So, glycogen will be spared, which in turn has been proposed to serve as a satiety signal (Flatt, 1996; Melanson *et al*, 1999; Westerterp-Plantenga & Kovacs, 2002). However, glycogen stores were not measured in the present study since a precise measurement of glycogen stores is not feasible in this kind of study.

Table 2 Cognitive restraint (Factor 1 of the TFEQ[a]), disinhibition (Factor 2 of the TFEQ[a]) and general hunger (Factor 3 of the TFEQ[a]) before the VLCD (−3), after VLCD but before intervention (week 0) and at the end of intervention (week 13) with conjugated linoleic acid (CLA, $n=27$) or placebo (oleic acid, $n=27$)

	CLA	*Placebo*	*CLA*[b]	*Dose*[b]	*Gender*[b]
F1 (−3)	5.0±2.8	4.9±2.1			
F1 (0)	5.2±2.7	6.5±2.7			
F1 (13)	5.6±2.8	6.9±3.0	−0.6 (−2.0 to 0.9)	0.7 (−0.7 to 2.1)	−0.1 (−1.5 to 1.3)
F2 (−3)	5.2±2.6	5.6±2.4			
F2 (0)	4.7±3.0	4.5±2.4			
F2 (13)	5.4±3.0	4.6±2.7	1.1 (−0 to 2.1)	−0.4 (−1.7 to 0.8)	−0.1 (−1.2 to 0.9)
F3 (−3)	4.0±3.1	3.9±2.8			
F3 (0)	3.1±2.8	3.3±3.3			
F3 (13)	3.2±2.4	3.2±2.9	0.1 (−1.1 to 1.3)	0.6 (−0.6 to 1.8)	−0.1 (−1.4 to 1.2)

[a]Three-Factor Eating Questionnaire (Stunkard & Messick, 1985).
[b]Multiple regression analysis. Regression coefficient (confidence interval).

Even though appetite was affected by CLA supplementation, this did not result in a decreased EI during breakfast. Also, 24 h EI seems not to be reduced, since body weight was similar in the CLA and placebo groups. Results on the effect of CLA on food intake in animals are controversial. In some studies, a lowered food intake by CLA has been observed (West *et al*, 1998; Park *et al*, 1999b; Miner *et al*, 2001), whereas in others CLA supplementation did not affect food intake (DeLany *et al*, 1999; Azain *et al*, 2000; West *et al*, 2000; Sisk *et al*, 2001). In our study, the appetite scores in the fasting state were not related to the energy consumed at breakfast. This implies that the improved parameters of appetite (lowered feeling of hunger, higher feeling of satiety and fullness) by CLA supplementation were not strong enough to lower EI compared to placebo. In fact, a relation between appetite parameters and subsequent EI is not always present (Mattes, 1990; Westerterp-Plantenga *et al*, 2001).

In this study, CLA did not enhance body-weight maintenance more than placebo. In animals, especially mice studies, the effects of CLA on body weight (Park *et al*, 1997; West *et al*, 1998, 2000; DeLany *et al*, 1999; Park *et al*, 1999a, b; Miner *et al*, 2001; Sisk *et al*, 2001) have been studied extensively. In humans, however, only a few studies have been conducted to study the effect of CLA supplementation and no study observed an effect on body weight. Blankson *et al* (2000) found that after 12 weeks, 1.7, 3.4, 5.1 or 6.8 g CLA/day did not affect body weight in overweight and obese subjects, although fat mass decreased with 1.7 and 5.1 g CLA/day. Also, in a study by Berven *et al* (2000) body weight remained unchanged. They showed that a daily consumption of 3.4 g CLA/day had no more effect on body weight, BMI or fat mass than placebo in obese subjects. Also, Zambell *et al* (2000) observed no effect of 3 g CLA/day on body weight or composition. Next to body weight and fat mass, Risérus *et al* (2001) studied the effect of 4.2 g CLA/day on abdominal obesity. Even though sagital abdominal diameter in obese men was lowered, body weight and fat mass were not affected by CLA. Owing to the action of CLA, that is, lowering fat uptake by adipose cells because of a lower lipoprotein lipase activity, it seemed to be of interest to investigate the effects of CLA on weight (re)gain. However, neither 1.8 nor 3.6 g CLA/day improved body-weight maintenance more than placebo, even when protein intake was supported as was done in the high-dosage study in the CLA as well as in the placebo group. However, CLA supplementation caused changes in body composition, that is, increase of fat-free mass (Kamphuis *et al*, submitted). Differences between humans and animals for the effect of CLA on body composition might be because of several factors, for example, dosage or length of intervention, although a recent publication suggests that differences in metabolic rate might be of more importance than other factors (Terpstra, 2001).

In the present study, subjects received either 1.8 or 3.6 g CLA/day, while in previous studies doses between 1.7 and 6.8 g CLA/day were used. A dosage as low as 1.7 g CLA/day was effective in lowering fat mass (Blankson *et al*, 2000), whereas in the present study a dose of 1.8 g CLA/day was effective in improving the appetite profile, but not energy intake at breakfast. Since the habitual intake of CLA was assessed between 0.1 and 0.3 g/day (Ens *et al*, 2001), even a high intake of milk, milk products or beef by a subject will most likely not have confounded the results of the present study.

There is growing evidence that the different isomers of CLA (c9,t11 and t10,c12) might have different effects (Park *et al*, 1999a; Choi *et al*, 2000; Halvorsen *et al*, 2000; Pariza *et al*, 2001). The c9,t11 isomer is the principal dietary form of CLA (80–90%) and seems to be the most active, because of its abundance and incorporation into membranes, but the t10,c12 isomer seems to be the most important in energy metabolism (Pariza *et al*, 2001). In our study, a mixture of equal amounts of both isomers was used, so the effects of this study could result from either or both isomers.

During the study, subjects completed three times a TFEQ to measure dietary restraint. At the start of the study, all subjects were unrestrained eaters measured with Factor 1 of the TFEQ, but dietary restraint increased by VLCD presumably since subjects are being forced to restrain their eating. Disinhibition (Factor 2 of TFEQ) decreased after VLCD compared to before. A possible explanation for this decrease might be that disinhibited eating is removed by the study. Factor 3 (hunger) remained unchanged, although short-term hunger feelings were increased after VLCD. The CLA and placebo interventions did not show to have an effect on any factor of the TFEQ. This implies that the effects observed are because of physiological effects rather than cognitive behavior.

In the present study, subjects of the high-dosage groups were asked to replace their habitual lunch by one meal of the VLCD to increase protein supply. It has been shown that a single meal replacement can be a tool for weight management (Ashley *et al*, 2001), however, this seems not to be the case in the present study. The increase in dietary restraint of the subjects of the low-dosage interventions compared to the high-dosage interventions was similar. Furthermore, when the average increase in energy intake above energy requirement during the weight regain period was calculated, there were no differences between subjects of the low-dosage intervention, who did not replace their lunch and subjects of the high-dosage intervention, who replaced their lunch (0.8 and 0.5 MJ, respectively). This means that the impact of one meal replacement was not significantly present during the weight regain period of this study.

In summary, 13-week supplementation with 1.8 or 3.6 g CLA/day after a 3-week VLCD was not effective in reducing EI at breakfast or improving body-weight maintenance compared to placebo (1.8 or 3.6 g oleic acid/day), but affected dose-independently parameters of appetite. CLA supplementation increased the feelings of fullness and satiety, and decreased the feeling of hunger.

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