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The influence of dietary conjugated linoleic acid isomers and Se on the fatty acid profile in rat blood plasma and selected tissues*

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ABSTRACT

The objective of this study was to determine the conjugated linoleic acid (CLA) isomer composition of blood plasma and other organ tissues of Wistar rats fed a diet enriched with 1 or 2% CLA isomer(s) and 2 ppm Se as sodium selenate for 4 weeks. The dietary supplement of CLA isomers significantly elevated the concentration of CLA isomers and other fatty acids containing conjugated double bonds in plasma, spleen, pancreas, heart, and kidney tissues. *cis9,trans11*CLA was preferentially accumulated in plasma and organ tissues of rats fed the CLA isomer mixture with or without Se, while the percentage contribution of *trans10,cis12*CLA in the tissues of organs tended to be lower than its percentage in the administered CLA isomer mixture. Dietary CLA isomer(s) increased the Ca content of whole blood, while the diet with the CLA isomer(s) and Se increased the concentration of Mg and Se in spleen tissue. The experimental diets did not produce any substantial changes in the Fe or Zn concentrations in blood or spleen tissue. The diet enriched in *trans10,cis12*CLA and Se significantly increased spleen, pancreas and heart weights. Regardless of the presence of Se, the diet enriched in CLA isomer(s) stimulated the accumulation of polyunsaturated fatty acids in blood plasma, and in pancreas, heart, and kidney tissues. The diets enriched in the CLA isomer(s) usually resulted in a decrease of the monounsaturated fatty acid concentration and Δ9-desaturase capacity in plasma, spleen, pancreas, and kidney tissues, but not in noticeably decreased concentrations in heart and brain tissues. The presence of both Se and the CLA isomer(s) produced less consistent changes in the capacity of Δ9 desaturase and monounsaturated fatty acid concentrations compared with the effect of the diet with the CLA isomer(s) only.

KEY WORDS: CLA isomers, fatty acids, Se, Zn, Fe, Ca, Mg, blood plasma, rat tissue

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INTRODUCTION

Conjugated linoleic acid (CLA) represents a mixture of geometrical and positional isomers of linoleic acid with conjugated double bonds located in positions 11,13; 10,12; 9,11; 8,10 or 7,9 on the carbon chain of the fatty acid (Park, 2005). Many reports have demonstrated health-promoting effects of dietary CLA, especially *cis9,trans11*CLA, in various animal species. These reports have shown that dietary CLA has anticarcinogenic and antidiabetogenic properties and that it delays the onset of atherosclerosis (Ostrowska et al., 2003; Ohashi et al., 2004). On the other hand, diets enriched in *trans10,cis12*CLA caused reduction of body fat in rodents and, probably, in humans. These health promoting effects seem to be the effect of increased energy expenditure associated with sympathetic nerve activation rather than a consequence of reduced food intake (Belury, 2002). Among other physiological effects, CLA isomers affect lipid metabolism, modify fatty acid (FA) oxidation, reduce the concentration of *cis9*C16:1, oleic acid (*cis9*C18:1), arachidonic acid, and inhibit formation of eicosanoids in tissues of laboratory animals. Little is known, however, about the influence of the individual CLA isomers on the accumulation of the above-mentioned compounds in selected organs of the examined animals.

Low molecular weight Se-compounds in the bodies of mammals were recognized in the late 1950's when it was found that Se replaces sulphur in methionine and cysteine. The human Se-proteome consists of twenty-five selenoproteins (Tapiero et al., 2003). Se-containing proteins (e.g., the glutathione-peroxidase (GPx) family) are essential in the metabolism of arachadonic acid, as well as in redox regulation (thioredoxin reductases) (Tapiero et al., 2003). Moreover, Se-amino acid-containing proteins have generally been shown to protect against the toxicity of heavy-metals (e.g., Hg, Pb, Sb or Cd) and enable peroxynitrite scavenging (Czauderna et al., 2004; Schomburg et al., 2004). Proteins containing Se-cysteine, in particular, protect cell membranes, lipids and tissues from oxidative stress and control cell redox status (Shweizer et al., 2005). In accordance with these findings, recent studies on experimental animals showed that the concentrations of mono- and polyunsaturated fatty acids (MUFA and PUFA) were positively correlated with the content of Se in the diet (Crespo et al., 1995; Czauderna et al., 2004a,b).

The current study was, therefore, conducted to determine the effect of dietary CLA isomers on the FA profile, in particular, of CLA isomers in selected organs of rats. Another aim of our study was to investigate the influence of dietary Se on the accumulation of the individual CLA isomers and other FAs in the analysed tissues.

MATERIAL AND METHODS

Animals and experimental design

Ten groups of 7-8 female rats, 8 weeks of age and an initial body weight of about 200 g (Table 1) were housed individually as described previously (Czauderna et al., 2004b). Rats (Wistar, Ifz:BOA) were fed *ad libitum* the Labofeed diet (Control) or diets enriched in CLA isomer(s) and/or 2 ppm Se (as Na_2SeO_4) (Table 1). After 4 weeks the rats were killed by CO_2 inhalation, their blood was collected and their spleen, pancreas, heart, kidneys and brain, removed. The organ tissues were freeze-dried immediately.

Chemicals

All reagents were analytical grade, whereas HPLC-grade organic solvents were purchased from Lab-Scan (Ireland). Sodium selenate (Na_2SeO_4) and all fatty acid standards were provided by Sigma (USA). The CLA isomer mixture (95-97%), *cis9,trans11*CLA (95-97%) and *trans10,cis12*CLA (95-97%) were supplied by Larodan Fine Chemicals AB (Sweden). The composition of *cis9,trans11*CLA (*c9,t11*CLA) and *trans10,cis12*CLA (*t10c12*CLA) was 99.9 and 99.8%, respectively, i.e. 0.1-0.2% - other *cis,cis* (*c,c*) and *trans*,*trans* (*t,t*) CLA isomers were detected. The composition of the CLA isomer mixture was, %: *t11t13* - 2.9; *t10t12* - 5.1; *t9t11* - 4.3; *t8t10* - 2.9; *c11t13* - 13.4; *t10c12* - 28; *c9t11* - 28.6; *c8t10* - 9.6; *c11c13* - 1.6, *c10c12* - 1.5; *c9c11* - 1.4; *c8c10* - 0.7. The ratio of the concentration of *c9t11*CLA to *t10c12*CLA in the CLA isomer mixture was 1.0242. The composition CLA isomer(s) was assessed using an Ag^+-HPLC system (Czauderna et al., 2003).

Preparation of blood plasma for fatty acid HPLC analysis

Blood samples from rats were collected into heparinized tubes (kept in an ice bath) and centrifuged at 1500-1700 *g* for 15 min (at 2-4°C). Blood plasma was stored at -28°C. On the day of analysis, 1 ml of plasma (0-1°C) was deproteinized with 1 ml of 7% cooled solution (0-1°C) of trichloroacetic acid and centrifuged at 2000 *g* for 10 min (at 2-4°C) and 200-450 µl of supernatant were used for saponification as below.

Saponification of samples

All freeze-dried organ tissue samples (45-55 mg) and deproteinized plasma (100 µl) from rats were hydrolysed with 3.5 ml of 2 M NaOH at ~85°C for 30 min.

Table 1. Initial average body weight of rats[1], composition of the diet enriched in the CLA isomers and Se, total feed intake, body weight gain, Se, Zn, Fe, Mg and Ca contents in whole blood as well as sum of all assayed fatty acids (ΣFAs) concentration in blood plasma and spleen

Group	Supplement	Level of additives in the diet	Total feed intake[2] g	Average body weight of rats, g	The number of rats per group	[2]Body weight gain, g	Se in whole blood[4]	Zn in whole blood[4]	Fe in whole blood[4]	Mg in whole blood[4]	Ca in whole blood[4]	ΣFAs[3] plasma	ΣFAs[3] spleen
							μg/g	μg/g	mg/g	mg/g	μg/g	μg/g	mg/g
1	-	-	435[abc]	184.9	8	59.4[a]	1.84	17.3	2.33	198	400	200[a]	26.6
2_{+Se}	Se	2 ppm	433	185.3	8	52.8[b]	2.10	16.4	2.22	183	407	355	15.6
3	CLA isomer mixture[3]	1 %	427[a]	184.4	7	54.8	1.96	17.7	2.37	200	442	456	19.2[A]
4	*cis-9trans-11* isomer *(c9t10)*	1 %	440[d]	185.6	7	59.0	2.04	24.7	2.47	222	465	297	12.3
5	*trans-10cis-12* isomer *(t10c12)*	1 %	420	184.4	7	54.1	1.96	18.0	2.42	207	421	274	20.1
6	CLA isomer mixture	2 %	413[bd]	183.3	7	56.8	2.02	17.6	2.25	214	480	270	30.2
3_{+Se}	Se CLA isomer mixture	2 ppm 1 %	425[d]	181.9	7	56.1	2.33	19.0	2.37	199	473	304	13.0[A]
4_{+Se}	Se *cis-9trans-11* isomer *(c9t11)*	2 ppm 1 %	430	184.2	7	55.5[a]	2.21	17.9	2.26	194	479	320	15.5
5_{+Se}	Se *trans-10cis-12* isomer *(t10c12)*	2 ppm 1 %	436	183.8	7	62.2[b]	1.96	17.7	2.27	172	402	551[a]	18.2
6_{+Se}	Se CLA isomer mixture	2 ppm 2 %	415[cd]	182.8	7	58.4	1.84	14.7	1.85	109	247	342	23.5

[1] body weight of individually adapted rats after one week of submaintenance feeding (in parenthesis - number of rats in a group). Initial body mass of rats did not differ statistically differ among group at the $P<0.1$ level

[2] during 4 weeks of feeding with the CLA and/or Se; means in columns with the same letter are significantly different at [a,b] $P<0.05$

[3] sum of detected saturated (SFA), mono- (MUFA), polyunsaturated fatty acids (PUFA) and CLA isomers

[4] determined in pooled samples from rats fed the same diets

The hydrolysates were acidified with 4 M HCl to pH ~2 and then free fatty acids were extracted four times with 4 ml portions of dichloromethane. The lower organic layer was dried with Na_2SO_4 (~100 mg) and then the organic solvent was removed under a stream of argon (Czauderna et al., 2005b). Afterwards the residue (I) was used for derivatization as below or re-dissolved in 1 ml of dichloromethane and 20-30 µl of the resulting solution were injected onto the silver-ion exchange columns (Ag+-HPLC system I). The mobile phase of 1.6% acetic acid and 0.0125% acetonitrile in n-hexane was chosen as the optimum mobile phase for fractionation of underivatized fatty acids containing conjugated double bonds (Czauderna et al., 2003). Analyses were performed using an isocratic elution program (flow-rate of 1 ml/min) and UV detection at 234 nm, column temperature 25-28°C.

Derivatization procedures

To a residue (I) in a reacti-vial, 0.5 ml of dibromacetophenone (48 g/l in acetone) and 60 µl of triethylamine were added. The resulting solution was mixed again and reacted for 30 min at 40°C (Czauderna et al., 2004b). The processed samples were then injected onto C_{18} Nova Pak columns (Czauderna et al., 2004b). The binary gradient elution program was used for analysis of all derivatized fatty acids in standards and biological samples (Czauderna et al., 2004b). Injection volumes were 5-20 µl. The maximum pressure of HPLC systems was 38.5 MPa. Fatty acid derivative peaks were identified by the retention time of processed standards injected separately and by adding standard solutions to biological samples. Moreover, saturated fatty acids were differentiated from unsaturated fatty acids and conjugated fatty acids (e.g., CLA isomers) by the use of a photodiode array detector (Czauderna et al., 2003, 2004b).

The concentrations of Se, Zn, Fe, Ca and Mg in whole blood and tissue of spleens were determined by atomic absorption spectrometry (AAS) (PU9100X Atomic Absorption Spectrometer, UNICAM, Philips).

Statistical analyses of the effects of Se or the CLA isomer(s) in the diets were conducted using the nonparametric Mann-Whitney U test for comparing pairs in an independent experimental group (one-factor analysis), while statistical analyses of the interaction between the CLA isomer(s) and Se were performed using two-factorial ANOVA (CLA isomer(s)×Se). The statistical analyses were performed using the Statistica v. 6 package (Statistica, 2002).

RESULTS AND DISCUSSION

Effect of experimental diets on mineral constituents in blood plasma and spleen

Although Se is an essential element for antioxidant and thyroid hormone function, supplementation of inorganic Se (as selenate) has also been shown to markedly alter the body weight gain, feed intake, and accumulation of Se, Zn, Fe, Mg, Ca, and several fatty acids in some organs of rats (Tables 1-6). No macroscopic lesions or toxic symptoms of adding 2 ppm Se (as selenate) or 1 or 2% CLA isomer(s) were observed. This is consistent with our previous studies (Czauderna et al., 2004a,b) corroborating that only chronic feeding of inorganic Se compounds at a rate of more than 5 ppm can be teratogenic and hepatotoxic in animals and humans (Tapiero et al., 2003). In contrast to selenite, selenate is not as effectively incorporated into the body of animals and is less reactive and toxic.

The concentrations of mineral elements in whole blood of rats fed the diet enriched in CLA isomers and/or Se are given in Table 1. Although the concentrations of Se, Zn, Fe, Mg and Ca in blood were not significantly affected by the dietary CLA isomer(s) and Se, some of them (Se, Zn, Ca and Mg) tended to decrease in the blood of rats fed the CLA isomers(s). Interestingly, the concentration of Ca showed the highest increase in the blood of rats fed the diet enriched in *c9t11*CLA with or without Se and the diet with only 2% of the CLA isomer mixture. The current results are thus consistent with our previous study, in which supplementing 1 or 2% of CLA isomer(s) showed a tendency to elevate the concentration of Ca in the rat liver (Korniluk et al., 2005). In addition, the concentration of Ca exhibited a tendency to increase in the brain and heart tissues of rats fed the diet enriched in 1 or 2% of the CLA isomer mixture (data not presented); another striking result of our studies was the significant increase ($P<0.01$) in the Ca concentration in the brain tissue of rats fed the diet supplemented with both Se and the CLA isomer(s). In conclusion, our studies and the results obtained by Belury (2002) confirm that CLA isomers modulate the accumulation of Ca in animal bodies and, therefore, also have a potent effect on bone formation.

The presence of *c9t11*CLA in the diet most efficiently elevated the concentrations of Se, Zn, Fe and Mg in blood in comparison with other CLA isomer(s) added to the rat diet. In our study, adding selenate with or without CLA isomer(s) to the diet for 4 weeks resulted in a small increase of the Se concentration in blood (Table 1) and an only slightly higher increase in spleen tissues (Table 4). This is in good agreement with our previous results in which supplementing Se, regardless of the presence or absence of CLA isomer(s), also resulted in a slight increase of Se accumulation (~10%) in the liver

Table 2. The concentrations of fatty acids (FAs) in blood plasma of rats fed diets supplemented with the CLA isomers and/or Se

Group	Sum of CLA[1] µg/g	non-dietary CLA[1] µg/g	CFA[1] µg/g	*c9*C18:1[2] µg/g	LA[2] µg/g	C18:0 µg/g	Desaturase index[3] Δ9-index	PUFA µg/g	MUFA µg/g	C12:0-C16:0[4] µg/g	C14:0 - C18:0[5] µg/g	SFA[6] µg/g	Ratio of SFA/ MUFA	Ratio of SFA/PUFA
1	0[7]	0	0	19.5	24.7^{ab}	50.6	0.278	64^{ABabcd}	22.2^{Aa}	84	135^{a}	136^{a}	6.129	$2.421^{ABCDEab}$
2_{+Se}	0	0	0	37.4	42.0	85.6	0.304	120	45.8^{A}	84	170	235	5.136	2.172
3	45.6	4.34	2.41^{A}	20.2	30.9	124	0.140	128	29.5	59	96	101	11.086^{b}	2.206^{c}
4	42.3	2.19	1.59	22.0	28.0	54.7	0.537	181^{a}	26.8	56	110	116	4.328	0.849^{A}
5	43.6	3.85	3.91	17.9	32.6	57.3	0.239	128	21.1	85	142	146	6.936	1.252^{B}
6	67.3	6.97	7.13^{a}	14.2	33.3	46.6	0.233	156^{b}	21.3	64	110	114	5.372^{b}	0.813^{Cc}
3_{+Se}	48.2	5.48	13.6^{A}	19.6	37.4	57.4	0.248	154^{A}	27.8	86	145	150	5.409	1.122^{a}
	-	(NS)[8]	(**)	-	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)
4_{+Se}	69.5	4.44	3.19	24.0	42.1^{a}	58.0	0.293	175^{B}	27.9	83	141	145	5.196	0.831^{D}
	-	(NS)	(NS)	-	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)
5_{+Se}	62.1	7.71	5.74	36.7	54.1^{b}	80.5	0.181	228^{c}	56.5^{a}	139	303^{a}	323^{a}	5.724	1.440
	-	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)
6_{+Se}	79.8	10.45	3.34^{a}	25.4	42.2	54.6	0.318	185^{d}	28.7	96	150	156	5.446	0.960^{E}
	-	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)	(NS)

[1] all diets enriched in CLA isomers, regardless of the presence of Se, statistically increased CLA isomers and CFA (i.e. non-CLA fatty acids containing conjugated double bonds) contents compared with the control rats (Group 1) and Group 2_{+Se} (the significant difference at the $P<0.01$)

[2] oleic acid (i.e. *cis9*C18:1); LA - linoleic acid (i.e. *cis9,cis12*C18:2)

[3] *cis9*C18:1/C18:0 + *cis9*C18:1, where: *cis9*C18:1 - oleic acid (*c9*C18:1); C18:0 - stearic acid (the abbreviation:Δ9-index)

[4] a sum of saturated fatty acids: C12:0, C14:0 and C16:0; [5] a sum of saturated fatty acids: C14:0, C16:0 and C18:0

[6] a sum of SFA: C8:0 - caprylic acid; C10:0 - capric acid; C12:0 - lauric acid; C14:0 - myristic acid; C16:0 - palmitic acid; C18:0 - stearic acid

[7] below quantification limit (3 × the detection limit)

[8] in parenthesis - statistical analysis of data by ANOVA for two-factorials design, e.g.: CLA isomer(s) × Se

Table 3. Fatty acid (FA) concentrations in spleen tissue[1] of rats fed diets supplemented with the CLA isomers and/or Se

Group	Sum of CLA[2] mg/g	non-dietary CLA[4] µg/g	CFA[3] µg/g	*c9*C18:1 mg/g	LA mg/g	C18:0 mg/g	Desaturase index Δ9-index	PUFA mg/g	MUFA mg/g	C12:0-C16:0 mg/g	C14:0-C18:0 mg/g	SFA mg/g	Ratio of SFA/MUFA	Ratio of SFA/PUFA
1	0.145	32	240	2.51^{Aa}	2.44	2.31^{ab}	0.520^{Aab}	18.6	2.95^{Aa}	5.52^{Aab}	7.81^{Aab}	7.95^{Aab}	2.696	0.764
2_{+Se}	0.145	14	436^{a}	1.62	2.40	2.10	0.436	10.0^{a}	5.37^{c}	3.41^{a}	5.50^{a}	5.60^{a}	1.044^{a}	0.872^{a}
3	2.84^{a}	319	838	1.91^{b}	3.17^{A}	2.39^{c}	0.445	11.8^{b}	2.47^{b}	4.94^{B}	7.32^{B}	7.42^{B}	3.002	0.653
4	1.51^{b}	179^{A}	611	1.42^{a}	1.85	1.91^{a}	0.426^{a}	6.8	1.80^{a}	3.46^{b}	5.36^{b}	5.45^{b}	3.021	0.815
5	3.51	402	1069	2.02	3.06	2.63	0.435	12.3	2.54	5.12	7.73	7.84	3.090	0.649
6	5.31	656	881	1.75	4.08	2.27	0.435^{b}	23.6	2.63	4.26	6.52	6.61	2.514	0.444
3_{+Se}	1.76^{a}	250	769^{a}	1.42^{Ab}	2.15^{A}	1.86^{bc}	0.433^{A}	7.8^{b}	1.78^{Abc}	3.34^{AB}	5.19^{AB}	5.27^{AB}	2.968^{a}	0.685
	-	(NS)[5]	(NS)	-	(NS)	-	(NS)	(NS)	(NS)	(NS)	(**)	(NS)	(NS)	(NS)
4_{+Se}	2.65^{b}	245^{A}	526	1.83	2.13	2.07	0.469	9.2	2.27	4.20	6.26	6.34	2.793	0.718
	-	(**)	(NS)	-	(NS)	-	(NS)	(NS)	(NS)	(**)	(NS)	(**)	(NS)	(NS)
5_{+Se}	3.10	339	728	1.86	2.56	2.33	0.443	10.8	2.44	4.96	7.27	7.39	3.025	0.736
	-	(NS)	(NS)	-	(NS)	-	(NS)	(NS)	(NS)	(NS)	(**)	(NS)	(NS)	(NS)
6_{+Se}	6.87	939	757	2.19	3.08	2.37	0.480	16.0^{a}	2.76	5.07	7.41	7.51	2.723	0.495^{a}
	-	(NS)	(NS)	-	(NS)	-	(NS)	(NS)	(NS)	(**)	(NS)	(**)	(NS)	(NS)

[1] lyophilized samples

[2] all diets enriched in CLA isomer(s), regardless the presence of Se, increased CLA isomers content compared with the control rat (Group 1) and group enriched in Se (2_{+Se}) (the significant difference at the $P<0.01$)

[3] all diets enriched in CLA isomer(s) or/and Se increased CFA isomers content compared with the control rat (Group 1) (the significant difference at the $P<0.01$)

[4] the sum of non-dietary CLA isomers detected in assayed rat organs

[5] in parenthesis - statistical analysis of data by ANOVA for two-factorials design, e.g.: CLA isomer(s) × Se

Table 4. Spleen mass, Se, Zn, Fe, Mg and Ca levels in spleen tissue[1], CLA isomer concentrations in blood plasma and spleen tissue of rats[2] fed experimental diets

Group	Spleen mass, g	Se	Zn	Fe	Mg	Ca	*t10c12*	*c9t11*	*ct/tc*	*c,c*	*t,t*	*t10c12*	*c9t11*	*ct/tc*	*c,c*	*t,t*
		content in spleen, µg/g					content of dietary CLA isomers in spleen, mg/g					content of dietary CLA isomers in plasma, µg/g				
1	0.464^{Aa}	1.15	41.9	3869	669	190	0.033	0.037	0.077	0.006	0.031	0	0	0	0	0
2_{+Se}	0.486^{b}	1.55	41.9	4012	682	168	0.041	0.043	0.097	0.005	0.029	0	0	0	0	0
3[3]	0.490	1.11	41.0	4285	681	133	0.838^{a}	0.709^{a}	1.84^{a}	0.053	0.633^{A}	10.0	12.1	23.5	2.66	15.2
4	0.479	1.07	45.3	3344	659	135	0.257	0.852	1.12	0.027^{A}	0.188^{B}	0.94	28.7	29.6	1.11	9.40
5	0.496	1.09	40.0	4360	669	141	2.03	0.064	2.61	0.084	0.408	31.2	0.53	32.0	1.27	6.55
6[3]	0.520	1.12	41.9	3887	689	130	1.73	1.22	3.65	0.169	0.840	17.0	24.3	44.1	2.99	13.3
3_{+Se}[3]	0.471	1.66	44.6	4829	740	176	0.533^{a}	0.396^{a}	1.12^{a}	0.066	0.306^{A}	12.7	16.3	30.9	2.34	9.51
4_{+Se}	0.512^{a}	1.66	46.1	4150	792	165	0.366	1.48	1.87	0.100^{A}	0.390^{B}	1.73	48.4	50.3	1.27	13.5
5_{+Se}	0.515^{Ab}	1.60	47.5	5195	792	162	1.68	0.093	2.34	0.085	0.338	33.0	1.82	37.1	2.12	15.2
6_{+Se}[3]	0.518	1.43	43.2	3806	724	159	2.14	1.63	4.46	0.222	1.25	20.6	25.5	49.0	3.97	16.4

[1] determined in samples obtaining by combination of spleen from each rats fed the same diets (i.e. the spleen rat sample); means in columns with the same letter are statistically different (the significant difference at the ^{a,b} $P<0.05$ or ^{A,B} $P<0.01$

[2] CLA isomer contents in spleen and plasma of control and 2_{+Se} rats are below quantification limit; *c,c* and *t,t* - *cis,cis* and *trans,trans* isomers

[3] dietary CLA isomer mixture containing: 15.2% - *t,t*CLA isomers; 5.2% - *c,c*CLA isomers; 79.6% - the sum of *c9t11*CLA and *t10c12*CLA

Table 5. The concentrations of CLA isomers and other conjugated non-CLA fatty acids (CFA) in pancreas and heart tissues of rats fed experimental diets[1]

Group	Sum of CLA isomers	CFA	Non-dietary CLA	*t10c12*	*c9t11*	*ct/tc*	*c,c*	*t,t*	Sum of CLA isomers	CFA	Non-dietary CLA	*t10c12*	*c9t11*	*ct/tc*	*c,c*	*t,t*
	content of dietary CLA isomers								content of dietary CLA isomers							
	concentration of conjugated FAs in pancreas, mg/g								concentration of conjugated FAs in heart, mg/g							
1	1.26	0.065	0.157	0.249	0.590	0.906	0.019	0.173	0.078	0.306	0.005	0.012	0.038	0.052	0.001	0.020
2_{+Se}	0.886	0.132	0.181	0.180	0.334	0.547	0.029	0.129	0.012	0.167	0.002	0.002	0.005	0.006	0.001	0.002
3	31.4	1.78	2.50	8.73	12.4	23.0	0	5.92	3.13^{A}	1.29	0.231	0.814	0.884	2.12	0.108	0.675
4	36.4	1.49	0.929^{a}	2.46	29.3	32.2	0.185^{a}	3.09	4.96	0.533	0.214	0.129	3.72	3.90	0.196	0.654
5	28.9	1.65	1.57	19.6	0.768^{a}	24.7	0.222	2.42	2.74	0.735	0.250	1.67	0.113	1.98	0.087	0.423
6	48.3	2.98	3.67	13.6	19.3	35.6	0.396	8.57	9.65^{a}	1.52	0.728	2.48	2.93	6.57	0.350	2.00
3_{+Se}	37.4	2.17	2.69	10.1	16.1	28.5	0.119	6.09	3.81^{A}	1.76	0.309	0.917	0.962	2.36	0.118	1.02
4_{+Se}	41.3	1.52	1.48^{a}	2.08	31.9	35.6	0.884^{a}	3.24	8.96	1.70	0.402	0.199	6.85	7.17	0.288	1.10
5_{+Se}	36.6	1.88	2.10	22.2	1.85^{a}	32.3	0.236	1.93	2.68	3.59	0.151	1.63	0.111	1.92	0.113	0.491
6_{+Se}	63.1	2.81	4.23	18.5	24.9	47.4	0.954	10.5	6.93^{a}	1.25	0.558	1.83	1.94	4.58	0.317	1.47

[1] means in columns with the same letter are statistically different (the significant difference at ^{a,b} $P<0.05$ or ^{A,B} $P<0.01$)

Table 6. The concentrations of CLA isomers and other conjugated non-CLA fatty acids (CFA) in kidney and brain tissues of rats fed experimental diets[1]

Group	Sum of CLA isomers	CFA	Non-dietary CLA	*t10c12*	*c9t11*	*ct/tc*	*c,c*	*t,t*	Sum of CLA isomers	CFA	Non-dietary CLA	*t10c12*	*c9t11*	*ct/tc*	*c,c*	*t,t*
	content of dietary CLA isomers								content of dietary CLA isomers							
	concentration of conjugated FAs in kidneys, mg/g								concentration of conjugated FAs in brain, µg/g							
1	0.170	0.053	0.017	0.048	0.065	0.117	0.010	0.026	0[2]	0	0	0	0	0	0	0
2_{+Se}	0.838	0.225	0.131	0.157	0.350	0.530	0.045	0.133	0	0	0	0	0	0	0	0
3	6.32	0.750	0.454	1.59	2.41	4.35	0.283	1.23	218	0	53	49	64	127	9	30
4	10.1	2.28	0.382	0.370	7.82	8.21	0.296	1.20	267	13.3	53	38	140	180	2	32
5	8.89[a]	0.720	0.619[a]	6.36[a]	0.293	6.99	0.229[a]	1.06[a]	340	0	60	117	115	248	0[a]	33
6	15.8	2.34[a]	1.29[b]	4.30	6.20[a]	11.4	0.486	2.62	423	0	92	120	114	254	16	61[a]
3_{+Se}	7.16	0.693	0.450	1.97	2.59	5.05	0.314	1.35	253	17.8	54	60	75	158	8	33
4_{+Se}	8.41	4.64	0.314	0.250	6.64	6.90	0.214	0.988	476	9.4	83	82	236	352	0	41
5_{+Se}	5.53[a]	2.07	0.434[a]	3.74[a]	0.368	4.33	0.139[a]	0.625[a]	342	0	56	124	93	238	12[a]	36
6_{+Se}	11.7	0.874[a]	0.750[b]	3.42	4.12[a]	8.41	0.509	2.03	289	0	81	48	81	155	15	38[a]

[1] means in columns with the same letter are statistically different (the significant difference at $^{a,b}P<0.05$ or $^{A,B}P<0.01$)

[2] CLA isomer contents in brain of control and 2_{+Se} rats are below quantification limit; *c,c* and *t,t* - *cis,cis* and *trans,trans* isomers

(Czauderna et al., 2005a) and femoral muscles of rats (Czauderna et al., 2004b) in comparison with the control animals. Surprisingly, Se supplementation to the diet enriched with a higher level of the CLA isomer mixture usually most efficiently decreased the concentration of all assayed elements in blood and the liver tissue (Czauderna et al., 2005a) in comparison with the control and other experimental groups. This result suggests that in rats, during simultaneous supplementation of 2% CLA isomer mixture and Se, major changes occurred in the metabolism of the administered nutrient. This influence on metabolism is consistent with the significant decrease in total feed intake as well as with the tendency to decrease the body weight gain of rats fed diets enriched with the 2% CLA isomer mixture and Se.

Influence of experimental diets on the fatty acid composition of rat organ tissues

In agreement with our previous studies (Czauderna et al., 2004a,b; Korniluk et al., 2006), massive changes of the concentration of fatty acids in the examined tissues of rat organs and blood plasma were observed. As can be seen from the results summarized in Table 1, all supplemented diets showed a tendency or statistically significant increase in the concentration of the sum of all assayed FAs in plasma. As could be expected, feeding the diet enriched in *t10c12*CLA and Se resulted in a higher increase of FAs in plasma; at the same time, these findings are in accordance with other studies (Czauderna et al., 2004a,b; Korniluk et al., 2006). Similarly, the diet containing *t10c12*CLA and Se significantly increased the concentration of total FAs in femoral muscles in rats (Czauderna et al., 2004b) as well as the weight of the liver, pancreas, spleen and heart (Czauderna et al., 2003), and, consequently, these supplements in the rat diets resulted in the highest body weight gain. The increased weight of these organs is probably due to stimulation of lipoprotein or protein synthesis in the examined rats (West et al., 1998; Czauderna et al., 2004a,b). It seems reasonable to assume that the interaction between Se and *t10c12*CLA or its metabolites resulted in the most efficient elevation of rat body weight accretion, consequently, most effectively decreased energy expenditure.

CLA isomer accumulation in blood plasma and examined organs

As could be expected, feeding the diets containing the CLA isomer(s) significantly stimulated the accumulation of all CLA isomers and other non-CLA conjugated FAs (CFA) in blood plasma and tissues of spleen, pancreas heart, kidneys and brain (i.e. dietary origin and due to endogenous synthesis) (Tables 2-6). This stimulating effect of the CLA isomer(s) is usually stronger in

the plasma, spleen and pancreas of rats fed the diets containing both the CLA isomer(s) and Se. Detailed analyses of our Ag^+-HPLC chromatograms revealed that the accumulation of CLA isomers in rat plasma and organs depended on the geometrical form of the administered isomer. Our results clearly demonstrate that the accumulation of *t10c12*CLA and *c9t11*CLA in all examined rat organs and plasma was selective. This is in good agreement with our previous studies (Czauderna et al., 2004a,b; Korniluk et al., 2006), in which *t10c12*CLA and *t10t12*CLA concentrations also tended to be lower than those of *c9t11* and *t9t11* isomers in the liver and femoral muscles of rats in comparison with the composition of the CLA isomer mixture supplemented to the diet (i.e. the *c9t11*CLA:*t10c12*CLA concentration ratio in the dietary CLA isomer mixture: 1.0242). In our current study, *c9t11*CLA was preferentially accumulated in plasma, pancreas, heart, kidneys and brain tissues of rats fed the diet enriched in 1 or 2% of the CLA isomer mixture, regardless of the presence of Se. Thus, we can suggest that in these rat organ tissues, *t10c12*CLA and *t10t12*CLA may be more efficiently metabolized by the cells than their 9,11 isomers. Interestingly, in spleen tissue, the abundance of *t10c12*CLA was higher in comparison with the concentration of *c9t11*CLA, regardless of whether Se was supplemented or not. Possible explanations may be that *t10c12*CLA is preferentially accumulated in spleen cells or that *c9t11*CLA is more rapidly metabolized to long-chain conjugated FAs (CFA), e.g., *cis6,cis9,cis11*C18:3, *cis6,trans10,cis12*C18:3, *cis8,cis11,trans13*C20:3, *cis8,trans12*, *cis14*C20:3, *cis5,cis8,cis11,trans13*C20:4 and *cis5,cis8,trans12,cis14*C20:4. Similarly, in all other examined organ tissues and plasma of rats fed the diet containing the CLA isomer(s) with/without Se, the lowest accumulation of CFA was found in brain tissue. Moreover, the accumulation of all CLA isomers in brain tissue was significantly lower as compared with the accumulation of all CLA isomers in plasma and other organ tissues of rats fed the diet enriched in the CLA isomers. Additionally, with regard to other fatty acids assayed in the brain, changes in the concentration of fatty acids in brain tissue of rats fed the experimental diets were smaller than the changes in other rat organ tissues and blood plasma. Interestingly, in all examined organs and plasma of rats fed the diet enriched in the CLA isomers (regardless of the presence of Se) the presence of CLA isomers possessing different positional and/or geometrical chemical formulas (i.e. non-dietary CLA isomers), as compared with the geometrical and positional chemical formula of dietary CLA isomers, was found. This is consistent with our previous results in rats (Czauderna et al., 2004a) showing that the supplementing CLA isomers also resulted in accumulation of non-dietary CLA isomers in the liver tissue.

Detailed analysis of the Ag^+-chromatograms and data summarized in Tables 4-6 (groups 3 and 6) revealed that the percentage of dietary *t,t*CLA isomers in

pancreas, kidney, brain and liver tissues (Czauderna et al., 2004a) is smaller (~10%) in comparison with their respective percentages in the dietary CLA isomer mixture (i.e. the administered CLA isomer mixture containing 15.2% *t,t*CLA isomers). This effect was also observed in the organ tissues of rats fed the diets enriched in both Se and the 1-2% CLA isomer mixture (groups 3_{+Se} and 6_{+Se}). Thus, our current experiment demonstrated that dietary *t,t*CLA isomers are preferentially metabolized in the cells of these organ tissues in comparison with other the dietary isomers (i.e. *c,c*; *c9t11* and *t10c12*CLA isomers). Considering the above results, it may be hypothesized that *t,t*CLA isomers are catabolized more slowly and are poor substrates for β-oxidation. This is consistent with the results obtained by Yang et al. (2002) showing that *t,t*CLA isomers are favourably incorporated into membrane phospholipids due to their geometrical configuration. In contrast, only in spleen tissue and blood plasma of rats fed the diet containing the CLA isomer mixture, regardless of the presence of Se, was the percentage of *t,t*CLA isomers similar to the percentage of the *t10c12*CLA isomer in the supplemented CLA isomer mixture. This effect could be related to more efficient metabolism of *c9t11*CLA in spleen tissue in comparison with the capacity of *t10c12* metabolism and that of other geometrical and positional CLA isomers.

Influence of experimental diets on the non-conjugated fatty acid constituent in rat tissues

The relationship between the experimental diets and the concentrations of other fatty acids are summarized in Tables 1-8. The plasma PUFA levels reflect the fact that all of the diets supplemented with CLA isomer(s) usually showed a tendency towards or significantly stimulated the accumulation of polyunsaturated fatty acids in pancreas, heart and kidney tissues of rats (Tables 2, 7 and 8). Interestingly, addition of Se to the diets containing CLA isomer(s) generally resulted in a higher increase of PUFA in plasma and pancreas tissue than when only CLA isomer(s) were supplemented. At the same time, the SFA/PUFA ratio was usually lower in the plasma, spleen, pancreas, heart, kidney and brain tissues of rats fed the diets enriched in the CLA isomer(s) in comparison with the control group. This is in good agreement with our previous study (Czauderna et al., 2004b) in rats showing that supplementing CLA isomer(s) to the diet, with or without Se, resulted in an increased PUFA concentration, as well as in a higher (MUFA+PUFA)/SFA ratio in femoral muscles of rats. Consequently, these changes in the concentration of MUFA and PUFA and the SFA/PUFA ratio generally lead to improvements in the nutritional quality of meat of monogastric farm animals in terms of human health. Surprisingly, only in brain tissue did the addition of Se to CLA-supplemented diets always increase the SFA/PUFA ratio, moreover, the value

Table 7. The concentrations of conjugated fatty acids (CFA), PUFA, MUFA, LA and selected saturated fatty acids (SFA) in pancreas and heart tissues of rats fed diets with ($_{+Se}$)/without ($_{-Se}$) Se[1]

Item / Group	Pancreas, mg/g					Heart, mg/g				
	1	3	4	5	6	1	3	4	5	6
CFA_{+Se}		2.17	1.52	1.88	2.81		1.71	1.70	3.59	1.25
Interaction[2]	0.06^{ABCD}	NS	NS	NS	NS	0.31^{ABCD}	NS	* *	* *	NS
CFA_{-Se}[3,4]		1.78^{A}	1.49^{B}	1.65^{C}	2.98^{D}		1.29^{A}	$.53^{B}$	0.73^{C}	1.51^{D}
$_{other}CLA_{+Se}$	0.16^{A}	2.69	1.48	2.10	4.23	0.01^{ABCD}	0.31	0.40	0.15	0.56
interaction	^{BCD}	NS	NS	NS	NS		NS	NS	NS	NS
$_{other}CLA_{-Se}$[3]		2.47^{A}	$.93^{B}$	1.57^{C}	3.67^{D}		0.23^{A}	0.21^{B}	0.25^{C}	0.73^{D}
LA_{+Se}		21.0	26.8	24.7	19.8		5.52	8.47	6.13	7.42
interaction	18.2	NS	NS	NS	NS	5.96^{A}	NS	NS	NS	NS
LA_{-Se}[3]		20.7	18.7	20.7	17.5		7.11	6.24	7.03	10.5^{A}
$\Delta 9\text{-index}_{+Se}$		0.77	0.80	0.78	0.76		0.40	0.68	0.36	0.46
interaction	83^{ABC}	NS	NS	NS	NS	0.43	NS	NS	NS	NS
$\Delta 9\text{-index}_{-Se}$[3]		0.76^{A}	0.82	0.74^{B}	0.72^{C}		0.42	0.56	0.43	0.46
$PUFA_{+Se}$		91	191	100	116		15.9	29.1	19.2	22.1
interaction	$54^{aA\ bc}$	NS	NS	NS	NS	15.0^{A}	* *	NS	NS	NS
$PUFA_{-Se}$[3]		83^{a}	85^{A}	78^{b}	93^{c}		18.1	20.0	16.5	33.2^{A}
$MUFA_{+Se}$		25.0	28.5	30.5	24.1		3.75	7.41	5.01	3.28
interaction	25.8	NS	NS	NS	NS	5.43	NS	NS	NS	* *
$MUFA_{-Se}$[3]		21.9	22.2	20.2	16.8		4.23	5.63	3.52	7.25
C12:0-16:0$_{+Se}$		21.3	28.0	24.2	19.5		4.93	5.26	6.21	4.52
interaction	22.6	NS	* *	NS	NS	5.50	NS	NS	* *	NS
C12:0-C16:0$_{-Se}$[3]		21.2	21.9	20.9	20.8		5.81	4.80	4.28	6.95
C14:0-C18:0$_{+Se}$		25.9	33.0	29.4	24.0		9.01	7.78	12.4	7.37
interaction	26.8	NS	* *	NS	NS	10.2	NS	* *	NS	NS
C14:0-C18:0$_{-Se}$[3]		25.9	25.8	25.5	25.5		9.91	8.03	7.49	11.9
SFA_{+Se}		26.3	33.5	29.9	24.4		9.15	7.89	12.5	7.41
interaction	27.2	NS	* *	NS	NS	10.3	NS	NS	* *	NS
SFA_{-Se}		26.2	26.2	25.9	25.8		10.0	8.14	7.57	12.0
$SFA/MUFA_{+Se}$		1.24	1.28	1.13	1.05		2.64	1.49	3.26	2.33
interaction	1.11^{a}	NS	NS	NS	NS	2.66	NS	NS	NS	NS
$SFA/MUFA_{-Se}$[3]		1.46	1.24	1.44	1.76^{a}		2.58	1.93	2.32	1.80
$SFA/PUFA_{+Se}$		0.31	0.30	0.31	0.22		0.60	0.31	0.74	0.33
interaction	0.52^{ABCa}	NS	NS	NS	NS	0.71^{ab}	NS	NS	* *	NS
$SFA/PUFA_{-Se}$[3]		0.34^{A}	0.32^{B}	0.35^{C}	0.32^{a}		0.59	0.51	0.46^{a}	0.37^{b}
ΣFAs_{+Se}		117	225	130	140		23.8	21.5	32.3	29.5
interaction	81^{a}	NS	NS	NS	NS	25.4	NS	NS	* *	NS
ΣFAs_{-Se}[3]		109^{a}	111^{a}	104	119^{a}		24.3	28.2	19.6	37.3

[1] abbreviation for FA(s) and other items see Tables 1, 2 and 3
[2] interaction: the CLA isomer(s) × Se (ANOVA analyses)
[3] the content of FA(s) in group 3, 4, 5 and 6, respectively; i.e. rats fed diets without Se ($_{-Se}$)
[4] means in rows with the same letter are statistically different

Table 8. The concentrations of conjugated fatty acids (CFA), PUFA, MUFA, LA and selected saturated fatty acids (SFA) in kidneys and brain tissues of rats fed diets with ($_{+Se}$)/without ($_{-Se}$) Se[1]

Item / Group	Kidneys, mg/g					Brain, mg/g				
	1	3	4	5	6	1	3	4	5	6
CFA_{+Se}		0.69	4.64	2.07	0.87		0.018	0.011	0	0
interaction[2]	0.05	NS	NS	**	NS	0^{a}	-	-	-	-
CFA_{-Se}[3,4]		0.75	2.28	0.72	2.34		0	0.013^{a}	0	0
$_{other}CLA_{+Se}$		0.450	0.314	0.434	0.750	0^{ABCD}	0.054	0.083	0.056	0.081
interaction	0.017^{ABCD}	NS	NS	NS	**		NS	NS	NS	NS
$_{other}CLA_{-Se}$		0.45^{A}	0.38^{B}	0.62^{C}	1.29^{D}		0.053^{A}	0.053^{B}	0.060^{C}	0.092^{D}
LA_{+Se}		7.39	5.16	6.15	6.43		0.50	0.68	0.56	0.48
interaction	6.87	NS	NS	NS	NS	0.46	NS	NS	NS	NS
LA_{-Se}		7.20	6.92	7.23	6.34		0.48	0.54	0.69	0.57
$\Delta 9\text{-index}_{+Se}$		0.682	0.752	0.647	0.650		0.622	0.634	0.625	0.620
interaction	0.785	NS	NS	NS	NS	0.619^{a}	NS	NS	NS	NS
$\Delta 9\text{-index}_{-Se}$		0.692	0.716	0.782	0.697		0.565	0.640^{a}	0.576	0.602
$PUFA_{+Se}$		28.1	29.8	24.6	31.5		34.5	37.8	33.9	35.0
interaction	24.9^{AB}	NS	NS	NS	NS	35.2	NS	NS	NS	NS
$PUFA_{-Se}$		25.3	34.4^{A}	28.2	35.6^{B}		36.2	40.6	32.0	35.4
$MUFA_{+Se}$		8.76	9.44	7.28	7.74		12.4	13.7	12.1	12.1
interaction	12.7^{aA}	NS	NS	NS	NS	12.1	NS	NS	NS	NS
$MUFA_{-Se}$		7.74^{a}	11.8	7.09^{A}	7.62^{b}		13.6	13.9	12.8	11.8
$C12:0–C16:0_{+Se}$		6.24	5.46	5.19	7.29		8.59	8.77	7.69	8.53
interaction	6.98	NS	NS	NS	NS	9.10^{A}	**	NS	**	NS
$C12:0–C16:0_{-Se}$		5.96	7.55	5.55	6.98		11.4^{A}	10.7	10.1	9.50
$C14:0\text{-}C18:0_{+Se}$		8.93	7.72	6.84	10.0		14.7	15.1	13.5	14.6
interaction	9.42	NS	NS	NS	NS	15.6^{a}	**	NS	**	NS
$C14:0\text{-}C18:0_{-Se}$		8.36	10.7	7.02	9.32		20.0^{a}	17.5	18.1	16.2
SFA_{+Se}		9.06	7.89	6.96	10.2		14.9	15.3	13.7	14.8
interaction	9.56	NS	NS	NS	NS	15.8^{a}	NS	NS	**	**
SFA_{-Se}		8.47	10.8	7.15	9.46		20.3^{a}	17.7	18.3	16.4
$SFA/MUFA_{+Se}$		1.06	0.86	0.97	1.38		1.20	1.11	1.13	1.22
interaction	$0.75^{ab\,A}$	NS	NS	NS	NS	1.31	**	NS	**	NS
$SFA/MUFA_{-Se}$		1.15	0.94^{a}	1.04^{b}	1.25^{A}		1.49	1.28	1.44	1.40
$SFA/PUFA_{+Se}$		0.33	0.27	0.28	0.39		0.43	0.40	0.40	0.42
interaction	0.38^{ab}	NS	NS	NS	NS	0.45^{a}	**	NS	**	NS
$SFA/PUFA_{-Se}$		0.34	0.33	0.26^{a}	0.27^{b}		0.57	0.44	0.64^{a}	0.46
ΣFAs_{+Se}		37.2	37.7	31.6	41.7		49	53	48	50
interaction	34.4^{ab}	NS	NS	NS	NS	51^{a}	NS	NS	NS	NS
ΣFAs_{-Se}		33.8	45.2^{a}	35.4	45.1^{b}		57^{a}	58	50	52

[1] abbreviation for FA(s) and other items see Tables 1-3 and 7
[2] interaction: the CLA isomer(s) x Se (ANOVA analyses)
[3] means in rows with the same letter are statistically different

of this ratio were greater, in contrast to their value in brain tissue of control rats and rats fed the diets containing only the CLA isomer(s).

As can be seen from the results in Tables 2, 3, 7 and 8, all of the diets with the CLA isomer(s) usually decreased the concentration of SFA in plasma, spleen, pancreas, heart and kidney tissues. On the other hand, no consistent influence on the SFA concentration was found in the brain tissue of rats fed the diets enriched in the CLA isomer(s), regardless of the presence of Se. Among quality parameters, two groups of saturated FAs are very important for the nutritional evaluation of animal fat: C12:0, C14:0, C16:0 (atherogenic) and C14:0, C16:0, C18:0 (thrombogenic), which are believed to be linked to coronary heart disease (CHD). In the present study, the concentrations of these two groups of fatty acids in plasma, spleen, pancreas, kidneys and brain tissues are summarized in Tables 2, 3, 7 and 8. These results clearly indicate that the dietary CLA isomer(s) usually decreased the concentration of both groups of SFA in plasma, pancreas, heart and kidney tissues. The importance of this data is due especially to the evidence for the physiological effects of CLA isomer(s), such as their antiatherogenic and antithrombotic action. The addition of Se to the diet containing the CLA isomer(s) slightly changed the accumulation of these fatty acids in comparison with supplementing the CLA isomer(s) alone. Surprisingly, the accumulation of these groups of fatty acids increased in the brain tissue of rats fed the diet with CLA isomer(s), but when both the CLA isomer(s) and Se were added, the concentration of both groups of SFA decreased in comparison with the control rats.

The diets enriched in the CLA isomer(s) usually showed a tendency or significantly decreased the concentration of MUFA as well as the capacity of Δ9-desaturase (i.e. Δ9-index) in plasma, spleen, pancreas and kidney tissues, while they did not result in any noticeable decrease in heart and brain tissues. The *t10c12*CLA isomer inhibits the activity and gene expression of Δ9-desaturase (Madron et al., 2002; Czauderna et al., 2004), thereby reducing endogenous synthesis of *c9t7*CLA and *c9t11*CLA, as well as *c9* monounsaturated fatty acids. Therefore, it seems necessary to assess the capacity of Δ9-desaturation using the Δ9 index (Tables 2, 3, 7 and 8). The current results are consistent with our previous studies (Czauderna et al., 2004a,b) showing that CLA isomer(s), *t10c12*CLA in particular, are responsible for the decrease in the concentration of monounsaturated fatty acids (especially *c9*C18:0 and *c9*C16:1) in the bodies of examined animals due to decreasing the Δ9 desaturation of such FAs as C16:0 and C18:0 (Wahle et al., 2004). The addition of Se to the diet enriched in the CLA isomer(s) usually resulted in a decrease in the value of the Δ9-index and of the MUFA concentration, however, the simultaneous presence of Se and the CLA isomer(s) exerted a less consistent influence on the capacity of Δ9 desaturation and MUFA concentration compared with the effect of the diet with only the

CLA isomer(s). Supplementation with *t10c12*CLA lowered the expression of the gene for this desaturase (Madron et al., 2002), thereby decreasing endogenous synthesis of *c9t11*CLA, e.g. in the mammary gland. Moreover, the *t10c12,* but not the *c9t11* isomer of CLA apparently also inhibited Δ6 and Δ5 desaturation of other unsaturated fatty acids like linoleic (LA) and α-linolenic acids (Wahle et al., 2004). Consequently, the concentrations of LA in plasma, spleen, heart, kidneys and brain tissues are higher in rats fed diets enriched with *t10c12*CLA or a CLA isomer mixture due to the lower yield of more unsaturated metabolites of LA.

CONCLUSIONS

The results point to the possibility that the physiological effects that CLA isoforms exert on rats are isomer-dependent. The *c9t11* isomer of C18:2 was preferentially accumulated in rat bodies, while the levels of *t10c12*CLA tended to be lower due to more efficient metabolism of 10,12 isoforms of CLA than of their 9,11 isomers. All diets containing CLA isomer(s), regardless of the presence of Se, resulted in a significant increase in the CLA isomer levels in the bodies of rats. Feeding Se and *t10c12*CLA considerably increased body weight gain, and spleen, heart, pancreas and liver weights, without any change in food intake. Moreover, these additives increased the accumulation of PUFA as well as CLA isomer(s) and their metabolites (CFA) in muscle, heart, pancreas tissues and in plasma. Therefore, we argue that diets enriched in these additives are able to improve feed conversion efficiency and the nutritive value of food for human and animal health. The changes in the plasma and organ tissue concentrations of FAs associated with atherogenicity and thrombogenicity in rats fed diets with CLA isomer(s) point to the beneficial nutritional value of meat from monogastric animals.

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REFERENCES

Belury M.A., 2002. Dietary conjugated linoleic acids in health: Physiological effects and mechanisms of action. Annu. Rev. Nutr. 22, 505-531

Crespo A.M., Reis M.A., Lanca M.J., 1995. Effect of selenium supplementation on polyunsaturated fatty acids in rats. Biol. Tr. Elem. Res. 47, 335-341

Czauderna M., Kowalczyk J., Bulska E., Boldižarova K., Niedźwiedzka K.M., Ruszczyńska A.,

Leng L'., 2005a. Effect of dietary CLA isomers on selenium, zinc, copper, chromium, magnesium and calcium levels in rat liver. J. Anim. Feed Sci. 14, Suppl. 1, 529-532

Czauderna M., Kowalczyk J., Korniluk K., Wąsowska I., 2005b. Improving the analysis of fatty acids using combination of gas chromatography and Ag^+ liquid chromatography. J. Anim. Feed Sci. 14, Suppl. 1, 263-266

Czauderna M., Kowalczyk J., Niedźwiedzka K.M., Wąsowska I., Pastuszewska B., Bulska E., Ruszczyńska A., 2004a. Liver and body mass gain, content of CLA isomers and other fatty acids in the liver of rats fed CLA isomers and selenium. J. Anim. Feed. Sci. 13, 353-369

Czauderna M., Kowalczyk J., Wąsowska I., Niedźwiedzka K.M., 2003. Determination of conjugated linoleic acid isomers by liquid chromatography and photodiode array detection. J. Anim. Feed Sci. 12, 269-382

Czauderna M., Kowalczyk J., Wąsowska I., Niedźwiedzka K.M., Pastuszewska B., 2004b. Conjugated linoleic acid (CLA) content and fatty acids composition of muscle in rats fed isomers of CLA and selenium. J. Anim. Feed Sci. 13, 183-196

Korniluk K., Czauderna M., Kowalczyk J., Mieczkowska A., Taciak M., Leng L'., 2006. Influence of conjugated linoleic acids isomers and selenium on growth performance, feed efficiency, and fatty acids profile of the liver in rats. J. Anim. Feed. Sci. 15, 131-146

Madrom M.S., Peterson D.G., Dwyer D.A., Corl B.A., Baumgard L.H., Beermann D.H., Bauman D.E., 2002. Effect of extruded full-fat soybeans on conjugated linoleic acid content of intramuscular, intermuscular, and subcutaneous fat in beef steers. J. Anim. Sci. 80, 1135-1143

Ohashi A., Matsushita Y., Shibata H., Kimura K., Miyashita K., Saito M., 2004. Conjugated linoleic deteriorates insulin resistance in obese/diabetic mice in association with decreased production of adiponectin and leptin. J. Nutr. Sci. Vitaminol. 50, 416-421

Ostrowska E., Suster D., Muralitharan M., Cross R.F., Leury B.J., Bauman E., Dushea E.R., 2003. Conjugated linoleic acid decreased fat accretion in pigs: evaluation by dual-energy X-ray absorptiometry. Brit. J. Nutr. 89, 219-229

Park Y., Storksonb J.M., Albrightb K.J., Liub W., Pariza M.W., 2005. Biological activities of conjugated fatty acids: conjugated eicosadienoic (conj. 20:2$\Delta^{c11,t13/t12,c14}$), eicosatrienoic (conj. 20:3$\Delta^{c8,t12,c14}$), and heneicosadienoic (conj. 21:2$\Delta^{c12,t14/c13,t15}$) acids and other metabolites of conjugated linoleic acid. Biochim. Biophys. Acta 1687, 120-129

Schweizer U., Streckfub F., Pelt P., Carlson B.A., Hatfield D.L., Köhrle J., Schomburg L., 2005. Hepatically derived selenoprotein P is a key factor to kidney but not for brain selenium supply. Biochem. J. 386, 221-226

Schomburg L., Schweizer U., Köhrle J., 2004. Selenium and selenoproteins in mammals: extraordinary, essential, enigmatic. CMLS, Cell Life Sci. 61, 1988-1995

Statistica by StatSoft, 2002. Web: www.statsoft.pl

Tapiero H., Townsend D.M., Tew K.D., 2003. The antioxidant role of selenium and seleno-compounds. Biomed. Pharmacotherapy 57, 134-144

Wahle K.W.J., Heys S.D., Rotondo D., 2004. Conjugated linoleic acids: are they beneficial or detrimental to health? (Review). Prog. Lipid Res. 43, 553-587

West D.B., DeLany J.P., Camet P.M., Blohm F., Truett A.A., Scimeca J., 1998. Effects of conjugated linoleic acid on body fat and energy metabolism in the mouse. Amer. J. Physiol. 275, R667-R672

Yang L., Yeung S.Y.V., Huang Y., Wang H.Q., Chen Z.Y., 2002. Preferential incorporation of trans,trans-conjugated linoleic acid isomers into the liver of suckling rats. Brit. J. Nutr. 87, 253-260