The Metabolic Conversion of 9,11,13-Eleostearic Acid (18:3) to 9,11-Conjugated Linoleic Acid (18:2) in the Rat

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Summary The presence of a significant amount of 9,11-conjugated linoleic acid (CLA, 9,11-18:2) was confirmed in the liver and plasma lipids of rats fed a 1% (w/w % of diet) eleostearic acid (ESA, 9,11,13-18:3) diet for 4 wk. The chemical structure of the 9,11-CLA apparent in the tissue lipids was identified by gas chromatography-electron impact mass spectrometry after its conversion to a 4,4-dimethyloxazoline derivative. The concentration of CLA in the total fatty acids of the liver and plasma lipids reached to about 1% for each in the CLA-supplemented rats, while reaching 3.2% and 2.5%, respectively, in the ESA-supplemented rats. The results suggest that α -ESA is metabolized partially to CLA via a delta 13-saturation reaction in the rat. Some biological activities observed in α -ESA-fed animals may be ascribed also to CLA that is formed from ESA in the body.

Key Words eleostearic acid, conjugated trienoic acid, conjugated linoleic acid, tung oil, conjugated fatty acid

Some plant seed oils are known to contain conjugated fatty acids such as α -eleostearic acid (α -ESA; 9c,11t,13t-18:3), calendic acid (8t,10t,12c-18:3; *n*-6) and parinaric acid (9c,11t,13t,15c-18:4) (1, 2). Other conjugated fatty acids, bosseopentaenoic acid (5c,8c,10t,12t,14c-20:5) and stellaheptaenoic acid (4c,7c,9t,11t,13c,16c,19c-22:7), have also been found in marine algae (3–5). Additionally, conjugated linoleic acid (CLA; 9,11-18:2, conjugated dienoic acid) is well known to be a component of several dairy products including milk and cheese (6, 7).

CLA has been shown to have properties as anti-carcinogenic and anti-obesity agent in animal studies (8–10). In our previous studies, conjugated trienoic fatty acids (conjugated linolenic acid, CLnA; conjugated eicosapentaenoic acid, CEPA; and conjugated docosahexaenoic acid, CDHA), prepared by alkaline isomerization from α -linolenic acid (α -LnA; 9,12,15-18:3), eicosapentaenoic acid (EPA; 5,8,11,14,17-20:5) and docosahexaenoic acid (DHA; 4,7,10,13,16,19-22:6), demonstrated a significantly higher cytotoxic effect on human tumor cells than did CLA (11). Furthermore, we observed that tung oil fatty acids (mainly consisting of eleostearic acids) induce cytotoxic actions against human tumor cells as efficiently as the conjugated trienoic fatty acids (12). Tung oil contains both α -ESA and β -eleostearic acid (β -ESA; 9t,11t,13t-18:3) which

together compose about 80% of the constituent fatty acids (3). Some researchers have reported the nutritional characteristics of tung oil-derived conjugated trienoic fatty acids in rats (13–15). However, the intestinal absorption and metabolism of conjugated trienoic acids (including tung oil ESA) in animals are not yet fully understood. In the present study, we examined the intestinal absorption of ESA, and found the novel metabolic conversion of ESA to CLA in rats. In the present study, we first succeeded in confirming the occurrence of CLA from ESA with gas-chromatograph/mass spectrometric analysis of the dimethyloxazoline derivative.

MATERIALS AND METHODS

Animals. Male Sprague-Dawley rats (4 wk of age) were obtained from Japan SLC (Hamamatsu, Japan). After acclimatization to a commercial diet for 1 wk, rats were randomly divided into four groups according to the test oils administered: a LA-fed group (*n*=10), an α -LnA-fed group (*n*=10), a CLA-fed group (*n*=10) and an ESA-fed group (*n*=10). The diet composition was as follows (g/kg diet): casein (Oriental Yeast Co., Tokyo), 200.0; α -cornstarch (Oriental Yeast), 397.5; sucrose, 70.0; oil mixture (see Table 1), 100.0; cellulose powder (Oriental Yeast), 50.0; AIN-96 mineral mix (Oriental Yeast), 35.0; AIN-96 vitamin mix (Oriental Yeast), 10.0; L-cystine, 3.0; and choline bitartrate, 2.5 (16). The food was stored at –20°C until feeding. The rats were caged individually, given free access to food and distilled water, and housed in a temperature- and humidity-controlled room with light cycles of 12 h on and 12 h off. During the feeding period, body weight and food consumption were measured every day. After 4 wk of test oil feeding, rats were sacrificed by decapita-

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Abbreviations: CLA, conjugated linoleic acid; DMOX, 4,4-dimethyloxazoline; ESA, eleostearic acid; FAME, fatty acid methyl ester; GC, gas chromatography; GC-EIMS, gas chromatography-electron impact mass spectrometry.

Table 1. Fatty acid composition of dietary oil mixtures and basal oils.

	Fatty acid composition (%)						
	16:0	18:8	18:1	18:2	CLA	α -LnA	ESA
Dietary oil mix							
α -LnA oil mix	8.8	3.2	14.6	62.5	N	10	N
ESA oil mix	8.5	3.7	13.7	63.2	N	N	10
LA oil mix	9.0	3.4	15.1	71.6	N	N	N
CLA oil mix	9.2	3.8	14.7	61.4	10	N	N
Basal oil							
Perilla oil	6.9	1.9	14.0	15.9	N	61.3	N
Tung oil	4.7	5.0	8.0	15.6	N	N	66.8
Safflower oil fatty acids	8.1	2.9	17.2	71.8	N	N	N
CLA prepared from safflower oil	9.3	5.8	14.6	0.7	69.6	N	N
Safflower oil	9.2	3.5	14.7	71.8	N	N	N

Tung oil was a gift from Nippon Oil and Fat Co. Perilla oil, safflower oil fatty acids, CLA prepared from safflower and safflower oil were obtained from Rinoru Oil Mills Co. ESA oil mix was prepared with safflower oil and tung oil (safflower oil:tung oil=85.0:15.0, vol/vol). α -LnA oil mix was prepared with safflower oil and perilla oil(safflower oil:perilla oil=83.7:16.3, v/v). CLA oil mixture was prepared with safflower oil and CLA prepared from safflower oil (safflower oil:CLA=83.7:16.3, v/v). LA oil mix was prepared with safflower oil and safflower oil fatty acids (safflower oil:safflower oil fatty acids=83.7:16.3, v/v)

tion, and the liver and plasma were collected and stored at -20°C until assaying. All procedures were performed in accordance with the Animal Experiment Guidelines of Tohoku University.

GC Analysis. The liver was homogenized with four volumes of ice-cold saline. Total lipids from the liver homogenate and plasma were extracted by Folch's procedure (17). Total lipids were added to a known amount of heptadecanoic acid (17:0, Sigma), as the internal standard, and submitted to methylation with 4% HCl/methanol for 20 min at 60°C to prepare their fatty acid methylesters. The fatty acid methylesters were analyzed by gas chromatography performed on a GC 353B gas chromatograph (GL Sciences Inc., Tokyo, Japan) equipped with a FID detector and a Supelcowax-10 fused silica capillary column (60 m $\times$ 0.32 mm i.d., Supelco, Bellefonte, PA, USA) (18). α - and β -ESA methylesters were analyzed at retention times of 45.5 min and 47.5 min, respectively (Fig. 1A). Table 1 shows the fatty acid composition of five test oils used in the present animal experiment. Tung oil contained 61.1% α -ESA and 5.7% β -ESA, while CLA prepared from safflower oil contained 34.4% 9c,11t/9t,11c-isomer, 35.6% 10t,12c-isomer, 2.5% 9c,11c-isomer and 1.4% 9t,11t/10t,12t-isomer. To prepare the test oil mixtures, safflower oil was mixed with one of the test oils to give a final oil mixture containing 10% of the tested fatty acid (see Table 1).

GC-EIMS analysis. To identify the unknown fatty acids, gas chromatographic-mass spectrometry was performed using a GC-17A gas chromatograph-electron impact mass spectrometer (Shimadzu, Kyoto, Japan) with a Supelcowax-10 fused silica capillary column. Helium was used as the carrier gas. The injector

and column oven temperature were programmed with the same conditions as the GC analysis mentioned above. The ion source temperature was 250°C . The mass spectra were recorded at 70 eV and analyzed by a CLASS-5000 data system (Shimadzu). The fatty acids prepared from the total lipid extracts from sample plasma and tissues were converted to 4,4-dimethyloxazoline (DMOX) derivatives prior to being submitted to GC-EIMS analysis (19).

Determination of lipid hydroperoxides. To examine the oxidation determination of tissue lipids possibly caused by conjugated fatty acid intake, membranous phospholipid hydroperoxides, phosphatidylcholine hydroperoxide (PCOOH), and phosphatidylethanolamine hydroperoxide (PEOOH), in the liver and plasma were determined by chemiluminescence detection-high performance liquid chromatography (CL-HPLC) as described previously (20, 21). The CL-HPLC unit consisted of a JASCO HPLC system (Japan Spectroscopic Co., Tokyo, Japan) combined with a CLD-100 chemiluminescence detector (Tohoku Electronic Industries Co., Sendai, Japan) and a JASCO UV detector (UV-970) equipped with a JASCO Finepak SIL NH₂-5 column (*n*-propylamine-bound silica column, 5 μM particle size, 250 $\times$ 4.6 mm). The mobile phase applied was 2-propanol/methanol/water (1,350:450:200, v/v/v) and the flow rate was 1.0 mL/min. Total lipid fractions extracted from the liver and plasma were used as samples. A calibration curve was prepared for PCOOH by photooxidation of synthetic phosphatidylcholine (1-hexadecanoyl-2-[9-*cis*-octadecadienoyl]-*sn*-glycero-3-phosphocholine, Sigma). A thiobarbituric acid (TBA) test was also performed by the method of Ohkawa et al. (22) using liver homogenates and plasma as samples.

Fig. 1. Gas chromatograms of (A) standard fatty acid methyl esters, (B) fatty acid methyl esters derived from liver total lipids of rats fed the 1% ESA diet, and (C) B plus commercial CLA (50% 9c,11t/50% 10t,12c-18:2).

Lipid determination. Triacylglycerol and total cholesterol were measured using commercially available enzymatic kits, Triacylglycerol Test Wako and Total Cholesterol Test Wako (Wako Pure Chemical, Tokyo, Japan), respectively. Phospholipid content was determined by the method of Bartlett (23).

Statistical analysis. Statistical analysis was performed using one-way ANOVA, followed by the Newman-Keules test for multiple comparisons among several groups. A difference was considered to be significant at $p < 0.05$.

RESULTS

Effects of dietary conjugated fatty acids on body weight and food intake of rats

Rats in the conjugated fatty acid-fed groups (ESA-fed

Fig. 2. The GC/MS spectrum of the 4,4-dimethyloxazoline derivatives of 9,11-CLA identified in liver total lipids of rats fed the 1% ESA diet.

group and CLA-fed group) exhibited no unusual effects in regards to growth or food intake when compared with those in the non-conjugated fatty acid-fed groups (α -LnA-fed group and LA-fed group) during the 4-wk feeding experiment. Moreover, there were no significant differences in liver weights among the rats in the four dietary groups.

Occurrence of 9,11-CLA in the rats fed the ESA diet

An ambiguous peak was detected in the liver and plasma fatty acid methyl esters from rats fed the 1% ESA diet (Fig. 1B, arrow). When the liver fatty acid methyl esters were analyzed by GC after the addition of authentic CLA (a mixture of 50% 9c,11t/9t,11c-CLA and 50% 10t,12c-CLA, Sigma), this ambiguous peak gave the same retention time as that of 9c,11t/9t,11c-CLA (Fig. 1C, arrow). The dimethyloxazolin derivatives of the total fatty acids derived from the liver and plasma lipids were then analyzed by GC-EIMS. The GC-EIMS spectrum (Fig. 2) of the dimethyloxazolin derivative of the ambiguous peak component (Fig. 1C, arrow) found in the liver lipids exhibited m/z 333 as a molecular ion, and m/z 182 and 262 as allylic cleavages of a fatty acyl chain. Furthermore, the mass spectrum exhibited a mass difference of 12 mass units between m/z 196 and 208, and between m/z 210 and 234, indicating that these are conjugated double bonds at 9 and 11 carbon numbers (Fig. 2). Consequently, the ambiguous peak (Fig. 1C, arrow) apparent in the liver and plasma lipids of rats fed the 1% ESA diet was confirmed to be a 9,11-CLA.

Fatty acid composition of the liver and plasma of rats fed conjugated fatty acids

Dietary α -ESA was found to be well absorbed from the digestive tract into the rat body after oral intake (Tables 2 and 3). However, β -ESA was not detected in the liver or plasma lipids of rats fed the 1% ESA diet (Tables 2 and 3). Furthermore, the 9,11-CLA, which might be converted from ESA, was composed of up to 3.2% and 2.5% of total fatty acids in the liver and plasma lipids, respectively, in the rats fed the 1% ESA diet. The CLA concentration in the liver and plasma lipids of ESA-fed rats was meaningfully higher than that of

Table 2. Fatty acid composition of liver total lipids of rats fed 1% conjugated fatty acid diets for 4 wk.

Diet group	1% α -LnA	1% ESA	1%LA	1% CLA
Fatty acid	Composition (%)			
16:0	29.9 $\pm$ 1.0	28.9 $\pm$ 0.9	29.7 $\pm$ 1.1	30.4 $\pm$ 0.9
16:1(<i>n</i> -7)	2.7 $\pm$ 0.2 ^a	2.2 $\pm$ 0.2 ^{ab}	2.3 $\pm$ 0.2 ^{ab}	1.9 $\pm$ 0.2 ^b
18:0	8.1 $\pm$ 0.6 ^a	8.3 $\pm$ 0.9 ^a	9.0 $\pm$ 0.7 ^a	12.2 $\pm$ 0.8 ^b
18:1(<i>n</i> -9)	11.7 $\pm$ 0.3 ^a	11.9 $\pm$ 0.3 ^{ab}	11.9 $\pm$ 0.3 ^{ab}	10.8 $\pm$ 0.4 ^b
18:1(<i>n</i> -7)	3.7 $\pm$ 0.2	4.0 $\pm$ 0.2	4.0 $\pm$ 0.3	3.6 $\pm$ 0.4
18:2(<i>n</i> -6)	30.2 $\pm$ 1.2	28.9 $\pm$ 1.3	30.9 $\pm$ 2.0	27.2 $\pm$ 1.9
18:3(<i>n</i> -3)	2.3 $\pm$ 0.2 ^a	0.1 $\pm$ 0.02 ^b	0.2 $\pm$ 0.02 ^b	0.1 $\pm$ 0.01 ^b
CLA				
9c,11t/9t,11c	ND ^a	3.2 $\pm$ 0.2 ^b	ND ^a	1.0 $\pm$ 0.1 ^c
10t,11c	ND ^a	ND ^a	ND ^a	0.4 $\pm$ 0.03 ^b
9c,11c	ND ^a	ND ^a	ND ^a	0.2 $\pm$ 0.04 ^b
9t,11t/10t,12t	ND ^a	ND ^a	ND ^a	0.1 $\pm$ 0.01 ^b
20:4(<i>n</i> -6)	8.5 $\pm$ 0.3 ^b	9.2 $\pm$ 0.5 ^{ab}	10.7 $\pm$ 0.9 ^b	9.6 $\pm$ 0.8 ^{ab}
α -ESA(<i>n</i> -5)	ND ^a	0.1 $\pm$ 0.02 ^b	ND ^a	ND ^a
β -ESA(<i>n</i> -5)	ND	ND	ND	ND
22:6(<i>n</i> -3)	1.5 $\pm$ 0.1 ^a	0.6 $\pm$ 0.1 ^b	0.6 $\pm$ 0.1 ^b	0.7 $\pm$ 0.1 ^b

Values are meant $\pm$ SE (*n*=9–10). Values not sharing a common superscript letter are significantly different at *p*<0.05. ND, not detected (below 0.1%).

Table 3. Fatty acid composition of plasma total lipids of rats fed 1% conjugated fatty acid diets for 4 wk.

Diet group	1% α -LnA	1% ESA	1%LA	1%CLA
Fatty acid	Coposition(%)			
16:0	25.0 $\pm$ 0.6	23.7 $\pm$ 0.7	24.4 $\pm$ 0.9	25.9 $\pm$ 1.0
16:1 (<i>n</i> -7)	2.2 $\pm$ 0.1	2.3 $\pm$ 0.2	2.0 $\pm$ 0.2	2.0 $\pm$ 0.3
18:0	9.9 $\pm$ 0.3 ^a	8.5 $\pm$ 0.4 ^b	10.5 $\pm$ 0.8 ^a	12.9 $\pm$ 0.4 ^c
18:1 (<i>n</i> -9)	9.8 $\pm$ 0.3	11.1 $\pm$ 0.4	10.0 $\pm$ 0.5	10.0 $\pm$ 0.8
18:1 (<i>n</i> -7)	3.9 $\pm$ 0.2	4.2 $\pm$ 0.2	3.6 $\pm$ 0.2	4.2 $\pm$ 1.2
18:2 (<i>n</i> -6)	34.3 $\pm$ 1.0	36.0 $\pm$ 1.8	32.9 $\pm$ 1.7	28.7 $\pm$ 3.3
18:3 (<i>n</i> -3)	2.3 $\pm$ 0.1 ^a	0.1 $\pm$ 0.01 ^b	0.1 $\pm$ 0.02 ^b	0.1 $\pm$ 0.02 ^b
CLA				
9c,11t/9t,11c	ND ^a	2.5 $\pm$ 0.2 ^b	ND ^a	1.0 $\pm$ 0.1 ^c
10t,12c	ND ^a	ND ^a	ND ^a	0.8 $\pm$ 0.1 ^b
9c,11c	ND ^a	ND ^a	ND ^a	0.01 $\pm$ 0.02 ^b
9t,11t/10t,12t	ND ^a	ND ^a	ND ^a	0.01 $\pm$ 0.02 ^b
20:4 (<i>n</i> -6)	10.2 $\pm$ 0.9 ^{ab}	9.2 $\pm$ 1.2 ^a	14.2 $\pm$ 1.7 ^b	12.7 $\pm$ 1.2 ^b
α -ESA (<i>n</i> -5)	ND ^a	0.5 $\pm$ 0.2 ^b	ND ^a	ND ^a
β -ESA (<i>n</i> -5)	ND	ND	ND	ND
22:6 (<i>n</i> -3)	1.0 $\pm$ 0.1 ^a	0.2 $\pm$ 0.03 ^b	0.3 $\pm$ 0.03 ^b	0.3 $\pm$ 0.04 ^b

Values are meant $\pm$ SE (*n*=9–10). Values not sharing a common superscript letter are significantly different at *p*<0.05. ND, not detected (below 0.1%).

rats fed the 1% CLA diet (Tables 2 and 3). No CLA was found in the tissue lipids of rats fed the α -LnA- and linoleic acid-diets (Tables 2 and 3).

Lipid peroxidation and lipid contents

Among the four dietary oils groups, no significant differences in the concentrations of membranous phospholipid hydroperoxides (PCOOH and PEOOH) (Table 4) or in the TBA values (Table 5) were confirmed in either the liver or plasma of the rats. The triacylglycerol, total cholesterol, and phospholipid contents in the liver and plasma of rats were not significantly different (Table 6).

DISCUSSION

In the present study, we observed that CLA is present in the liver and plasma of rats fed a 1% ESA-supplemented diet. This finding may indicate the metabolic conversion of ESA to CLA, since CLA was not contained in the ESA-supplemented diet.

Leukotriene B₄ (LTB₄), a known activator of leukocytes, has a conjugated triene structure in its configuration. Although ω -oxidation is the major deactivation pathway of LTB₄ in leukocytes, another pathway by the reduction of conjugated double bonds has been

Table 4. The contents of phospholipids hydroperoxide in the liver and plasma of rats fed conjugated trienoic and dienoic fatty acid diets for 4 wk.

Diet group	Liver		Plasma	
	PCOOH (μ mol/mol PL)	PEOOH	PCOOH (mmol/mol PL)	PEOOH
1% α -LnA	24.1 $\pm$ 3.8	21.5 $\pm$ 3.3	50.6 $\pm$ 8.4	ND
1%ESA	26.9 $\pm$ 5.2	26.6 $\pm$ 2.2	68.6 $\pm$ 17.0	ND
1%LA	26.8 $\pm$ 4.0	25.8 $\pm$ 5.8	55.0 $\pm$ 15.1	ND
1%LA	26.8 $\pm$ 4.0	25.8 $\pm$ 5.8	55.0 $\pm$ 15.1	ND
1%CLA	37.8 $\pm$ 5.0	22.9 $\pm$ 1.9	68.7 $\pm$ 30.0	ND

Values are meant $\pm$ SE ($n=9-10$). ND, not detected.

Table 5. The contents of TBA values in the liver and plasma of rats fed conjugated trienoic and dienoic fatty acid diets for 4 wk.

Diet group	Liver (nmol/g tissue)	Plasma (nmol/mL)
1% α -LnA	99 $\pm$ 7.0	14.0 $\pm$ 2.1
1%ESA	103 $\pm$ 9.0	9.2 $\pm$ 2.3
1%LA	157 $\pm$ 32.0	10.5 $\pm$ 1.1
1%CLA	125 $\pm$ 15.0	9.4 $\pm$ 0.5

Values are meant $\pm$ SE ($n=9-10$).

reported (24–26). In the reductive pathway, LTB₄ is converted to 12-oxo-LTB₄ by LTB₄ 12-hydroxydehydrogenase, which is distributed in various tissues and leukocytes of porcine (24, 25). 12-Oxo-LTB₄ is known to be converted further by another enzyme, which has not been identified, to 10,11,14,15-tetrahydro-12-oxo-LTB₄ (conjugated dienoic acid) (24–26). Accordingly, we suppose that, in our rat model, either a novel enzyme recognizing conjugated trienoic acid or the enzyme active in the LTB₄ reductive pathway may convert ESA (a conjugated triene) to CLA (a conjugated diene) with delta 13-saturation (reduction).

ESA absorption from the digestive tract was confirmed in rats fed the 1% ESA-supplemented diet. In our previous reports (11, 12), conjugated trienoic fatty acids induced apoptosis via membrane phospholipid hydroperoxidation in human tumor cells (DLD-1, MCF-

7, HepG2, A549, MKN-7), but not in normal cells (MRC-5, TIG-103, KMS-6) (11, 12). In the present study, α -ESA (conjugated trienoic fatty acid) feeding did not increase lipid hydroperoxide in therats (Tables 4 and 5), agreeing with our previous study (14), in which in vivo lipid peroxidation was induced only in tumor cells by conjugated trienoic fatty acids. Here, α -ESA (conjugated trienoic fatty acid) intake did not affect rat growth, food intake, or the liver or plasma lipid compositions of the rat (Table 6). Accordingly, dietary supplementation of α -ESA (conjugated trienoic fatty acids) may not be harmful for the growth of rats.

CLA is known to provide several beneficial effects, such as anti-carcinogenesis, anti-atherosclerosis, and decreases in body weight and body fat pad (8–10). In the present study, CLA was found to be efficiently accumulated in the liver and plasma lipids when an ESA diet was given to the rats (Tables 2 and 3). Tung oil ingestion might induce several physiological effects mediated not only by ESA itself, but also by CLA metabolically formed from ESA in the rat body (8–10). Ha et al. reported that the antioxidant potency of CLA is stronger than that of α -tocopherol or BHT (27). On the other hand, some test-tube studies have demonstrated that CLA accelerates lipid peroxidation (28, 29). Our previous study indicated that CLA is not a pro-oxidant in HepG2, but rather might act as an antioxidant (18). In the present study, CLA supplementation did not increase the concentrations of phospholipid hydroperoxides (Table 4) or TBARS (Table 5) of the liver and plasma in the rat. Therefore, we recognized that CLA does not facilitate lipid peroxidation in vivo.

Furthermore, the concentration of 10t,12c-CLA in the liver, but not in the plasma, was lower than that of 9c,11t/9t,11c-CLA in rats fed the 1% CLA diet (Table 2). Similarly, the 10t,12c-CLA content was half as much as that of 9c,11t/9t,11c-CLA in the cellular lipid fraction of the CLA-supplemented HepG2 (18). These results show that the 10t,12c-isomer is metabolized and decomposed faster than 9c,11t/9t,11c-isomers in the liver. It might be related that the 10t,12c-isomer is more active than the 9t,11c-isomer in regard to decreased body weight and growth inhibition of tumor cells (30, 31).

In summary, we have demonstrated here the in vivo conversion to CLA from ESA, and provide evidence that

Table 6. Lipid contents of the liver and plasma of rats fed 1% conjugated trienoic and dienoic fatty acid diets for 4 wk.

iet group	Liver			Plasma		
	TG	TC (mg/g tissue)	PL	TG	TC (mg/dL)	PL
1% α -LnA	24.0 $\pm$ 2.1	2.1 $\pm$ 0.1	56.2 $\pm$ 3.3	232 $\pm$ 20	81 $\pm$ 5	132 $\pm$ 9
1%ESA	29.2 $\pm$ 3.5	2.8 $\pm$ 0.2	53.4 $\pm$ 2.0	329 $\pm$ 35	86 $\pm$ 6	162 $\pm$ 12
1%LA	29.0 $\pm$ 2.4	2.6 $\pm$ 0.1	63.3 $\pm$ 6.2	239 $\pm$ 22	93 $\pm$ 7	127 $\pm$ 11
1%CLA	22.1 $\pm$ 2.7	2.4 $\pm$ 0.2	62.5 $\pm$ 2.7	233 $\pm$ 36	90 $\pm$ 7	143 $\pm$ 14

Values are meant $\pm$ SE ($n=9-10$).

dietary conjugated trienoic acid (ESA) and conjugated dienoic acid (CLA) do not meaningfully cause membrane lipid peroxidation in the rat.

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