ABSTRACT

Two experiments were performed to assess the effect of different amounts of dietary polyunsaturated fatty acids (PUFA) on fatty acid composition of chickens. The contribution of endogenous fatty acid synthesis to fatty acid profile was also estimated. In trial 1, different fat sources were blended in different ratios allowing a gradient of dietary PUFA (from 15 to 61 g/kg), keeping added fat level constant (9%). In trial 2, PUFA-rich oil was added in increasing inclusion levels (2, 4, 6 and 8 %), achieving a dietary PUFA content ranging between 27 and 59 g/kg.. Increasing dietary PUFA inclusion resulted in an increase of PUFA deposition, with higher efficiency when dietary fat also provided saturated (SFA) and monounsaturated (MUFA) fatty acids (trial 1). Increasing dietary PUFA in both trials resulted in a decrease of SFA and MUFA concentration in the whole body. The estimated deposition of fatty acids from endogenous synthesis is reduced when dietary fat increases from 0 to 10%, varying between 35.34 % and 17.66 % for SFA; and between 52.70 % and 7.01 % for MUFA in the whole body. The higher variation range for the MUFA supports the existence of a mechanism maintaining the SFA: (MUFA+PUFA) ratio inside a specific range in biological membranes.

INTRODUCTION

Due to the concerns of consumers about fat content and composition of the meat, numerous studies have been carried out regarding fat deposition and dietary modification of fatty acids in monogastric animals such as chickens (Lopez-Ferrer et al., 2001a, b) and pigs (Kouba et al., 2003). Moreover, it has been observed that dietary fatty acid composition affects not only fat composition but also total body fat deposition in rats and in chickens (Shimomura et al., 1990; Sanz et al., 2000). Given the close relationship between dietary and tissue polyunsaturated fatty acids (PUFA) content, and their reported health benefits compared to saturated fatty acids (SFA) (Kinsella et al., 1990; Srinath & Katan, 2004), many of these works focus on the enrichment of animal tissues with PUFA. However, the relationship between dietary and tissue concentration of SFA and monounsaturated fatty acids (MUFA) is more complex because these fatty acids in animals have a double origin: exogenous (dietary) and endogenous (fatty acid synthesis). This synthesis in the case of chickens is carried out in the liver, and lipoproteins carry these fatty acids to the rest of the tissues. That is, fatty acid composition of the animal depends on the balance between dietary and endogenous fatty acids. To our knowledge, there are no works in the literature quantifying the contribution of endogenous fatty acid synthesis to tissue fatty acid composition in response to different proportions between the three families of fatty acids (SFA, MUFA and PUFA) included in the diet.

The aim of this study was to assess the lipid composition (SFA, MUFA and PUFA) and the contribution of endogenous fatty acid synthesis to whole body fatty acid profile in response to a dietary polyunsaturation gradient achieved through two different nutritional strategies: by keeping added fat constant and by increasing added fat inclusion level.

MATERIALS AND METHODS

Animals and diets

The two experimental trials received prior approval from the Animal Protocol Review Committee of the Universitat Autònoma de Barcelona. All animal housing and husbandry conformed to the European Union guidelines.

A total of 192 Ross 308 female broiler chickens of one day of age (Granja Solé, Tarragona, Spain in trial 1 and Terra-Avant S.A., Girona, Spain in trial 2) were used in each trial. The animals were housed in groups of four in 48 cages under controlled conditions of temperature, humidity and ventilation. The diets were formulated according to the requirements recommended by the NRC (1994) on the basis of cereals (more than 50%) and soybean meal. The composition of the diets is shown in tables 1 and 2.

[table 1 and table 2]

In trial 1, the four experimental treatments were the result of blending a mixture of linseed and fish oil (in a ratio of 4 to 1) with tallow in different proportions keeping added fat constant (9%) achieving a dietary PUFA content of 15 (PU15), 34 (PU34), 45 (PU45) and 61 (PU61) g PUFA/kg. The PUFA to non-PUFA ratio ranged between 0.18 and 1.69.

In trial 2, the four experimental treatments resulted from different oil inclusion levels: 2% (O2), 4% (O4), 6% (O6) and 8% (O8). The concentration of PUFA in the experimental diets was 27, 38, 48 and 59 g PUFA/kg of diet for the O2, O4, O6 and O8 diets respectively. The PUFA to non-PUFA ratio of the diets was relatively constant (between 1.55 and 1.80). The added oil used was a mixture of linseed and fish oil in a

ratio of 4 to 1, similar to the one used in trial 1. Almond husk was added in different amounts to the feeds in order to get isoenergetic diets (table 2).

Tallow and linseed oil were obtained from Cailà-Parés, S.A. (Barcelona, Spain), fish oil was kindly supplied by Agrupación de Fabricantes de aceites marinos, S.A. (Vigo, Spain).

In both trials feed and water were provided *ad libitum*. Body weight and food consumption were measured during the experimental period for each cage. Individual food intake was inferred from group measurements. Feed samples were taken during the experiments for Weende analysis (AOAC, 1995) and fatty acid content.

Sample collection

In both trials at the end of the experimental period (44 days, 2318±16.1 g of final weight for trial 1 and 40 days, 2240±14.6 g of final weight days for trial 2), two animals per cage were killed by lethal injection (sodium pentobarbitate, 200 mg/kg). The whole animals were frozen, cut, and ground with a cutter (Tec-Maq model cut-20, INTEFISA). After that, samples from each animal were taken, freeze-dried, ground and stored at –20°C until further analyses. The other two animals per cage were killed in a commercial slaughterhouse and quartered. Thighs with skin and breast muscle from these animals were destined to a parallel experiment (Cortinas et al., 2004). Nevertheless, fatty acid profile of the breast muscle of these animals is used in this paper to assess the potential of endogenous fat synthesis in intramuscular fat.

Fatty acid concentration

Fatty acid content from feeds was determined by GC following the methodology described by Sukhija & Palmquist (1988) Fatty acid content of the diets is presented in table 3. Fatty acid profile of the whole body and breast muscle was determined as described previously by Carrapiso et al. (2000). Nonodecanoic acid (C19, Sigma-Aldrich) was used as internal standard in both cases.

[table 3]

Statistics and calculations

Regression analyses were performed for both trials using the REG procedure of SAS® (SAS Institute, 2002) between PUFA intake (g/animal/day) and SFA, MUFA and PUFA content in the tissues (% of total fatty acids). As established by Crespo & Esteve-Garcia (2002a), the ratio between SFA, MUFA and PUFA of the whole chicken and SFA, MUFA and PUFA of the feeds (% of total fatty acids) was calculated as an indicator of endogenous synthesis. These ratios were analyzed by one-way ANOVA using the GLM procedure of SAS, where the input factor was dietary polyunsaturation.

In order to quantify the potential of endogenous synthesis, linear regression analyses were performed using the REG procedure of SAS between the SFA and MUFA intakes (g/animal/day) and their respective concentration in the tissue (% of total fatty acids). In all cases, p values lower than 0.05 were considered significant.

RESULTS

Effect of dietary polyunsaturation on lipid composition

In table 4, linear regression equations between PUFA intakes (g/animal/day) and SFA, MUFA and PUFA deposition (% of total fatty acids) are presented. PUFA deposition increases as their intake increases in both trials, but the slope of deposition is higher in

trial 1 (7.54) than in trial 2 (5.99). SFA and MUFA have lower concentrations with higher PUFA intakes. In both trials, the slopes of SFA and MUFA equations are negative (-2.19 and -1.91 for SFA, for trial 1 and 2 respectively; and -5.44 and -4.03 for MUFA for trial 1 and 2 respextively).

[table 4]

Effect of dietary polyunsaturation on potential endogenous synthesis

In table 5, the ratios between SFA, MUFA and PUFA concentrations in whole animal and their concentration in the diets are shown. As established by Crespo and Esteve-Garcia, 2002a, ratio values above 1 indicate net fatty acid synthesis and values lower than 1 show net fatty acid beta-oxidation. Regarding SFA, in the first trial the ratio was lower than 1 in the PU15 and PU34 treatments, 1 in the PU45 and higher than 1 in the PU61. This suggests that in the latter case dietary SFA had to be synthesized. In the second trial the ratio decreased with increasing added oil but never reached values lower than 1; this means that in trial 2 there was always a net synthesis of SFA, even in animals fed the diets with a relatively high percentage of added fat. In the case of MUFA, there was a net synthesis for all treatments in both trials. Whereas in the first trial the ratio was relatively constant among treatments, in the second trial the ratio decreased as dietary oil increased (from 1.91 to 1.29, $p<0.001$), similarly to SFA ratio. Finally, PUFA ratio was in all cases lower than one, indicating that there was always a net oxidation. In trial 1, the ratio was not different among treatments, but in trial 2 the ratio increased from 0.5 to 0.8 ($p<0.001$) with added oil.

[table 5]

As it was previously mentioned, regression analyses were performed between SFA and MUFA intakes (g/animal/day) and their respective content (% of total fatty acids) in the

whole body in order to estimate the endogenous synthesis potential (figure 1). The intercept of the estimated equations from the first trial show the theoretical endogenous synthesis of SFA or MUFA when dietary fat level is high (approximately 10%) but there is no intake of SFA or MUFA. In the second trial, it represents the SFA or MUFA synthesis when no fat is added to the diets. Based in this information, the endogenous synthesis potential of SFA was 17.66% (confidence interval: from 16.86 to 18.45) for the first trial and 35.34% (confidence interval: from 33.94 to 36.74) for the second one. Concerning MUFA, the endogenous synthesis potential seen in the whole body in trial 1 was 7.01% (confidence interval: from 3.98 to 10.03) and 52.70% in trial 2 (confidence interval: from 49.38 to 56.03).

DISCUSSION

Fatty acid composition in chicken tissues is a combination of endogenous synthesis of fatty acids, from carbohydrate and protein precursors, and direct deposition from the diet. SFA and MUFA have this double origin, whilst PUFA deposition depends almost exclusively on dietary supplementation when no essential fatty acids deficiency exists. The main fatty acids resulting from hepatic lipogenesis are 16:0, 18:0, 18:1n9 and 16:1n7 (Bartov, 1979; Crespo & Esteve-Garcia, 2002b).

In both trials, PUFA deposition increased with increasing dietary PUFA inclusion, whilst both SFA and MUFA deposition decreased. The reduction in SFA and MUFA concentration is due to the inverse relationship between PUFA and SFA and MUFA deposition, already described in the literature (Ajuyah et al., 1991; Lopez-Ferrer et al., 2001a, b). In trial 1 this reduction can be attributed to the lower intakes of SFA and

MUFA when dietary PUFA increase. In trial 2, when animals consumed a low fat diet (O2, O4), endogenous synthesis of SFA and MUFA plays an important role, but when fat consumption increases, the contribution of endogenous fatty acid synthesis to body fat decreases (Donaldson, 1985), even if the added fat is rich in PUFA and low in SFA and MUFA. The decrease in fatty acid synthesis was possibly due to a lower availability of carbohydrate precursors as dietary fat increased and to an inhibition of lipogenic enzymes by dietary fatty acids, as suggested by Mourot & Hermier (2001). For this reason, SFA and MUFA concentrations in the body in trial 2 were lower when added fat inclusion increased, in spite of higher SFA and MUFA intakes.

The slope of the MUFA equations is bigger than in the SFA ones. This suggests that, in the case of high PUFA intakes, SFA are preferred to MUFA for deposition in order to maintain a relatively constant unsaturated (MUFA + PUFA) to saturated fatty acids ratio in cellular membranes (Asghar et al., 1990; Bou et al., 2004). The fact that MUFA and PUFA slopes are lower in trial 2 ($p<0.05$), whereas the slopes of SFA equations between the two trials do not statistically differ ($p>0.05$), further supports the idea that MUFA are exchanged for PUFA when necessary and SFA deposition is more independent of the effect of high PUFA intakes. Other authors have reported the lower manipulation of SFA compared to MUFA and PUFA in broiler meat (Lopez-Ferrer et al., 1999) and in eggs (Baucells et al., 2000).

Regarding the ratios between body-to-dietary SFA, MUFA and PUFA, it is interesting to note that there is still a net synthesis of SFA and MUFA when the level of dietary fat is high (10.2% for trial 1, table 1; and 9.9% for the O8 treatment in trial 2, table 2). This

could indicate that hepatic lipogenesis is not completely inhibited by dietary fat. In trial 1, this net synthesis of SFA and mainly MUFA is higher in the PU61 treatment than in the rest ($p<0.05$) which could mean that high PUFA diets compared to SFA and MUFA rich diets exhibit a lower inhibition effect upon hepatic lipogenesis, as it was suggested by Crespo & Esteve-Garcia (2002a). Concerning body-to-dietary PUFA, the ratio values are always under 1, because PUFA come mainly from the diet. In trial 1, this is not different among treatments, but in trial 2 the ratio increases with added oil. This suggests that in the low fat treatments, PUFA are oxidized in order to obtain energy, substrate or both to synthesize SFA and MUFA. This idea is supported by the fact that the slope of the regression equations described above (table 4) between PUFA intake and PUFA deposition is higher in trial 1 than in trial 2 ($p<0.001$), suggesting that when added fat level is high, increasing dietary PUFA are deposited more readily from the diet, whereas in trial 2 the efficiency of deposition is lower, possibly due to their contribution to SFA and MUFA synthesis in the low-fat treatments.

[figure 1]

Looking at the information provided by the regression equations between SFA and MUFA intake and their deposition in the body, the intervals marked by the intercept values from trial 1 (lower values, when no SFA/MUFA is consumed, 10% dietary fat) and trial 2 (higher values, when no fat is consumed) show the variation of endogenous synthesis when dietary fat is increased from 0% to 10%. In figure 1 we show not only the contribution of endogenous synthesis to fatty acid profile in whole body but also in breast (representing intramuscular fat). That is, increasing from 0 to 10% of dietary fat, SFA from endogenous synthesis found in the whole body and in breast decreased from 35 to 17% and from 39 to 23% of total fatty acids respectively; and MUFA from

endogenous synthesis decreased from 53% to 7% and from 42 to 10 % respectively. It can be observed that the variation range of SFA proportion is lower than the range of variation of MUFA both in the whole body and in breast. This further supports the hypothesis already mentioned that there is a homeostatic mechanism in the cellular membranes to keep the SFA: unsaturated fatty acids ratio inside a relatively narrow range to maintain membrane fluidity. Also, breast muscle has a lower range of variation of SFA and MUFA than the whole body. This shows that fat composition of intramuscular fat (main fat depot present in breast muscle) is less modifiable by the diet than storage fat (main fat present in the whole body), which seems logical given that intramuscular fat is comprised mainly of membrane phospholipids, and phospholipid composition affects the execution of different metabolic activities (Merrill & Schroeder, 1993). Changes in this composition could seriously affect cell metabolism, hence phospholipid fatty acids are less affected by diet composition than triglyceride fatty acids, whose main role is to store energy.

In conclusion, increasing dietary PUFA inclusion results in a linear increase in PUFA deposition in the whole body of chickens. The rate of deposition (represented by the slope of the equations between dietary PUFA and tissue PUFA) is higher when added fat provides higher amounts of SFA and MUFA (trial 1). Also, increasing dietary PUFA, both modifying the added fat source or increasing added PUFA-rich oil level, results in a linear decrease of SFA and MUFA concentration, more marked in the case of MUFA. Regarding endogenous synthesis potential, the estimated deposition of fatty acids coming from endogenous synthesis is reduced when dietary fat increases from 0 to 10%. Despite a high inclusion level of fat, SFA and MUFA synthesis are not completely

inhibited. The lower variation range found in breast muscle suggests that intramuscular fat is less modifiable by the diet than total body fat. The higher variation range for MUFA supports the existence of a mechanism maintaining the SFA: (MUFA+PUFA) ratio inside a specific range in biological membranes.

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TABLE 1. Composition and chemical analysis of the diets. Trial 1.

Ingredients	**g/kg diet**
Wheat	393.0
Soya 48	340.9
Barley	133.9
Added fat[1]	90.0
Bicalcium phosphate	21.7
Calcium carbonate	9.8
Salt	4.5
Vitamin mineral mix[2]	4.0
DL-Methionine	2.8
L-Lysine	0.4
Chemical analysis	
Dry matter	907.8
Crude protein	229.8
Crude fat	101.7
Crude fibre	34.7
Ash	60.8

[1]PU15: 90 g/kg tallow; PU34: 55 g/kg tallow, 30 g/kg linseed oil, 5 g/kg fish oil; PU45: 35 g/kg tallow, 45 g/kg linseed oil, 10 g/kg fish oil; PU61: 70 g/kg linseed oil, 20 g/kg fish oil.

[2]Vitamin and mineral mix per kg of feed: Vitamin A: 12000 UI; Vitamin D_3: 2400 UI; Vitamin E: 176 IU; Vitamin K_3: 3 mg; Vitamin B_1: 2.2 mg; Vitamin B_2: 8 mg; Vitamin B_6: 5 mg; Vitamin B_{12}: 11 µg; Folic acid: 1.5 mg; Biotin: 150 µg; Calcium pantotenate: 25 mg; Nicotinic acid : 65 mg; Mn: 60 mg; Zn: 40 mg; I: 0.33 mg; Fe: 80 mg; Cu: 8 mg; Se: 0.15 mg.

TABLE 2. Composition and chemical analysis of the diets[1]. Trial 2.

Ingredients (g/kg diet)	Dietary treatments*			
	O2	O4	O6	O8
Maize	584.9	526.8	468.6	410.2
Soya 48	354.9	364.2	373.4	382.4
Added oil[2]	20.0	40.0	60.0	80.0
Almond husk	0.0	29.2	58.4	87.6
Bicalcium phosphate	17.5	17.6	17.6	17.7
Calcium carbonate	10.8	10.7	10.6	10.5
Salt	5.7	5.7	5.7	5.7
Vitamin mineral mix[3]	4.0	4.0	4.0	4.0
DL-Methionine	1.8	1.9	1.9	2.0
L-Lysine	0.5	0.3	0.2	0.0
Chemical analysis				
Dry matter	882.6	890.8	894.2	902.8
Crude protein	213.1	218.2	214.8	218.5
Crude fat	44.5	63.0	84.0	99.1
Crude fibre	32.2	54.5	79.3	98.6
Ash	57.9	61.0	58.5	62.0

[1]O2: 2% of added oil (27 g PUFA/kg); O4: 4% of added oil (38 g PUFA/kg); O6: 6% of added oil (48 g PUFA/kg); O8: 8% of added oil (59 g PUFA/kg).

[2]Linseed and fish oil mixture in a ratio 4:1.

[3]Vitamin and mineral mix per kg of feed: Vitamin A: 12000 IU; Vitamin D_3: 2400 IU; Vitamin E: 176 IU; Vitamin K_3: 3 mg; Vitamin B_1:2.2 mg; Vitamin B_2: 8 mg; Vitamin B_6: 5 mg; Vitamin B_{12}: 11 µg; Folic acid: 1.5 mg; Biotin: 150 µg; Calcium pantotenate: 25 mg; Nicotinic acid : 65 mg; Mn: 60 mg; Zn: 40 mg; I: 0.33 mg; Fe: 80 mg; Cu: 8 mg; Se: 0.15 mg.

TABLE 3. Fatty acid composition of the experimental diets, expressed as g per kg.

Fatty Acid[3]	Trial 1[1]				Trial 2[2]			
	PU15	PU34	PU45	PU61	O2	O4	O6	O8
% Added oil	9	9	9	9	2	4	6	8
g PUFA/kg diet	15	34	45	61	28	38	48	59
Total FA	100.45	98.81	99.57	96.89	45.37	60.29	75.70	91.63
SFA	43.75	32.38	26.22	15.74	7.89	9.79	11.65	13.66
C 10:0	0.05	0.03	0.02	0.00	0.08	0.08	0.08	0.08
C 14:0	2.72	2.01	1.79	1.45	0.30	0.53	0.76	0.99
C 15:0	0.44	0.30	0.23	0.11	0.03	0.05	0.07	0.09
C 16:0	23.80	18.15	15.25	10.31	5.82	6.84	7.84	8.96
C 17:0	1.19	0.77	0.53	0.14	0.06	0.08	0.09	0.11
C 18:0	14.64	10.23	7.68	3.33	1.39	1.92	2.44	2.98
C 20:0	0.12	0.16	0.17	0.17	0.16	0.19	0.22	0.26
MUFA	41.30	32.55	28.32	20.31	9.91	12.83	15.90	19.07
C 16:1t	0.20	0.15	0.12	0.07	0.02	0.03	0.03	0.04
C 16:1	2.25	1.73	1.65	1.52	0.40	0.71	1.01	1.32
C 18:1 n9[4]	35.62	27.76	23.59	15.69	8.67	10.85	13.21	15.67
C 18:1 n7t	1.60	1.37	1.29	1.12	0.49	0.67	0.86	1.05
C 20:1	0.28	0.29	0.31	0.35	0.15	0.23	0.30	0.38
C 24:1	0.09	0.46	0.81	1.46	0.10	0.11	0.12	0.14
PUFA	15.40	33.77	45.03	60.84	27.60	37.66	48.08	58.79
C 18:2 n6	13.16	16.23	17.98	20.17	17.79	18.72	19.87	21.31
C 18:3 n3	1.55	16.45	24.62	36.27	8.57	16.65	24.63	32.69
C 18:4 n3	0.27	0.11	0.23	0.43	0.14	0.27	0.41	0.53
C 20:4 n6	ND	ND	0.13	0.19	ND	ND	0.12	0.14
C 20:5 n3	ND	0.81	1.77	3.35	0.72	1.38	2.07	2.75
C 22:6 n3	ND	0.07	0.18	0.33	0.28	0.53	0.76	1.01
PUFA:SFA	0.35	1.04	1.72	3.87	3.50	3.85	4.13	4.30

[1]PU15: 15 g polyunsaturated fatty acids /kg of feed; PU34: 34 g/kg dietary polyunsaturated fatty acids; PU45: 45 g/kg dietary polyunsaturated fatty acids; PU61: 61 g/kg dietary polyunsaturated fatty acids.

[2]O2: 2% of added oil; O4: 4% of added oil; O6: 6% of added oil; O8: 8% of added oil.

[3]Total FA: total fatty acids; SFA: saturated fatty acids; MUFA: monounsaturated fatty acids; PUFA: polyunsaturated fatty acids.

[4]C 18:1 n9 includes sum of cis and trans forms.

ND: Not detected.

TABLE 4. Regression equations between polyunsaturated fatty acid intake (g/animal/day, X) and the content of saturated (SFA), monounsaturated (MUFA) and polyunsaturated (PUFA) fatty acids (g/100g of total fatty acids, Y) in the whole body.

		Dependent variable (Y)	Equation	p value	R^2	CV (%)
Trial 1	Whole body	% SFA	Y = 36.95 – 2.19·X	<0.001	0.94	3.51
		% MUFA	Y = 63.68 – 5.44·X	<0.001	0.96	4.81
		% PUFA	Y = 7.54·X	<0.001	0.96	8.02
Trial 2	Whole body	% SFA	Y = 32.36 – 1.91·X	<0.001	0.87	4.07
		% MUFA	Y = 51.87 – 4.03·X	<0.001	0.85	6.67
		% PUFA	Y = 15.46 + 5.99·X	<0.001	0.89	6.27

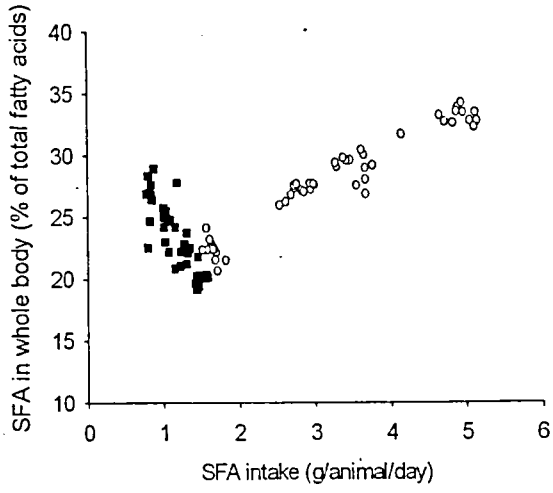
TABLE 5. Body-to-dietary fatty acid ratio[1] of saturated fatty acids (SFA), monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA) in response to increasing levels of polyunsaturation.

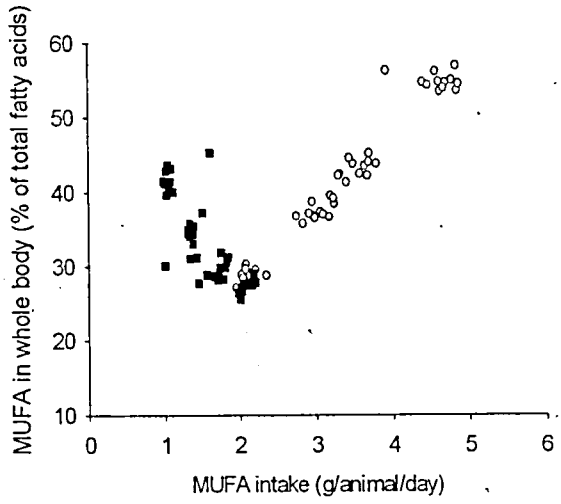
	Trial 1				p-value	RSD
% Added fat	**9**	**9**	**9**	**9**	**Dietary**	
PUFA (g/kg)	**15**	**34**	**45**	**61**	**PUFA**	
SFA	0.76^{d}	0.89^{c}	1.03^{b}	1.39^{a}	<0.001	0.041
MUFA	1.33^{b}	1.32^{b}	1.32^{b}	1.37^{a}	<0.001	0.046
PUFA	0.79	0.80	0.78	0.78	0.093	0.040
	Trial 2					
% Added fat	**2**	**4**	**6**	**8**	**Added oil**	
PUFA (g/kg)	**27**	**38**	**48**	**59**		
SFA	1.58^{a}	1.54^{a}	1.44^{b}	1.36^{c}	<0.001	0.068
MUFA	1.91^{a}	1.62^{b}	1.40^{c}	1.29^{d}	<0.001	0.086
PUFA	0.50^{d}	0.65^{c}	0.76^{b}	0.82^{a}	<0.001	0.035

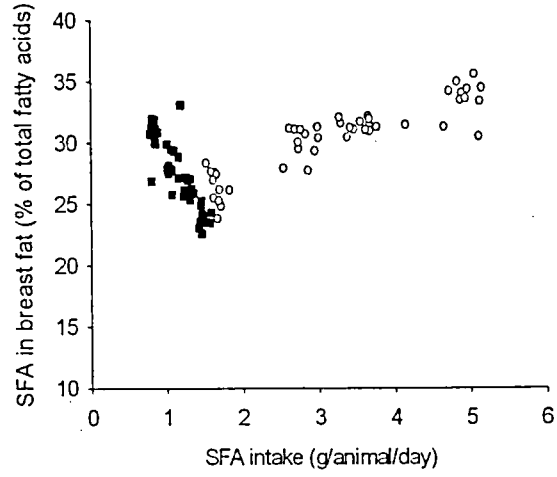
[1]Calculated as the ratio of SFA, MUFA and PUFA concentration in whole body (g/100 g of total fatty acids) between their respective concentrations in the diet (g/100 g of total fatty acids).

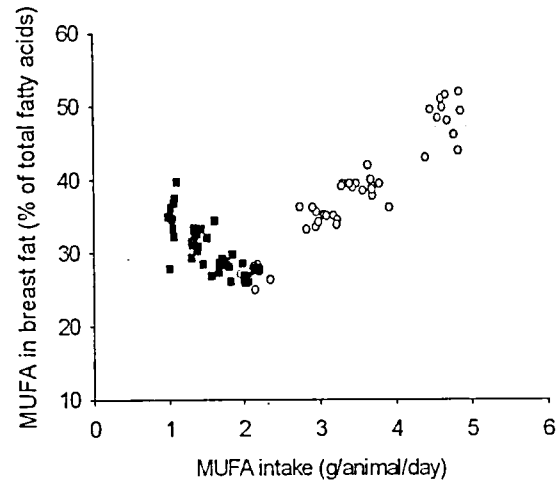
[2]Values given in this table correspondence to least-squares means obtained from ANOVA (n=24) and their RSD. Means in a row not sharing a superscript letter differ ($p<0.05$).

Figure 1: Regression equations between SFA and MUFA intake (g/animal/day) and their respective concentrations (g/100g of total fatty acids) in the whole body (upper graphs) and in breast muscle (lower graphs). Data represented by ◆ are from trial 1 (gradient of polyunsaturation achieved keeping added fat constant) and data represented by □ are from trial 2 (gradient of polyunsaturation achieved increasing added fat inclusion level).









Trial	Independent variable	Tissue	Equation	R^2	VC (%)[1]
1	SFA intake	Body	y = 17.66 + 3.20·x	0.94	3.43
2	SFA intake	Body	y = 35.34 - 10.18·x	0.87	4.1
1	SFA intake	Breast	y = 23.26 + 2.20·x	0.83	3.94
2	SFA intake	Breast	y = 39.22 - 10.32·x	0.89	3.35
1	MUFA intake	Body	y = 7.01 + 10.25·x	0.98	3.54
2	MUFA intake	Body	y = 52.70 - 12.62·x	0.84	6.84
1	MUFA intake	Breast	y = 9.81+ 8.30·x	0.95	4.88
2	MUFA intake	Breast	y = 42.56 - 7.72·x	0.75	5.52

[1]Variation coefficient