British Journal of Nutrition (2010), **104**, 204–213
doi:10.1017/S0007114510000541

Effect of diet, sex and age on fatty acid metabolism in broiler chickens: SFA and MUFA

R. Poureslami[1], G. M. Turchini[2], K. Raes[1]†, G. Huyghebaert[3] and S. De Smet[1]*

[1]*Laboratory for Animal Nutrition and Animal Product Quality, Department of Animal Production, Ghent University, Proefhoevestraat 10, 9090 Melle, Belgium*
[2]*School of Life and Environmental Sciences, Deakin University, Warrnambool, Vic Australia*
[3]*Institute for Agricultural and Fisheries Research, Animal Science Unit, Scheldeweg 68, 9090 Melle, Belgium*

(Received 28 July 2009 – Revised 28 January 2010 – Accepted 29 January 2010 – First published online 4 March 2010)

The present study was conducted to evaluate the effect of different dietary lipid sources, age and sex on the SFA and MUFA metabolism in broiler chickens using a whole body fatty acid balance method. Four dietary lipid sources (palm fat, Palm; soyabean oil, Soya; linseed oil, Lin; and fish oil, Fish) were added at 3 % to a basal diet containing 5 % Palm. Diets were fed to female and male chickens from day 1 to either day 21 or day 42 of age. The accumulation (percentage of net intake and *ex novo* production) of SFA and MUFA was significantly lower in broilers fed on Palm than in broilers fed on the other diets (85·7 *v.* 97·4 %). Conversely, β-oxidation was significantly higher in Palm-fed birds than the average of the other dietary treatments (14·3 *v.* 2·6 %). On average, 33·1 % of total SFA and MUFA accumulated in the body were elongated, and 13·8 % were Δ-9 desaturated to longer chain or more unsaturated metabolites, with lower proportions being elongated and desaturated for the Palm and Fish diets than for the Soya and Lin diets. Total *in vivo* apparent elongase activity decreased exponentially in relation to the net intake of SFA and MUFA, while it increased with age. Total *in vivo* apparent Δ-9 desaturase activity was not significantly affected by dietary treatment or age. Total *ex novo* production and β-oxidation of SFA and MUFA showed a negative and positive curvilinear relationship with net intake of SFA and MUFA, respectively. Sex had no effect on SFA and MUFA metabolism.

Fatty acid metabolism: Broiler chickens: Elongation: Desaturation: Oxidation

Dietary SFA are associated with an increase in LDL and a decrease in HDL as a risk factor for CVD, obesity and associated disorders[1,2]. The current dietary recommendations for reducing the risk of CVD underline reducing SFA intake (mainly 16 : 0) by increasing MUFA (mainly 18 : 1*n*-9) in the diet[3]. The ratio of TAG:cholesterol in postprandial TAG-rich lipoprotein of healthy men is inversely correlated with the ratio of 18 : 1*n*-9 over 16 : 0 and with the MUFA:SFA ratio in dietary fat[3]. High MUFA content in the diet favours lipid oxidation and reduces risk of obesity development[4]. MUFA are also of particular interest for their protective role against SFA-induced insulin resistance and cell death[2].

White meat such as chicken is commonly preferred to red meat, supposedly as a more healthy choice, due to its lower SFA content and consequently healthier unsaturated:saturated ratio[5]. However, poultry nutritionists are increasingly urged by the industry to provide even better fatty acid composition in chickens. In attempting to optimise fatty acid composition of chicken products, previous studies reported that different broiler chicken tissues reflect dietary fatty acid composition[6,7]. However, information on metabolism (deposition, elongation, Δ-9 desaturation and β-oxidation) of individual SFA and MUFA in broiler chickens is limited. An early *ex vivo* study in chickens using isotopic dilution technique to study the metabolism of 16 : 0, 18 : 0 and 18 : 1*n*-9 in liver is available[8]. However, one of the drawbacks of the *ex vivo* methods is that the knowledge deduced from this method is restricted to a distinct tissue[9]. Several *in vivo* methods are able to give an indication about the fatty acid metabolism at the whole body (WB) level[10]. The WB fatty acid balance method described by Turchini *et al.*[9] proved to be a reliable method to estimate SFA and MUFA metabolism in fish[11], and a capable method to describe the fate of each individual dietary fatty acid.

It is believed that dietary fatty acid composition may affect WB fat partitioning for storage (accumulation) or energy (β-oxidation) utilisation[12]. The objective of the current experiment was to examine the effect of dietary fat source together with slaughter age and sex on the SFA and MUFA metabolism in a commercial strain of broiler chicken using the WB fatty acid balance method.

Abbreviations: Fish, fish oil; Palm, palm fat; Soya, soyabean oil; WB, whole body.

* **Corresponding author:** Stefaan De Smet, fax +32 9 264 9099, email stefaan.desmet@ugent.be
† Present address: Research Group EnBiChem, Department of Industrial Engineering and Technology, University College West-Flanders, Graaf Karel de Goedelaan 5, 8500 Kortrijk, Belgium.

Materials and methods

Animals and diets

The experiment was carried out according to the guidelines of the Ethics Committee of Ghent University (Belgium) for the humane care and use of animals in research. Details concerning the experimental design and analytical methods can be found in Poureslami *et al.*[13]. Briefly, four hundred 1-d-old male and female Ross 308 chickens were subjected to one of the four dietary treatments: palm fat (Palm, SFA source); soyabean oil (Soya, 18 : 2*n*-6 source); linseed oil (Lin, 18 : 3*n*-3 source) or fish oil (Fish, *n*-3 long-chain PUFA source). The four test oils were added at 3 % to the same basal diet containing 5 % Palm. The fatty acid composition of the diets is given in Table 1. Mass ingested and excreted fatty acids were recorded in two subsequent periods: 7–21 d and 21–42 d of age, and the WB fatty acid content was quantified as the sum of seven anatomical compartments of the broiler chickens, which were analysed individually[7].

Whole body fatty acid balance calculations

The WB fatty acid balance method and calculation model described by Turchini *et al.*[9,11,14] were employed to estimate the different fates (excretion, net intake, body accumulation, *ex novo* production, elongation, Δ-9 desaturation and β-oxidation) of individual dietary SFA and MUFA. Briefly, in the WB fatty acid balance method, upon a feeding experiment, initial and final WB fatty acid mass as well as accumulation was quantified. Then, ingested and excreted fatty acids (apparent digestibility) were determined, and the net fatty acid intake (fatty acid absorption) was computed. The difference between fatty acid accumulation in the WB and the net intake was considered as the total appearance or disappearance of an individual fatty acid. At this step, the balance of SFA and MUFA was calculated. Backward calculations were made along the known SFA and MUFA metabolic pathway (Fig. 1). It is worth noting that some of the fatty acids involved in the metabolic pathway (namely 22 : 1*n*-9, 24 : 1*n*-9, 20 : 1*n*-11 and 22 : 1*n*-11) were not available in a detectable quantity in chickens' WB. Hence,

Table 1. Fatty acid profile of the diets (mg/g feed)

Fatty acid	Palm	Soya	Lin	Fish
12 : 0	0·16	0·09	0·09	0·12
14 : 0	0·84	0·50	0·46	2·27
16 : 0	35·9	23·7	21·2	26·2
17 : 0	0·11	0·09	0·08	0·19
18 : 0	3·93	3·42	3·07	3·24
20 : 0	0·37	0·33	0·33	0·30
22 : 0	0·10	0·17	0·11	0·11
14 : 1*n*-5	0·01	–*	–	0·01
16 : 1*n*-7	0·17	0·14	0·12	2·02
18 : 1*n*-7	0·76	0·86	0·62	1·22
18 : 1*n*-9	31·4	23·9	21·2	21·1
20 : 1*n*-9	0·17	0·17	0·15	0·38
ΣPUFA	15·6	26·5	28·6	22·3
Total fatty acids	89·5	79·9	76·1	79·4

Palm, palm fat diet; Soya, soyabean oil diet; Lin, linseed oil diet; Fish, fish oil diet; ΣPUFA, sum of PUFA.
* –, not detected.

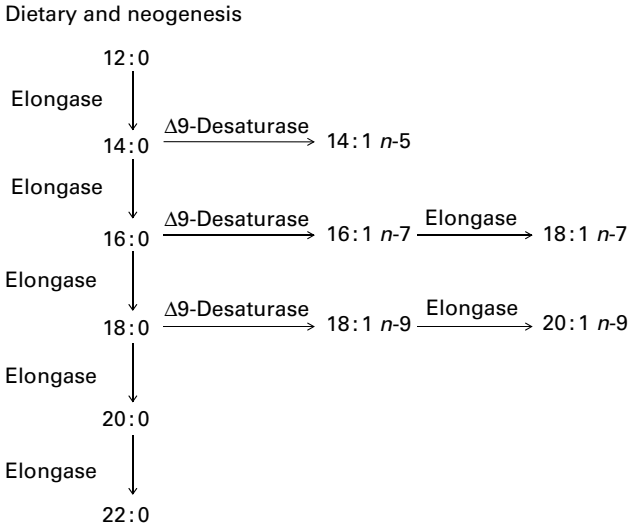

Fig. 1. Schematic representation of the SFA and MUFA elongation and desaturation pathways considered in the present study for the computation of the whole body fatty acid balance method.

in the present study, they were not considered in the computations of the model. In the backward computations, the number of μmol of longer chain or more unsaturated fatty acids that appeared was subtracted from the numbers of μmol of the previous fatty acids in the elongation and Δ-9 desaturation pathways, and so forth, backward until 12 : 0 (the shortest fatty acid identified in the present study). In this way, a new fatty acid balance was obtained, in which any fatty acid appearance was ascribable to *ex novo* production and any fatty acid disappearance was ascribable to β-oxidation. Hence, total *ex novo* production was computed. Subsequently, fatty acid accumulation and β-oxidation were calculated and expressed as '% of *ex novo* production plus net intake', while the proportion of fatty acids elongated and/or Δ-9 desaturated was expressed as percentage relative to total fatty acid accumulation. Finally, the bioconversion (i.e. elongation and/or Δ-9 desaturation) and β-oxidation of a specific fatty acid (μmol of fatty acid per g body weight per d) were quantified. The overall accretion of elongation and Δ-9 desaturation for a given fatty acid was expressed as an indicator of the overall apparent *in vivo* elongase and Δ-9 desaturase activities (μmol/g per d).

Data analyses

Data were analysed using S-PLUS for Windows (version 6.1, Insightful, Seattle, WA, USA). A linear model was used to analyse the fixed effects of diet, age and sex and their interaction terms. In case of a significant diet effect, the mean values were compared with the Tukey's *post hoc* test ($P<0·05$). Regression analysis was performed to examine the effect of SFA and MUFA net intake on the total *in vivo* apparent elongase and Δ-9 desaturase activities, *ex novo* production and β-oxidation of SFA and MUFA.

Results

Table 2 shows fatty acid intake and apparent digestibility in broiler chickens. A higher 12 : 0, 16 : 0, 18 : 0, 20 : 0, ΣSFA,

Table 2. SFA and MUFA intake (mg/d) and apparent digestibility (%) in broiler chickens

	Diet (*n* 12)				Age (*n* 24; d)		Sex (*n* 24)			*P*			
	Palm	Soya	Lin	Fish	7–21	21–42	Male	Female	RMSE	Diet	Age	Sex	Interactions*
Intake (mg/d)													
12 : 0	16·5^{a}	9·07^{b}	9·11^{b}	12·1^{c}	7·03	16·3	12·5	10·8	0·51	<0·001	<0·001	<0·001	D × A, D × S, A × S
14 : 0	88·2^{b}	51·7^{b}	49·6^{c}	230^{a}	62·4	147	113	97·1	5·53	<0·001	<0·001	<0·001	D × A, D × S, A × S, D × A × S
16 : 0	3778^{a}	2434^{c}	2279^{d}	2662^{b}	1677	3899	2994	2582	120	<0·001	<0·001	<0·001	D × A, A × S
18 : 0	412^{a}	351^{b}	329^{c}	329^{c}	213	497	382	329	15·1	<0·001	<0·001	<0·001	D × A, A × S
20 : 0	39·2^{a}	33·5^{b}	35·0^{b}	30·0^{c}	20·7	48·2	37·0	32·0	1·46	<0·001	<0·001	<0·001	D × A, A × S
22 : 0	10·3^{d}	17·6^{a}	12·3^{b}	11·2^{c}	7·71	18·0	13·8	11·9	0·55	<0·001	<0·001	<0·001	D × A, D × S, A × S, D × A × S
ΣSFA	4345^{a}	2897^{c}	2714^{d}	3275^{b}	1989	4626	3553	3063	142	<0·001	<0·001	<0·001	D × A, A × S
14 : 1*n*-5	0·56^{b}	0·13^{c}	0·00^{d}	1·43^{a}	0·31	0·75	0·57	0·49	0·03	<0·001	<0·001	<0·001	D × A, D × S, A × S, D × A × S
16 : 1*n*-7	18·2^{b}	14·4^{b,c}	12·7^{c}	205^{a}	37·0	88·3	67·4	57·7	4·46	<0·001	<0·001	<0·001	D × A, D × S, A × S, D × A × S
18 : 1*n*-7	79·6^{c}	88·2^{b}	66·8^{d}	124^{a}	53·6	126	96·3	82·9	3·93	<0·001	<0·001	<0·001	D × A, D × S, A × S
18 : 1*n*-9	3302^{a}	2465^{b}	2272^{c}	2146^{d}	1532	3560	2734	2358	109	<0·001	<0·001	<0·001	D × A, A × S
20 : 1*n*-9	17·5^{b}	17·5^{b}	16·1^{c}	38·2^{a}	13·3	31·4	24·0	20·7	1·03	<0·001	<0·001	<0·001	D × A, D × S, A × S
ΣMUFA[5]	3418^{a}	2586^{b}	2368^{c}	2515^{b}	1636	3807	2923	2520	116	<0·001	<0·001	<0·001	D × A, A × S
Apparent digestibility (%)													
12 : 0	85·6	87·0	85·7	86·1	85·9	86·2	86·2	86·0	3·95	0·803	0·804	0·924	–
14 : 0	75·4	81·4	77·9	81·3	78·3	79·7	79·2	78·8	6·05	0·055	0·421	0·826	–
16 : 0	72·2	77·4	73·3	70·3	73·0	73·5	73·6	73·0	7·00	0·104	0·814	0·783	–
18 : 0	69·7^{a,b}	74·3^{a}	70·3^{a,b}	66·2^{b}	69·2	71·1	70·3	70·0	6·53	0·037	0·314	0·882	–
20 : 0	75·0^{a,b}	77·5^{a}	78·0^{a}	72·8^{b}	75·7	76·0	75·8	75·8	4·26	0·017	0·813	0·997	–
22 : 0	78·8^{b}	82·0^{b}	89·3^{a}	78·6^{b}	78·9	85·6	82·0	82·4	3·99	<0·001	<0·001	0·692	D × A
ΣSFA	76·1	80·0	79·1	76·0	76·8	78·7	77·8	77·7	5·10	0·138	0·218	0·921	–
16 : 1*n*-7	63·4^{a,b}	70·7^{a}	57·0^{b}	88·3^{c}	68·6	71·0	69·2	70·5	8·44	<0·001	0·335	0·597	D × A, D × S
18 : 1*n*-7	70·0^{b}	78·9^{a}	72·2^{b}	79·1^{a}	73·8	76·2	74·6	75·5	4·86	<0·001	0·100	0·529	D × A, D × S
18 : 1*n*-9	81·0^{b}	88·2^{a}	85·5^{a,b}	85·3^{a,b}	81·3	88·6	85·1	84·9	4·80	0·008	<0·001	0·848	D × A
20 : 1*n*-9	60·7^{c}	70·4^{b}	66·3^{b,c}	76·9^{a}	63·5	73·6	68·0	69·1	5·45	<0·001	<0·001	0·489	D × S
ΣMUFA	68·8^{b}	77·0^{a}	70·2^{b}	82·4^{a}	71·8	77·4	74·2	75·0	5·16	<0·001	<0·001	0·615	D × A, D × S

Palm, palm fat diet; Soya, soyabean oil diet; Lin, linseed oil diet; Fish, fish oil diet; RMSE, root mean squares error; D, diet; A, age; S, sex; ΣSFA, sum of SFA; ΣMUFA, sum of MUFA.
[a,b,c,d] Mean values for diets within a row with unlike superscript letters were significantly different ($P<0·05$).
* Significant interaction terms at $P<0·05$.

https://doi.org/10.1017/S0007114510000541 Published online by Cambridge University Press

18 : 1*n*-9 and ΣMUFA intake was recorded in the birds fed with Palm diet than in the birds fed with the other diets ($P<0·001$). Intake of 14 : 0, 16 : 1*n*-7, 18 : 1*n*-7 and 20 : 1*n*-9 was 3·6-, 13-, 1·6- and 2·2-fold higher, respectively, in birds fed on Fish diet than in birds fed on the other diets ($P<0·001$). The effect of age and sex was significant for the intake of all the given fatty acids, with a higher fatty acid intake in 21–42-d-old chickens and in male broiler chickens always.

Apparent digestibility of 12 : 0, 14 : 0, 16 : 0 and ΣSFA was not influenced by diet, age and sex ($P>0·05$). Digestibility of 18 : 0 was higher for the Soya diet than for the Fish diet ($P<0·05$), with intermediary values for the Palm and Lin diets. Digestibility of 20 : 0 was higher for the Soya and Lin diets than for the Fish diet ($P<0·05$), whereas the digestibility of 22 : 0 was higher for the Lin diet than for the three other diets ($P<0·05$). Digestibility of 16 : 1*n*-7 and 20 : 1*n*-9 was larger for the Fish diet than for the other diets ($P<0·001$). Digestibility of 18 : 1*n*-7 was higher for the Soya and Fish diets than for the Palm and Lin diets ($P<0·001$), and digestibility of 18 : 1*n*-9 was higher for the Soya diet than for the Palm diet ($P<0·01$). The ΣMUFA digestibility was higher in the Soya- and Fish-fed birds than in the Lin- and Palm-fed birds ($P<0·001$). Digestibility of 22 : 0, 18 : 1*n*-9 and 20 : 1*n*-9 and ΣMUFA was higher in the birds slaughtered at day 42 than in those slaughtered at day 21 ($P<0·001$). Sex had no effect on FA digestibility ($P>0·05$).

The SFA and MUFA content of WB broiler chickens and fatty acid appearance/disappearance are presented in Table 3. Diet had a significant effect on the content of most individual SFA and MUFA, except for the major SFA (16 : 0) and for ΣSFA. The 14 : 0 and 14 : 1*n*-5 content was higher in the Fish diet than in the three other diets ($P<0·001$). The 18 : 0 content was higher in the Fish diet than in the Palm diet ($P<0·05$). The 16 : 1*n*-7 was higher for the Fish diet than for the Soya and Lin diets. Birds fed on the Palm diet had a higher 18 : 1*n*-7, 18 : 1*n*-9 and 20 : 1*n*-9 content than the birds fed on Lin, Fish, and Soya diets, respectively ($P<0·001$). The ΣMUFA content was higher in Palm-fed birds than in Fish-fed birds ($P<0·05$), with intermediary values in Soya- and Lin-fed birds. The effect of age was significant on the SFA and MUFA content of broiler chickens (except for 22 : 0), with a higher fatty acid content at 21–42 d of age always. The effect of sex was significant for 16 : 0, 18 : 0, ΣSFA, 18 : 1*n*-9, 20 : 1*n*-9 and ΣMUFA, with a higher fatty acid content in the female birds than in the male birds.

A net disappearance was observed in all dietary treatments for 12 : 0, 16 : 0, 20 : 0 and 22 : 0. For 14 : 0, a net disappearance was observed in birds fed on the Palm and Fish diets that differed from the net appearance that was observed in birds fed on the Soya and Lin diets ($P<0·001$). Birds fed on the Palm diet had a lower 18 : 0 appearance but a higher 18 : 1*n*-7 and 20 : 1*n*-9 appearance in contrast with the birds fed on the other diets ($P<0·001$). Appearance of 16 : 1*n*-7 was lower for the Fish diet than for the other diets ($P<0·001$). Age significantly affected SFA and MUFA appearance/disappearance, with higher values at 7–21 d for all the given fatty acids, except for 18 : 0 and 18 : 1*n*-9. The effect of sex was only significant on 18 : 0 appearance, with higher values in female birds ($P<0·05$).

The different fates of ΣSFA and MUFA are summarised in Table 4. Across diets, sex and age, on average 94·5 % of total SFA and MUFA from the net intake and *ex novo* production were accumulated in the WB, while the rest (5·5 %) were β-oxidised for energy production. Accumulation was lower (85·7 *v.* 97·4 %) and thus β-oxidation was higher (14·3 *v.* 2·6 %) for the Palm diet than for the three other diets ($P<0·001$). Accumulated SFA and MUFA may have either of the three fates in the WB, i.e. deposition as such in the tissues, Δ-9 desaturation of SFA to MUFA and elongation to longer chain metabolites (Fig. 1). Across diets, sex and age, 33·1 % of accumulated ΣSFA and MUFA were elongated to longer chain products, whereas 13·8 % were Δ-9 desaturated. Elongation as a proportion of accumulation was higher for the Lin diet than for the Palm diet ($P<0·01$), and desaturation was higher for the Lin diet than for the Palm and Fish diets ($P<0·01$).

The accretion of elongated 12 : 0 and 14 : 0 (namely 14 : 0 and 16 : 0, respectively) and the accretion of total elongated fatty acids were lower in the Fish- and Palm-fed birds than in the Soya- and Lin-fed birds ($P<0·05$). The apparent elongation of 18 : 1*n*-9 to 20 : 1*n*-9 was higher in birds fed on Palm diet than in those fed on the other diets ($P<0·001$). The apparent elongation of 18 : 1*n*-9 to 20 : 1*n*-9 was slightly higher in birds at 7–21 d of age than in the older birds ($P<0·001$), whereas elongation of all the given SFA was always higher in birds at 21–42 d of age ($P<0·05$).

The accretion of Δ-9 desaturated 14 : 0 (net appearance of 14 : 1*n*-5) was higher for the Fish diet than for the other diets, whereas the opposite trend was observed for 16 : 0 desaturation (net appearance of 16 : 1*n*-7; $P<0·001$). Total Δ-9 desaturation activity and Δ-9 desaturation of 18 : 0 were not significantly affected by dietary manipulation ($P>0·05$). The apparent activity of Δ-9 desaturase on 16 : 0 was higher in 7–21-d-old birds than in the older birds, while the opposite was observed for 18 : 0 ($P<0·05$). Sex did not influence apparent elongase and Δ-9 desaturase activities of the given fatty acids ($P>0·05$).

The β-oxidation of SFA and MUFA is presented in Table 5. Total β-oxidation of ΣSFA and MUFA and β-oxidation of 12 : 0, 16 : 0 and 18 : 1*n*-9 were higher for birds fed on the Palm diet than for those fed on the other diets ($P<0·001$; $P<0·05$ for 18 : 1*n*-9). β-Oxidation of 14 : 0 was higher for the Fish diet than for the Soya and Lin diets. β-Oxidation of 20 : 0 was higher for the Palm and Lin diets than for the Soya and Fish diets ($P<0·001$). Birds fed on the Soya diet had a higher 22 : 0 β-oxidation than those fed on the other diets ($P<0·001$). Age influenced β-oxidation of the fatty acids (except for 18 : 1*n*-7), with greater values in 7–21-d-old birds always ($P<0·05$). Sex did not influence β-oxidation of the fatty acids ($P>0·05$).

The total *ex novo* production and β-oxidation of ΣSFA and MUFA were plotted against the net intake of these fatty acids expressed as μmol/g per d (Fig. 2). *Ex novo* production of SFA and MUFA showed a negative curvilinear relationship with the net intake of these fatty acids (R^2 0·56). A similar trend was observed for individual SFA and MUFA (data not shown). The β-oxidation of ΣSFA and MUFA indicated a strong positive curvilinear relationship with the net intake of these fatty acids (R^2 0·97). A sharp increase in β-oxidation was observed by elevating the net intake of SFA and MUFA from 23 to 30 μmol/g per d. Similar strong relationships were observed for individual SFA and MUFA (data not shown).

Table 3. SFA and MUFA content (mg/g broiler chicken) and fatty acid appearance/disappearance (μmol/g per d) in whole body broiler chickens

	Diet (*n* 12)				Age (*n* 24; d)		Sex (*n* 24)			*P*			
	Palm	Soya	Lin	Fish	7–21	21–42	Male	Female	RMSE	Diet	Age	Sex	Interactions*
Fatty acid content (mg/g broiler chicken)													
12 : 0	0·08^{a,c}	0·07^{b}	0·07^{b,c}	0·09^{a}	0·07	0·09	0·08	0·08	0·01	<0·001	<0·001	0·699	–
14 : 0	0·74^{b}	0·66^{b}	0·66^{b}	1·63^{a}	0·73	1·11	0·89	0·96	0·12	<0·001	<0·001	0·051	D × A
16 : 0	24·1	24·1	23·1	23·5	19·2	28·2	22·5	25·0	3·73	0·889	<0·001	0·029	–
18 : 0	4·57^{b}	5·26^{a,b}	5·09^{a,b}	5·36^{a}	4·30	5·84	4·73	5·41	0·69	0·041	<0·001	0·001	–
20 : 0	0·12^{b}	0·16^{a}	0·08^{c}	0·11^{b}	0·09	0·14	0·12	0·12	0·01	<0·001	<0·001	0·140	D × A
22 : 0	0·03^{b}	0·04^{a}	0·04^{a}	0·04^{a}	0·03	0·04	0·03	0·04	0·01	<0·001	0·201	0·277	D × A
ΣSFA	29·7	30·3	29·1	30·7	24·4	35·5	28·3	31·5	4·51	0·830	<0·001	0·019	–
14 : 1*n*-5	0·14^{b}	0·11^{b}	0·12^{b}	0·19^{a}	0·11	0·17	0·14	0·14	0·03	<0·001	<0·001	0·808	D × A
16 : 1*n*-7	4·52^{a,b}	3·92^{b}	4·12^{b}	5·09^{a}	3·72	5·10	4·35	4·48	0·80	0·005	<0·001	0·402	D × A
18 : 1*n*-7	1·95^{a}	1·74^{a,b}	1·54^{b}	1·71^{a,b}	1·57	1·90	1·69	1·79	0·23	0·001	<0·001	0·129	D × A
18 : 1*n*-9	40·3^{a}	37·9^{a,b}	36·3^{a,b}	32·0^{b}	30·0	43·3	34·8	38·5	5·40	0·005	<0·001	0·023	A × S
20 : 1*n*-9	0·34^{a}	0·31^{a,b}	0·26^{b}	0·32^{a}	0·27	0·34	0·29	0·32	0·04	<0·001	<0·001	0·029	–
ΣMUFA	47·3^{a}	44·0^{a,b}	42·4^{a,b}	39·3^{b}	35·7	50·8	41·3	45·2	6·35	0·031	<0·001	0·038	A × S
Total fatty acids†	89·6	99·1	99·4	87·4	76·7	111	89·8	98·0	12·7	0·051	<0·001	0·032	–
Fatty acid appearance/disappearance (μmol/g per d)													
12 : 0	−0·04^{a}	−0·01^{c}	−0·01^{c}	−0·02^{b}	−0·03	−0·02	−0·02	−0·02	0·00	<0·001	<0·001	0·950	D × A
14 : 0	−0·08^{a}	0·02^{b}	0·04^{b}	−0·04^{c}	−0·16	−0·04	−0·11	−0·09	0·04	<0·001	<0·001	0·121	D × A
16 : 0	−4·85^{a}	−0·90^{b}	−0·31^{b}	−1·38^{b}	−3·30	−0·42	−2·19	−1·53	1·34	<0·001	<0·001	0·099	D × A
18 : 0	0·05^{b}	0·38^{a}	0·42^{a}	0·52^{a}	0·27	0·41	0·26	0·42	0·20	<0·001	0·019	0·013	–
20 : 0	−0·071^{a}	−0·048^{b}	−0·070^{a}	−0·050^{b}	−0·077	−0·043	−0·061	−0·059	0·005	<0·001	<0·001	0·436	D × A
22 : 0	−0·020^{c}	−0·036^{a}	−0·024^{b}	−0·019^{c}	−0·028	−0·021	−0·025	−0·025	0·002	<0·001	<0·001	0·684	D × A
14 : 1*n*-5	0·04^{b}	0·04^{b}	0·04^{b}	0·06^{a}	0·04	0·04	0·04	0·04	0·01	<0·001	0·539	0·490	–
16 : 1*n*-7	1·28^{a}	1·12^{a}	1·15^{a}	0·71^{b}	1·16	0·98	1·09	1·04	0·27	<0·001	0·032	0·169	–
18 : 1*n*-7	0·30^{a}	0·19^{b}	0·21^{b}	0·06^{c}	0·22	0·16	0·18	0·19	0·07	<0·001	0·011	0·823	–
18 : 1*n*-9	0·45^{b}	1·93^{a,b}	2·26^{a}	1·22^{a,b}	0·86	2·08	1·10	1·84	1·59	0·042	0·012	0·116	–
20 : 1*n*-9	0·05^{a}	0·03^{b}	0·02^{b}	−0·02^{c}	0·02	0·01	0·02	0·02	0·01	<0·001	0·003	0·352	D × A, A × S

Palm, palm fat diet; Soya, soyabean oil diet; Lin, linseed oil diet; Fish, fish oil diet; RMSE, root mean squares error; D, diet; A, age; S, sex; ΣSFA, sum of SFA; ΣMUFA, sum of MUFA.
^{a,b,c} Mean values for diets within a row with unlike superscript letters were significantly different ($P<0·05$).
* Significant interaction terms at $P<0·05$.
† Sum of SFA, MUFA and PUFA.

https://doi.org/10.1017/S0007114510000541 Published online by Cambridge University Press

Table 4. Fate of sum of SFA and MUFA (% net intake + *ex novo* production), fate of accumulated sum of SFA and MUFA (% of accumulation), and accretion of elongated and desaturated fatty acids (μmol/g per d) in broiler chickens

	Diet (*n* 12)				Age (*n* 24; d)		Sex (*n* 24)			*P*			
	Palm	Soya	Lin	Fish	7–21	21–42	Male	Female	RMSE	Diet	Age	Sex	Interactions*
Fate of ΣSFA and MUFA (% net intake+ *ex novo* production)													
Accumulation	85·7^{b}	96·7^{a}	98·8^{a}	96·8^{a}	90·8	98·2	94·2	94·8	6·37	<0·001	<0·001	0·751	D × A
β-Oxidation	14·3^{a}	3·27^{b}	1·20^{b}	3·20^{b}	9·19	1·78	5·78	5·19	6·38	<0·001	<0·001	0·751	D × A
Fate of accumulated fatty acids (% of accumulation)													
Elongation	14·7^{b}	42·0^{a,b}	51·7^{a}	24·2^{a,b}	18·7	47·5	26·3	40·0	26·8	0·008	<0·001	0·086	–
Δ9-Desaturation	11·1^{b}	15·6^{a,b}	18·0^{a}	10·5^{b}	10·9	16·6	12·6	15·0	5·90	0·009	0·002	0·168	A × S
Accretion of elongated fatty acid (μmol/g per d)													
12 : 0 → 14 : 0	0·99^{b}	3·45^{a}	3·96^{a}	1·36^{a}	1·40	3·47	1·79	3·09	2·80	0·029††	0·015	0·117	–
14 : 0 → 16 : 0	0·98^{b}	3·40^{a}	3·90^{a}	1·54^{b}	1·44	3·47	1·81	3·09	2·77	0·040††	0·016	0·120	–
16 : 0 → 18 : 0	1·22	2·47	2·71	1·76	1·53	2·55	1·61	2·47	1·60	0·111	0·033	0·071	–
18 : 1*n*-9 → 20 : 1*n*-9	0·05^{a}	0·03^{b}	0·02^{b}	0·00^{c}	0·03	0·02	0·02	0·03	0·01	<0·001	<0·001	0·571	D × A
Total	3·53^{b}	9·55^{a}	10·8^{a}	4·71^{b}	4·62	9·67	5·43	8·88	7·21	0·048††	0·020	0·107	–
Accretion of Δ9-desaturated fatty acid (μmol/g per d)													
14 : 0 → 14 : 1*n*-5	0·04^{b}	0·03^{b}	0·04^{b}	0·06^{a}	0·04	0·04	0·04	0·04	0·01	<0·001	0·539	0·490	–
16 : 0 → 16 : 1*n*-7	1·57^{a}	1·31^{a}	1·36^{a}	0·77^{b}	1·37	1·14	1·27	1·24	0·34	<0·001	0·022	0·715	–
18 : 0 → 18 : 1*n*-9	1·08	2·10	2·30	1·24	1·22	2·14	1·31	2·04	1·42	0·112	0·031	0·084	A × S
Total	2·70	3·45	3·69	2·07	2·63	3·32	26·3	3·25	1·73	0·112	0·180	0·174	A × S

Palm, palm fat diet; Soya, soyabean oil diet; Lin, linseed oil diet; Fish, fish oil diet; RMSE, root mean squares error; D, diet; A, age; S, sex.
[a,b,c] Mean values for diets within a row with unlike superscript letters were significantly different at $P<0·05$.
* Significant interaction terms at $P<0·05$.
† Mean values for diets were significantly different ($P<0·1$).

NS British Journal of Nutrition

Table 5. β-Oxidation of SFA and MUFA in broiler chickens (μmol/g per d)

	Diet (*n* 12)				Age (*n* 24; d)		Sex (*n* 24)			*P*			
	Palm	Soya	Lin	Fish	7–21	21–42	Male	Female	RMSE	Diet	Age	Sex	Interactions*
β-Oxidation of fatty acid (μmol/g per d)													
12 : 0	0·038[a3]	0·006[b]	0·001[b]	0·011[b]	0·024	0·004	0·014	0·014	0·012	<0·001	<0·001	0·967	D × A × S
14 : 0	0·057[a,b]	0·004[b]	0·000[b]	0·151[a]	0·092	0·014	0·051	0·055	0·113	0·008	0·024	0·916	–
16 : 0	3·03[a]	0·50[b]	0·14[b]	0·38[b]	1·83	0·20	1·12	0·91	1·03	<0·001	<0·001	0·493	D × A
20 : 0	0·071[a]	0·049[b]	0·070[a]	0·051[b]	0·077	0·043	0·061	0·059	0·005	<0·001	<0·001	0·436	D × A
22 : 0	0·020[c]	0·035[a]	0·024[b]	0·019[c]	0·028	0·021	0·025	0·025	0·002	<0·001	<0·001	0·684	D × A × S
18 : 1*n*-7	<0·001	<0·001	<0·001	<0·001	<0·001	<0·001	<0·001	<0·001	0·001	0·405	0·324	0·324	–
18 : 1*n*-9	0·58[a]	0·13[a,b]	0·00[b]	0·02[b]	0·33	0·04	0·19	0·17	0·48	0·021	0·049	0·923	–
Total	3·88[a]	0·72[b]	0·23[b]	0·66[b]	2·43	0·32	1·50	1·25	1·48	<0·001	<0·001	0·573	D × A

Palm, palm fat diet; Soya, soyabean oil diet; Lin, linseed oil diet; Fish, fish oil diet; RMSE, root mean squares error.
[a,b] Mean values for diets within a row with unlike superscript letters were significantly different ($P<0·05$).
*Significant interaction terms at $P<0·05$.

Fig. 2. Total *ex novo* production and β-oxidation of total SFA and MUFA in relation to the sum of SFA and MUFA net intake (μmol/g per d) across diets, age and sex (*n* 16). Broken line regression equation for *ex novo* production: $Y = 0$, if $X > 24·51$, and $Y = 24·51 + 0·4076 \times (24·51 - X)$, if $X \leq 24·51$; R^2 0·56; root mean square means (RMSE) = 1·53 (*n* 16). Broken line quadratic regression equation for β-oxidation: $Y = 0·078$, if $X < 15·28$, and $Y = 0·078 + 0·0362 \times (X - 15·28)^2$, if $X \geq 15·28$; R^2 0·97; RMSE = 0·403. ■, *Ex novo*; ⁎, β-oxidation.

The relationship between total apparent elongase activity and the net intake of ΣSFA and MUFA (μmol/g per d) was exponential (R^2 0·70; Fig. 3). It was observed that increasing the SFA and MUFA net intake is associated with decreasing total elongase activity on SFA and MUFA. A slight negative linear relationship between total apparent Δ-9 desaturase activity and SFA net intake was noticed (r −0·48; n 16; data not shown).

Significant interaction terms are given in the tables, but these are not further discussed here since they were negligible compared with the main effects. *P*-values for the interaction effects were considerably smaller than those for the main effects. In addition, the significant interaction effects were in most cases the result of scale differences, and only in a few cases, they were the result of opposite effects. The most marked diet × age interaction effect was for the digestibility of 16 : 1*n*-7 and 18 : 1*n*-7, which was higher in the young birds than in the older birds fed on the Lin diet, whereas no difference or the opposite was found in the birds fed on the other diets.

Fig. 3. Total apparent elongase activity on total SFA and MUFA in relation to the sum of SFA and MUFA net intake (μmol/g per d) across diets, age and sex. Exponential regression equation: $Y = 146 \times e^{-0·17X}$; R^2 0·70; root mean square means 4·23 (*n* 16).

Discussion

The experiment underlying the present study was primarily designed to examine the effect of different dietary PUFA sources on PUFA metabolism[13]. Hence, diets differed more in the content and source of PUFA than in those of SFA and MUFA. Nevertheless, we assumed that the experimental data are appropriate for making inferences on SFA and MUFA metabolism also, which is the topic of the present manuscript.

Apparent digestibilities of 12 : 0, 14 : 0, 16 : 0, 18 : 0, ΣSFA and 18 : 1*n*-9 in this experiment were in the same range as that reported previously[15–17]. We found no data on apparent digestibilities of long-chain SFA and MUFA in the literature, e.g. 20 : 0 and 20 : 1*n*-9, to compare with the present results. It has to be mentioned that the digestibility of SFA may be underestimated due to endogenous biosynthesis of SFA and biohydrogenation of unsaturated fatty acids in the chicken hindgut, while the opposite should be considered for MUFA[18]. A relatively higher fatty acid digestibility in birds slaughtered at 42 d of age than in those slaughtered at 21 d of age might be due to a low bile salt concentration and lower formation of mixed micelles and lower lipid absorption in younger broiler chickens[17]. An interesting finding of the present study was that across dietary treatments, the ΣSFA content of broiler chickens was almost constant (approximately 30 mg/g), suggesting that to increase the MUFA:SFA ratio in the broiler chicken tissues or final products, the prime strategy is to enhance dietary MUFA than to decrease SFA.

Thanks to the WB fatty acid balance method implemented in the present study, it was possible to estimate and follow the fate of dietary fatty acids. In Palm-fed birds, intake and total β-oxidation of SFA and MUFA were highest, whereas accumulation (percentage of net intake and *ex novo* production) was lowest when compared with the other groups. In Lin-fed birds, intake of SFA and MUFA was minimal, while apparent elongation and Δ-9 desaturation activities were higher when compared with the other groups. It can thus be stated that the dietary intake of SFA and MUFA has a negative relationship with the accumulation of these fatty acids, but a positive relationship with β-oxidation.

The Δ-9 desaturase (stearoyl-CoA desaturase) enzyme is a rate-limiting lipogenic enzyme, and is considered to modulate *de novo* fatty acid biosynthesis[19–21]. An earlier study suggested that Δ-9 desaturase in chickens is up-regulated by low-fat high-carbohydrate diets, and is down-regulated by the addition of dietary PUFA[22]. In general, it has been reported that Δ-9 desaturase activity could be influenced by different factors including nutrition[23]. In the present study, the dietary fat level was constant, and Δ-9 desaturase activity was not influenced by the PUFA content of the diets. Across diets, sex and age, 18 : 0 followed by 16 : 0 were the most Δ-9 desaturated fatty acids in the present study. This corresponds with a previous finding that the preferred substrates for Δ-9 desaturase are palmitoyl-CoA and stearoyl-CoA[22].

The elongation process is involved in many aspects of endogenous fatty acid synthesis by inserting two carbon units at the carboxyl terminal of fatty acids[19]. In the current experiment, accretion of an elongated fatty acid was used as an indication of the total apparent *in vivo* elongase activity on this fatty acid. Comparatively, accretion of elongated fatty acids was numerically higher than that of Δ-9 desaturated fatty acids. This is in harmony with a recent report in rainbow trout using a similar WB fatty acid balance method[11]. According to another study, elongation activity in bovine adipose tissue is 3-fold greater than desaturation activity[24]. In bovine, the desaturation of 18 : 0 to form 18 : 1*n*-9 is proposed as the limiting process in the biosynthesis of 18 : 1*n*-9 from 16 : 0[24]. Hence, the addition of a double bond appears to be the rate-limiting step in MUFA biosynthesis rather than the chain elongation across species.

Total apparent *in vivo* elongase activity was significantly reduced when SFA and MUFA were abundantly provided (Palm diet). This, together with the fact that total Δ-9 desaturase activity seemed to be unaffected by dietary fatty acid composition, suggests that a dietary surplus of SFA and MUFA may be an obstacle in the biosynthesis of some MUFA, especially 18 : 1*n*-9, by suppressing elongase activity. However, in the birds supplied with abundant SFA and MUFA, the actual accumulation of MUFA directly originating from the diet coped with the inhibited endogenous biosynthesis of these fatty acids and therefore recorded the highest accumulation in the WB.

Comparing Fish- and Lin-fed birds in the present study with rainbow trout fish given similar dietary treatments[11], it was found that accretion of elongated SFA (14 : 0 and 16 : 0) is higher in broiler chickens than in rainbow trout fish, whereas elongation of MUFA (18 : 1*n*-9) in chickens seems to be half of that in trout. Interestingly, accretion of Δ-9 desaturated 16 : 0 and 18 : 0 in Fish and Lin groups of chickens seems to be up to 7-fold higher than that in similarly fed rainbow trout.

It is known that fatty acids with a chain length ranging from eight to twenty carbon atoms, both SFA and MUFA, are good substrates for mitochondrial β-oxidation[25]. The effect of diet on β-oxidation of SFA and MUFA in this experiment confirms isotopic studies in rats and human subjects arguing that the nature of the ingested fat affects β-oxidation of the fatty acids[2]. In the present study across diets, 16 : 0 showed a higher β-oxidation rate than the other fatty acids. It has been explained by Sanz *et al.*[26] that fatty acids are not utilised on an equal basis for oxidation. Our finding regarding higher β-oxidation of 16 : 0 over 18 : 1*n*-9, 20 : 0 and 22 : 0 is in general harmony with a previous report that the β-oxidation rate drops sharply when the fatty acid chain length increases from eighteen to twenty carbon atoms[25].

In the present experiment, the β-oxidation of ΣSFA seemed to be higher than that of ΣMUFA in broiler chickens. This is in agreement with an earlier study indicating a preferential β-oxidation of SFA over MUFA in rat muscle, heart, and liver, and beef liver[12]. Across diets, age and sex, β-oxidation of both SFA and MUFA in the present study followed the same trend as fatty acid net intake, suggesting that despite the possible existence of a preferential order of oxidation for some fatty acids over others, the fatty acid oxidation is primarily directed towards those provided in surplus by the diet. On the other hand, it has been reported that diets containing high amounts of PUFA result in greater rates of β-oxidation of fatty acids[15,26,27]. This was not apparent in the present study. Total PUFA intake was in the order Lin > Soya > Fish > Palm diets. The β-oxidation of ΣSFA and ΣMUFA (percentage of net intake and *ex novo* production) was similar for the Soya, Lin and Fish diets, and was lower for these diets

than for the Palm diet. In the accompanying paper[13] on the PUFA metabolism, the β-oxidation of 18 : 2*n*-6 and 18 : 3*n*-3 was also not related to their proportions in the diet. Hence, higher proportions of PUFA in the diet did not increase β-oxidation of fatty acids in the present study, in contrast with the above-mentioned studies. In these studies, high amounts of PUFA in the diet also reduced abdominal fat pad weights. This was also not apparent in the present study. However, it should be mentioned that in the present study, 3 % of a PUFA source was included in the diet, whereas this was 8–10 % in the above-mentioned studies[15,26,27].

Opposite to the ΣSFA and MUFA accumulation, elongation and Δ-9 desaturation, β-oxidation in 7–21-d-old birds was higher than that in the older birds. Accordingly, as reported previously by Kloareg *et al.*[28], it is known that the stage of growth and development influences β-oxidation of the fatty acids. Young fast-growing chickens possess a higher rate of metabolism than chickens approaching slaughter age.

Similar to isotopic methods, the WB method applied in the present study was unable to estimate the multidirectional metabolism of some fatty acids. For instance, 18 : 1*n*-9 can be elongated to form 20 : 1*n*-9 or saturated to form 18 : 0. Conversely, 18 : 1*n*-9 can be formed from these fatty acids[29]. The WB fatty acid balance method estimates fatty acid metabolism based on the fatty acid detection in the WB pool over a specific time frame. Besides the drawbacks, the employed WB fatty acid balance method has proven to be reliable to study the turnover of SFA and MUFA in the WB[11,30].

In conclusion, the present study has shown that increasing the intake of SFA and MUFA is associated with a lower accumulation of SFA and MUFA relative to the net intake and *ex novo* production in WB broiler chickens, a higher rate of β-oxidation and a lower rate of apparent elongase activity. Apparent elongase and Δ-9 desaturase activities increase with age, but sex has no effect on this.

Acknowledgements

The authors wish to acknowledge the Iranian Ministry of Science and Technology for providing the first author the opportunity to do this research. The authors also thank the technical staff of the Laboratory of Animal Nutrition and Animal Product Quality and of the Animal Science Unit of the Institute for Agricultural and Fisheries Research involved in this experiment for their skilled assistance. None of the authors has any financial or personal conflict of interest to disclose. The study was conducted by own funding. R. P. conducted the laboratory analysis, and was involved in data analysis and writing of the manuscript. G. M. T. assisted in data analysis and manuscript writing. G. H. was involved in setting up and carrying out the animal experiment. K. R. and S. D. S. were involved in the experimental setup, data analysis and manuscript writing.

References

1. Lesna K, Suchanek P, Kover J, *et al.* (2008) Replacement of dietary saturated FAs by PUFAs in diet and reverse cholesterol transport. *J Lipid Res* **49**, 2414–2418.
2. Bergouignan A, Momken I, Schoeller DA, *et al.* (2009) Metabolic fate of saturated and mono-unsaturated dietary fats: the Mediterranean diet revisited from epidemiological evidence to cellular mechanisms. *Prog Lipid Res* **48**, 128–147.
3. Lopez S, Bermudez B, Pacheco YM, *et al.* (2007) Dietary oleic and palmitic acids modulate the ratio of triacylglycerols to cholesterol in postprandial triacylglycerols-rich lipoproteins in men and cell viability and cycling in human monocytes. *J Nutr* **137**, 1999–2005.
4. Mendez MA, Popkin BM, Jakszyn P, *et al.* (2006) Adherence to a Mediterranean diet is associated with reduced 3-year incidence of obesity. *J Nutr* **136**, 2934–2938.
5. De Smet S & Raes K (2004) Meat fatty acid composition as affected by fatness and genetic factors: a review. *Anim Res* **53**, 81–98.
6. Rymer C & Givens DI (2005) *n*-3 Fatty acid enrichment of edible tissues of poultry: a review. *Lipids* **40**, 121–130.
7. Poureslami R, Raes K Huyghebaert G, *et al.* (2010) Effect of diet, age, and gender on the polyunsaturated fatty acid composition of broiler anatomical compartments. *Br Poult Sci* (In the Press).
8. Infield JM & Annison EF (1973) The metabolism of palmitic, stearic, oleic, and linoleic acids in broiler chickens. *Br J Nutr* **30**, 545–554.
9. Turchini GM, Francis DS & De Silva SS (2007) A whole body, *in vivo*, fatty acid balance method to quantify PUFA metabolism (desaturation, elongation and beta-oxidation). *Lipids* **42**, 1065–1071.
10. Cunnane S (2001) Application of new methods and analytical approaches to research on polyunsaturated fatty acid homeostasis. *Lipids* **36**, 975–979.
11. Turchini GM & Francis DS (2009) Fatty acid metabolism (desaturation, elongation and β-oxidation) in rainbow trout fed fish oil- or linseed oil-based diets. *Br J Nutr* **102**, 69–81.
12. Jones PJH & Pencharz PB (1985) Whole body oxidation of dietary fatty acids: implications for energy utilization. *Am J Clin Nutr* **42**, 769–777.
13. Poureslami R, Raes K, Turchini GM, *et al.* (2010) Effect of diet, sex and age on fatty acid metabolism in broiler chickens: *n*-3 and *n*-6 PUFA. *Br J Nutr* **104**, 189–197.
14. Turchini GM, Francis DS & De Silva SS (2008) A whole body *in vivo*, fatty acid balance method to quantify PUFA metabolism (desaturation, elongation and beta-oxidation). *Lipids* **43**, 977, Erratum to Lipids (2007) **42**, 1065–1071.
15. Ortiz LT, Rebole A & Alzueta C (2001) Metabolisable energy value and digestibility of fat and fatty acids in linseed determined with growing broiler chickens. *Br Poult Sci* **1**, 57–63.
16. Crespo N & Esteve-Garcia E (2002) Nutrient and fatty acid deposition in broilers fed different dietary fatty acid profiles. *Poult Sci* **81**, 1533–1542.
17. Smits CHM, Moughan PJ & Beynen AC (2000) The inhibitory effect of a highly viscous carboxymethylcellulose on dietary fat digestibility in the growing chicken is dependent on the type of fat. *J Anim Physiol Anim Nutr* **83**, 231–238.
18. Smink W, Gerrits WJJ, Hovenier R, *et al.* (2008) Fatty acid digestion and deposition in broiler chickens fed diets containing either native or randomized palm oil. *Poult Sci* **87**, 506–513.
19. Jeffcoat R (1979) The biosynthesis of unsaturated fatty acids and its control in mammalian liver. *Essays Biochem* **15**, 1–36.
20. Garcia-Fernandez M, Gutierrez-Gil B, Garcia-Gamez E, *et al.* (2009) Genetic variability of the stearoyl-CoA desaturase gene in sheep. *Mol Cell Probes* **23**, 107–111.
21. Flowers MT & Ntambi JM (2009) Stearoyl-CoA desaturase and its relation to high-carbohydrate diets and obesity. *Biochim Biophys Acta* **1791**, 85–91.
22. Lefevre P, Tripon E, Plumelet C, *et al.* (2001) Effects of polyunsaturated fatty acids and clofibrate on chicken stearoyl-CoA desaturase 1 gene expression. *Biochem Biophys Res Commun* **280**, 25–31.

23. Brenner RR (1989) Factors influencing fatty acid chain elongation and desaturation. In *The Role of Fats in Human Nutrition*, 2nd ed., pp. 45–79 [AJ Vergroesen and M Crawford, editors]. London: Academic Press.
24. John LCS, Lunt DK & Smith SB (1991) Fatty acid elongation and desaturation enzyme activities of bovine liver and subcutaneous adipose tissue microsomes. *J Anim Sci* **69**, 1064–1073.
25. Bremer J & Norum KR (1982) Metabolism of very long-chain monounsaturated fatty acids (22 : 1) and adaptation to their presence in the diet. *J Lipid Res* **23**, 243–256.
26. Sanz M, Lopez-Bote CJ, Menoyo D, *et al.* (2000) Abdominal fat deposition and fatty acid synthesis are lower and β-oxidation is higher in broiler chickens fed diets containing unsaturated rather than saturated fat. *J Nutr* **130**, 3034–3037.
27. Newman RE, Bryden WL, Fleck E, *et al.* (2002) Dietary *n*-3 and *n*-6 fatty acids alter avian metabolism: metabolism and abdominal fat deposition. *Br J Nutr* **88**, 11–18.
28. Kloareg M, Noblet J & Milgen JV (2007) Deposition of dietary fatty acids *de novo* synthesis and anatomical partitioning of fatty acids in finishing pigs. *Br J Nutr* **97**, 35–44.
29. Lin DS, Connor WE & Spenler CW (1993) Are dietary saturated, monounsaturated, and polyunsaturated fatty acids deposited to the same to the same extent in adipose tissue or rabbits? *Am J Clin Nutr* **58**, 174–179.
30. Francis DS, Peters DJ & Turchini GM (2009) Apparent *in vivo* Δ-6 desaturase activity, efficiency, and affinity are affected by total dietary C_{18} PUFA in the freshwater fish Murray Cod. *J Agric Food Chem* **57**, 4381–4390.