Dietary Conjugated Linoleic Acid Consumption during Pregnancy and Lactation Influences Growth and Tissue Composition in Weaned Pigs

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ABSTRACT We evaluated the effects of conjugated linoleic acid (CLA) on growth performance, tissue fatty acid composition and ex vivo lipogenic enzyme activity in piglets ($n = 40$) reared on sows fed diets supplemented with CLA or linoleic acid (LA). Weaned offspring of both sow groups were offered either a CLA- or LA-enriched starter diet for 35 d. The starter diets were formulated to contain 2 g CLA (containing 58.9 g CLA/100 g total fatty acids) or LA per 100 g feed. All piglets were slaughtered at 70 d of age and tissue samples of the back fat, omental fat and longissimus dorsi were collected. Irrespective of the dietary fat supplied in the starter period, piglets reared on the CLA sows had greater final body and warm carcass weights ($P < 0.01$), and greater feed intake ($P = 0.02$) than piglets reared on the LA sows. The dietary effect on the fatty acid composition was similar for the adipose and muscle tissues. Compared with the LA-enriched diets, CLA increased the level of total saturated fatty acids ($P < 0.05$), whereas that of monounsaturated fatty acids was decreased ($P < 0.05$). Dietary CLA increased glucose-6-phosphate dehydrogenase ($P < 0.01$) and malic enzyme activities ($P < 0.06$) in the fat tissues, but did not affect fatty acid synthase activity. The shift toward a higher deposition of saturated fatty acids and a lower deposition of monounsaturated fatty acids is the result of down-regulation of Δ9-desaturase activity that was induced by CLA rather than an altered rate of de novo synthesis. J. Nutr. 130: 2981–2989, 2000.

KEY WORDS: • *piglets* • *conjugated linoleic acids* • *adipose tissue* • *lipogenesis*

Conjugated linoleic acids (CLA)[1] refer to a group of octadecadienoate isomers with a set of conjugated diene double bonds and a combination of *cis* and/or *trans* spatial configurations. They have been reported to profoundly affect lipid metabolism (Belury and Kempa-Steczko 1997, Chin et al. 1994, West et al. 1998) and to act as a repartitioning agent in growing-finishing pigs (Dugan et al. 1997, Ostrowska et al. 1999). Dietary CLA infused into the abomasum of dairy cows has been reported to increase CLA content of milk fat, to alter milk fatty acid composition and to markedly depress the total content and yield of milk fat (Chouinard et al. 1999). Furthermore, CLA infusion dramatically reduced palmitic, linoleic and arachidonic acids and increased stearic acid content of cow's milk (Loor and Herbein 1998). Results of a recent study identified *t*10,*c*12–18:2 as the CLA isomer responsible for the inhibition of milk fat synthesis by reducing de novo lipid synthesis (Baumgard et al. 2000).

Neonatal growth in pigs depends primarily on milk produced by the sow as the sole source of energy during early development. Piglets are born with extremely little body fat (Le Dividich et al. 1994) and low reserves of stored glycogen (Boyd et al. 1978); thus, sufficient energy uptake after birth is important. Suckling newborn piglets are unable to synthesize fatty acids from carbohydrate (Le Dividich et al. 1994); deposition of body fat depends in large part on the amount of fat intake (Le Dividich et al. 1997). The most promising strategy to improve neonatal piglet growth rate is to increase the fat content of the maternal milk (Averette et al. 1999). Both the yield and lipid composition of the colostral and mature milk can be manipulated by the amount and origin of dietary fat provided to pregnant and lactating sows (Fritsche et al. 1993). In agreement with the latter studies, we reported that compared with a control treatment (linoleic acid), dietary CLA in the sow lactation diet significantly increased the level of saturated fatty acids and decreased that of monounsaturated fatty acids in the milk lipids (Bee 2000). In addition, dietary CLA isomers (*c*9,*t*11; *t*10,*c*12; *t*9,*t*11/*t*10,*t*12; *c*9,*c*11) were excreted in the milk and, therefore, were available to the suckling pigs. The question arises whether the modified fatty acid composition and the amount of CLA in the milk lipids affect growth performance and tissue composition of neonatal pigs. In view of the results reported by Chin et al. (1994), indicating the possibility that CLA may act as a growth factor on development and growth of rats, the objective of this study was to establish, by feeding CLA to sows during pregnancy and lactation and further offering CLA in a starter diet after weaning, to what extent growth and fat metabolism of the piglets were influenced.

[1] Abbreviations used: CC, pigs reared on sows fed the CLA diet and then fed the CLA starter diet; CL, pigs reared on sows fed the CLA diet and then fed the LA starter diet; CLA, conjugated linoleic acids; FAME, fatty acid methyl esters; FAS, fatty acid synthase; G6PDH, glucose-6-phosphate dehydrogenase; LA, linoleic acid–enriched oil; LC, pigs reared on sows fed the LA diet and then fed the CLA starter diet; LL, pigs reared on sows fed the LA diet and then fed the LA starter diet; ME, malic enzyme; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids.

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MATERIALS AND METHODS

Animals and experimental diets. The 40 piglets used in this study were progeny of multiparous Swiss Large White sows that were fed from the day of mating, during pregnancy and lactation either a diet supplemented with 2 g of oil enriched with linoleic acid per 100 g of diet (LA; $n = 4$) or a diet supplemented with 2 g of oil enriched with a commercially available CLA per 100 g of diet (CLA; $n = 6$; SELIN-CLA, Grünau Jllertissen GmbH, Jllertissen, Germany). The linoleic acid–enriched oil, derived from sunflower oil, served as source material to produce CLA (Grünau Jllertissen GmbH). Details regarding the housing, nutrition, management and experimental conditions of sows are described elsewhere (Bee 2000).

Two females and two castrates from each of 10 litters were selected on the basis of the mean body weight of the litter at d 35 of lactation. After 35 d of rearing, piglets from two sows fed LA and piglets from three sows fed CLA were randomly assigned to each of two starter diets. The piglets were given for 35 d free access to a starter diet shown in **Table 1** that was supplemented with CLA or LA (2 g/100 g diet). The same lots of LA- and CLA-containing oils were used for the sow diet and the starter diet. The experimental groups were denoted in the text and tables as follows: treatment LL ($n = 8$) and LC ($n = 8$), pigs reared on sows fed the LA diet and then fed the LA or CLA starter diet, respectively; and treatment CL ($n = 12$) and CC ($n = 12$), pigs reared on sows fed the CLA diet and then fed the LA or CLA starter diet, respectively. Body weight of each pig was recorded at birth, weaning and d 35 of the starter period. From weaning until slaughter, the four selected piglets from each individual litter were housed in a separate pen under normal husbandry conditions. Feed consumption was recorded for each pen and average total feed intake and average feed efficiency ratio were calculated. Grouping piglets from different litters in a single pen was avoided to prevent losses of piglets due to piglet scour or edema in the first 10 d after birth. Furthermore, it was imperative to exclude medical treatments with antibiotics during the postweaning period because that would have questioned or confounded possible growth-promoting effects of CLA. All procedures involving animals were approved by the Swiss Federal Committee for Animal Care and Use.

Sampling of tissues at slaughter. The diets were withdrawn 12 h before the pigs were brought to the abattoir of the research station, where they were electrically stunned and exsanguinated. Internal organs were removed and the warm carcasses were weighed. The semitendinosus muscle was removed from the right side of each carcass and the weight, girth and length was recorded. The loin eye area was determined at the level of the 13th rib of the longissimus dorsi muscle. Furthermore back fat and omental fat were collected from each pig and stored at −20°C until fatty acid profiles were determined. Samples of longissimus dorsi muscle were lyophilized, homogenized and stored at −20°C for further analysis.

Sample analysis. Adipose tissue and longissimus dorsi muscle were analyzed for total lipid content by the method of Winter (1963) and fatty acid methyl esters (FAME) were prepared as reported earlier (Bee 2000). CLA and FAME were determined by gas chromatography (HP 5860 A GC, Urdorf, Switzerland). Methyl tridecanoate (91560, Fluka, Buchs, Switzerland) was used as internal standard to quantify the FAME. CLA standards (Matreya,, Pleasant Gap, PA) were used to identify each CLA peak. A beef-pig blend reference standard (CRM 163: Commission of the European Communities, Community Bureau of Reference, Brussels, Belgium) was used to validate the measurements. The following CLA isomers were determined: *cis*9,*trans*11 (*c*9,*t*11)-, *trans*10,*cis*12 (*t*10,*c*12)-, *cis*9,*cis*11 (*c*9,*c*11)- and *cis*10,*cis*12 (*c*10,*c*12)-18:2. The *trans*9,*trans*11 (*t*9,*t*11)- and *trans*10,*trans*12 (*t*10,*t*12)-18:2 could not be separated and are reported as the sum of both isomers.

At the abattoir, weighted quantities of back fat and omental fat were homogenized in an ice-cooled 0.25 mol/L sucrose buffer (in 0.1 mol/L phosphate buffer, pH 7.4). The samples were centrifuged twice at 15,000 × *g* for 10 min and the supernatant recentrifuged at 30,000 × *g* for 40 min in the same buffer. The supernatants were stored at −70°C to assess lipogenic enzyme activities using standard photometric methods. Samples were analyzed in duplicate for glucose-6-phosphate dehydrogenase (G6PDH, EC 1.1.1.49), malic enzyme (ME, EC 1.1.1.40) and fatty acid synthase (FAS, EC 2.3.1.85) using the methods of Löhr and Waller (1974), Hsu and Lardy (1969) and Roncari (1981), respectively. NADPH formation (G6PDH, ME) or oxidation (FAS) was measured at 37°C by absorbance at 340 nm. A commercial protein dye-binding assay kit, using bovine γ-globulin as a standard, was used to measure the soluble protein concentration in the supernatant fraction (Bio-Rad Protein Assay, Bio-Rad, Glattbrugg, Switzerland). The enzyme activities were expressed as μmol NADPH produced or oxidized · min^{-1} · mg^{-1} protein.

Statistical analysis. Data analyses were performed with the PROC GLM procedure of SAS (1998). The experimental model was a 2 × 2 × 2 factorial randomized ANOVA. Least-square values were obtained assuming fixed models that included the effects of sex, rearing (R) and starter (S) period. Growth performance data in the rearing period were analyzed as a 2 × 2 randomized ANOVA with R

TABLE 1

Ingredient composition of starter diets[1]

Item	Starter diet	
	LL and CL	LC and CC
	g/100 g	
Oat	20.00	
Wheat bran	20.00	
Apple pomace	20.00	
Barley	16.20	
Wheat	7.60	
Dextrose	5.00	
Yeast	1.90	
Potatoe protein	2.10	
Whey powder	2.50	
NaCl	0.20	
Lysine-HCl	0.04	
DL-Methionine	0.06	
L-Threonine (98%)	0.06	
Tryptophan	0.03	
Vitamin-mineral premix[2]	2.00	
Color grit[3]	0.23	
Linoleic acid (60%)[4]	2.00	
Conjugated linoleic acid[5]		2.00

[1] Starter diet was formulated to contain 12.4 MJ digestible energy, 152 g crude protein, 11.1 g lysine, 6.7 g methionine-cystine, 7.2 g threonine and 2.1 g tryptophan per kg air-dried diet.

[2] Supplied the following nutrients per kilogram of diet: 1.2 mg all-*trans* retinol, 0.013 mg cholecalciferol, 16 mg vitamin E, 2.8 mg riboflavin, 2.8 mg vitamin B-6, 0.020 mg vitamin B-12, 0.2 mg vitamin K-3, 102 mg pantothenic acid, 19 mg niacin, 0.48 mg folic acid, 116 mg Fe as Fe-sulfate, 0.16 mg I as $Ca(IO)_3$, 0.2 mg Se as Na_2Se, 6.0 mg Cu as $CuSO_4$, 96 mg Zn as ZnO_2, 1.5 mg Mn as MnO_2, 196 g choline, 0.96 mg biotin.

[3] Color supplement added to the diets to avoid feeding mistakes.

[4] The linoleic (LA)-enriched oil is derived from sunflower oil and served as source material to produce the conjugated linoleic acid (CLA)-enriched oil (SELIN-CLA: Grünau Jllertissen GmbH, Germany). The fatty acid composition of the linoleic-enriched oil expressed in g per 100 g total fatty acids: myristic (14:0): 0.14, palmitic (16:0): 5.59, stearic (18:0): 2.37, eicosanoic (20:0): 0.24, behenic (22:0): 0.42, palmitoleic [16:1(n-7)]: 0.13, oleic [18:1(n-9)]: 23.86; eicosenoic [20:1(n-9)]: 0.18, erucic [22:1(n-9)]: 0.21, linoleic [18:2(n-6)]: 65.79 and linolenic acid [18:3(n-3)]: 0.75. LL, piglets of LA-fed sows fed LA starter; CL, piglets of CLA-fed sows fed LA starter.

[5] The fatty acid composition of the CLA-enriched oil (SELIN-CLA) expressed in g per 100 g total fatty acids: myristic (14:0): 0.24, palmitic (16:0): 4.37, stearic (18:0): 1.67, eicosanoic (20:0): 0.18, behenic (22:0): 0.27, palmitoleic [16:1(n-7)]: 0.17, oleic [18:1(n-9)]: 29.48; eicosenoic [22:1(n-9)]: 0.13, and linoleic [18:2(n-6)]: 3.99. The following CLA isomers were detected: *cis* (*c*), *trans* (*t*) *c*9,*t*11, 20.33; *c*9,*c*11, 5.59; *t*10,*c*12, 21.73; *c*10,*c*12, 1.33; and *t*9,*t*11/*t*10,*t*12, 9.96. LC, piglets of LA-fed sows fed CLA starter; CC, piglets of CLA-fed sows fed CLA starter.

TABLE 2

Lipid concentrations and fatty acid composition of the starter diet[1]

	Starter diet			
Item[2]	LL and CL	LC and CC	CLA isomer[3]	Isomer[4]
Lipid, *g/100 g diet*	5.19	5.05		
	g/100 g total fatty acids			
14:0 (myristic)	0.3	0.3		
16:0 (palmitic)	12.8	12.0		
18:0 (stearic)	3.9	2.6		
20:0 (eicosanoic)	0.4	0.4		
22:0 (behenic)	0.4	0.2		
SFA	17.7	15.5		
16:1(n-7) (palmitoleic)	0.3	0.3		
18:1(n-9) (oleic)	25.5	28.4		
20:1(n-9) (eicosenoic)	0.5	0.5		
22:1(n-9) (erucic)	0.6	0.5		
MUFA	26.9	29.7		
18:2(n-6) (linoleic)	52.6	29.1		
18:3(n-3) (α-linolenic)	2.4	2.2	*c*9,*t*11	8.0 (34.3%)
PUFA	55.4	54.8	*c*9,*c*11	2.1 (9.0%)
Total CLA	ND[5]	23.3	*t*10,*c*12	8.5 (36.5%)
16:1(n-7)/16:0	0.02	0.03	*c*10,*c*12	0.5 (2.2%)
18:1(n-9)/18:0	6.50	10.85	*t*9,*t*11/*t*10,*t*12	4.2 (18.0%)

[1] Only fatty acids that accounted for ≥0.1 g/100 g of total are presented.
[2] Fatty acids were designated by the number of carbon atoms followed by the number of double bonds. The position of the first double bond relative to the methyl (n) end of the molecule was also indicated. The sums of the main fatty acid series are represented as: SFA, saturated fatty acids, MUFA, monounsaturated fatty acids, PUFA, polyunsaturated fatty acids. Values are expressed as weight percentages of total fatty acids.
[3] *c*, *cis; t*, *trans.*
[4] Amount of conjugated linoleic acid (CLA) isomer (g/100 g) in the total fatty acid methyl ester and in round brackets the relative composition (%) in the total CLA.
[5] ND, not detected. See Table 1 for diet abbreviations.

and sex as main factors. The individual pig values were considered as the experimental unit of all response variables. Significant sex and R × S effects were indexed in the tables. No significant R × sex, S × sex and R × S × sex interactions were found. Differences of $P < 0.05$ were considered significant.

RESULTS

The oil added to the starter diet fed in the treatments LL and CL was composed mainly of linoleic [18:2(n-6): 65.79 g/100 g total fatty acids] and oleic acid [18:1(n-9): 23.86 g/100 g total fatty acids]. The CLA isomers detected in the CLA-enriched oil were *c*9,*t*11; *t*10,*c*12; *c*9,*c*11; *c*10,*c*12 and *t*9,*t*11/*t*10,*t*12 and amounted to 58.9 g/100 g of total fatty acids. The fatty acid profile of the dietary lipid reflected the fat supplemented **(Table 2)**. The CLA starter diets (LC, CC) were lower in palmitic (16:0), stearic (18:0) and linoleic acids and slightly higher in oleic acid compared with the respective control (LL, CL). None of the CLA isomers were detected in the diets supplemented with the linoleic-enriched oil.

Growth performance in the rearing and starter period. Body weight at birth and weaning were not affected by the diet fed to the dams during gestation and lactation (**Table 3**). However, irrespective of the dietary fat supplied in the starter period, piglets reared on sows fed CLA had greater total feed intake ($P = 0.02$), higher daily weight gain ($P < 0.01$), and higher final body ($P < 0.01$) and warm carcass weights ($P < 0.01$) than piglets reared on sows fed LA. Feed efficiency did not differ between groups ($P = 0.42$).

Morphometric measurements of dissected muscles. Loin eye area and girth and weight of semitendinosus muscle of piglets reared on dams fed CLA were significantly larger and heavier than those of piglets suckling sows fed LA (**Table 4**). However, compared with the respective controls, the loin eye area was smaller ($P = 0.01$) and the semitendinosus muscle was shorter, if weaned piglets were fed the CLA supplemented starter diets (LC and CC).

Fatty acid profiles of the back fat and omental fat. The effect of dietary CLA on the fatty acid composition was similar for back fat (**Table 5**) and omental fat (**Table 6**). Supplementation with CLA in the preweaning period markedly increased the levels of lauric (12:0), stearic, and total saturated fatty acids (SFA) ($P < 0.01$ for each), whereas those of palmitoleic acid [16:1(n-7)], eicosenoic acid [20:1(n-9)] and total monounsaturated fatty acids (MUFA) were decreased in both tissues ($P < 0.05$ for each); oleic acid was increased and heptadecanoic [17:0] and linoleic acids were decreased only in the back fat ($P < 0.05$). Feeding of the CLA-enriched starter diet had even more pronounced effects on these same fatty acids and also on myristic (14:0) and palmitic acids. However, palmitoleic acid in back fat and omental fat and oleic acid and MUFA levels in omental fat were not affected by CLA in the starter diet. The presence of CLA in the starter diet caused linoleic, eicosadienoic [20:2(n-6)], arachidonic [20:4(n-6)] and linolenic acids [18:3(n-3)] and total polyunsaturated fatty acid (PUFA) concentrations to be significantly decreased in both fat tissues. The ratios of palmitoleic to palmitic acid [16:1(n-7)/16:0] and oleic to stearic acid [18:1(n-9)/18:0] were markedly decreased by dietary CLA ingested during both the lactation and starter periods. In contrast to the fatty acid

TABLE 3

Effects of consumption of conjugated linoleic acid (CLA) in the lactation and postweaning period, or in the postweaning period only on growth performance and carcass weight of progeny of sows fed a CLA- or linoleic acid (LA)-fortified diet during pregnancy and lactation[1]

	Treatment					*P*-values[2]	
Item	LL	LC	CL	CC	SEM	R	S
Rearing period							
Birth weight, *kg*	1.41	1.70	1.67	1.71	0.18	0.19	
Weaning weight, *kg*	9.79	9.41	10.36	9.70	0.35	0.28	
Weight gain, *g/d*	233	242	262	240	2	0.22	
Post-weaning period							
Final weight, *kg*	21.1	20.6	24.8	22.9	0.5	<0.01	0.19
Weight gain, *g/d*	337	325	417	394	3	<0.01	0.47
Total feed intake,[3] *kg*	22.0	20.8	26.9	24.3	0.9	0.02	0.16
Feed efficiency,[3] *g food/g gain*	1.96	1.87	1.85	1.84	0.23	0.42	0.55
Carcass weight, *kg*	14.94	14.16	16.94	16.02	0.45	<0.01	0.19

[1] Results are presented as least-square means and SEM, $n = 8$ (LL and LC) or 12 (CL and CC). See Table 1 for diet abbreviations.
[2] Effects of rearing (R) and starter period (S).
[3] Average value of the pen ($n = 10$).

profile, total lipid content in both back fat and omental fat was unaffected by diet.

None of the CLA isomers were detected in the fat tissues of piglets of the LL group (Tables 5 and 6). By contrast, the piglets fed CLA during the rearing and/or starter periods incorporated CLA isomers, dependent on the supply. The most abundant was *c*9,*t*11 and in decreasing order *t*10,*c*12 > *t*9,*t*11/*t*10,*t*12 > *c*9,*c*11 > *c*10,*c*12–18:2. The lowest detectable levels of isomers were found in the CL and the highest in the CC group. The *c*10,*c*12–18:2 was present only at trace levels in the CL group. The CLA contents of the back fat and omental fat were similar and do not indicate a preferential incorporation into either tissue.

Due to significantly elevated levels of lauric, myristic, palmitic and palmitoleic acids (0.14 vs. 0.13; 2.07 vs. 1.87; 28.89 vs. 26.96; 2.50 vs. 2.20 g/100 g total fatty acids, respectively), barrows showed a more saturated fatty acid pattern than gilts in the omental fat, whereas that of the back fat did not differ between sexes. The higher concentrations were compensated mainly by slightly but not significantly lower depositions of oleic and linoleic acids ($P = 0.41$ and $P = 0.17$), respectively. Additionally, barrows had significantly lower levels of *c*9,*t*11; *t*10,*c*12 and *t*9,*t*11/*t*10,*t*12 (1.11 vs. 1.31; 1.04 vs. 1.26; 0.54 vs. 0.64 g/100 g total fatty acids). No significant interactions occurred between experimental treatments and sex for any of the fatty acids.

Fatty acid profile of lipids in longissimus dorsi muscle. The total lipid content of the loin muscle was significantly affected by the preweaning treatment (**Table 7**), with lower levels found in the CL and CC groups. However, the fatty acid composition was affected in a similar way as previously presented for the adipose tissue. An exception was the level of palmitoleic and linolenic acids, which were elevated in the piglets fed the CLA-enriched starter diet (LC, CC). Total PUFA content was not influenced by dietary treatment. In contrast to the fat pads, *c*10,*c*12–18:2 could not be detected, *t*10,*c*12 was the most abundant CLA isomer in the longissimus muscle instead of *c*9,*t*11 in the fat tissues, and the overall CLA level was markedly lower.

As reported for omental fat, the levels of myristic, palmitic stearic and palmitoleic acids (1.42 vs. 1.22; 25.25 vs. 23.70; 11.34 vs. 10.94; 3.18 vs. 2.73 g/100 g total fatty acids, respectively) were greater in the longissimus dorsi fat of barrows, and those of linoleic and arachidonic acids (19.62 vs. 22.23; 0.91 vs. 1.10 g/100 g total fatty acids, respectively) were lower than in the gilts, $P < 0.05$). CLA levels did not differ between sexes. No significant interactions occurred between treatments and sex for any of the fatty acids.

TABLE 4

Morphometric measurements of longissimus dorsi and semitendinosus muscle as affected by the consumption of conjugated linoleic acid (CLA)-fortified diet in the lactation and postweaning period, or in the postweaning period only[1]

	Treatment					*P*-values[2]	
Item	LL	LC	CL	CC	SEM	R	S
Longissimus muscle							
Loin eye area, *cm*^{2}	16.0	13.2	17.1	15.8	0.5	0.02	0.01
Semitendinosus muscle							
Weight, *g*	75.0	70.7	89.3	81.3	1.3	0.02	0.23
Length, *mm*	133	125	140	130	1.1	0.09	0.03
Girth, *mm*	113	112	125	120	0.9	<0.01	0.23

[1] Results are presented as least-square means and SEM, $n = 8$ (LL and LC) or 12 (CL and CC). See Table 1 for diet abbreviations.
[2] Effects of rearing (R) and starter period (S).

TABLE 5

Effects of consumption of conjugated linoleic acid (CLA) in the lactation and postweaning period, or in the postweaning period only on the lipid content and fatty acid composition of back fat tissue in progeny of sows fed a CLA- or linoleic acid (LA)-fortified diet during pregnancy and lactation[1]

	Treatment					*P*-values[2]	
Item[3]	LL	LC	CL	CC	SEM	R	S
Lipid, *g/100 g*	52.2	58.6	62.0	57.1	3.56	0.30	0.85
			g/100 g total fatty acids				
12:0	0.06	0.13	0.08	0.17	0.05	<0.01	<0.01
14:0[4]	1.78	2.10	1.58	2.48	0.14	0.15	<0.01
16:0[4]	24.58	26.53	23.60	28.59	0.40	0.27	<0.01
17:0	0.42	0.59	0.50	0.76	0.01	0.01	<0.01
18:0	8.12	12.30	11.57	14.03	0.37	<0.01	<0.01
SFA	35.25	42.80	37.64	46.48	0.50	<0.01	<0.01
16:1(n-7)	4.55	3.97	1.80	1.95	0.30	<0.01	0.46
18:1(n-9)[4]	32.18	29.64	31.74	26.85	0.36	<0.01	<0.01
20:1(n-9)[4]	0.62	0.62	0.72	0.63	0.07	0.02	0.02
MUFA	37.70	34.48	34.58	29.68	0.42	<0.01	<0.01
18:2(n-6)	24.24	15.52	24.27	14.64	0.40	0.41	<0.01
20:2(n-6)	0.62	0.45	0.75	0.42	0.12	0.17	<0.01
20:4(n-6)[4]	0.42	0.28	0.50	0.23	0.10	0.63	<0.01
18:3(n-3)	1.22	0.87	1.05	0.92	0.16	0.48	<0.01
CLA isomers[5]							
*c*9,*t*11[6]	ND[7]	2.00	0.42	2.67	0.16	<0.01	<0.01
*c*9,*c*11[4]	ND	0.51	0.05	0.68	0.07	<0.01	<0.01
*t*10,*c*12[4,6]	ND	1.86	0.33	2.58	0.15	<0.01	<0.01
*c*10,*c*12[4]	ND	0.14	ND	0.22	0.05	<0.01	<0.01
*t*9,*t*11/*t*10,*t*12[4,6]	ND	0.98	0.16	1.38	0.10	<0.01	<0.01
PUFA	27.05	22.72	27.78	23.84	0.44	0.14	<0.01
16:1(n-7)/16:0	0.18	0.15	0.08	0.07	0.06	<0.01	0.06
18:1(n-9)/18:0[4]	4.03	2.48	2.75	1.94	0.21	<0.01	<0.01

[1] Results are presented as least-square means and SEM, $n = 8$ (LL and LC) or 12 (CL and CC). See Table 1 for diet abbreviations.
[2] Effects of rearing (R) and starter period (S).
[3] Fatty acids were designated by the number of carbon atoms followed by the number of double bonds. The position of the first double-bond relative to the methyl (n) end of the molecule was also indicated. The sums of the main fatty acid series are represented as: SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.
[4] Interaction between rearing (R) and starter period (S) is significant ($P < 0.05$).
[5] *c*, *cis; t*, *trans.*
[6] Sex effect is significant ($P < 0.05$).
[7] ND, not detected.

Lipogenic enzyme activities; Irrespective of the dietary treatment, the activity of key lipogenic enzymes was higher in the omental fat than in the back fat (**Table 8**). The presence of CLA in the starter diet, but not in the rearing period, markedly increased the activity of G6PDH and ME in both tissues. The effect of the CLA supplement was more pronounced when piglets were weaned from sows receiving linoleic acid than from sows receiving CLA (R × S interaction $P < 0.05$). In contrast, FAS activity was unaltered by the fat supplemented to the diet.

DISCUSSION

The experimental design of this study did not allow us to include the effect of litter in the statistical model. However, because most of the reported treatment effects were significant, we consider the results not to be confounded by litter effects. Irrespective of the starter diet fed in the 35-d postweaning period, pigs born to and reared by the sows fed CLA had greater feed intake, daily weight gain, and final body and warm carcass weights than pigs reared by sows fed the LA diet. In contrast to recent observations in growing pigs fed CLA (Ostrowska et al. 1999), feed efficiency was similar for both groups (Dugan et al. 1997). In addition, neonatal growth was unaffected by the nutrition of the dams. We can only speculate on the reasons for these surprising results. Previously, we observed that compared with sows fed a control diet, sows fed a CLA-enriched diet tended to have lower fat content in the mature milk (unpublished observations). However, on the basis of the growth performance of those litters, which depends mainly on the sow's milk production (King et al. 1998), we concluded that milk yield might not have been decreased by the supplementation of CLA to the dams. The observation that piglets reared by sows gain less weight than those reared artificially provides evidence that sow-reared neonates have unrealized growth potential (King et al. 1998). The two major reasons for the slower growth rate are the availability and composition of the milk. One can question whether the supply of milk CLA during lactation might have impeded the maximal possible growth rate, which then resulted in compensatory growth in the postweaning period when feed was freely accessible. In addition to enhanced growth performance, pigs reared on sows supplemented with CLA also had improved morphometric muscle measurements for semitendinosus (+17% heavier, *P*

TABLE 6

Effects of consumption of conjugated linoleic acid (CLA) in the lactation and postweaning period, or in the postweaning period only on the lipid content and fatty acid composition of the omental fat in progeny of sows fed a CLA- or linoleic acid (LA)-fortified diet during pregnancy and lactation[1]

	Treatment					*P*-values[2]	
Item[3]	LL	LC	CL	CC	SEM	R	S
Lipid, *g/100 g*	71.1	71.7	64.6	70.7	2.5	0.07	0.10
			g/100 g total fatty acids				
12:0[4]	0.10	0.16	0.11	0.17	0.05	0.01	<0.01
14:0[4]	1.60	2.24	1.65	2.40	0.15	0.15	<0.01
16:0[4]	24.91	29.55	26.14	31.10	0.52	0.11	<0.01
17:0	0.46	0.58	0.49	0.67	0.11	0.15	<0.01
18:0	11.20	15.03	15.36	16.80	0.47	<0.01	<0.01
SFA	38.65	47.95	44.15	51.57	0.70	<0.01	<0.01
16:1(n-7)[4]	2.97	3.19	1.57	1.67	0.21	<0.01	0.26
18:1(n-9)	29.11	26.54	26.95	24.54	0.72	0.20	0.13
20:1(n-9)	0.56	0.52	0.62	0.57	0.09	0.04	0.04
MUFA	32.89	30.48	29.39	26.98	0.71	0.03	0.14
18:2(n-6)	26.09	14.31	23.68	13.27	0.25	0.03	<0.01
20:2(n-6)	0.58	0.31	0.59	0.31	0.08	0.97	<0.01
20:4(n-6)	0.48	0.25	0.49	0.21	0.09	0.37	<0.01
18:3(n-3)	1.01	0.83	0.97	0.80	0.02	0.45	<0.01
CLA isomers[5]							
*c*9,*t*11[4]	ND[6]	2.15	0.28	2.42	0.03	<0.01	<0.01
*c*9,*c*11	ND	0.53	ND	0.61	0.09	0.08	<0.01
*t*10,*c*12[4]	ND	2.02	0.21	2.37	0.18	<0.01	<0.01
*c*10,*c*12[4,7]	ND	0.15	ND	0.18	0.05	<0.01	<0.01
*t*9,*t*11/*t*10,*t*12[4,7]	ND	1.00	0.11	1.25	0.10	<0.01	<0.01
PUFA	28.46	21.57	26.46	21.45	0.56	0.28	<0.01
16:1(n-7)/16:0	0.12	0.11	0.06	0.05	0.05	<0.01	0.17
18:1(n-9)/18:0	2.62	1.84	1.84	1.48	0.21	<0.01	<0.02

[1] Results are presented as least-square means and SEM, $n = 8$ (LL and LC) or 12 (CL and CC). See Table 1 for diet abbreviations.
[2] Effects of rearing (R) and starter period (S).
[3] Fatty acids were designated by the number of carbon atoms followed by the number of double bonds. The position of the first double-bond relative to the methyl (n) end of the molecule was also indicated. The sums of the main fatty acid series are represented as: SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.
[4] Sex effect is significant ($P < 0.05$).
[5] *c*, *cis, t*, *trans.*
[6] ND, not detected.
[7] Interaction between rearing (R) and starter period (S) is significant ($P < 0.05$).

= 0.02; +9% larger, $P < 0.01$; +5% longer, $P = 0.09$) and longissimus dorsi (+14% larger loin eye, $P = 0.02$), resulting in greater warm carcass weights that were influenced mainly by the rearing period rather than by the postweaning diet. Further investigation is required to determine to what extent these effects were due to hypertrophy of muscle fibers.

CLA was reported to reduce the catabolic responses induced by immune stimulation without adversely affecting immune function (Miller et al. 1994). These responses are mediated by cytokines and regulated by prostaglandin E_2 synthesis. Recently, CLA was found to lower the level of prostaglandin E_2 in both serum (Sugano et al. 1997) and bone organ cultures (Li and Watkins 1998) and reduce cytokine production (Sugano et al. 1997). In addition to affecting the immune response, cytokines induce catabolic processes in skeletal muscle (Hotamisligil and Spiegelman 1994). Therefore, reducing the immune response may have provided more available energy for anabolic processes. Additionally, the concept of CLA as a nutrient with growth-promoting effects might be supported by the observation that CLA had insulin-sensitizing effects in Zucker rats (Houseknecht et al. 1998). Dietary CLA could have acted similarly to fats rich in 20/22-carbon fatty acids (e.g., fish oil) by enhancing peroxisomal fatty acid oxidation due to induced acyl Co-A oxidase expression in skeletal muscle, which in turn is accompanied by enhanced expression of glucose transporter-4 gene and improved glucose uptake in skeletal muscle. Therefore, anabolic effects on muscles reported in this study for piglets reared on sows fed CLA could be due to reduced immune stimulation in the early stage of development of the neonate and/or elevated fuel supply to the muscle resulting from improved glucose utilization.

Previous studies have shown that only trace amounts of free fatty acids cross the swine placental tissues during gestation; therefore, their contribution to fetal energy supply or lipid storage appears limited (Thulin et al. 1989). From a number of observations reviewed by Leskanich and Noble (1999), it appears that de novo lipogenesis at birth, the principal mechanism of fatty acid accretion, is low. This deficit in body lipid content is largely rectified through the piglet's access to a rich lipid source in the form of maternal milk. In view of the fact that in our housing system and under the present experimental conditions, the suckling pigs did not have access to the sow's diet, the presence of CLA isomers (*c*9,*t*11; *t*10,*c*12; and *t*9,*t*11/*t*10,*t*12) in the fat of CL pigs clearly shows that CLA in the milk lipids were absorbed by the suckling piglets and incorporated in the early stage of fat accretion. In the mature milk of

TABLE 7

Effects of consumption of conjugated linoleic acid (CLA) in the lactation and postweaning period, or in the postweaning period only on the lipid content and fatty acid composition of the longissimus dorsi muscle in progeny of sows fed a CLA- or linoleic acid (LA)-fortified diet during pregnancy and lactation[1]

	Treatment					*P*-values[2]	
Item[3]	LL	LC	CL	CC	SEM	R	S
Lipid, *g/100 g*	1.10	1.24	0.93	0.86	0.49	<0.01	0.69
			g/100 g total fatty acids				
14:0[4]	1.22	1.51	1.08	1.48	0.15	0.23	<0.01
16:0[4]	22.95	26.11	23.18	25.67	0.34	0.77	<0.01
17:0	0.39	0.49	0.47	0.51	0.10	0.11	0.04
18:0[4,5]	9.83	11.55	11.35	11.83	0.24	<0.01	<0.01
SFA[4]	34.38	39.72	36.21	39.52	0.39	0.12	<0.01
16:1(n-7)[4]	3.33	3.76	2.09	2.64	0.24	<0.01	<0.01
18:1(n-9)	29.32	28.05	28.48	25.05	0.49	0.02	<0.01
20:1(n-9)	0.46	0.41	0.46	0.41	0.09	0.99	0.05
MUFA	33.38	32.45	31.28	28.26	0.53	<0.01	0.03
18:2(n-6)[4]	22.14	18.18	22.58	20.65	0.23	0.03	<0.01
20:2(n-6)	0.71	0.39	0.62	0.38	0.09	0.06	<0.01
20:4(n-6)	6.33	4.32	5.80	4.52	0.37	0.70	<0.01
22:4(n-6)[4]	1.28	0.75	1.14	0.83	0.15	0.67	<0.01
18:3(n-3)	0.60	0.65	0.60	0.71	0.08	0.16	<0.01
CLA isomers[6]							
*c*9,*t*11	ND[7]	0.98	0.26	1.24	0.19	0.03	<0.01
*c*9,*c*11	ND	0.21	0.02	0.13	0.10	0.35	<0.01
*t*10,*c*12	ND	1.35	0.52	1.93	0.21	<0.01	<0.01
*t*9,*t*11/*t*10,*t*12	ND	0.31	0.03	0.30	0.11	0.80	<0.01
PUFA	32.24	27.83	32.65	32.22	0.60	0.04	0.04
16:1(n-7)/16:0	0.15	0.14	0.09	0.10	0.05	<0.01	0.33
18:1(n-9)/18:0	2.98	2.44	2.51	2.13	0.16	<0.01	<0.01

[1] Results are presented as least-square means and SEM, $n = 8$ (LL and LC) or 12 (CL and CC). See Table 1 for diet abbreviations.
[2] Effects of rearing (R) and starter period (S).
[3] Fatty acids were designated by the number of carbon atoms followed by the number of double bonds. The position of the first double-bond relative to the methyl (n) end of the molecule was also indicated. The sums of the main fatty acid series are represented as: SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.
[4] Sex effect is significant ($P < 0.05$).
[5] Interaction between rearing (R) and starter period (S) is significant ($P < 0.05$).
[6] *c*, *cis; t*, *trans.*
[7] ND, not detected.

CLA-fed sows, the *c*10,*c*12–18:2 isomer was not detected and the amount of *c*9,*c*11–18:2 was low [0.5 g/100 g total fatty acids; (Bee 2000)], which explains the absence of these isomers in the tissue lipids of the CL group. Although both the rearing and starter period lasted for 35 d each, compared with the milk (CL), the CLA intake from the starter diet (LC) was higher and resulted in marked differences in CLA deposition between the groups. In the tissue lipids of piglets fed CLA during the rearing and starter periods, the highest amounts of CLA isomers were incorporated and were on average the sum of those found in the CL and LC group. For the piglets of CL and CC treatments and in agreement with recent pig data (Kramer et al. 1998), the relative absorption of all the isomers seemed to be similar because there was little difference in general between the distribution of CLA isomers in the commercial CLA preparation fed to pigs and the adipose tissue. If CLA was supplied only by the milk (*c*9,*t*11, 38 g; *c*9,*c*11, 11 g; *t*10,*c*12, 34 g; *c*10,*c*12, 1 g; *t*9,*t*11/*t*10,*t*12: 17 g/100 g of total milk CLA), relatively more *c*9,*t*11–18:2 (43%) and less *c*9,*c*11–18:2 (5%) was deposited, which might be due to differences in absorption rates and therefore availability of the milk lipids rather then selectivity in deposition. However, a certain tissue selectivity for the main isomers is suggested by the pattern of CLA enrichment because compared with the adipose tissue, *t*10,*c*12 CLA was the most abundant isomer in the muscle. These findings corroborate previous results in rats (Li and Watkins 1998) and were independent of CLA supply by the milk or diet. The tissue fatty acid composition is a dynamic system constantly receiving, oxidizing and incorporating dietary fatty acids. The metabolic turnover in adipose tissue is lower than in other tissues (Otten et al. 1993); therefore, differences in CLA incorporation might also be due to differences in turnover rates of certain isomers or selective discrimination.

Irrespective of the starter diet, intramuscular fat content of piglets reared on sows fed CLA were lower than those of the control group. We assume that the lower fat content was associated mainly with reduced triacylglycerols rather than differences in phospholipid deposition because the latter seems to remain constant after postnatal development (Lebret et al. 1999). The present data are in contrast with a study showing increased fat deposition in the longissimus thoracis in growing-finishing pigs (Dugan et al. 1999), but corroborate the reported CLA effects on body composition (Ostrowska et al. 1999).

TABLE 8

Effects of the consumption of conjugated linoleic acid (CLA) in the lactation and postweaning period, or in the postweaning period only on the activity level of lipogenic enzymes in the back fat and omental fat of progeny of sows fed a CLA- or linoleic acid (LA)-fortified diet during pregnancy and lactation[1]

	Treatment					*P*-values[2]	
	LL	LC	CL	CC	SEM	R	S
			$\mu mol \cdot min^{-1} \cdot mg\ protein^{-1}$				
Back fat[3,4]							
G6PDH[5]	42.6	70.9	56.0	59.1	1.2	0.86	<0.01
ME[5]	21.7	53.7	51.5	50.2	1.4	0.05	0.02
FAS	5.7	5.6	5.4	3.9	0.4	0.12	0.18
Omental fat[3,4]							
G6PDH	67.5	92.5	62.3	84.5	1.5	0.34	<0.01
ME	48.7	83.2	74.2	79.8	1.8	0.28	0.05
FAS	11.0	11.8	11.5	10.6	0.6	0.72	0.99

[1] Results are presented as least-square means and SEM, $n = 8$ (LL and LC) or 12 (CL and CC). See Table 1 for diet abbreviations.
[2] Effects of rearing (R) and starter period (S).
[3] G6PDH (glucose-6-phosphate dehydrogenase) and ME (malic enzyme) activities are expressed in μmol NADPH produced $\cdot$ $min^{-1} \cdot mg^{-1}$ protein.
[4] FAS (fatty acid synthase) activities are expressed in μmol NADPH oxidized $\cdot$ $min^{-1} \cdot mg^{-1}$ protein.
[5] Interaction between rearing (R) and starter period (S) is significant ($P < 0.05$).

The question arises whether differences in the *t*10,*c*12 CLA level, the most abundant isomer found in the muscle, were responsible for or triggered the differences in fat deposition (Baumgard et al. 2000, Park et al. 1999). However, this does not explain why the intramuscular fat content of the LC group was similar to that of the CC group.

The fatty acid composition of the pig tissues was significantly modified by the dietary lipids. CLA consumption in the rearing period increased the concentration of stearic acid and total SFA, but decreased that of palmitoleic and oleic acids and total MUFA. CLA feeding in the postweaning period further enhanced the differences. In addition, linoleic, linolenic and arachidonic acids and total PUFA were markedly decreased. The linoleic acid–enriched diet could only partly overcome the effect of the maternal milk CLA on the tissue fatty acid composition (CL), which might be due to the fact that already deposited CLA isomers affected fat metabolic pathways.

G6PDH and ME are the main enzymes involved in supplying NADPH for the reductive biosynthesis of fatty acids (Mourot et al. 1995), but they also contribute to metabolic pathways other than lipogenesis. Both fat tissues of pigs receiving CLA in the starter diet had greater G6PDH and ME activities than those in pigs receiving the LA starter diet. The G6PDH activity was higher than ME in both tissues and seemed to be the main producer of NADPH. This observation agrees with comparative studies in newborns (Le Dividich et al. 1994) and growing pigs (Mourot et al. 1995). Furthermore, the inclusion of CLA in the starter diet had a greater effect on enzyme activities in the back fat of pigs reared on sows fed LA in the diet than in pigs reared on sows fed CLA, suggesting that the presence of CLA isomers during rearing had already altered fat metabolism before pigs received the starter diet. Perhaps the effects of dietary CLA on activities of ME and G6PDH are indirect rather than direct. Increasing concentrations of glucose have been reported to result in the progressive induction of ME (Mariash and Oppenheimer 1984) and G6PDH (Boll et al. 1996). Assuming that CLA has insulin-sensitizing effects and improves glucose utilization by the adipocytes, the effects found on ME and G6PDH activities could in fact be related to elevated availability of glucose induced by the dietary CLA rather than a direct effect of CLA on the enzymes.

The activity of FAS was markedly higher in the omental fat than the back fat, and could explain the differences in the fat content of the two tissues. However, the fatty acid composition was somewhat contradictory to the enzyme activity. Dietary CLA supply markedly increased tissue SFA, fatty acids that derive primarily from the diet and de novo synthesis, but had no effect on FAS activity. The lower ratio of palmitoleic to palmitic and oleic to stearic acid in all tissues of the LC, CL and CC experimental groups compared with the LL group could indicate a down-regulation of Δ9-desaturase activity, which was proposed by other authors (Li and Watkins 1998, Pariza et al. 2000) and could explain the high amount of SFA in the tissues. The low tissue level of arachidonic acid of animals fed CLA further suggested that other desaturases (Δ6-, Δ5-desaturase) might be affected by dietary CLA as was observed in research with rodents (Belury and Kempa-Steczko 1997).

In conclusion, piglets reared on sows fed CLA during pregnancy and lactation grew faster in the postweaning period, irrespective of the starter diet. Furthermore, they deposited higher amounts of SFA, which was compensated mainly by a lower content of MUFA in the adipose and muscle tissue. When weaned piglets were fed a CLA-enriched starter diet, the effects on fatty acid composition were even more pronounced. The elevated SFA deposition due to dietary CLA was related to an inhibition of the Δ9-desaturase activity and probably other desaturases, rather than elevated de novo synthesis rate.

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