Effects of a high-fat diet on the growth performance, lipid metabolism, and the fatty acids composition of liver and skin fat in Pekin ducks aged from 10 to 40 days

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ABSTRACT This study aimed to investigate the effect of a high-fat diet on the growth performance, serum, liver, and skin lipid metabolism as well as the fatty acids composition of liver and skin fat in Pekin ducks from 10 to 40 d of age based on a pair-fed group. Two hundred forty healthy male ducks (10 d old, 470.53 ± 0.57 g) were randomly divided into 3 groups (8 replicates per cage of 10 ducks): a normal diet (**ND**, 3% fat), a high-fat diet (**HFD**, 9% fat), and a pair-fed diet (**PFD**, given the ND in an amount equal to that consumed of the HFD to eliminate the effects of feed intake). The results were as follows: compared to ND feeding, HFD feeding significantly decreased ($P < 0.05$) the feed intake and feed:gain ratio (F:G), along with serum triglyceride and nonesterified fatty acid contents. When compared with the ND and PFD, the HFD significantly decreased ($P < 0.05$) the liver weight and inhibited hepatic de novo lipogenesis (glucose-6-phosphate dehydrogenase and malate dehydrogenase activities), β-oxidation (carnitine palmitoyltransferase-1 content), and decreased saturated fatty acids and monounsaturated fatty acids deposition. Moreover, the HFD significantly increased ($P < 0.05$) the total fat content, lipid droplet area, and polyunsaturated fatty acids (**PUFA**s) content in the liver, as well as the abdominal fat weight, subcutaneous fat weight, the total fat and PUFAs content in skin fat. These results suggested that the HFD improved feed efficiency, which was related to HFD feeding inhibiting hepatic de novo lipogenesis and β-oxidation and promoting the deposition of fat in skin as well as altering the fatty acids composition of the liver and skin fat in Pekin ducks.

Key words: fatty acid composition, high-fat diet, lipid metabolism, Pekin duck

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INTRODUCTION

In the commercial production of poultry, to achieve a lower feed-to-gain ratio (**F:G**) and improve the utilization efficiency of unconventional feeds, high levels of fat are often added to the completed feed (Leeson and Summers, 2008; Jalaludeen et al., 2022). For broiler chickens, high levels of fat in diets cause excessive fat deposition, especially in the liver and abdominal cavity, which reduces the survival rate and carcass yield (Brue and Latshaw, 1985). Compared with broiler chickens, waterfowl have unique lipid metabolism processes and a high tolerance toward the dietary fat content (Lu et al., 2015; Wei et al., 2021). The main site of de novo lipogenesis (**DNL**) in waterfowl is the liver, and fat from DNL is often preferentially stored in the liver, which easily leads to fatty liver when waterfowl are fed a high-carbohydrate diet (Mourot et al., 2000; Lu et al., 2015). Moreover, unlike broiler chickens, which mainly deposit abdominal fat, ducks mainly deposit fat under the skin (Liu et al., 2019). Ducks have higher body fat levels than other bird species (Baéza et al., 2002). However, there is little information about the effect of a high-fat diet (**HFD**) on lipid metabolism in the serum, liver, and skin fat of Pekin ducks.

More importantly, when studying the effects of a HFD on lipid metabolism in poultry, it is often uncertain whether the metabolizable energy concentration of a HFD affects the feed intake (**FI**) or the fats themselves. Ducks can regulate the amount of energy ingestion via FI. The feed intake and F:G in meat ducks increase when receiving low-energy diets compared to high-energy diets

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(Zeng et al., 2015; Baéza. 2016). Bai et al. (2019) further mirrored the instinct of meat ducks to regulate FI based on diet energy concentration. Thus, eliminate the influence of FI on lipid metabolism is particularly important when studying the role of dietary fat content. Chartrin et al. (2006) found that overfeeding stimulated the hepatic activity of the main enzymes involved in lipogenesis from glucose in ducks. Cai et al. (2021) suggested that feed restriction may promote the utilization of fatty acids in active metabolic tissues through adiponectin to guarantee the energy homeostasis of the body in broiler chickens. Hence, in the current study, we used the pair-fed model to investigate the effect of a HFD on the growth performance, body fat deposition, and lipid metabolism in serum, liver, and skin fat, as well as the fatty acids (**FA**) composition of liver and skin fat in Pekin ducks.

MATERIALS AND METHODS

The Institutional Animal Care and Use Committee (**IACUC**) of Sichuan Agricultural University approved all procedures used in the study.

Birds, Experimental Design, Diet, and Management

A total of 240 one-day-old Pekin ducklings were fed a standard starter diet with 1.23% fat added from 1 to 9 d of age. On D 10, all ducks were randomly allocated by body weight (**BW**, 470.53 ± 0.57 g) into 3 groups, each consisting of 8 replicate cages with 10 ducks per cage. The diet treatments were a normal diet (**ND**, 3% fat), a high-fat diet (**HFD**, 9% fat), and a pair-fed diet (**PFD**, given the ND in an amount equal to that consumed of the HFD to eliminate the effects of feed intake). These diets were formulated to meet or exceed the nutrient requirements of meat ducks according to the NRC (1994) and China Agricultural Industry Standards (2012). The moisture, crude protein, crude fat, crude fiber, and crude ash of the experimental diets were analyzed as described by the AOAC (2005) (Table 1). The fatty acid composition of the experimental diets was analyzed as described by Matsumoto et al. (2018) and is also shown in Table 1. Birds were reared in cages (length 100 × width 80 × height 60 cm) in a humidity- and temperature-controlled room and had free access to water and feed (ND and HFD), except for the PFD group. The diet was offered in pelleted form and the diameter of the feed particles was 3.8 mm.

Table 1. Composition and nutrient levels of the experimental diet (on an as-fed basis).

Ingredient, %	Normal diet (ND)	High-fat diet (HFD)
Corn	40.69	40.69
Soybean meal	23.00	23.00
Corn starch	6.00	-
Flour	10.00	10.00
Rice bran meal	14.00	14.00
Duck fat	3.00	9.00
Dicalcium phosphate	1.28	1.28
Limestone	0.86	0.86
Sodium chloride	0.35	0.35
DL-Methionine	0.14	0.14
Choline chloride	0.15	0.15
Mineral premix[1]	0.50	0.50
Vitamin premix[2]	0.03	0.03
Nutrient levels[3], %		
Metabolizable energy (ME), MJ/kg	12.13	13.55
Moisture[3]	12.52	12.31
Crude protein[3]	16.48	16.39
Crude fat[3]	4.13	9.73
Crude fiber[3]	5.27	5.59
Crude ash[3]	5.24	5.52
Calcium	0.70	0.70
Nonphytate phosphorus	0.35	0.35
Lysine	0.85	0.85
Methionine	0.40	0.40
Fatty acid profiles (g/100 g of total fatty acid) on an analyzed basis[3]		
Palmitic acid (C16:0)	19.82	21.07
Stearic acid (C18:0)	4.45	5.18
Oleic acid (C18:1 n−9)	38.40	43.76
Linoleic acid (C18:2 n−6)	37.33	29.99
Saturated fatty acid (SFA)	24.27	26.25
Monounsaturated fatty acid (MUFA)	38.40	43.76
Polyunsaturated fatty acid (PUFA)	37.33	29.99
U:S ratio[4]	3.12	2.81

[1]The trace mineral premix provided the following (per kg of diet): Fe ($FeSO_4 \cdot H_2O$), 80 mg; Cu ($CuSO_4 \cdot 5H_2O$), 10 mg; Mn ($MnO_4 \cdot H_2O$), 100 mg; Zn ($ZnSO_4 \cdot H_2O$), 60 mg; I (KI) 0.45 mg; and Se (Na_2SeO_3), 0.30 mg.

[2]The vitamin premix provided the following (per kg of diet): vitamin A 8,000 IU; vitamin D_3, 2,000 IU; vitamin E, 20 IU; vitamin K_3, 1.0 mg; vitamin B_1, 0.6 mg; vitamin B_2, 8.0 mg; vitamin B_6, 3.5 mg; vitamin B_{12} 0.01 mg; niacin, 10.0 mg; niacin, 35.0 mg; folic acid, 0.55 mg; and biotin 0.18 mg.

[3]The moisture, crude protein, crude fat, crude fiber, and crude ash contents and the fatty acid profiles were measured values, while the others were calculated values.

[4]Unsaturated fatty acid:Saturated fatty acid = UFA:SFA.

Data and Sample Collection

At 40 d of age, after 12 h of feed withdrawal, ducks were weighed, and feed consumption was obtained by cages. Body weight, body weight gain (**BWG**), FI, F:G, and total crude fat intake were calculated. Then, one bird per cage with the weight closest to the cage was selected, 5 mL of blood was obtained via the jugular vein and centrifuged at 3,000 × g for 15 min at 4°C to collect serum and the bird was euthanized by cervical dislocation. The total crude fat intake was the arithmetic product of the feed intake and the measured value of crude fat in the diets. The liver, abdominal fat, subcutaneous fat, uropygial gland, and gallbladder weights were recorded and expressed in g/100 g BW. After that, the serum, liver, and subcutaneous fat were excised and stored at −20°C until further analysis.

Serum Biochemical Analyses

Serum glucose (**Glu**), triglyceride (**TG**), total cholesterol (**TC**), total bile acids (**TBA**), high-density lipoprotein-C (**HDL-C**), low-density lipoprotein-C (**LDL-C**), and very-low-density lipoprotein-C (**VLDL-C**) were analyzed using an automatic biochemical analyzer

(HATICHI7180, Japan). Serum biochemical parameters such as nonesterified fatty acid (**NEFA**) and leptin (**LEP**) were determined spectrophotometrically with commercial ELISA kits (Nanjing Jiancheng Bioengineering Institute, Nanjing, China).

Liver and Skin fat Chemical Parameters and Fatty Acid Composition

The moisture contents of the liver and skin fat were determined by a freeze dryer (FDU-2110; Tokyo Rikakikai CO., Ltd, Tokyo, Japan). The fat contents of the liver and skin fat were measured by the Soxhlet extraction method described by the AOAC (2005). The compositions of fatty acids in the feed, liver, and skin fat (as dry matter basis) were analyzed as described by Matsumoto et al. (2018). In brief, 3-mL samples were prepared and homogenized in an extraction buffer (chloroform: methanol:H_2O=8:4:3; v/v). The chloroform layer was collected and dried after centrifugation (1,500 × g for 15 min at 4°C). The samples were dried using a vacuum drying oven (DZG-6020; Shanghai Sumsung Laboratory Instrument Co., Ltd, Shanghai, China). The dry residue was resolved in 2.0 mL of 0.5 mol/L KOH-methanol solution and boiled (10 min at 60°C). After cooling to room temperature, the solution was mixed with 2 mL of 14% boron trifluoride-methanol and boiled (2 min at 80°C). After cooling, the solution was mixed with 3 mL of 0.9% NaCl and 1 mL of hexanes and centrifuged (2,000 × g for 5 min at 4°C). The hexane layer was collected for analysis. The fatty acid compositions were determined using a gas chromatography analyzer (GS-2010 Plus, Shimadzu Co., Ltd, Kyoto, Japan).

Liver Histology and Oil Red O Staining

The fixed hepatic tissues were embedded in paraffin and cut into 2-μm-thick sections by staining with hematoxylin and eosin (**HE**). Histology and three HE (HE, 100 × and 400 ×) images of each hepatic slice were evaluated in a blinded manner to treatment by 3 independent pathologists based on the procedures described by Brunt et al. (1999) and Zeng et al. (2014). Oil red O staining was conducted as described by Mehlem et al. (2013). In brief, the sections (4.5-μm thick) were stained with oil red O. Images of HE- or oil red O-stained sections were captured with a BA210Digital microscope (Motic China Group Co., Ltd, Xiamen, China), and the lipid droplet area of the liver was measured by Image-Pro Plus 6.0 (Media Cybernetics, Inc, MD). Three different visual field images made for each hepatic slice were captured for each replicate duck.

Liver and Skin Fat Lipid Metabolism Analyses

Liver lipid metabolism parameters such as TG, TC, glucose-6-phosphate dehydrogenase (**G-6-PD**), malate dehydrogenase (**MDH**), fatty acid synthase (**FAS**), acetyl-CoA carboxylase (**ACC**), carnitine palmitoyltransferase 1 (**CPT-1**), hormone sensitive lipase (**HSL**) and lipoprotein lipase (**LPL**), as well as the skin fat LPL activity and skin FAS, HSL, and ACC contents, were determined spectrophotometrically with commercial ELISA kits (Nanjing Jiancheng Bioengineering Institute, Nanjing, China).

Statistical Analysis

Data were analyzed using the SAS software package (version 9.4, SAS Institute Inc., Cary, NC). Student's t test was applied when the dietary treatment showed a significant difference at $P < 0.05$ as evaluated using Student's t test. Data are shown as the means and standard errors of the means (**SEM**s). Graphs were generated using GraphPad Prism 8.3 software.

RESULTS

Growth Performance, Total Crude Fat Intake, and Caloric Conversion

As shown in Table 2, no significant effect ($P > 0.05$) on the BWG of Pekin ducks was observed among the 3 dietary treatments. Compared to the ND, the HFD significantly decreased ($P < 0.05$) the FI and F:G and markedly increased ($P < 0.05$) the total crude fat intake and caloric conversion of ducks from 10 to 40 d of age. Moreover, compared to the PFD, the HFD significantly increased ($P < 0.05$) the total crude fat intake and caloric conversion of birds from 10 to 40 d of age.

Table 2. Effect of a high-fat diet on the growth performance and total crude fat intake of ducks from 10 to 40 d of age.[1]

Item	ND	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Body weight gain (BWG), g/bird	2702	2665	2594	21.35	0.412	0.204
Feed intake (FI), g/bird	5802	5451	5451	44.26	<0.001	>0.999
Feed-to-gain ratio (F/G), g/g	2.15	2.05	2.10	0.02	0.003	0.104
Total crude fat intake, g/bird[3]	239.6	530.4	225.2	29.38	<0.001	<0.001
Caloric conversion/(Mcal/kg), g/bird[4]	6.23	6.63	6.09	0.05	<0.001	<0.001

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).
[3]Arithmetic product of the feed intake (g/bird) and the measured value of crude fat in the diet (%).
[4]The caloric conversion was calculated by the formula:caloric conversion (Mcal ME/kg weight gain) = diet ME density (Mcal/kg) × feed intake (kg) ÷ weight gain (kg); the calculation formula refers to Zeng et al. (2015).

Table 3. Effect of a high-fat diet on the body fat deposition in ducks at 40 d of age.[1]

Item	NC	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Abdominal fat weight, g	28.93	34.94	24.41	1.31	0.039	<0.001
Abdominal fat yield, %	0.92	0.94	0.91	0.01	0.043	0.006
Skin fat weight, g	452.6	549.0	441.0	15.13	0.006	0.005
Skin fat yield, %	20.83	24.69	20.64	0.58	0.007	0.003
Uropygial gland weight, g	5.05	5.97	5.04	0.27	0.184	0.817
Uropygial gland yield, %	0.16	0.19	0.16	0.01	0.207	0.280
Gallbladder weight, g	3.39	2.88	3.29	0.15	0.191	0.164
Gallbladder yield, %	0.11	0.09	0.11	0.01	0.184	0.076

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).

The Body Fat Deposition

As shown in Table 3, the abdominal fat weight, abdominal fat yield, skin fat weight, and skin fat yield were increased ($P < 0.05$) in ducks fed the HFD compared to Pekin ducks fed the ND or PFD.

Serum Lipid Metabolism

As shown in Table 4, the lipid metabolism-related biomarkers in the serum, such as the TG and NEFA contents were significantly decreased ($P < 0.05$) in birds fed a HFD compared to those fed a ND. Moreover, the serum TBA content was increased ($P < 0.05$) and the HDL-C content was decreased ($P < 0.05$) in ducks fed a HFD compared to meat ducks fed a PFD.

Liver Chemical Parameters, Lipid Metabolism, and Fatty Acid Composition

Figure 1 depicts that liver weight and liver yield were decreased ($P < 0.05$) and the liver total fat content was increased ($P < 0.05$) in ducks fed a HFD compared to ducks fed a ND. However, the weight, yield, moisture and total fat content were unaffected in the livers of meat ducks fed a HFD compared to ducks fed a PFD.

As shown in Table 5, the lipid metabolism-related biomarkers in the liver, such as G-6-PD and MDH activity and the CPT-1 content, were reduced ($P < 0.05$) in ducks fed a HFD compared to ducks fed a ND or PFD. In addition, liver LPL activity was lower ($P < 0.05$) in birds fed a HFD than in those fed the PFD.

As shown in Table 6, the contents of palmitic acid, linoleic acid, polyunsaturated fatty acids (**PUFAs**) and the U:S ratio were increased ($P < 0.05$), and the contents of stearic acid, oleic acid, saturated fatty acids (**SFAs**) and monounsaturated fatty acids (**MUFAs**) were decreased ($P < 0.05$) in the livers of ducks fed a HFD compared to the ND and PFD groups.

Liver Histopathological Score and Lipid Droplet Area

Figure 2 shows that no treatment effect ($P > 0.05$) on the histopathological score of the liver in ducks fed a HFD was observed compared with those fed a ND and PFD. However, the liver lipid droplet area was higher ($P < 0.05$) in ducks fed the HFD than in those fed the ND, but it was similar between the PFD and HFD groups.

Skin Fat Lipid Metabolism and Fatty Acid Composition

As shown in Table 7, the content of skin fat was higher ($P < 0.05$) in meat ducks fed a HFD than in those fed a ND or PFD. The lipid metabolism-related biomarkers in the skin, such as the LPL activity were lower

Table 4. Effect of a high-fat diet on lipid metabolism in the serum of ducks at 40 d of age.[1]

Item	ND	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Glucose (Glu), mmol/L	8.26	8.28	8.38	0.25	0.482	0.417
Triglyceride (TG), mmol/L	0.85	0.66	.65	0.03	0.002	0.153
Total cholesterol (TC), mmol/L	3.70	3.77	3.98	0.07	0.924	0.066
Total bile acids (TBA), μmol/L	17.81	22.09	10.03	1.50	0.141	0.008
High-density lipoprotein-C (HDL-C), mmol/L	2.24	2.27	2.52	0.05	0.779	0.027
Low-density lipoprotein-C (LDL-C), mmol/L	1.15	1.21	1.18	0.03	0.572	0.563
Very-low-density lipoprotein-C (VLDL-C), mmol/L	0.35	0.35	0.29	0.02	0.903	0.202
Nonesterified fatty acid (NEFA), mmol/L	1.09	0.84	0.77	0.05	0.007	0.790
Leptin (LEP), ng/ml	1.92	1.69	1.96	0.07	0.117	0.150

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).

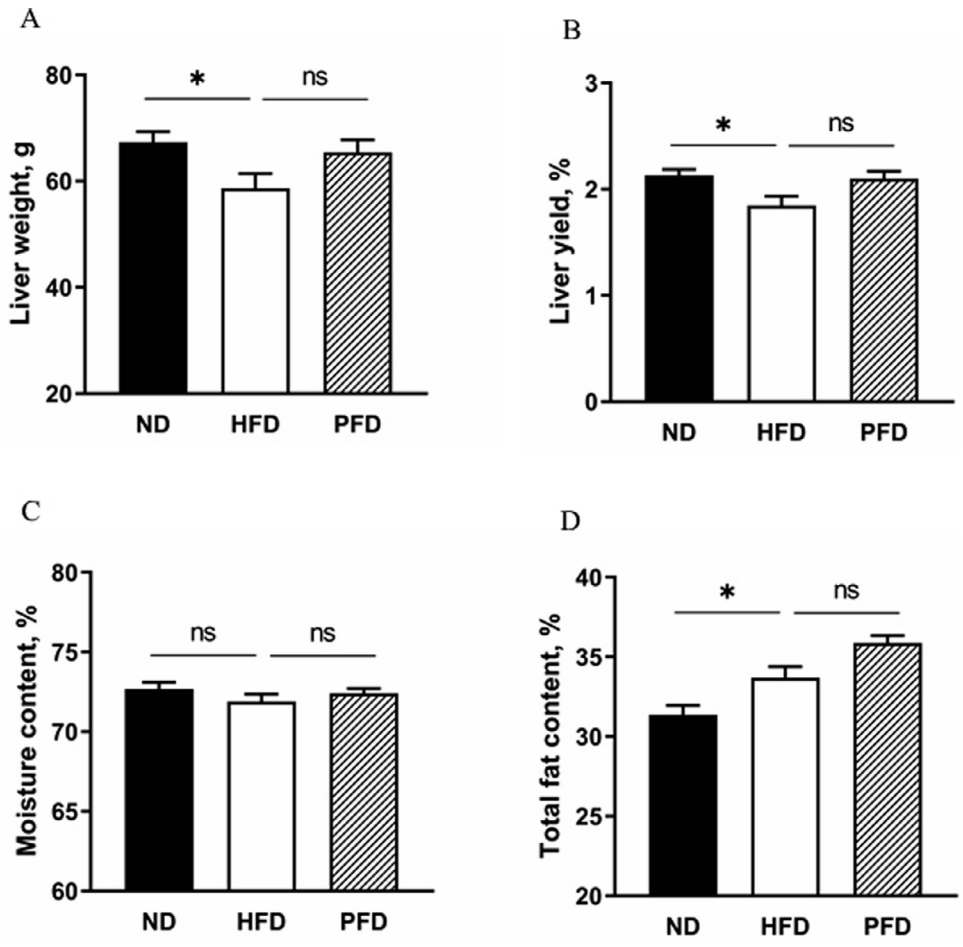

Figure 1. Effect of a high-fat diet on the weight, moisture content and total fat content of the liver in ducks at 40 days of age. (A) Liver weight; (B) Liver yield; (C) Moisture content; (D) Total fat content. PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD). Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10). Error bars represent SEM. $^*P < 0.05$; ns, not significant.

($P < 0.05$) in ducks fed a HFD than in meat ducks fed other treatments.

As shown in Table 8, the contents of oleic acid and MUFAs were decreased ($P < 0.05$) and linoleic acid and PUFAs were increased ($P < 0.05$) in the skin of ducks fed a HFD compared to other treatments. Moreover, stearic acid and SFA levels were decreased ($P < 0.05$) in the skin of birds fed a HFD compared with those of meat ducks fed the PFD.

DISCUSSION

In the current study, birds fed a HFD had a significantly lower FI and F:G than ducks fed a ND. This was consistent with our previous research (Bai et al., 2019) in which a high dietary energy content, that is, when the fat content in the diet increased from 0.9% to 7.63%, did not affected the BW and BWG, but the FI was decreased in ducks from 15 to 21 d of age. Ducks meet

Table 5. Effect of a high-fat diet on the lipid metabolism in the liver of ducks at 40 d of age.[1]

Item	ND	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Glucose-6-phosphate dehydrogenase (G-6-PD), U/g protein	26.99	17.31	32.52	2.39	0.020	<0.001
Malate dehydrogenase (MDH), U/mg protein	12.51	10.18	13.05	0.44	0.007	0.004
Triglyceride (TG), mmol/g protein	0.31	0.29	0.29	0.01	0.432	0.863
Total cholesterol (TC), μmol/g protein	14.71	13.19	13.52	0.54	0.420	0.872
Fatty acid synthase (FAS), ng/mg protein	0.88	0.78	0.88	0.03	0.133	0.173
Acetyl-CoA carboxylase (ACC), ng/mg protein	0.15	0.13	0.14	0.01	0.172	0.870
Carnitine palmitoyltransferase 1 (CPT-1), ng/mg protein	0.74	0.67	0.77	0.02	0.016	0.025
Hormone sensitive lipase (HSL), ng/mg protein	1.41	1.34	1.49	0.03	0.407	0.115
Lipoprotein lipase (LPL), U/mg protein	0.19	0.24	0.37	0.03	0.170	0.022

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).

Table 6. Effect of a high-fat diet on the fatty acid composition in the liver of ducks at 40 d of age (dry matter basis).[1]

Item (g/100 g of total fatty acid)	ND	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Palmitic acid (C16:0)	27.76	28.87	26.79	0.23	<0.001	<0.001
Stearic acid (C18:0)	20.53	18.01	21.04	0.36	<0.001	<0.001
Oleic acid (C18:1 n−9)	38.76	33.31	40.41	0.81	<0.001	<0.001
Linoleic acid (C18:2 n−6)	12.95	19.81	11.76	0.95	<0.001	<0.001
Saturated fatty acid (SFA)	48.29	46.88	47.83	0.17	<0.001	<0.001
Monounsaturated fatty acid (MUFA)	38.76	33.31	40.41	0.81	<0.001	<0.001
Polyunsaturated fatty acid (PUFA)	12.95	19.81	11.76	0.95	<0.001	<0.001
U:S ratio[3]	1.07	1.13	1.09	0.01	<0.001	<0.001

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).
[3]Unsaturated fatty acid:Saturated fatty acid = UFA:SFA.

their energy requirement for growth at a fairly wide range of diet energy levels (Leeson and Summers. 2008). Many previous studies have reported improved feed efficiency when birds are fed a high-fat diet (Coon et al., 1981; Sanz et al., 2000; Fan et al., 2008; Zeng et al., 2015). Furthermore, after eliminating the effect of FI (PFD vs. HFD), we found that the dietary fat content did affect the total crude fat intake and caloric conversion, along with causing a higher BWG (+71 g) and a lower F:G (−0.05) of meat ducks. One reason was that when ducks were fed a HFD, a component of the lower heat increase and the "extra caloric" influence of fat was observed (Brue and Latshaw, 1985). The other reason is that a high dietary fat content also affects the lipid metabolism in the whole body of meat ducks.

Indeed, in the current study, serum TG and NEFA levels were significantly decreased in birds fed a HFD compared to a ND, which indicated a higher rate of dietary lipid clearance from the bloodstream to tissues. This result was also in agreement with the results of

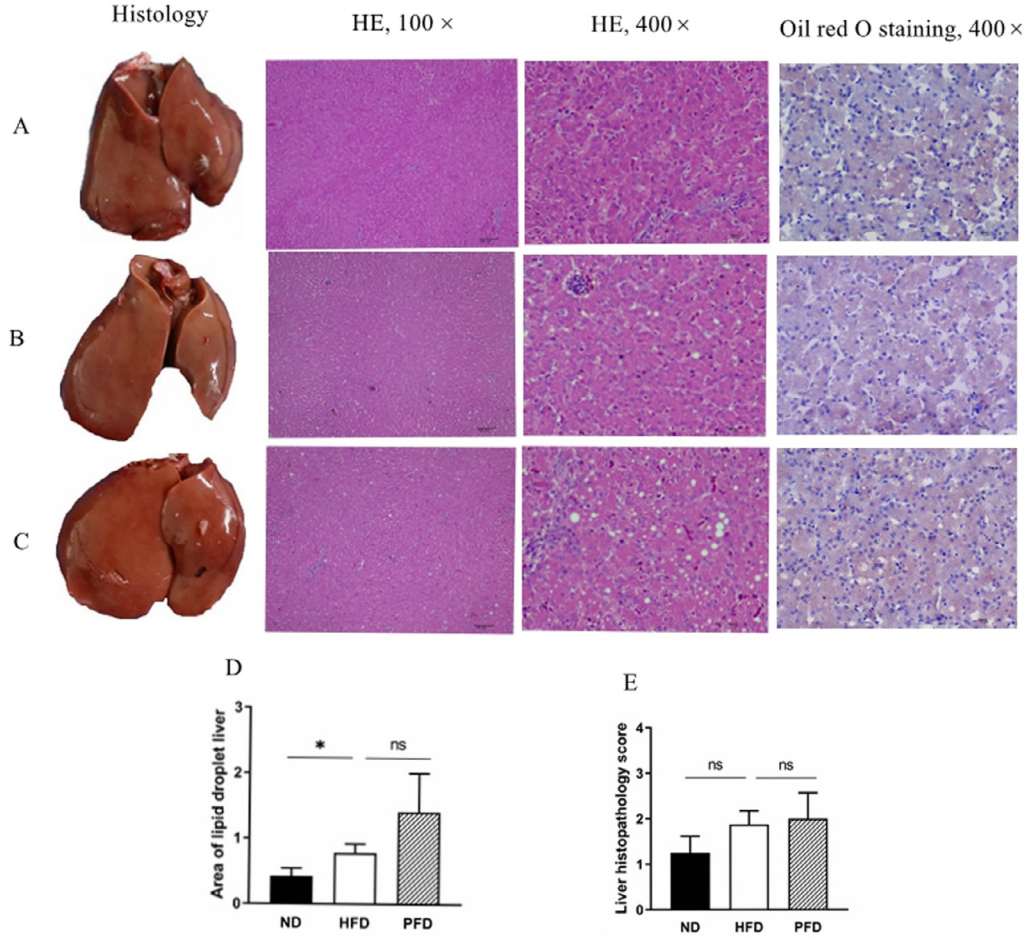

Figure 2. Effect of a high-fat diet on the histopathological changes and lipid deposition in the liver of ducks at 40 days of age. (A) Normal diet, ND; (B) High-fat diet, HFD; (C) Pair-fed diet, PFD; (D) Liver HE histopathological score, grading the histological lesions with reference to Brunt et al. (1999) and Zeng et al. (2014); (E) Analysis results of the lipid droplet area stained with oil red O staining. Scale bar = 100 μm (100 ×) and 10 μm (400 ×). PFD, pair-fed diet in which ducks on the ND were pair-fed the same feed intake as ducks on the HFD. Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10). Error bars represent SEM. $^*P < 0.05$; ns, not significant.

Table 7. Effect of a high-fat diet on the parameters and lipid metabolism of skin fat in ducks at 40 d of age.[1]

Item	ND	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Moisture content, %	4.83	4.74	5.97	0.47	0.949	0.145
Total fat content, %	85.52	89.69	89.61	0.65	0.004	0.957
Lipoprotein lipase (LPL), U/mg protein	12.14	8.42	11.50	0.64	0.013	0.026
Fatty acid synthase (FAS), ng/mg protein	59.00	52.86	56.24	1.78	0.116	0.494
Hormone sensitive lipase (HSL), ng/mg protein	47.01	46.61	51.97	2.74	0.955	0.458
Acetyl-CoA carboxylase (ACC), ng/mg protein	6.28	6.23	6.66	0.21	0.938	0.472

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).

Table 8. Effect of a high-fat diet on the fatty acid composition in the skin fat of ducks at 40 d of age (dry matter basis).[1]

Item, g/100 g of total fatty acid	ND	HFD	PFD[2]	SEM	*P* value	
					HFD vs. ND	HFD vs. PFD
Palmitic acid (C16:0)	25.05	24.55	24.92	0.17	0.244	0.282
Stearic acid (C18:0)	6.60	6.17	7.05	0.13	0.134	0.003
Oleic acid (C18:1 n−9)	51.78	48.24	51.09	0.39	<0.001	<0.001
Linoleic acid (C18:2 n−6)	16.57	21.04	16.94	0.44	<0.001	<0.001
Saturated fatty acid (SFA)	31.65	30.72	31.97	0.24	0.147	0.015
Monounsaturated fatty acid (MUFA)	51.78	48.24	51.09	0.39	<0.001	<0.001
Polyunsaturated fatty acid (PUFA)	16.57	21.04	16.94	0.44	<0.001	<0.001
U:S ratio[3]	2.16	2.26	2.13	0.02	0.139	0.013

[1]Values are the means of 8 cages per treatment of 10 ducks per pen (n = 10).
[2]PFD, pair-fed diet in which ducks on the normal diet (ND) were pair-fed the same feed intake as ducks on the high-fat diet (HFD).
[3]Unsaturated fatty acid:Saturated fatty acid = UFA:SFA.

Hakim et al. (2021), who demonstrated that a HFD may reduce TG concentrations in the blood of broiler chickens due to a lower proportion of carbohydrates. Furthermore, Nguyen et al. (2008) noted that serum TG may originate from four sources: DNL, cytoplasmic TG stores, lipoprotein remnants directly taken up by the liver, and plasma NEFA released by adipose tissue. These results indicated that a HFD can inhibit DNL and lipolysis in adipose tissue. Similarly, in our study, we also found that a HFD inhibited hepatic DNL (as evidenced by the decrease in G-6-PD and MDH activity) and fatty acid β-oxidation (as evidenced by the decrease in the CPT-1 content) as well as the LPL activity in the skin fat, which resulted in a decrease in liver weight and yield and an increase in abdominal fat and skin fat weight and yield of ducks. Gilbert et al. (1975) showed that a HFD reduces the DNL capacity of poultry livers and that waterfowl eating diets rich in carbohydrates can increase liver weight (Chartrin et al., 2006), which indicated that the increase in liver weight may be proportional to that in DNL of the liver.

Moreover, numerous studies have shown that animals fed a HFD can increase the fat content of the liver (Liu et al., 2016). It is generally accepted that the fat in the liver mainly comes from 2 sources, the DNL from carbohydrates in the feed and the fat contained in the feed itself. TGs can be synthesized from glucose resulting from the digestion of the carbohydrates. The composition of the fatty acids synthesized through this route is characterized by an elevated content of palmitic acid, stearic acid, and oleic acid, which constitute 90% of the total fatty acids (Baião and Lara, 2005). Jalaludeen et al. (2022) stated that a diet rich in carbohydrates promotes hepatic lipogenesis, and consequently, the synthesis in SFAs and MUFAs, but PUFAs may be mainly provided by fat in the diet of ducks. Similarly, in the present study, we observed that the contents of SFAs and MUFAs were decreased and the contents of PUFAs as well as U:S ratio were increased in the liver and skin fat of ducks fed a HFD. There results further indicated that the PUFA deposition in liver or skin fat mainly came from dietary fat itself, which could increase the fluidity of adipocyte cell membrane and promote more fat deposition in adipose tissues when ducks fed a HFD. This may be the unique lipid metabolism of meat duck, which needs more concern.

Furthermore, after eliminating the effect of FI (HFD vs. PFD), we found that the dietary fat content itself increased the content of TBA and decreased the content of HDL-C in serum. The reason may be due to the difference in the composition of FAs in the HFD; the composition of PUFA in the HFD was lower than that in the ND. A previous study showed that consuming more omega-3 FAs can promote HDL-C formation (Rader, 2003; Siri and Krauss, 2005). Cao et al. (2022) also found that the serum HDL-C level of geese fed a HFD was lower than that of geese in the NC group. Additionally, a HFD promotes permeability in the intestine and elevates cecum and blood bile acid concentrations because intestinal farnesoid X receptor expression and ursodeoxycholic acid synthesis may affect bile homeostasis by altering intestinal permeability (Stenman et al., 2012; Murakami et al., 2016; Lin et al., 2019). More importantly, after eliminating the effect of FI (HFD vs. PFD), we confirmed that a high dietary fat content indeed inhibited hepatic DNL, fatty acid β-oxidation, and lipolysis of adipose tissue, along with significantly increasing the fat deposition in the whole body of ducks.

CONCLUSIONS

In conclusion, these results confirmed that a HFD reduced the FI and F:G of Pekin ducks, which was related to the increased fat deposition by inhibiting hepatic DNL and β-oxidation as well as lipolysis of adipose tissue and the promotion of bile acid secretion. Moreover, HFD could increase the PUFA deposition in liver or skin fat which could increase the fluidity of adipocyte cell membrane and promote more fat deposition in adipose tissues in ducks. The data in the present study provide some valuable information for understanding the regulation of lipid metabolism and subcutaneous fat deposition in Pekin ducks.

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DISCLOSURES

No conflicts of interest exist in the submission of this manuscript, and the manuscript has been approved by all authors for publication.

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