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Effect of dietary fat source and bird's sex on performance and peroxisome proliferator-activated receptor-γ gene expression in broiler chicks

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ABSTRACT

The present study was conducted to investigate the effects of different sources and levels of dietary fat and bird's sex on performance and gene expression of peroxisome proliferator-activated receptor-gamma (PPARγ), as an adipose differentiation and development regulatory key, in broiler chicks. One-week-old broiler chicks (840: 420 males and 420 females) were randomly allocated into a factorial arrangement of treatments including 3 different fat sources (soybean oil, yellow grease, and poultry fat), 3 different fat levels (0, 20, and 40 g/kg of diet), and 2 sex categories (male and female) based on a completely randomized design with 4 replicates of 15 birds each. In addition to performance parameters, abdominal fat tissue was collected from 6 wk-old chicks and total RNA was extracted by RNX reagent. A semi-quantitative reverse transcription PCR was performed to investigate the effects of dietary treatments on PPARγ gene expression. Results showed that dietary fat source had no effect on body weight gain (BWG); however, supplementation of 20 g/kg fat into the broiler diets improved feed conversion ratio (FCR). Male chicks had higher feed intake and BWG, as well as the better FCR values than female broilers. The highest expression of PPARγ gene was detected in the abdominal fat of birds fed on diets containing soybean oil and poultry fat, and females expressed PPARγ gene higher than males. Our findings indicated that different dietary fat sources and sex might affect the development of broiler abdominal fat by interfering in PPARγ gene expression.

Key words: Broiler chick, Dietary fat source, Gene expression, Poultry fat, PPARγ

Digestibility of dietary fat sources, included in broiler diets to achieve high energy density, however, depends on fatty acid profile, which affects the performance parameters of birds (Crespo and Esteve-Garcia 2002) and also expression of some genes involved in the modulation of energy metabolism (Rosen *et al*. 2000). One of these genes is peroxisome proliferator-activated receptor-gamma (PPARγ), which modulates glucose and lipid metabolism (Rosen and Spiegelman 2001). In mammals, PPARγ regulates several gene expressions that are involved in proliferation and differentiation of adipose tissues (Tontonoz *et al*. 1995, Saladin *et al*. 1999). In chickens, PPARγ is expressed in various tissues; however, the highest expression was detected in the abdominal fat deposits. It seems that PPARγ has a critical role in differentiation of chickens' adiposities (Meng *et al*. 2005, Wang *et al*. 2008).

In rodent, dietary fatty acids are associated with the higher expression of PPARγ (Vidal-Puig *et al*. 1996) and nutritional deprivation in porcine could reduce the expression of adipocyte's PPARγ gene (McNeel *et al*. 2000). In broiler chicks, increase in dietary concentrations of polyunsaturated fatty acids (PUFA) markedly enhanced the expression of PPARγ (Sato *et al*. 2004). Investigations with mammals showed that sexual hormones affect the expression of this gene, directly or indirectly. Estrogen and androgen receptors were identified in the adipose tissue of mammals (Dieudonne *et al*. 2000) and it was suggested that sex hormones could affect adipose tissue and lipid metabolism through these specific receptors (Ma *et al*. 1998). Data with avian species are scarce for PPARγ gene expression and factors affecting it.

Economically, there is a real concern in poultry industry to reduce the fat content of products by dietary or genetic manipulations. Also, the limitation and high costs of dietary energy sources (especially human-consumed oil sources) have encouraged recycling of some waste products such as poultry fat (PF). The present study was conducted to investigate the effects of different fat sources including soybean oil (SO), PF, and yellow grease (YG) on performance and PPARγ gene expression in abdominal adipose tissue of male and female broiler chicks.

MATERIALS AND METHODS

Birds, diets and experimental procedures: The present trial was performed at the Poultry Research Station of the university, and all procedures used were approved by Isfahan University of Technology Animal Care and Use

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Committee. one-week-old broiler chicks (840: 420 males and 420 females) were randomly distributed among different dietary treatments according to a 3×3×2 factorial arrangement of treatments consisting 3 different fat sources (SO, YG, and PF), 3 different fat levels (0, 20, and 40 g/kg of diet), and 2 sex categories (male and female) based on a completely randomized design with 4 replicates of 15 birds. Yellow grease used was of plant origin, which was obtained from food industry. Experimental diets were formulated using analyzed (AOAC 2002) feed ingredients to meet nutrient requirements according to NRC (1994) recommendations for broiler chicks during the starter (7 to 21 d of age) and grower (22 to 42 d of age) periods (Tables 1 and 2, respectively). The metabolizable energy values of used fat sources were considered as 38.92, 37.46, and 33.90 MJ/kg for SO, YG, and PF, respectively (Jahanian 2014). Chicks had free access to water and experimental diets throughout the duration of study. Body weight and feed consumption were measured periodically as starter and grower periods. Probable mortalities were recorded daily to adjust feed: gain data. Different fat sources were analyzed for their fatty acid profile by gas chromatography (GC) (López-Ferrer *et al*. 1999).

On the final day of trial (day 42 of age), 2 birds / pen nearest to the average weight of pen were selected to evaluate PPARγ gene expression. Feed was removed 3 h before slaughtering. Each bird was exsanguinated by cutting the jugular vein and allowed to bleed for approximately 2 min. Abdominal fat samples were removed immediately and total RNA was extracted with RNX reagent and stored at –80 °C until further analysis could be performed.

Primers were referred to chicken PPARγ (accession number: AF470456) and GAPDH (accession number: K01458). These primers were PPARγ F: 52 -TAC ATA AAG TCC TTC CCG CTG ACC-32 R: 52 -TCC AGT GCG TTG AAC TTC ACA GC-32 and GAPDH F: 52 -TGA CGT GCA GCA GGA ACA C-32 , R: 52 -CAG TTG GTG GTG CAC GAT G- 32, respectively.

Reverse transcriptions (RT) were performed with 7 µg of total RNA extracted from chicken abdominal fat. The cDNA was amplified by polymerase chain reaction (PCR) in a 20 µl reaction volume containing 2 µl of 10 X PCR buffer, 0.4 µl of each 10 mMdNTP mixture, 0.5 unit of Ex Taq DNA polymerase, 2 µl of cDNA, and 1 µl of each 10 pM primers. In the present study, the condition of PCR amplification was as fallows: one cycle at 97 °C for 5 min, 25 cycles at 95 °C for 30 sec, 67 °C for 30 sec and 72 °C for 1 min, and a final extension cycle at 72 °C for 8 min. The PCR-amplified fragments were run beside molecular weight markers on 2% agarose gel stained with ethidium bromide.

Statistical analysis: Data related to body weight gain (BWG), feed intake (FI), and feed conversion ratio (FCR)

Table 1. Ingredients and nutrient composition of experimental diets during starter period (7–21d)

Ingredients (g/kg unless stated otherwise)	Control	20 g/kg soybean oil	20 g/kg yellow grease	20 g/kg poultry fat	40 g/kg soybean oil	40 g/kg yellow grease	40 g/kg poultry fat
Corn, yellow	611.6	541.1	544.3	552.2	466.1	472.6	488.3
Soybean meal	304.5	304.6	305.4	307.3	298.9	300.4	304.3
Fish meal	50	50	50	50	50	50	50
Wheat bran	5	40.4	36.4	26.6	96.4	88.3	68.6
Fat supplement	–	20	20	20	40	40	40
Limestone	10.3	10.3	10.3	10.3	10.3	10.3	10.2
Dicalcium phosphate	8.1	8	8	8.1	7.8	7.8	7.9
Common salt	3.9	3.9	3.9	3.9	3.8	3.9	3.9
Mineral premix[1]	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Vitamin premix[2]	2.5	2.5	2.5	2.5	2.5	2.5	2.5
DL-Methionine	1.6	1.7	1.7	1.6	1.7	1.7	1.7
Washed builder sand	–	15	15	15	20	20	20
Nutrient composition							
ME (MJ/kg)	12.18	12.18	12.18	12.18	12.18	12.18	12.18
Crude protein	209	209	209	209	209	209	209
Calcium	9.2	9.2	9.2	9.2	9.2	9.2	9.2
Available phosphorus	4.1	4.1	4.1	4.1	4.1	4.1	4.1
Methionine + cysteine	8.8	8.8	8.8	8.8	8.8	8.8	8.8
Lysine	12.5	12.6	12.6	12.6	12.6	12.6	12.6

[1]Mineral premix provided per kilogram of diet: Mn, 65 mg; Zn, 55 mg; Fe, 50 mg; Cu, 8 mg; I (from Ca $(IO_3)_2.H_2O$), 1.8 mg; Se, 0.3 mg. [2]Vitamin premix provided per kilogram of diet: vitamin A (from vitamin A acetate), 9,800 IU; cholecalciferol, 2,100 IU; vitamin E (from DL-α-tocopheryl acetate), 22 IU; riboflavin, 4.4 mg; niacin (as nicotin amide), 40 mg; pantothenic acid (as calcium pantothenate), 35 mg; menadione, 1.5 mg; folic acid, 0.8 mg; thiamine, 3 mg; pyridoxine, 10 mg; biotin, 1 mg; choline (as choline chloride), 560 mg; ethoxyquine, 125 mg.

Table 2. Ingredients and nutrient composition of experimental diets during growing period (21–42 d)

Ingredients (g/kg unless stated otherwise)	Control	20 g/kg soybean oil	20 g/kg yellow grease	20 g/kg poultry fat	40 g/kg soybean oil	40 g/kg yellow grease	40 g/kg poultry fat
Corn, yellow	674.7	604.2	604.2	615.3	529.3	535.7	551.5
Soybean meal	254	254.6	254.6	257.3	248.8	250.4	254.2
Fish meal	40	40	40	40	40	40	40
Wheat bran	5	40.2	40.2	26.3	96.2	88.1	68.4
Fat supplement	–	20	20	20	40	40	40
Limestone	11.8	11.8	11.8	11.8	11.8	11.8	11.7
Dicalcium phosphate	5.6	5.5	5.5	5.6	5.3	5.4	5.5
Common salt	3	3	3	3	3	3	3
Mineral premix[1]	2.5	2.5	2.5	2.5	2.5	2.5	2.5
Vitamin premix[2]	2.5	2.5	2.5	2.5	2.5	2.5	2.5
DL-Methionine	0.8	0.7	0.7	0.7	0.7	0.7	0.7
Washed builder sand	–	15	15	15	20	20	20
Nutrient composition							
ME (MJ/kg)	12.43	12.43	12.43	12.43	12.43	12.43	12.43
Crude protein	185.6	185.6	185.6	185.6	185.6	185.6	185.6
Calcium	8.6	8.6	8.6	8.6	8.6	8.6	8.6
Available phosphorus	3.3	3.3	3.3	3.3	3.3	3.3	3.3
Methionine + cysteine	7.4	7.4	7.4	7.2	7.2	7.2	7.2
Lysine	10.8	10.8	10.8	10.8	10.9	10.8	10.9

[1]Mineral premix provided per kilogram of diet: Mn, 65 mg; Zn, 55 mg; Fe, 50 mg; Cu, 8 mg; I (from Ca $(IO_3)_2.H_2O$), 1.8 mg; Se, 0.3 mg. [2]Vitamin premix provided per kilogram of diet: vitamin A (from vitamin A acetate), 9800 IU; cholecalciferol, 2100 IU; vitamin E (from DL-α-tocopheryl acetate), 22 IU; riboflavin, 4.4 mg; niacin (as nicotin amide), 40 mg; pantothenic acid (as calcium pantothenate), 35 mg; menadione, 1.5 mg; folic acid, 0.8 mg; thiamine, 3 mg; pyridoxine, 10 mg; biotin, 1 mg; choline (as choline chloride), 560 mg; ethoxyquine, 125 mg.

were analyzed by the General Linear Models procedures of SAS software (SAS Institute 2001) based on a 3×3×2 factorial arrangement of treatments including dietary fat source, fat level, and bird's sex as the main effects and respective two- and three-ways interactions. Least significant difference test was used to compare treatment means at $P<0.05$ statistical level.

RESULTS AND DISCUSSION

The fatty acid profile of the used fat sources revealed marked differences (Table 3). Gas chromatography analysis showed that SO had more PUFA than YG and PF; however, the long chain fatty acids (especially C20: 4 and C23) were higher in PF than other supplemental fat sources.

Although BWG was not affected by the source and level of supplemental fats during the entire trial period, using PF decreased ($P<0.01$) BWG during the starter phase (Table 4). Also, the chick's sex affected BWG, so that males gained greater ($P<0.001$) weights compared to females throughout the trial period.

Our data reinforced the results of some previous studies which reported that total BWG was not affected by dietary fat source as long as the energy to protein ratio and other nutrients are well-balanced (Sanz *et al*. 1999). Nonetheless, in the present study the birds fed on SO- and YG-containing diets had the higher BWG in starter period than those fed PF. Wongsuthavas *et al*. (2007) reported that unsaturated fatty acid (UFA) profile of the mentioned oils can affect

Table 3. Fatty acid composition of supplemental fat sources (mg/g)

Fatty acids	Soybean oil	Poultry fat	Yellow grease
C 16: 0	105.9	227.8	109.5
C 16: 1	nd[1]	59.9	nd
C 17: 0	nd	nd	nd
C 17: 1	nd	nd	nd
C 18: 0	43.0	49.9	34.3
C 18: 1	209.4	349.6	183.1
C 18: 2	497.8	145.1	336.6
C 18: 3	63.1	6.8	35.4
C 20: 0	3.4	nd	nd
C 20: 4 (n-6)	1.8	2.5	nd
C 22	3.3	nd	nd
C 20: 5 (n-3)	nd	nd	nd
C 23	2.4	4.2	nd
C 22: 6 (n-3)	nd	nd	nd

[1]nd: not detected.

digestibility and micelle formation; consequently, diets supplemented with SO had the better efficiency of ingested metabolizable energy (ME) for weight gain than diets containing higher levels of saturated fatty acids (SFA), as described by Sanz *et al*. (2000). Similarly, Jahanian and Rasouli (2014) reported that laying hens on SO-supplemented diets had better egg production percentage than those on palm fatty acid powder (enriched of SFA). It was demonstrated that younger birds do not have complete

Table 4. Effects of different sources and levels of dietary fat and broiler sex on body weight gain (g/d per bird)

Fat sources	Levels (g/kg)	Sex	Starter (2–3 wk of age)	Grower (4–6 wk of age)	Overall (2–6 wk of age)
Control	–	Male	38.78	68.81	57.00
		Female	34.77	65.85	53.32
Soybean oil	20	Male	37.03	69.40	56.43
		Female	34.14	59.31	49.23
	40	Male	36.33	70.88	57.03
		Female	32.35	62.34	50.60
Yellow grease	20	Male	36.75	72.15	57.92
		Female	33.24	62.89	50.62
	40	Male	36.60	67.99	56.06
		Female	33.42	60.26	49.11
Poultry fat	20	Male	30.95	69.80	54.15
		Female	31.21	61.60	49.51
	40	Male	34.92	69.47	55.65
		Female	32.73	63.44	50.84
Fat source					
Soybean oil			35.14[a]	65.49	53.72
Yellow grease			35.01[a]	66.53	53.93
Poultry fat			32.29[b]	65.97	52.54
Fat level (g/kg)					
0			36.78	67.33	55.18
20			33.89	66.01	53.08
40			34.46	66.02	53.64
Sex					
Male			35.95[a]	69.85[a]	56.42[a]
Female			33.15[b]	62.43[b]	50.58[b]
Probability					
Fat source			***	ns	ns
Fat level			ns	ns	ns
Sex			***	***	***
Fat source × Fat level			***	*	*
Fat source × Sex			**	ns	ns
Fat level × Sex			ns	ns	ns
Fat source × Fat level × Sex			ns	ns	ns
SEM			1.31	3.79	1.65

[ab]Means with different superscripts within the column of each classification (fat source, fat level, and sex) are significantly different (P<0.05). SEM, standard error of means; [*]P<0.05; [**]P<0.01; [***]P<0.001; ns, not significant.

Table 5. Effects of different sources and levels of dietary fat and broiler sex on feed intake (g/d per bird)

Fat sources	Levels (g/kg)	Sex	Starter (2–3 wk of age)	Grower (4–6 wk of age)	Overall (2–6 wk of age)
Control	–	Male	62.54	193.73	141.51
		Female	56.89	181.33	131.55
Soybean oil	20	Male	60.83	188.34	137.12
		Female	55.61	177.51	128.59
	40	Male	58.66	187.88	136.29
		Female	54.63	181.59	130.66
Yellow grease	20	Male	60.82	190.88	138.55
		Female	58.47	175.96	130.78
	40	Male	61.50	192.35	139.66
		Female	55.11	174.85	127.65
Poultry fat	20	Male	54.42	185.43	133.21
		Female	52.13	176.16	126.39
	40	Male	60.58	186.63	135.91
		Female	58.56	182.80	133.29
Fat source					
Soybean oil			57.35[b]	182.98	132.46
Yellow grease			59.01[a]	184.30	135.15
Poultry fat			56.28[b]	183.05	132.31
Fat level (g/kg)					
0			59.71[a]	186.64	135.82
20			56.98[b]	182.40	132.64
40			58.13[a]	184.72	134.09
Sex					
Male			59.95[a]	188.78[a]	136.86[a]
Female			55.71[b]	178.19[b]	129.57[b]
Probability					
Fat source			***	ns	ns
Fat level			*	ns	ns
Sex			***	***	***
Fat source × Fat level			***	ns	ns
Fat source × Sex			ns	*	*
Fat level × Sex			ns	ns	ns
Fat source × Fat level × Sex			ns	ns	ns
SEM			1.90	5.62	4.15

[ab]Means with different superscripts within the column of each classification (fat source, fat level, and sex) are significantly different (P<0.05). SEM, standard error of means; [*]P<0.05; [***]P<0.001; ns, not significant.

physiological ability to digest and absorb fat, and they are very sensitive to the profile of fatty acids (Noy and Sklan 1995). This factor might be a main reason why BWG was affected during the starter period but not at the remaining trial period.

Feed intake was influenced by bird's sex (P<0.001), and male chicks consumed more feed than females (Table 5). Birds fed on diets supplemented with YG had greater (P<0.001) FI during the starter period. These findings are consistent with those of Noy and Sklan (1979), who reported that a lower ME was provided by an UFA-enriched diet than by a diet containing SFA; consequently birds on diets containing UFA had higher FI. Shimomura *et al*. (1990) reported a higher post-prandial muscle lipoprotein lipase activity in rats fed on safflower oil than those fed tallow, suggesting a higher rate of muscle β-oxidation for PUFA than for SFA. In contrast to our results, Azman *et al*. (2004) reported that in isocaloric and isonitrogenous diets, FI of birds fed on diets with high levels of SFA was higher than birds on UFA-enriched diets.

Supplementation of experimental diets with different fat sources and levels decreased (P<0.05) FI compared with control birds. The absence of supplemental fat in control group and subsequent lower feed retention time in digestive tract might have affected FI of control chicks in the present study.

Feed conversion ratio was not affected by supplemental fat source during the entire trial period (Table 6). However, the best (P<0.001) FCR values during the starter period were obtained with SO-containing diets. Also, inclusion of

Table 6. Effects of different sources and levels of dietary fat and broiler sex on feed conversion ratio (g feed/g gain)

Fat sources	Levels (g/kg)	Sex	Starter (2–3 wk of age)	Grower (4–6 wk of age)	Overall (2–6 wk of age)
Control	–	Male	1.59	2.72	2.26
		Female	1.61	2.79	2.32
Soybean oil	20	Male	1.63	2.72	2.29
		Female	1.60	2.75	2.30
	40	Male	1.62	2.65	2.23
		Female	1.65	2.97	2.44
Yellow grease	20	Male	1.65	2.65	2.25
		Female	1.67	2.74	2.29
	40	Male	1.61	2.68	2.28
		Female	1.68	2.74	2.31
Poultry fat	20	Male	1.75	2.61	2.26
		Female	1.91	2.67	2.27
	40	Male	1.70	2.76	2.35
		Female	1.73	2.84	2.40
Fat source					
Soybean oil			1.63[c]	2.80	2.33
Yellow grease			1.65[b]	2.71	2.28
Poultry fat			1.73[a]	2.72	2.32
Fat level (g/kg)					
0			1.59	2.76[a]	2.29[ab]
20			1.66	2.69[b]	2.28[b]
40			1.67	2.78[a]	2.34[a]
Sex					
Male			1.66	2.68[b]	2.28[b]
Female			1.65	2.79[a]	2.34[a]
Probability					
Fat source			***	ns	ns
Fat level			ns	*	**
Sex			ns	*	***
Fat source × Fat level			ns	*	ns
Fat source × Sex			ns	ns	ns
Fat level × Sex		**	*	ns	
Fat source × Fat level × Sex			ns	*	ns
SEM			0.001	0.005	0.002

[abc]Means with different superscripts within the column of each classification (fat source, fat level, and sex) are significantly different (P<0.05). SEM, standard error of means; [*]P<0.05; [**]P<0.01; [***]P<0.001; ns , not significant.

20 g/kg fat into the diets improved FCR values during both grower (P<0.05) and entire (P<0.01) trial period. The bird's sex affected (P<0.001) feed conversion efficiency, so that male chicks revealed better FCR values than females.

Unsaturated fatty acid profile of diets containing SO might be the reasonable explanation for the improvement of FCR values during the starter period, because the digestibility of fats increases with the degree of unsaturation (Sanz *et al*. 2000, Wongsuthavas *et al*. 2007). This improvement is also related to the higher BWG (than birds on PF) and concomitant lower FI (compared with YG-supplemented birds) in SO-supplemented chicks. As noted

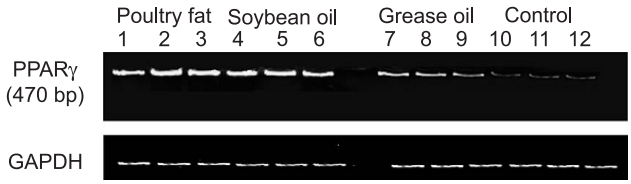

Fig. 1. Effect of different dietary fat sources on the expression of peroxisome-proliferator-activated receptor-γ (PPARγ) gene.

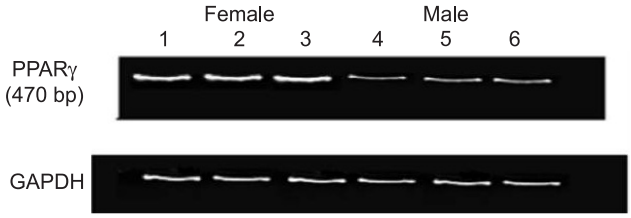

Fig. 2. Effect of broiler's sex on the expression of peroxisome-proliferator-activated receptor-γ (PPARγ) gene.

in Table 6, the best FCR values in the grower and entire period were obtained with diets supplemented with 20 g/kg fat. Increase in wheat bran and washed sand in diets containing 40 g/kg supplemental fat might be a reason why FCR values were higher in the birds on diets containing 40 g/kg fat.

The expression of PPARγ gene was affected by dietary fat source (Fig.1). The highest expressions were detected in fat tissue of birds fed on diets containing SO and PF. In addition, the bird's sex affected the expression of PPARγ gene, so that expression was higher in females than that of male chicks (Fig. 2).

It is hypothesized that dietary fatty acid profile may, to a certain extent, play a significant role in the induction of adipocyte differentiation in chicks. There are several studies, which demonstrated that dietary fatty acid profile affect the expression of PPARγ gene (Itoh *et al*. 1999, Chambrier *et al*. 2002, Sato *et al*. 2004). Consistent with the previous report (Sato *et al*. 2004), the expressions of PPARγ in the adipose tissue of chicks on SO- and PF-containing diets were higher than those on YG and/or control diets. The concentrations of linoleic acid and long chain fatty acids were greater in SO and PF, respectively, which could affect PPARγ expression in abdominal fat tissue (Table 3).

Chambrier *et al*. (2002) reported that linolenic acid, docosahexaenoic acid, and ω-6 PUFA had no considerable effect on human adipocytes, but eicosapentaenoic acid increased PPARγ mRNA levels. Also, Sato *et al*. (2004) showed that PPARγ gene expression was higher in chicks fed linoleic acid-enriched diets than those fed oleic acid, and the fat deposition was correlated with the level of PPARγ expression. Generally, diets containing YG have possibly the higher levels of oxidized fatty acids and free radicals, and birds fed on YG-diets are exposed to oxidative stress. Oxidized fatty acids of YG could affect the PPARγ gene expression, because these fatty acids induce oxidative stress by producing some inflammatory elements such as tumor necrosis factor-alpha, which could suppress the

expression of PPARγ (Austin *et al*. 1998, Itoh *et al*. 1999). Our findings suggested that like in mammals, administration of exogenous fatty acids (especially PUFA) can affect deposition of abdominal fat by alteration of adipose tissue PPARγ gene expression in chickens.

Additionally, it was demonstrated that the sex hormones can influence energy metabolism and adipose tissue turnover (Dieudonne *et al*. 2000). Some research groups (Ma *et al*. 1998, Dieudonne *et al*. 2000) concluded that sex hormones could affect adipose tissue PPARγ gene expression and change energy metabolism. Ma *et al*. (1998) reported that estrogens induce formation and expression of PPARγ. Also, it was indicated that androgens inhibit differentiation and proliferation of pre-adipocytes (Garcia *et al*. 1999), and castration resulted in an increase in proliferation of adipocytes, while administration of androgen had an opposite effect (McTernan *et al*. 2000). Our findings reinforced the previous reports that the male sexual hormones reduce deposition of fat and this effect might be mediated through PPARγ gene expression.

Our findings indicated that different dietary fat sources had no effect on growth performance of broiler chicks; however, supplementation of 20 g/kg fat into the broiler diets improved growth performance and feed conversion efficiency. Diets containing higher levels of PUFA and long chain fatty acids (SO and PF) induced PPARγ gene expression in abdominal fat tissue. The PPARγ expression was higher in females than male chicks, indicating sex hormones might influence energy metabolism and adipose tissue accretion, in part, by regulation of PPARγ gene expression.

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