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Effect of dietary conjugated linoleic acid on body composition and energy balance in broiler chickens

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The effect of dietary conjugated linoleic acid (CLA) on body composition and energy metabolism was investigated in broiler chickens. Male broiler chicks were assigned to receive either a control diet (1 % sunflower oil) or a diet containing CLA (1 % of a 1:1 mixture of *trans*-10, *cis*-12 and *cis*-9, *trans*-11 isomers of octadecadienoic acid). The diets were fed *ad libitum* for 3 weeks and there were eight replicates per diet, each replicate including four chickens so that each treatment had thirty-two animals. The proportion of body fat was lower in the control group than in the CLA group. No significant differences as to the proportions of body water, ash and protein were observed. Feed and energy intake were significantly lower in CLA-fed birds. The percentage of ingested energy lost in excreta was higher after CLA feeding and heat expenditure as a percentage of ingested energy was lower in the CLA-fed group. The CLA-fed group showed a higher percentage of SFA and lower percentages of MUFA and PUFA in carcass fat. It is concluded that CLA stimulated *de novo* fatty acid synthesis and lowered desaturase activity.

Conjugated linoleic acid: Body composition: Energy metabolism: Broilers

The term conjugated linoleic acid (CLA) is used to designate a mixture of positional and geometric isomers of linoleic acid in which the double bonds are conjugated. Considerable attention has been paid to the potential, beneficial health effects of dietary CLA. CLA was found to act as a growth factor[1], as a fat-to-lean repartitioning agent[2–7], and it has anticarcinogenic[8,9], hypocholesterolaemic and antiatherogenic[10,11] properties. Food intake is usually not affected by incorporation of CLA in the diets and, therefore, the body fat-lowering effect of CLA is most likely mediated by enhanced energy expenditure. Measuring the energy expenditure of mice in metabolic chambers fed CLA indeed demonstrated an increase of energy expenditure[12].

Szymczyk *et al.*[13] showed that feeding CLA to broiler chickens resulted in subtantial incorporaton of CLA isomers into their tissue lipids, thus providing a potential CLA-rich source for human consumption. In their study, feeding CLA significantly decreased feed intake during the starter (8–21 d) period, but no effect was noted during the grower–finisher (22–42 d) period. Abdominal fat deposition was significantly reduced whereas the relative proportion of breast muscles was unaffected and that of leg muscles significantly increased[13]. It could be suggested that CLA feeding influences body composition and energy metabolism of broiler chickens.

The objective of the present study was to test whether the earlier observed CLA-induced reduction of abdominal fat in chickens[13] is associated with enhanced energy expenditure by investigating the influence of dietary CLA on growth, body composition and energy balance in broilers. In addition, the fatty acid composition of total carcass lipid was evaluated.

Experimental methods

The experimental protocol was approved by the animal experiments committee of the Faculty of Veterinary Medicine, Utrecht University, The Netherlands.

Animals and diets

One-day-old male broiler chickens (Ross 308) were purchased from a local hatchery. On arrival, they were wing-banded, weighed and housed in wire-floored, suspended cages. The temperature of the animal house was controlled and continuous lighting used throughout the entire experimental period. There were two dietary treatments, each consisting of eight replicates. A replicate was identical to a cage with four birds so that each treatment had thirty-two animals. Ten birds were killed at the beginning of the study to determine pre-experimental body composition. Sixty-four broilers were used for the feeding trial. The base diet was in pelleted form (Table 1) and fed for 7 d. To produce the experimental

Abbreviation: CLA, conjugated linoleic acid.

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Table 1. Composition of the diets (g/kg)†

Ingredients	Control diet	CLA-containing diet
Wheat (+ xylanase)	250·0	250·0
Maize	321·0	321·0
Soyabean meal (47·6 % cp)	225·0	225·0
Peas	50·0	50·0
Sunflower meal (32·0 % cp)	40·0	40·0
Potato protein	15·0	15·0
Fish meal (72·0 % cp)	25·0	25·0
Sunflower oil	10·0	–
CLA	–	10·0
Soya oil	30·0	30·0
Premix‡	5·0	5·0
Limestone	16·0	16·0
Monocalcium phosphate	7·0	7·0
Natuphos 5000G (phytase)	0·1	0·1
Salt	1·7	1·7
Sodium bicarbonate	1·7	1·7
L-Lysine HCl	0·8	0·8
DL-Methionine	1·7	1·7

cp, crude protein.
† The diets were prepared by Research Diets Services (Wijk bij Duurstede, The Netherlands).
‡ The 5 g Premix consisted of 12 000 IU vitamin A (4·1 mg retinol acetate); 2400 IU vitamin D_3 (0·06 mg cholecalciferol); 30 mg vitamin E; 1·5 mg vitamin K_3; 2·0 mg vitamin B_1; 7·5 mg vitamin B_2; 3·5 mg vitamin B6; 20 μg vitamin B_{12}; 35 mg niacin; 10 mg D-pantothenate; 460 mg choline chloride; 1·0 mg folic acid; 0·2 mg biotin; 80 mg Fe; 12 mg Cu; 85 mg Mn; 60 mg Zn; 0·4 mg Co; 0·8 mg I; 0·1 mg Se; 200 mg Ca; 125 mg anti-oxidant (Oxytrap PXN).

diet, sunflower oil was replaced by 1 g CLA per 100 g diet. The fatty acid composition of the diets is given in Table 2. CLA was purchased from Lipid Nutrition B.V. (Wormerveer, The Netherlands). It came in the form of a Clarinol G-80™ preparation that contained 79·5 % of CLA as TAG. The CLA preparation consisted of *cis*-9, *trans*-11 and *trans*-10, *cis*-12 CLA in equal amounts. The birds were fed the experimental diet for a period of 21 d. Feed and water were provided *ad libitum*. Individual body weight and feed intake per replicate were monitored weekly. From day 7 on, excreta were collected quantitatively.

Carcass analysis

At the end of the experiment, the birds were weighed and killed by cervical dislocation. Carcasses were cut in pieces and ground (Retsch, SM 2000, Haan, Germany) and the carcasses for each replicate were mixed, sampled, weighed and then dried in a forced-hot air oven at 60°C for a period of 3 d. The dried carcasses were weighed again and the percentage of water was calculated. Subsequently, the dried carcasses were ground in a coffee grinder and the homogenised samples were stored in plastic containers until analysed. The excreta were collected during the experimental period of 3 weeks and were also dried for a period of 3 d and homogenised in a coffee grinder.

Total lipids in the dried, homogenised carcasses and excreta were extracted as described previously[14]. Total lipids were saponified and methylated according to Metcalfe *et al.*[15] followed by GLC for determination of the fatty acid composition of feed and carcasses. The protein content of the dried carcasses was determined with the macro Kjeldahl method[16]. For the determination of the ash content, about 0·5 g dried, homogenised carcass was added to a small porcelain crucible and put in an oven that was programmed as follows: 1 h at 200°C, 2 h at 300°C, 3 h at 400°C and 10 h at 500°C[17].

Table 2. Selected fatty acids (% of FAME) in the diets†

	Control	CLA
C16: 0	12·13	12·02
C18: 0	3·59	3·26
C18: 1-*n*-9	21·39	19·08
C18: 1-*n*-7	1·2	1·2
C18: 2-*n*-6	51·86	44·04
C18: 3-*n*-3	4·28	4·31
CLA	–	9·27
Other	5·6	6·8
ΣSFA	17·2	16·7
ΣMUFA	23·3	21.0
ΣPUFA-*n*-6	51·9	44·1
ΣPUFA-*n*-3	5·1	5·2
ΣPUFA total	57.0	49·3

† Total fat content of the diets: 7·53 and 7·32 % for the control and the CLA-containing diet, respectively.
CLA, conjugated linoleic acid.

Bomb calorimetry

The gross energy content in dried, homogenised carcasses, faeces, diets and oils was determined with a bomb calorimeter (IKA Calorimeter C4000 Adiabatic, IKA Analystechnik, Heitersheim, Germany). As a thermochemical standard benzoic acid (BDH Ltd, Poole, UK) was used[18]. The total amount of energy that was lost as heat (heat production or energy expenditure) was calculated with the formula: energy lost as heat = energy in food − energy in excreta − energy stored in body. Energy stored in the body was determined as total energy at the end of the 21 d feeding period minus energy in the body at the beginning (= mean body weight × energy content) of the 21 d feeding period. The same procedure was used to calculate the retention of water, protein, fat and ash.

Statistical analysis

Four birds in a cage were considered as one experimental unit. This resulted in eight experimental units per dietary treatment. Mean data per cage were used in a one-way ANOVA with diet (sunflower oil *v.* CLA) as an independent variable. The level of statistical significance was preset at $P<0·05$.

Results

Body weight and body composition

Food intake was lower in CLA-fed birds than in controls, the lowering almost reaching statistical significance (Table 3). There was no difference in body weight gain and feed conversion rate between CLA-fed birds and controls. The proportion of body fat was higher in the CLA-fed group than in the control group ($P=0·044$). There were no differences in the proportions of body water, protein and ash between the two groups.

Table 3. Body composition and energy balance in broiler chickens fed the control diet or a conjugated linoleic acid (CLA)-containing diet for 21 d

(Mean values with their standard errors for eight units with each unit including four birds)

	CLA		Control			
Parameter	Mean	SE	Mean	SE	SEM (pooled)	*P* value
Feed intake (g/21 d)	1769	81·5	1897	149·1	42·4	0·051
Feed conversion (g feed/g weight gain)	1·44	0·033	1·48	0·078	0·02	0·133
Initial body weight (g)	144·3	7·5	140·4	9·4	3·0	0·372
Final body weight (g)	1376	62·0	1418	57·7	21·2	0·191
Weight gain in 21 d (g)	1232	63·4	1277	56·5	21·2	0·156
Apparent fat digestibility (%)	75·93	3·43	79·65	2·44	1·05	0·026
Energy metabolisability (%)	72·57	2·19	75·70	1·17	0·62	0·003
Body composition after 21 d						
Fat (g)	161·71	13·27	152·2	13·16	4·67	0·171
Water (g)	923	58·5	962	47·3	18·8	0·166
Protein (g)	237·0	11·72	241·7	10·65	3·96	0·415
Ash (g)	32·99	3·998	36·33	3·906	1·40	0·114
Fat (%)	11·76	1·07	10·73	0·78	0·33	0·044
Water (%)	67·03	1·67	67·86	1·07	0·50	0·258
Protein (%)	17·24	1·07	17·05	0·30	0·28	0·634
Ash (%)	2·41	0·35	2·57	0·30	0·12	0·345
Deposition in body						
Fat (g/21 d)	150·9	13·26	141·6	13·16	4·67	0·183
Water (g/21 d)	815	58·96	856	47·96	19·0	0·141
Protein (g/21 d)	218·6	12·12	223·8	10·38	4·01	0·375
Ash (g/21 d)	30·10	3·995	33·51	3·846	1·39	0·104
Energy (kJ/21 d)	11 186	750·6	10 941	675·0	252·4	0·504
Energy balance						
Intake (MJ/21 d)	30·06	1·37	32·22	2·53	1·02	0·052
Retained in body (MJ/21 d)	11·18	0·75	10·94	0·67	0·25	0·504
Excreta (MJ/21 d)	8·23	0·557	7·83	0·654	0·22	0·204
Heat production (MJ/21 d)	10·65	1·25	13·46	1·68	0·52	0·002

Energy balance

Energy intake was lower in CLA-fed birds, lowering almost reaching statistical significance (Table 3). Apparent fat digestibility and energy metabolisability were higher in the control group ($P=0·026$ and 0·003, respectively). Energy expenditure was calculated as the difference between the energy intake and the energy stored and excreted in the excreta. The higher heat production calculated for the control group differed from that for the CLA group ($P=0·002$). Energy storage was not affected by CLA feeding. The proportion of energy intake that was stored in the body was lower in controls than in the CLA-fed group (0·34 (SEM 0·02) and 0·37 (SEM 0·02), respectively; $P=0·007$).

Feed and carcass fatty acid composition and feed efficiency

As CLA was added to the experimental diet at the expense of sunflower oil, the ingested amounts of SFA, MUFA, PUFA-*n*-6 and PUFA-*n*-3 dropped (Table 4). The amounts of fatty acid

Table 4. Selected fatty acids as ingested in broiler chickens fed a control or a conjugated linoleic acid (CLA)-containing diet for a period of 21 d (g/21 d)

(Mean values with their standard errors for eight units with each unit including four birds)

	CLA		Control			
Fatty acid†	Mean	SE	Mean	SE	SEM	*P* value
C16: 0	14·69	0·673	16·46	1·294	0·4	0·006
C18: 0	4·01	0·1827	4·876	0·3832	0·1	<0·001
C18: 1*n*-9	23·48	1·069	29·02	2·281	0·63	<0·001
C18: 2*n*-6	54·2	2·466	70·4	5·53	1·51	<0·001
C18: 3*n*-3	5·305	0·2415	5·808	0·4565	0·1291	0·016
ΣSFA	20·48	0·932	23·39	1·838	0·515	0·001
ΣMUFA	25·86	1·177	31·61	2·484	0·687	<0·001
ΣPUFA-*n*-6	54·26	2·469	70·45	5·537	1·52	<0·001
ΣPUFA-*n*-3	6·36	0·289	6·95	0·5466	0·155	0·017
ΣCLA	11·398	0·519	–			
ΣPUFA total	60·62	2·759	77·40	6·083	1·67	0·001

† Total fatty acid contents were calculated as follows: total fat measured × 0·95 × percentage of selected fatty acid.

Table 5. Selected fatty acids stored in the body of broiler chickens fed a control or a conjugated linoleic acid (CLA)-containing diet for a period of 21 d (g/21 d)

(Mean values with their standard errors for eight units with each unit including four birds)

	CLA		Control			
Fatty acid†	Mean	SE	Mean	SE	SEM	*P* value
C16: 0	42·48	3·33	24·91	2·96	1·10	<0·001
C18: 0	19·62	1·57	8·36	0·54	0·40	<0·001
C18: 1*n*-9	36·37	3·23	45·19	5·80	1·66	0·002
ΣSFA	64·52	4·99	34·73	3·42	1·51	<0·001
ΣMUFA	43·01	3·89	53·23	7·17	2·04	0·003
ΣPUFA-*n*-6	26·33	9·45	41·26	2·62	2·45	<0·001
ΣPUFA-*n*-3	2·20	1·06	3·36	0·25	0·27	0·009
ΣCLA	2·70	1·53	0	0	0·38	<0·001
ΣPUFA total	28·53	10·50	44·61	2·83	2·72	<0·001

† Total fatty acid contents were calculated as follows: total fat measured × 0·95 × percentage of selected fatty acid.

stored in the carcasses are shown in Table 5. The amount of SFA in carcasses was increased in the CLA-fed group ($P<0·001$) and the amount of MUFA and PUFA were decreased ($P=0·003$ and $P<0·001$, respectively).

CLA consumption markedly increased the efficiency of incorporation (fatty acid deposited/fatty acid ingested) of SFA and decreased the incorporation of PUFA-*n*-3 (Table 6). Taking into account the amounts of fatty acids in the body at the start of the experiment, the ingested amounts of fatty acids and the amounts of fatty acids at the end of the experiment, one can estimate the minimal rate of *de novo* fatty acid synthesis during 21 d or the maximal rate of fatty acid degradation/disappearance in that period. The data indicated that CLA feeding preferentially induced SFA synthesis and that degradation/disappearance of PUFA is unaffected (Table 6).

Discussion

The effect of CLA on body composition and energy expenditure was studied in broiler chickens fed 1 g CLA/100 g diet. CLA feeding depressed feed intake, fat digestibility and energy metabolisability. This must result in a lower amount of metabolisable energy in the CLA treated group. However, weight gain during the experimental period did not differ between the dietary treatments. Moreover, deposition of fat, water, protein, ash and energy was not different (Table 3). CLA feeding had no negative effect on body fat deposition. Feed conversion was non-significantly lower in the CLA-fed birds, which is consistent with the finding by Szymczyk *et al.*[13]. The fat proportion, however, was higher in the body of birds fed CLA when compared to controls. This result is consistent with the finding by Du & Ahn[19] who found that feeding a diet containing 0·5 % CLA to broilers at 3 weeks of age, for a period of 3 weeks, resulted in an increase in abdominal fat content. Several studies have shown that incorporation of 1 % or less CLA in the diet can substantially reduce the proportion of body fat in mice[7,12,20], rats[21,22], chickens[13] and man[23,24]. The effects in mice appear more striking than in other species[25]. Badinga *et al.*[26] found that feeding CLA at the level of 5 % to 1-d-old broiler chickens for a period of 21 d significantly lowered the proportion of body fat and increased the proportion of body water. Szymczyk *et al.*[13] found lower abdominal fat in the body when they fed birds a diet with 1 % CLA. As mentioned earlier, Du & Ahn[19] observed an increase in abdominal fat in broilers fed CLA. Thus, experimental conditions such as age, genotype and metabolic status of the animal, as well as the level, the type of isomer and duration of CLA treatment may play an integral role in how CLA affects body composition. The lack of agreement between previous works suggests mechanisms involved are complicated and multiple.

Table 6. Minimum rate of *de novo* fatty acid synthesis, maximum rate of fatty acid disappearance and efficiency of incorporation of selected fatty acids in the body of broiler chickens fed a control diet or a conjugated linoleic acid (CLA)-containing diet for a period of 21 d

(Mean values for eight determinations per treatment group)

Fatty acid	Diet	Intake (g/21 d)	Retained (g/21 d)	Efficiency†	Minimum synthesis‡ (g/21 d)	Maximum disappearance§ (g/21 d)
C16: 0	CLA	14·7**	42·5***	2·87***	27·8***	
	Control	16·5	24·9	1·52	8·4	
C18: 0	CLA	4·0***	19·6***	4·9***	15·6***	
	Control	4·9	8·4	1·72	3·5	
C18: 1*n*-9	CLA	23·5***	36·4**	1·55	12·9	
	Control	29·0	45·2	1·56	16·2	
ΣSFA	CLA	20·5***	64·5***	3·15***	44·0***	
	Control	23·4	34·7	1·49	11·3	
ΣMUFA	CLA	25·9***	43·0**	1·66	17·1	
	Control	31·6	53·2	1·68	21·6	
ΣPUFA-*n*-6	CLA	54·3***	26·3***	0·48		28·0
	Control	70·5	41·3	0·59		29·2
ΣPUFA-*n*-3	CLA	6·40*	2·2**	0·35*		4·2
	Control	6·95	3·4	0·48		3·6
ΣPUFA total	CLA	60·6***	28·5***	0·47		32·1
	Control	77·4	44·6	0·58		32·8

Mean values were significantly different from those of the control diet: *$P<0·05$, **$P<0·01$, ***$P<0·001$.

† Efficiency is expressed as the ratio of fatty acid deposited in the body and dietary fatty acid.

‡ The values for the minimum amount of fatty acid synthesised are obtained by subtracting the amount of intake from the amount of retained.

§ The values for the maximum amount of fatty acid disappearing are obtained by subtracting the amount of retained from the amount of intake.

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Much to our surprise, the energy balance indicated that the calculated heat production was about 20 % lower in CLA-fed birds compared to the controls. This is opposite to what happens in mice after CLA consumption[7]. The present study does not give any information on the mechanism responsible for the decrease in energy expenditure in broilers fed CLA. However, it is tempting to speculate that the decrease in energy expenditure is related to the increase in the proportion of body fat. Another possibility is an effect of CLA on non-shivering thermogenesis, which is quite different in birds as compared to mammals where brown adipose tissue is the site for non-shivering thermogenesis. Birds lack brown adipose tissue[27].

Lee *et al.*[28] observed that CLA has the ability to alter the fatty acid composition of tissues by reducing the levels of MUFA which is consistent with the present findings. Choi *et al.*[29] observed that the ratio of SFA to MUFA in mice fed CLA was increased and indicated that this was related to a lipid-lowering effect of CLA. Studies in rats[30,31] and chickens[13,32] have shown that the percentage of SFA in the body increases whereas those of MUFA and PUFA decrease. The same was true for egg yolks of eggs produced by hens fed CLA[33,34]. In the present study, we also found a marked increase in SFA, but no lipid-lowering effect of CLA was observed. Carcasses of rats fed CLA also contained a higher proportion of SFA and less PUFA[35]. The reduction in MUFA (oleic acid) may be the result of a reduced Δ-9 desaturase activity due to feeding CLA[36–38]. The arachidonic acid concentration decreased in the carcasses. The present results are consistent with those of Belury & Kempa-Steczko[39] who proposed that CLA, acting as a substrate for Δ-6 desaturase, inhibited the conversion of linoleic acid into arachidonic acid. Consistent with the present observations is the finding that CLA dramatically reduced the percentages of MUFA in all tissues investigated through inhibition of Δ-9 desaturase[29,36,40,41]. The *trans*-10, *cis*-12 CLA isomer has been shown to have the highest biological activity in this respect, whereas *cis*-9, *trans*-11 CLA does not reduce the activity of Δ-9 desaturase[40,42].

The changes in fatty acid composition greatly increase the melting point for fat retained in the CLA-fed group (from 21 to around 35°C). What kind of effects this will trigger in the broilers is unknown yet. A similar change in fatty acid composition results in complete loss of hatchability of eggs from CLA-fed chickens[33,34]. In the broilers such a change in fatty acid composition makes chicken meat harder and drier[19].

When we calculate the amounts of fatty acids stored in the body during the experimental period and also the amounts of fatty acids ingested, we can determine the efficiency of fatty acid incorporation into the body. Calculation revealed a dramatically higher efficiency for SFA and a lower efficiency for PUFA-*n*-3 (Table 6). The CLA-induced differences in efficiency of incorporation might be related to preferential effects on synthesis or degradation of certain fatty acids. If the incorporation ratio was higher than 1·0, then the minimum amount of *de novo* synthesis of a specific fatty acid was calculated as deposited amount (g/21 d) minus the ingested amount (g/21 d). If the incorporation ratio was lower than 1·0, then the maximum amount of oxidation (or degradation) of a specific fatty acid was calculated as the ingested amount (g/21 d) minus the deposited amount (g/21 d). Both calculations can only indicate the lower and upper limit, respectively, because actual information about digestibility of individual fatty acids and the efficiency of incorporation of dietary fatty acids in deposited fatty acids is not available in the present experiment. The calculations show that CLA feeding preferentially induced SFA synthesis. This may explain the CLA-induced increase in body fat, which was statistically significant when expressed as percentage of the body. Much to our surprise the oxidation of PUFA was unaffected. This is contrary to many observations indicating preferential oxidation of PUFA[39,43,44]. In contrast, some studies have shown that CLA may have a modest enhancing effect on the level of PUFA[45,46]. Yet other studies, like the present one, show no effect of CLA on PUFA levels[31,47–49]. It appears that the ability of CLA to alter PUFA levels is tissue and species dependent. Consistent with the present results on fatty acid synthesis and degradation are our earlier observations showing CLA-induced activity of the lipogenic pathway in mice as evidenced by enhanced activities of acetyl-CoA carboxylase and fatty acid synthase[50]. In that same study it was shown that CLA did not alter the activities of 3-hydroxy-acyl-CoA dehydrogenase and citrate synthase, suggesting that fatty acid oxidation was not affected by CLA feeding[50].

It might be argued that differences in fatty acid composition between the control and CLA-containing diet may have caused differences in fatty acid deposition. There are indeed differences in fatty acid intake between the two experimental diets, as can be seen from Table 2 with the analysed fatty acid composition of the experimental diets. These differences are due to the fact that CLA was added at the expense of sunflower oil. The differences are minor except for linoleic acid. However, earlier work[14] indicates that the difference in linoleic acid intake cannot have caused the diet effects observed in the present study. The difference in ingested fatty acids as calculated in Table 4 is mainly caused by the difference in feed intake between the treatments.

Several studies have shown that the specific mechanisms by which dietary CLA reduces the body fat content are likely to vary from one animal species to another. Whether reduced accumulation of liver lipid in broilers fed CLA as observed by Badinga *et al.*[26] reflected enhanced β-oxidation or reduced *de novo* lipid synthesis warrants further investigation, but the present observation indicates higher *de novo* synthesis and lower desaturase activity. Measurement of enzyme expression and/or activity would complement the present data.

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