Dietary linseed oil and selenate affect the concentration of fatty acids in selected tissues of sheep

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ABSTRACT: The aim of the study was to determine the influence of the addition of 5% linseed oil (LO) and 2 μg Se as selenate (SeVI) per g of diet on body mass gain (BMG), feed conversion efficiency (FCE), and concentrations of fatty acids (FA), especially conjugated linoleic acid (CLA) isomers, in the liver, heart, *m. longissimus dorsi* (*MLD*), *m. biceps femoris* (*MBF*), subcutaneous fat (SF), perirenal fat (PF), and intermuscular fat (IF) of sheep. Each group comprised 5 lambs with an average initial body weight of 25 ± 2 kg. For 35 days the sheep were fed the control or experimental diets enriched with 5% LO, SeVI, or LO and SeVI combined. The diets with LO, regardless of the presence of SeVI, increased BMG, FCE, and the mass of *MBF, MLD*, and the liver. The LO treatment increased the concentration of total CLA isomers in the liver, SF, PF, and *MLD* in comparison with the control. Dietary LO with or without SeVI resulted in negligible changes in the concentration of total CLA isomers in *MBF.* The diet enriched with LO, irrespective of the presence of SeVI, increased the concentrations of *cis*- monounsaturated fatty acids (*cis*-MUFA), polyunsaturated fatty acids (PUFA), as well as unsaturated fatty acids (UFA) in the liver, heart, and especially in SF and PF (i.e. energy storage tissues) compared with the control group and SeVI-fed sheep. Dietary SeVI more effectively increased the capacity of Δ9-desaturation in all adipose tissues as the concentration of *cis9,trans11*CLA increased in these tissues. The addition of SeVI to the diet decreased the yield of the catabolism of fatty acids in these adipose tissues and both muscles compared with the control sheep or other experimental groups. The diets enriched with LO with or without SeVI increased the nutritional value of *MBF, MLD,* and adipose tissues as the ratio of unsaturated FA/saturated FA increased. Our results of a short-term study provide useful information for nutritionists carrying out further investigations to improve the nutritional quality of feed for ruminants as well as humans.

Keywords: selenium; linseed oil; fatty acid; lamb; liver; heart; muscle; adipose tissue

Most lipids added to rations can modify the fatty acid composition of body lipids, but not to a large extent (Mir et al., 2002; Demirel et al., 2004; Raes et al., 2004). According to Mir et al. (2002) oil can be added to diets for cattle to change the fatty acid composition of adipose tissues, but not of intramuscular fat. Dietary vegetable oils have substantially increased the concentration of conjugated linoleic acid (CLA) isomers in the bodies of examined animals (Aharoni et al., 2004; Czauderna et al., 2004a). Recent research has shown that supplementing high-concentrate finishing ratios with soyabean oil or other sources of linoleic acid (LA) is not an efficacious method for increasing

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*cis9,trans11*CLA in tissues (Beaulieu et al., 2002). On the other hand, in numerous experiments it has been found that feeding linseed oil (LO) rich in polyunsaturated fatty acids (PUFA) effectively decreased the content of saturated fatty acids (SFA) in meat and milk and increased the content of valuable monounsaturated fatty acids (MUFA) (*cis*- and *trans*-MUFA), PUFA, and other fatty acids (FA) containing conjugated double bonds (e.g. CLA isomers). Consequently, the percentage of undesirable FA, especially atherogenic and thrombogenic SFA (A-SFA and T-SFA), in ruminant meat or milk decreased. The main aim of modifying the fatty acid composition of meat or milk is to produce high-quality products meeting dietary recommendations for increased intake of PUFA, especially n-3PUFA, and reduced intake of A-SFA (C12:0, C14:0, and C16:0) and T-SFA (C14:0, C16:0, and C18:0). Many epidemiological and experimental studies (Niedźwiedzka et al., 2006; Murphy et al., 2007) documented that these SFA possess atherogenic and thrombogenic properties, while α-linolenic acid (αLNA) and its elongation and desaturation products (i.e. long-chain n-3PUFA; n-3LPUFA) improved anti-inflammatory status, immune response, and benefited the cardiovascular system by decreasing platelet aggregation, serum triglycerides, and cholesterol (Raes et al., 2004; Flachowsky et al., 2006). Considering the above facts, we hypothesized that dietary LO (a precursor of CLA isomers) stimulates the accumulation of UFA, especially fatty acids containing conjugated double bonds (CFA) in the body of sheep.

Diverse inorganic Se compounds like selenate or selenite are used as nutritional sources of Se. These compounds are metabolized to intermediates and then utilized for synthesis of proteins containing Se-cysteine (Se-Cys). The principal physiological roles of half of the Se-proteins are to maintain the appropriate metabolism of arachidonic acid and low concentrations of pre-oxides or free radicals within cells, thus decreasing oxidative stress and peroxidative damage of unsaturated fatty acids and lipids in living organisms (Tapiero et al., 2003; Shweizer et al., 2005; Yu et al., 2008).

Therefore, the aim of the present study was to explore the short-term effect of dietary LO and selenate (SeVI) on the profile of FA, particularly CFA, as well as interactions between dietary LO and SeVI in the liver, heart, muscles, and selected adipose fat tissues of sheep.

MATERIAL AND METHODS

Animals, housing, diets, and experimental design

Twenty male Polish Merino lambs with an average initial body mass (BM) of 25 ± 2 kg were allotted to 4 groups of 5 animals and housed individually. The animals were housed and handled in accordance with protocols approved by the Local Animal Care and Use Committee. During a 1-week preliminary period the sheep were given *ad libitum* access to a standard concentrate-hay diet with a vitamin and mineral premix (basal diet; Table 1). This basal diet contained: crude protein 120 g, crude fibre 12 g, and 11 MJ metabolizable energy in 1 kg dry mater. The total concentration of Se in the basal diet was 0.1 μg/g. For the next 35 days following the preliminary period the sheep were fed the basal diet (control group), the basal diet enriched with either 5% LO (LO group) or sodium selenate (SeVI) (total concentration of Se: 2 mg Se/kg diet; SE group), or the experimental diet with combined addition of 5% LO and SeVI (total concentration of Se: 2 mg Se/kg diet; LOSE group). The fatty acid profile of LO and other components of the diet are shown in Table 1. Weekly the rations accessible *ad libitum* were adjusted and the sheep weighed. At the end of the 35-day experiment the lambs were killed. Liver, heart, *m. longissimus dorsi* (*MLD*), *m. biceps femoris* (*MBF*), subcutaneous fat (SF), perirenal fat (PF), and intermuscular fat (IF) were removed, weighed, and frozen. Samples of liver, heart, and muscles were lyophilized and the obtained residues were stored in sealed tubes at –20°C until analysis. Concentrations of all fatty acids were calculated from lyophilized liver, heart, and muscle samples (i.e. dry matter; DM), whereas the fatty acid concentration in adipose tissues (SF, PF, and IF) was derived from fresh masses.

Chemicals

All of the chemicals used for saponification of samples, solvents used for eluting, and Waters HPLC equipment (Waters Corp., Milford, USA) were as described previously (Czauderna and Kowalczyk, 2002).

A CLA isomer mixture, *cis9,trans11*CLA (*c9t11*CLA), *trans10,cis12*CLA (*t10c12*CLA) (95–97%), and other fatty acid standards and sodium

Table 1. Chemical composition of the concentrate-hay diet with vitamins and mineral mixture[1] (the basal diet) and linseed oil fed to sheep

Item	Meadow hay	Concentrate			Linseed oil
		barley meal	soybean meal	wheat starch	
Fatty acids (μg/g)					
C8:0	29.5	5.27	4.00	3.10	26.0
C10:0	3.71	10.8	3.95	2.75	35.3
C12:0	4.17	6.50	0.85	10.0	9.62
C14:0	6.29	4.78	3.08	3.47	18.0
C16:0	199	149	301	51.1	1 405
C18:0	25.3	43.7	67.9	39.2	872
C20:0	5.31	1.07	2.98	0.54	26.8
C22:0	5.85	1.03	3.56	0.28	20.1
SFA	278	222	388	110	2 413
A-FA	208	160	305	64.6	1 432
T-FA	230	197	372	93.8	2 295
αLNA	1.60	1.72	0.58	4.36	5 892
γLNA[2]	113	105	315	15.9	894
*c9c12*C18:2[3]	161	294	807	44.0	4 962
*c9*C18:1	41.0	88.1	209	46.7	4 169
*c6*C18:1	4.50	130	64.2	108	539
MUFA	50.3	221	274	157	4 718
PUFA	276	401	1 123	64.3	11 749
ΣFA	681	856	1 880	339	19 070
Dry mass (%)	91.48	87.51	91.18	87.32	–
Crude protein (%)	13.20	9.56	45.08	0.88	–
Crude fibre (%)	24.59	4.27	6.09	–	–
Crude fat (%)	3.29	1.51	2.31	0.087	–
Ash (%)	6.26	1.80	6.33	0.12	–

[1]25 g/1 kg the basal diet; 1 kg of vitamin and mineral mixture containing: 285 g calcium, 16 g phosphorus, 56 g sodium, 42 mg cobalt as carbonate, 10 mg iodine as iodate, 1 g iron as sulphate, 6 mg selenium as sodium selenite, 0.5 g copper as cupric sulphate, 5.8 g manganese as sulphate, 7.5 g zinc as sulphate; vitamins: A (500 000 IU/kg), D_3 (125 000 IU/kg), and E as α-tocopherol (25 000 IU/kg)

[2]*C6c9c12*C18:3 (γ-linolenic acid)

[3]linoleic acid

selenate (Na_2SeO_4) were provided by Sigma-Aldrych Co. (St. Louis, USA). Acetone, dichloromethane, glacial acetic acid, and methanol were purchased from POCH (Gliwice, Poland) while acetonitrile from J.T. Baker (Deventer, the Netherlands). Triethylamine and 2,4´-dibromoacetophenone were from Merck (Darmstadt, Germany). Linseed oil was provided by APA Polska (Kobylnica near Poznan, Poland). All other chemicals were of analytical grade and organic solvents were of HPLC grade. Water used for the preparation of mobile phases and chemical reagents was prepared using an Elix Water Purification system (Millipore, Canada). The mobile phases were filtered through a 0.45 μm membrane filter (Millipore, Canada) and then degassed for 2–3 min in vacuum with ultrasonication prior to use.

Chromatographic equipment and analytical methods

A HPLC 625LC system (Waters Corp., Milford, USA) for quantification of underivatized CLA isomers and other CFA was used. The system comprised a 515 pump, a 712 WISP autosampler, a 996 photodiode array detector, and two ion-exchange columns loaded with silver ions (250 × 4.6 mm ChromSpher 5-μm Lipids columns; ChromPack, Middelburg, the Netherlands) in conjunction with a 10 × 3 mm guard column (HPLC system I; Waters Corp., Milford, USA). These conjugated fatty acids in hydrolysates were determined directly according to Czauderna et al. (2003a). Other FA were derivatized and then quantified according to Czauderna et al. (2002). Concentrations of fatty acids in biological samples were calculated using fatty acid standards and an internal standard (undecanoic acid) as a measure of extraction and derivatization yield. The instrument used (all Waters Corp., Milford, USA) consisted of: an Alliance separation module (model 2690), a 996 photodiode array detector, and two Nova-Pak C_{18} columns (4 μm, 250 × 4.6 mm I.D.) in conjunction with a guard column (HPLC system II).

Selenium (via Se^{82}) in assayed tissues of sheep was determined by the CPI-MS method after digestion of tissue samples using a mixture of 65% HNO_3 and 30% H_2O_2 (2 : 1, v/v) (Wysocka et al., 2003).

Statistical analyses

Results are presented as means of 5 individually analysed lyophilized liver, heart, and muscle samples, and fresh adipose tissue (i.e. SF, PF, and IF) of sheep. Mean values in columns having the same superscripts are significantly different at $^{a,b}P < 0.05$ and $^{A,B}P < 0.01$, while differences at $^{\alpha,\beta}P = 0.1$ are indicated as tendencies.

These one-factorial statistical analyses of the effects of LO or SeVI in the diets were conducted using the non-parametric Mann-Whitney U test for comparing independent experimental groups. Statistical analyses of interactions between LO and SeVI (i.e. LO × SeVI) were performed using two factorial ANOVA analysis; the interactions were significant at the $^{x,y}P < 0.05$ and $^{X,Y}P < 0.01$ levels, respectively. Statistical analyses were performed using the STATISTICA version 6 software package (StatSoft, 2002).

RESULTS AND DISCUSSION

In the current study, neither macroscopic lesions nor pathological changes were found in the liver, muscles or adipose tissues or in any of the other internal organs of sheep fed the diets enriched with LO and/or SeVI. Indeed, diets containing up to 2 mg selenium per kg would not be toxic for animals, especially for ruminants (Tinggi, 2003; McDowell et al., 2005). Moreover, only chronic dietary inorganic Se compounds, selenite (SeIV) in particular, at rates of more than 5 mg Se per kg can be hepatotoxic and teratogenic in humans and animals (Tanguy et al., 2003; Tapiero et al., 2003; Tinggi, 2003). As can be seen from the results summarized in Tables 2 and 3, the diet enriched with SeVI significantly increased the accumulation of Se in the liver, heart, and both muscles in comparison with the control group or LO-fed sheep; the addition of LO to the diet with SeVI did not reduce the concentration of Se in these organs and muscles compared with SeVI-fed sheep. Considering the above, we argue that dietary LO is not associated with oxidative stress in the body of sheep.

Experimental data on the relative body mass gain (BMG, %) of sheep, feed conversion efficiency (FCE; kg body mass gain/kg feed intake), and masses of the liver, heart, *MBF*, and *MLD* of sheep after 35 days of feeding the experimental diets are summarized in Table 2. Feeding the SeVI treatment statistically insignificantly increased the FCE and BMG of sheep, while the diet enriched with LO significantly increased ($P < 0.05$) the FCE and BMG of sheep compared with the control sheep. Thus, our results document that short-term feeding of the diet enriched with 2 μg Se as SeVI/g, irrespective of the addition of LO, does not have a detrimental effect on the growth performance of sheep. Our current investigations are in agreement with our previous studies in which no macroscopic lesions or pathological changes were found in any internal organs or tissues of rats fed diets enriched with 2 μg Se as SeVI/g diet (Czauderna et al., 2003b, 2004b). Considering the above, we argue that diets with higher concentrations of Se than 2 μg as SeVI/g diet detrimentally affect the growth performance of sheep. Indeed, recent studies support the concept that the Se pro-oxidative effect of diets containing higher concentrations of Se is due to the catalysis of hydrosulphide oxidation that results in an inhibitory effect on the yield of protein synthesis (Navarro-Alarcon and Cabrera-Vique, 2008).

Table 2. Effect[1] of dietary linseed oil and sodium selenate on relative body mass gain (BMG)[2], feed conversion efficiency (FCE)[3], concentrations of Se, *t11*C18:1, CLA isomers, sum of CFA (ΣCLA), selected saturated and unsaturated fatty acids (μg/g DM), and average masses[4] of the liver and heart of sheep

Item	Group			
	control	LO	SE	LOSE
BMG (%)	27.5^{ab}	38.9^{a}	27.8^{α}	$35.7^{b\alpha x}$
FCE	0.166^{a}	0.255^{a}	0.178^{b}	0.244^{b}
Liver (g)	618	625	$563^{a\alpha}$	659^{ax}
Se	0.53^{A}	0.41^{B}	4.92^{A}	4.43^{BX}
*t10c12*CLA	13.5^{Aa}	34.4^{AB}	2.9^{Ca}	10.5^{BCx}
*c9t11*CLA	635	397	841^{A}	184^{Ax}
$R_{c9t11CLA/t10c12CLA}$[5]	47.0^{Aa}	11.5^{a}	290.0^{AB}	17.5^{Bx}
*tt*CLA	176^{ab}	751^{a}	440^{b}	470
*cc*CLA	$177^{a\alpha}$	313^{α}	394^{ab}	199^{b}
Σ*ct/tc*CLA	683	504	900^{A}	252^{Ax}
ΣCLA	$1\,036^{\alpha}$	1 568	$1\,733^{\alpha a}$	921^{a}
ΣCFA	$2\,402^{A}$	2 355	853^{AB}	$2\,125^{B}$
ΣCLA + ΣCFA	$3\,438^{\alpha}$	3 923	$2\,586^{\alpha}$	3 046
*t11*C18:1	157^{a}	802^{a}	153^{A}	$1\,154^{Ax}$
C12:0	7.8	8.0	6.1	4.8
C14:0	217	137	189	157
C16:0	5 572	3 061	$5\,367^{a}$	$2\,622^{a}$
C18:0	6 629	5 019	5 475	3 835
C20:0	31.7	34.1	34.9	25.8
cis-MUFA[6]	$21\,817^{A}$	$48\,800^{AB}$	$19\,081^{BC}$	$35\,823^{C}$
trans-MUFA[7]	386	446	541	394
UFA/SFA[8]	4.116^{A}	12.797^{A}	4.128	11.977
PUFA	$29\,351^{\alpha}$	$57\,352^{\alpha\beta}$	$26\,376^{\beta}$	43 895
UFA[9]	$51\,553^{a}$	$106\,598^{ab}$	$45\,998^{Ab}$	$80\,112^{A}$
Heart (g)	272	248	256	267
Se	0.41^{A}	0.32^{B}	0.72^{A}	0.73^{Bx}
*t10c12*CLA	14.0^{a}	26.5^{a}	7.9	5.9
*c9t11*CLA	103	144.3	161	211^{x}
$R_{c9t11CLA/t10c12CLA}$	7.36^{A}	5.45	20.38^{A}	35.76^{x}
*tt*CLA	63.4^{Aa}	124.7^{a}	7.1^{AB}	78.5^{B}
*cc*CLA	103	87	92	88
Σ*ct/tc*CLA	133	212	173	228
ΣCLA	299	423	272^{α}	395^{α}
ΣCFA	94.6^{a}	34.5^{a}	75.8	73.7
ΣCLA + ΣCFA	394	458	348	469
*t11*C18:1	151^{Aa}	63^{a}	25^{A}	177
C12:0	29.7^{a}	44.4^{a}	25.4^{α}	51.5^{α}
C14:0	268	315	177^{a}	674^{ax}

Table 2 to be continued

Item	Group			
	control	LO	SE	LOSE
C16:0	1 893	2 660	1 846	2 359
C18:0	1 840[a]	2 710[a]	2 006	2 520
C20:0	14.3	19.1	15.1	20.3
cis-MUFA	6 984[a]	11 831[a]	5 145[A]	14 557[A]
trans-MUFA	219	130	126[a]	446[ax]
UFA/SFA	4.240	4.891	3.359	6.068
PUFA	10 114[a]	16 308[a]	8 516[A]	19 499[A]
UFA	17 316[a]	28 269[a]	13 787[A]	34 501[Ax]

LO = linseed oil, SE = sodium selenate, LOSE = linseed oil + sodium selenate, DM = dry matter

[1]mean values in columns having the same superscripts are significantly different at $^{a,b}P < 0.05$ and $^{A,B}P < 0.01$; differences at $^{\alpha,\beta}P = 0.1$ are indicated as tendencies; interactions were significant at the $^{x,y}P < 0.05$ and $^{X,Y}P < 0.01$ levels, respectively

[2]BMG (%) = $[(m_{35days} - m_{initial}) \times 100]/m_{initial}$, where: m_{35days} = average body mass of sheep fed the experimental diets for 35 days of study, $m_{initial}$ = average initial body mass (kg)

[3]kg body mass gain/kg diet intake

[4]masses derived from fresh organs and fresh muscle tissues

[5]concentration ratio of *c9t11*CLA and *t10c12*CLA

[6]concentration sum of *cis*-MUFA (i.e. *c7*C16:1, *c9*C16:1, *c6*C18:1, *c7*C18:1, *c9*C18:1, *c11*C18:1, *c12*C18:1, *c14*C18:1, *c11*C20:1, *c11*C22:1, *c13*C22:1, and *c15*C24:1)

[7]concentration sum of *trans*-MUFA (i.e. *t6*C18:1, *t7*C18:1, *t9*C18:1, and *t11*C18:1)

[8]concentration ratio of unsaturated fatty acids (MUFA and PUFA) and SFA

[9]concentration sum of MUFA and PUFA

It was found that the diet containing LO and SeVI (LOSE group) increased the BMG of sheep compared with the control group and sheep fed the diet containing SeVI; there was a statistically significant LOSE supplementation × BMG interaction ($P < 0.05$) that resulted in an increase in the body mass of sheep (Table 2). The effect of LO or LOSE treatment can be attributed to better utilization of these diets than the control diet or that enriched with SeVI. Therefore, an increase in FCE was observed for LO or LOSE treatments compared with the control diet and the diet containing SeVI. Moreover, the results presented in Tables 2 and 3 document that LO and LOSE treatments stimulated the growth of the liver as well as *MBF* and *MLD* more than the control diet and the diets enriched with SeVI.

Effect of the diet enriched with LO and SeVI on CLA isomer profile in assayed tissues

Chromatographic analyses using HPLC system I (Waters Corp., Milford, USA) and detection at 234 nm demonstrated that CLA isomers and non-CLA fatty acids containing conjugated double bonds (CFA) were detected in all assayed tissues (Tables 2–5). Moreover, detailed chromatographic investigations using HPLC system II (Waters Corp., Milford, USA) and photodiode detection at 234 and 256 nm documented that in all assayed tissues these unidentified non-CLA fatty acids containing conjugated double bonds (CFA) had shorter retention times (i.e. 23.2 ± 0.3, 24.8 ± 0.3, 38.3 ± 0.4, and 40.3 ± 0.4 min) than CLA isomers (i.e. 50.1–51.4 min and 53.6–55.3 min) (Czauderna and Kowalczyk, 2002). Considering the above, as well as the UV spectra of these species having an absorbance maximum at 234 nm (Czauderna and Kowalczyk, 2002; Banni et al., 2004), we argue that these fatty acids containing conjugated double bonds (CFA) contained more than two double bonds because of their shorter elution times compared with the retention times of the CLA isomers possessing two conjugated double bonds. Studies by Gnädig et al. (2001) and our recent studies carried out on rats also reinforce the finding that CLA isomers could be metabo-

Table 3. Effect[1] of dietary linseed oil and sodium selenate on concentrations of Se, *t11*C18:1, CLA isomers, ΣCFA, selected saturated and unsaturated fatty acids (μg/g DM), and average masses of *m. biceps femoris* and *m. longissimus dorsi* of sheep

Item	Group			
	control	LO	SE	LOSE
m. biceps femoris (kg)	4.1^{α}	4.7^{α}	4.3	4.6^{x}
Se	0.19^{A}	0.17^{B}	0.36^{A}	0.40^{BX}
*t10c12*CLA	$3.4^{a\alpha}$	4.5^{α}	2.3^{ab}	9.6^{b}
*c9t11*CLA	198^{Aa}	87^{a}	61^{A}	90
$R_{c9t11CLA/t10c12CLA}$	58.2^{Aa}	19.4^{A}	26.5^{a}	9.4^{x}
*tt*CLA	41.3^{a}	83.1^{a}	32.1^{b}	89.6^{b}
*cc*CLA	77	110	80	101
Σ*ct/tc*CLA	236^{Aa}	114^{a}	66^{A}	115
ΣCLA	355^{α}	307	178^{α}	305
ΣCFA	$1\ 16.2^{\alpha}$	73.4^{α}	91.6^{α}	156.7^{α}
ΣCLA + ΣCFA	471^{α}	380	$270^{\alpha a}$	462^{a}
*t11*C18:1	$1\ 260^{a}$	1 005	492^{a}	686
C12:0	22.4^{a}	12.6^{a}	29.6	15.7
C14:0	182^{A}	104	77^{A}	110
C16:0	$1\ 740^{Aa}$	$1\ 235^{a}$	$1\ 150^{A}$	1 582
C18:0	1 116	882	781	1 119
C20:0	6.7	4.8	5.5	6.8
cis-MUFA	$5\ 862^{a}$	5 933	$3\ 204^{ab}$	$6\ 981^{bx}$
trans-MUFA	$121^{a\alpha}$	72^{a}	63^{α}	119
UFA/SFA	2.927	3.650	2.597	3.481
PUFA	$8\ 020^{a}$	8 255	$4\ 823^{ab}$	$9\ 877^{b}$
UFA	$9\ 087^{a}$	8 272	$5\ 313^{a\alpha}$	$9\ 986^{\alpha}$
m. longissimus dorsi (g)	371^{α}	417^{α}	373	385
Se	0.10^{A}	0.08^{B}	0.20^{A}	0.25^{B}
*t10c12*CLA	$2.3^{A\alpha}$	9.6^{A}	3.1^{α}	5.4
*c9t11*CLA	254^{A}	254	91^{A}	151
$R_{c9t11CLA/t10c12CLA}$	110^{ab}	26.5^{a}	29.4^{b}	28.0
*tt*CLA	66.0	87.3	87.1	70.0
*cc*CLA	6.6^{Aa}	39.9^{A}	20.7^{a}	30.9
Σ*ct/tc*CLA	273^{A}	273	96^{A}	161^{x}
ΣCLA	345^{a}	400	204^{a}	262
ΣCFA	107.6	67.0	67.5	79.6
ΣCLA + ΣCFA	453^{a}	467	272^{a}	342
*t11*C18:1	$1\ 063^{A\alpha}$	$1\ 546^{\alpha}$	356^{AB}	$1\ 187^{B}$
C12:0	14.1^{α}	11.9	9.5^{α}	11.0
C14:0	181^{a}	115	99^{a}	108
C16:0	$2\ 238^{\alpha}$	$1\ 574^{\alpha}$	1 563	1 411
C18:0	1 378	1 060	1 076	1 026
C20:0	9.0	7.3	7.5	6.1
cis-MUFA	$6\ 465^{a}$	7 496	$3\ 959^{a}$	5 598
trans-MUFA	110	96	90	91
UFA/SFA	3.848^{α}	6.072^{α}	3.396	4.876
PUFA	$8\ 236^{\alpha}$	9 355	$5\ 380^{\alpha}$	6 931
UFA	$14\ 810^{a}$	16 948	$9\ 430^{a}$	12 620

LO = linseed oil, SE = sodium selenate, LOSE = linseed oil + sodium selenate, DM = dry matter

[1]mean values in columns having the same superscripts are significantly different at $^{a,b}P < 0.05$ and $^{A,B}P < 0.01$; differences at $^{\alpha,\beta}P = 0.1$ are indicated as tendencies; interactions were significant at the $^{x,y}P < 0.05$ and $^{X,Y}P < 0.01$ levels, respectively

lized *in vivo* into long-chain conjugated PUFA (via elongation and desaturation products of CLA isomers) using the same pathway as linoleic acid (LA). These studies revealed the presence of metabolites of CLA isomers in rat tissues (i.e. *c6c9c11*C18:3, *c6t10c12*C18:3, *c8c11t13*C20:3, *c8t12c14*C20:3, *c5c8c11t13*C20:4, and *c5c8t12c14*C20:4). As can be seen from the results summarized in Tables 2–5, all experimental diets fed to sheep resulted in several changes in the concentrations of CLA isomers and the sum of CLA isomer metabolites (ΣCFA) in all assayed tissues. In sheep fed the diet enriched with LO, irrespective of the presence of SeVI, decreased the concentration of *c9t11*CLA and the sum of *ct/tc*CLA isomers (Σ*ct/tc*CLA) in the liver in comparison with the control group or animals fed the diet with SeVI. Consequently, these diets decreased the ratio of *c9t11*CLA and *t10c12*CLA ($R_{c9t11\text{CLA}/t10c12\text{CLA}}$) in this organ compared with the control sheep and animals fed the diet enriched with only SeVI. Surprisingly, the diet with SeVI considerably decreased the concentration of *t10c12*CLA in the liver, heart, *MBF*, SF, and PF in comparison with the control sheep and, usually, the other experimental group (i.e. LO and LOSE). Thus, our current studies also revealed that dietary SeVI elevated the metabolism of *t10c12*CLA. Thus, our present data are in agreement with the results of Alasnier et al. (2002) and our previous investigations (Czauderna et al., 2003a, 2004b, c) in which *t10c12*CLA and *t10t12*CLA were also more efficiently driven through β-oxidation in the cells of the liver, heart, pancreas, brain, kidneys, adipose tissue, and femoral muscles of rats than their *9,11* CLA homologues. In our previous study conducted on rats, we also found that SeVI added to diets reduced the yield of *t10c12*CLA metabolism compared with *c9t11*CLA in the liver (Czauderna

Table 4. Effect[1] of dietary linseed oil and sodium selenate on concentrations of *t11*C18:1, CLA isomers, ΣCFA, selected saturated and unsaturated fatty acids (μg/g DM) of intermuscular fat of sheep[2]

Item	Group			
	control	LO	SE	LOSE
*t10c12*CLA	86	113	98^{a}	176^{a}
*c9t11*CLA	2 673^{a}	1 275^{a}	3 418^{A}	1 697^{A}
$R_{c9t11\text{CLA}/t10c12\text{CLA}}$	31^{a}	11^{a}	35^{A}	10^{A}
*tt*CLA	258	275	362$^{\alpha}$	596$^{\alpha}$
*cc*CLA	658$^{A\alpha}$	384$^{\alpha}$	154^{Ab}	338^{b}
Σ*ct/tc*CLA	3 640^{a}	1 795^{a}	4 030^{b}	2 548^{b}
ΣCLA	4 556^{a}	2 453^{a}	4 547^{b}	3 482^{b}
ΣCFA	117^{A}	534^{A}	183^{B}	625^{B}
ΣCLA + ΣCFA	4 673^{a}	2 987^{a}	4 730	4 107
*t11*C18:1	838	540	1 203^{A}	751^{A}
C12:0	265	196	306	271
C14:0	2 475^{a}	1 093^{a}	2 476^{A}	1 451^{A}
C16:0	12 941^{a}	6 741^{a}	11 732	8 308
C18:0	9 303$^{\alpha}$	5 697$^{\alpha}$	8 680	6 995
C20:0	71.4	41.7	65.7	58.7
cis-MUFA	72 358	60 179	75 882	71 749
trans-MUFA	2 198$^{A\alpha}$	744^{A}	1 668$^{\alpha B}$	980^{B}
UFA/SFA	6.320$^{\alpha}$	9.326$^{\alpha}$	7.081	9.091
PUFA	84 963$^{\alpha}$	68 547$^{\alpha}$	88 433	83 971
UFA	159 519	129 471	165 983	156 701

LO = linseed oil, SE = sodium selenate, LOSE = linseed oil + sodium selenate, DM = dry matter

[1]mean values in columns having the same superscripts are significantly different at $^{a,b}P < 0.05$ and $^{A,B}P < 0.01$; differences at $^{\alpha,\beta}P = 0.1$ are indicated as tendencies; interactions were significant at the $^{x,y}P < 0.05$ and $^{X,Y}P < 0.01$ levels, respectively

[2]concentration of Se in intermuscular fat is below the detection limit

Table 5. Effect[1] of dietary linseed oil and sodium selenate on concentrations of *t11*C18:1, CLA isomers, ΣCFA, and selected saturated fatty acids (μg/g fresh mass) in subcutaneous fat and perirenal fat of sheep[2]

Item	Group			
	control	LO	SE	LOSE
Subcutaneous fat				
*t10c12*CLA	108^{AB}	294^{A}	43^{Ca}	204^{Cx}
*c9t11*CLA	2 594	3 350	$2\,780^{\alpha}$	$3\,437^{\alpha}$
$R_{c9t11\text{CLA}/t10c12\text{CLA}}$	24^{ab}	11^{a}	65^{Ab}	17^{Ax}
*tt*CLA	$539^{a\alpha}$	$1\,144^{a}$	$301^{\alpha b}$	899^{b}
*cc*CLA	52^{AB}	979^{A}	555^{Ba}	917^{a}
Σ*ct/tc*CLA	3 087	3 986	$2\,881^{a}$	$3\,908^{a}$
ΣCLA	$3\,678^{a}$	$6\,109^{a}$	$3\,738^{b}$	$5\,724^{b}$
ΣCFA	75.4^{AB}	203.3^{A}	48.3^{BC}	116.2^{C}
ΣCLA + ΣCFA	$3\,753^{A}$	$6\,312^{A}$	$3\,786^{a}$	$5\,840^{a}$
*t11*C18:1	$1\,288^{A}$	$2\,480^{A}$	1 597	$1\,989^{x}$
C12:0	134^{α}	216^{α}	143^{a}	252^{a}
C14:0	$1\,532^{a}$	1 878	$1\,994^{a}$	2 455
C16:0	$11\,152^{ab}$	$15\,025^{a}$	$15\,532^{b}$	$15\,636^{x}$
C18:0	$7\,951^{A}$	$12\,525^{A}$	10 524	12 982
C20:0	63.3^{α}	67.2	81.8^{α}	83.3
cis-MUFA	$74\,265^{A}$	$164\,852^{A}$	$80\,708^{B}$	$153\,577^{B}$
trans-MUFA	$2\,880^{A}$	$4\,260^{A}$	3 398	3 809
UFA/SFA	7.884^{A}	12.093^{A}	6.313^{a}	10.782^{ax}
PUFA	$87\,949^{A}$	$191\,683^{A}$	$95\,868^{B}$	$181\,131^{B}$
UFA	$165\,093^{A}$	$360\,794^{A}$	$179\,975^{B}$	$338\,516^{B}$
Perirenal fat				
*t10c12*CLA	27^{A}	121^{A}	24^{a}	125^{a}
*c9t11*CLA	721^{AB}	$1\,363^{A}$	$1\,877^{B}$	$1\,085^{x}$
$R_{c9t11\text{CLA}/t10c12\text{CLA}}$	27^{Aa}	11^{a}	78^{Ab}	9^{b}
*tt*CLA	260^{A}	741^{A}	244^{a}	649^{a}
*cc*CLA	501^{α}	680^{α}	573	564
Σ*ct/tc*CLA	772^{Aa}	$1\,627^{a}$	$1\,945^{A}$	$1\,323^{x}$
ΣCLA	$1\,533^{\alpha}$	$3\,047^{\alpha}$	2 761	$2\,536^{x}$
ΣCFA	63.1^{AB}	112.7^{A}	$1\,722.7^{Ba}$	456.5^{Xa}
ΣCLA + ΣCFA	$1\,596^{AB}$	$3\,160^{A}$	$4\,484^{Ba}$	$2\,993^{Xa}$
*t11*C18:1	942	1 040	974	973
C12:0	$1\,808^{A}$	$1\,290^{A}$	$2\,335^{B}$	493^{Bx}
C14:0	$2\,362^{a\alpha}$	$1\,105^{a}$	$1\,041^{\alpha}$	1 513
C16:0	$10\,803^{\alpha}$	7 828	$6\,558^{\alpha a}$	$9\,887^{a}$
C18:0	9 203	6 872	8 602	7 506
C20:0	74.4	75.4	85.2^{α}	55.9^{α}
cis-MUFA	$79\,822^{\alpha}$	$104\,842^{\alpha}$	78 188	91 228
trans-MUFA	1 931	1 640	2 326	1 739
UFA/SFA	7.029^{a}	12.734^{a}	7.615	12.324
PUFA	$89\,743^{\alpha a}$	$113\,939^{a}$	$92\,196^{\alpha}$	101 877
UFA	$171\,496^{a}$	$220\,421^{a}$	172 710	194 843

LO = linseed oil, SE = sodium selenate, LOSE = linseed oil + sodium selenate, DM = dry matter

[1]mean values in columns having the same superscripts are significantly different at $^{a,b}P < 0.05$ and $^{A,B}P < 0.01$; differences at $^{\alpha,\beta}P = 0.1$ are indicated as tendencies; interactions were significant at the $^{x,y}P < 0.05$ and $^{X,Y}P < 0.01$ levels, respectively

[2]concentrations of Se in subcutaneous fat and perirenal fat are below the detection limit

et al., 2004c), femoral muscles (Czauderna et al., 2004b), heart, kidneys (Niedźwiedzka et al., 2006), and adipose tissues (Czauderna et al., 2003b). On the other hand, SeVI added to the diet, irrespective of the presence of LO, stimulated the accumulation of *t10c12*CLA in *MLD* and IF compared with the control group (Tables 3 and 4). Similarly, SeVI added to the diet increased the concentration of *tt*CLA in the liver, *MLD,* and IF. Moreover, this diet significantly increased the concentration of *cc*CLA and *ct/tc*CLA isomers in the liver (Table 2). Based on the above results, we argue that dietary SeVI reduced the metabolism of *tt*CLA, *cc*CLA, and *ct/tc*CLA isomers in the liver in comparison with the control animals and sheep fed the diet enriched with LO. Consequently, SeVI added to the diet most efficiently decreased the liver accumulation of CLA isomer metabolites (i.e. CFA), while most efficiently increased the sum of CLA isomers (ΣCLA) in the liver compared with the control group or other experimental groups.

The results summarized in Table 2 indicate that *tt*CLA isomers are catabolized more slowly and are poor substrates for β-oxidation. Indeed, these isomers are incorporated into liver membrane phospholipids due to their geometrical configuration (Yang et al., 2002; Czauderna et al., 2004c). On the other hand, the *ct/tc*CLA isomers in the liver of sheep fed the diet with LO, irrespective of the presence of SeVI (Table 2), are rapidly metabolized to form long-chain fatty acids containing conjugated double bonds (CFA), partly β-oxidized and, due to their unsuitable geometrical configuration, less efficiently incorporated into liver phospholipids.

As can be seen from the results in Tables 2 and 3, dietary SeVI considerably decreased the concentration of unsaturated fatty acids (UFA) in the liver, heart, and both muscles (*MBF* and *MLD*) in comparison with the control or other experimental groups. Especially important is the observation that dietary SeVI decreased the sum of CLA isomers and CFA (ΣCLA+ΣCFA) in these tissues compared with the control group. Taken together, these results suggest that dietary SeVI stimulated the biohydrogenation of unsaturated fatty acids in the rumen of sheep, as well as the β-oxidation of fatty acids in the liver, heart, and both muscles. Indeed, our studies indicate that dietary SeVI, irrespective of the addition of LO, resulted in a considerable increase in the concentration of Se in the organs and tissues of these sheep (Tables 2 and 3) compared with the control animals and sheep fed the diet with LO. On the other hand, dietary SeVI did not reduce the concentration of UFA, including CLA isomers in adipose tissues (i.e. IF, SF, and PF) of sheep, compared with the control animals. Concomitantly, we found that the concentration of Se in the adipose tissues of sheep fed the diet with SeVI was considerably lower (i.e. below the detection limit; Tables 4 and 5) than in the liver, heart, and both muscles. For this reason, we argue that dietary SeVI increased the biosynthesis of Se-Cys-proteins in the liver, heart, and muscles. Se-proteins formed especially in the liver, heart, and muscles effectively stimulated the catabolism of fatty acids (via β-oxidation) in these organs and tissues of sheep fed the diet containing SeVI.

As can be seen from our results summarized in Tables 2–5, the diet enriched with LO, irrespective of the presence of SeVI, stimulated the accumulation of *tt*CLA isomers in all assayed tissues compared with the control animal and sheep fed the diet with SeVI, with the exception of IF. These observations are consistent with the ability of ruminal bacteria to change the geometrical and positional configuration of *cis*-unsaturated fatty acids present in LO added to sheep diets (Aharoni et al., 2004). As the result of bacterial geometrical and positional isomerization of unsaturated fatty acids of dietary LO origin, *tt*CLA isomers are formed in tissues of sheep fed the diet enriched with this oil; unfortunately, SeVI added to the diet with LO showed an uneven, statistically significant influence on the formation of *tt*CLA isomers in assayed tissues of sheep compared with the diet with only LO. Similarly, all experimental diets, particularly LO treatment, stimulated the positional transformation of unsaturated fatty acids into *cc*CLA isomers in the liver, *MBF*, *MLD*, SF, and PF in comparison with the control group. In particular, a significant increase in the accumulation of *cc*CLA isomers was found in SF of sheep fed the diet enriched with LO with or without SeVI (ca. 17-fold). These results show that SF is indeed the tissue that most preferentially accumulates *cc*CLA isomers as well as very effectively incorporates *cis*-MUFA, *trans*-MUFA, and PUFA compared with other tissues of sheep fed the diet containing LO, irrespective of the presence of SeVI. Indeed, SF, stored energy, provides the fatty acids that sheep use during periods of high activity or starvation. On the other hand, the concentrations of CLA isomers, CFA, and UFA in PF and IF of sheep fed the diet enriched with LO, irrespective of the presence of SeVI, are lower in comparison

with the control group, as are the concentrations of these fatty acids in the SF of sheep from groups LO or LOSE.

Effect of the diet enriched with LO and SeVI on concentrations of non-conjugated fatty acids in assayed tissues

As can be seen from data summarized in Tables 2–5, the addition of LO to the diet elevated the concentration of *cis*-MUFA and PUFA in the liver, heart, *MLD*, PF, and especially in SF compared with the control group or sheep fed the diet with SeVI. Similarly, dietary LO increased the concentration of *trans11*C18:1 (*t11*C18:1) in the liver, *MLD,* PF, and, in particular, SF in comparison with the control sheep or SeVI-fed animals. The present study showed that the increase in the concentration of *t11*C18:1 resulted in elevation of the level of *c9t11*CLA in PF and SF of LO-fed sheep. This suggests that *c9t11*CLA in these fat tissues is in part the product of Δ9-desaturation of *t11*C18:1. On the other hand, the concentration of *t11*C18:1 in the liver negatively correlated with the concentrations of *c9t11*CLA (Table 2) and *c9*C18:1 (Czauderna et al., 2004a) in the liver of sheep fed the diet enriched with LO, irrespective of the presence of SeVI. This leads us to argue that the capacity of Δ9-desaturation is reduced in the liver of LO-fed sheep. Indeed, our recent studies (Czauderna et al., 2004a) documented that the Δ9-desaturase index is reduced in the liver of sheep fed the diet enriched with LO. Similarly, LO and LOSE treatments decreased the concentration of *c9t11*CLA (Tables 3 and 4) and *c9*C18:1 (Czauderna et al., 2004a) in *MBF* and IF. In fact, our previous studies (Czauderna et al., 2004a) documented that the yield of Δ9-desaturation in these tissues is reduced in sheep fed the diet enriched with LO, irrespective of the presence of SeVI. Moreover, we suggest that the yield of β-oxidation of *t11*C18:1 and other *trans*-MUFA as well as saturated fatty acids in *MBF* and IF of sheep was stimulated by the diet containing LO, regardless of the presence of SeVI. Similarly, addition of SeVI to the diet decreased the capacity of Δ9-desaturation in the *MBF* and *MLD* compared with the control sheep. Indeed, the current study and our previous work revealed that the concentrations of *c9*C18:1, *c9t11*CLA, and a value of Δ9-desaturase index in both muscles of SeVI-fed sheep decreased (Czauderna et al., 2004a; Niedźwiedzka et al., 2008). Moreover, a similar effect of dietary SeVI on the capacity of Δ9-desaturation was found in femoral muscles of rats (Czauderna et al., 2004b). On the other hand, the diet enriched with SeVI stimulated the accumulation of *c9t11*CLA (Tables 4, 5) and *c9*C18:1 (Czauderna et al., 2004a; Niedźwiedzka et al., 2008) in adipose tissues (IF, PF, and PF). These results documented that the effect of dietary SeVI on the capacity of Δ9-desaturation depends on the type of the tissue in sheep. Our current results and our previous studies (Czauderna et al., 2004a; Niedźwiedzka et al., 2008) demonstrated that the addition of SeVI to the diet efficiently increased the catabolism of fatty acids in both muscles, while decreasing the yield of Δ9-desaturation. On the other hand, dietary SeVI more effectively increased the capacity of Δ9-desaturation in adipose tissue, while decreasing the yield of fatty acid catabolism. Indeed, these results again show that adipose tissue, especially SF, is the stored energy (fatty acids) that sheep use during period of high activity or starvation. The current results are in agreement with our previous investigation in which the diet enriched with SeVI both increased the capacity of Δ9-desaturation in adipose tissues of rats and decreased the yield of fatty acid catabolism in these tissues (Czauderna et al., 2003b).

The diet enriched with LO, irrespective of the presence of SeVI, increased the concentrations of *cis*-MUFA, PUFA as well as UFA in the liver, heart and, especially, in SF and PF (i.e. energy storage tissues) compared with the control group and SeVI-fed sheep. Indeed, fatty acids derived from dietary LO collected in the liver in large vacuoles as triglycerides that accumulate in liver cells via the process of steatosis. As can be seen from the data summarized in Tables 2–5, the diet containing LO raised the value of the UFA/SFA ratio in all assayed tissues compared with the control group, although a particularly large increase was found in the liver. On the other hand, the addition of SeVI to the diet with LO usually decreased the UFA/SFA ratio in all examined organs and tissues compared with LO-fed sheep, although these ratios were higher than in the control sheep. Interestingly, the diet enriched with SeVI revealed a negligible effect on the UFA/SFA ratio in the liver, heart, and other assayed tissues of sheep in comparison with the control group.

Interestingly, the presented results are in agreement with our previous investigation in which the diet enriched with LO, regardless of presence of extra SeVI, also increased the accumulation of total

fatty acids (ΣFA) in the liver, SF, and PF of sheep compared with the control group and SeVI-fed sheep (Czauderna et al., 2004a; Niedźwiedzka et al., 2008). The results summarized in Table 5 showed that *trans*-MUFA most preferentially accumulated in SF compared with other assayed tissues of sheep. Our current study thus demonstrates that SF is the preferred tissue that also stores unsaturated fatty acids having a double bond with *trans*-geometrical configuration.

Interestingly, all experimental diets showed a negligible influence on the concentrations of the sum of CLA isomers, *cis*-MUFA, PUFA as well as UFA in IF and both muscles, while the concentrations of these fatty acids were preferentially increased in the heart of sheep fed the diets enriched with LO with or without SeVI.

CONCLUSION

Short-term addition of 2 μg Se as selenate per g diet can be used to increase the concentration of Se in tissues of farm ruminants without adversely influencing performance. The experimental diets enriched with linseed oil with or without selenate increased the nutritional value of *m. biceps femoris*, *m. longissimus dorsi*, adipose tissues, as the UFA/SFA ratio increased. Thus, the presented results of our short-term study constitute important information for nutritionists carrying out further investigations to improve the nutritional quality of feed for ruminants and humans. On the other hand, further research is necessary to determine if dietary selenate and other vegetable oils induce changes in the profiles of fatty acids and other essential elements in ruminant meat that are beneficial to human health.

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