Caponization and Testosterone Implantation Effects on Blood Lipid and Lipoprotein Profile in Male Chickens

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ABSTRACT To understand the role of lipid metabolism in increasing body fat accumulation after caponization of male chickens, trials were conducted to determine the effects of levels of testosterone implantation on lipoprotein composition. Male chickens were caponized at 12 wk and selected at 16 wk for a 10-wk feeding experiment. Fifteen male and 15 caponized (capon) chickens were used in trial 1. Ten sham operated chickens (sham) and 40 capons were randomly divided among 4 treatments in trial 2; the treatments were as follows: implantation of cholesterol (1.62 mm i.d. × 3.16 mm o.d., 9.24 ± 0.36 mg) or implantation of testosterone at low (1 mm i.d. × 3 mm, o.d., 5.88 ± 0.23 mg), medium (1.62 mm i.d. × 3.16 mm, o.d., 9.81 ± 0.17 mg), or high (2 mm i.d. × 4 mm, o.d., 16.7 ± 0.24 mg) dose. The results of trial 1 showed that caponization decreased ($P < 0.05$) blood testosterone concentrations and increased ($P < 0.05$) abdominal fat weight and relative abdominal fat weight in capons. Caponization also increased low density lipoprotein (LDL), high density lipoprotein (HDL), LDL protein, and HDL protein and decreased LDL-free cholesterol (LDL-FC), HDL-FC, and HDL-phospholipid (HDL-PL) percentages ($P < 0.05$). In trial 2 capons implanted with increasing testosterone levels exhibited proportional increases in blood testosterone concentration, although blood testosterone concentration in implanted capons were not fully restored to those of the sham group. High dose testosterone implantation inhibited abdominal fat accumulation and increased glucose and glycerol concentrations compared with the cholesterol implantation. Caponization of male chickens decreased the androgen level and increased the blood triacylglyceride content. Caponization also changed the lipoprotein profiles, which resulted in increased lipid storage capacity. The testosterone concentration, therefore, must achieve threshold concentrations to inhibit lipid accumulation in the testosterone implanted capon.

(*Key words*: caponization, testosterone implantation, lipoprotein, lipid metabolism)

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INTRODUCTION

Capons are male chickens whose testes have been surgically removed. Because of the resultant androgen deficiency, secondary male sexual characters including the comb, wattle, fighting behavior, and vocalization degenerate, and maturity regresses to an immature stage. Lipids also begin to accumulate in the body, enhancing flavor, texture, and meat juiciness compared with that of intact cockerels (Chen et al., 2000b).

Lipid accumulation after caponization (Fennell and Scanes, 1992; Chen et al., 2000a) increases subcutaneous and intercellular lipid accumulations (Hsieh et al., 2001). This lipid accumulation, as indicated by Hsieh et al. (2001), is attributed to increased hepatic lipogenesis capability and, hence, increased blood lipid concentration. Electrophoretic analyses of the blood lipoproteins have demonstrated that capons exhibit altered proportions of lipoprotein and thus have alterations in their serum lipoprotein transport mechanisms. Regardless of the changes in lipoprotein ratios, the lipoprotein composition does not change (Hsieh et al., 2001). Changes in the lipoprotein proportion mean that some lipoproteins increase while other lipoproteins decrease. This, however, does not reflect changes in the content of various lipoproteins. Compositions of various lipoproteins change according to physiological state, feeding regimen, and genotype (Hermier et al., 1984). The plasma concentration of very low density lipoprotein (VLDL) concentrations in birds give a good indication of the body lipid content in live broilers

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Abbreviation Key: CE = cholesterol esters; CHOL = total cholesterol; FC = free cholesterol; HDL = high density lipoprotein; LDL = low density lipoprotein; NEFA = nonesterified fatty acid; PL = phospholipid; SCWL = Single Comb White Leghorns; TG = triacylglyceride; VLDL = very low density lipoprotein.

TABLE 1. The basal diet composition

Ingredient	%
Yellow corn, grain	68.95
Soybean meal, 44%	14.2
Wheat bran	10.0
Fish meal, 65%	2.5
Limestone, pulverized	1.4
Dicalcium phosphate	1.6
Vitamin premix[1]	0.1
Mineral premix[2]	0.1
Salts	0.3
DL-Methionine	0.06
L-Lysine	0.025
Total	100
Calculated analysis	
Crude protein, %	15.9
ME, kcal/kg	2,873
Calcium, %	0.8
Available phosphorus, %	0.35

[1]The vitamin premix supplied per kilogram of diet: vitamin A, 12,000 IU; vitamin D_3, 3,125 ICU; vitamin E, 37.5 IU; vitamin K_3, 6.25 mg; vitamin B_1, 3.75 mg; vitamin B_2, 12.5 mg; vitamin B_6, 10.0 mg; calcium-pantothenate, 18.8 mg; niacin, 50 mg; biotin, 0.06 mg; folic acid, 1.25 mg; vitamin B_{12}, 0.05 mg.

[2]The mineral premix supplied per kilogram of diet: Cu ($CuSO_4 \cdot 5H_2O$, 25.45% Cu), 6 mg; Fe ($FeSO_4 \cdot 7H_2O$, 20.09% Fe), 50 mg; Mn ($MnSO_4 \cdot H_2O$, 32.49% Mn), 40 mg; Zn (ZnO, 80.35% Zn), 60 mg; Se ($NaSeO_3$, 45.56% Se), 0.075 mg.

and could serve as a basis for genetic selection criterion (Whitehead et al., 1984).

This study was, therefore, aimed at studying changes in lipoprotein composition and structure on lipid transportation in male chickens after caponization. Testosterone was also implanted to determine its role in changes in blood lipoprotein composition and structure.

MATERIALS AND METHODS

Bird Management and Experimental Design

Healthy male Single Comb White Leghorn (SCWL) chickens were caponized at 12 wk of age and housed in individual 40 × 30 × 38 cm cages for a 4-wk adaptation period. Fifteen male and 15 caponized (capons, prominent degenerated comb) chickens were selected at 16 wk of age for a 10-wk feeding experiment (trial 1). In trial 2, 10 sham operated chickens (sham) and 40 capons were randomly divided into 4 treatments: cholesterol implantation (1.62 mm i.d. × 3.16 mm o.d., 9.24 ± 0.36 mg) or testosterone dosages at low (1 mm i.d. × 3 mm, o.d., 5.88 ± 0.23 mg), medium (1.62 mm i.d. × 3.16 mm, o.d., 9.81 ± 0.17 mg), or high (2 mm i.d. × 4 mm, o.d., 16.7 ± 0.24 mg) for the 10-wk experiment (feeding to 26 wk of age). Feed (Table 1) and water were provided ad libitum during the feeding period.

Testectomy

The testectomy procedure was performed according to Chen et al. (2000a). Restricted to feed and water for 12 h before the surgical operation, male chickens were restrained, and the incision site was sterilized with iodine-alcohol. A 1-cm lateral incision was made at the second to last rib. The testes were then removed. Iodine-alcohol was applied again to the incision site.

Testosterone Implantation

The testosterone implantation procedure was performed according to the modified method of Fennell et al. (1990). A 1-cm implantation tube (Tygon Clear Tubing R-3603[2]) was used in this trial with different inner diameter sizes to control the testosterone dose. Testosterone was implanted subcutaneously at the back of a chicken's neck at 16, 20, and 24 wk of age.

Measurements

Feed intake and live weight were measured at 16, 21, and 26 wk of age. At 26 wk of age the chickens were euthanized, and the abdominal fat pad was removed for weighing.

Blood Constituents. Blood samples were taken from the brachial vein following a 12-h feed and water withdrawal at the end of the trial. The testosterone concentration was determined according to the method of Li et al. (1987), and estradiol concentration was determined using a γ-counter (COBRAII-D5003[3]) with a Webster radioimmunoassay kit, DSL estradiol RIA kit (DSL-4400[4]). Blood serum glucose, triacylglyceride (TG), and total cholesterol (CHOL) concentrations were analyzed with an automatic blood chemical analyzer with Roche testing kits (Cobas Mira Plus[5]). Nonesterified fatty acids (NEFA) were determined according to the modified method of Chromy et al. (1977). Phospholipid (PL) and glycerol were determined according to the method of Dryer et al. (1956) and Imai (1967), respectively. Serum lipoproteins were analyzed with a titian gel kit (3045[6]), and an electrophoresis data center scanning densitometry (Helena Laboratories[6]) was used to estimate the area of each fraction.

Lipoprotein Composition. Lipoproteins were isolated from plasma on the basis of their hydrated density in a single step according to the ultracentrifugation gradient procedure described by Chapman (1980) and modified by Hermier et al. (1985).

Ultracentrifugation was performed in a Beckman L8-70M ultracentrifuge[7] using a Beckman SW41 rotor[7] at 40,000 rpm for 24 h at 4°C. The resulting density limits reflected the alternation of the pigmented bands on the gradient and allowed fractionation of all plasmas as follows: VLDL ≤ density 1.006 > low density lipoprotein (LDL) ≤ 1.044 > high density lipoprotein (HDL) ≤ 1.21. The following components were quantified in each lipoprotein fraction. Protein was determined according to the method of Lowry et al. (1951). TG, CHOL, and PL were

[2]Staint-Gobain Performance Plastics Corp., Akron, OH.
[3]Packard Instrument Co., Downers Grove, IL.
[4]Diagnostic Systems Laboratories, Inc., Webster, TX.
[5]Roche Diagnostic Systems, Inc., Branchburg, NJ.
[6]Helena titian gel lipoprotein electrophoresis system, Helena Laboratories, Beaumont, TX.
[7]www.beckman.com.

TABLE 2. Caponization effects on growth performance and blood sex steroid concentrations in chickens (trial 1)

Parameter	Male	Capon	SEM
Body weight, g	1,722	1,803	41.2
Feed intake, g/day per bird	74.8	72.2	3.25
Abdominal fat weight, g	5.02[b]	46.3[a]	4.49
Relative abdominal fat weight, g/100 g of BW	0.28[b]	2.53[a]	0.22
Testosterone, pg/mL	885[a]	106[b]	148
Estradiol, pg/mL	20.1	25.9	2.04

[a,b]Means within the same row with different superscripts are significantly different ($P < 0.05$).

TABLE 3. Caponization effects on blood lipid constituents and lipoprotein proportions in chickens (trial 1)

Parameter	Male	Capon	SEM
Glucose, mg/dL	302	277	20.8
Triacylglycerol, mg/dL	42.7	52.8*	4.05
Cholesterol, mg/dL	155	152	7.07
Phospholipids, mg/dL	53.9	57.6	2.76
Nonesterified fatty acid, μM	136	139	2.59
Glycerol, mg/dL	9.70	9.21	0.64
VLDL,[1] %	4.88	4.53	0.52
LDL,[1] %	37.4	34.4	1.50
VLDL + LDL,[1] %	42.2	39.0	1.55
HDL,[1] %	57.7	60.9	1.55

[1]Percentages of total lipoprotein. VLDL = very low density lipoprotein; LDL = low density lipoprotein; HDL = high density lipoprotein.
*$P < 0.08$

determined according to blood constituents. Free cholesterol was determined with a kit[8] (free cholesterol C, Germany). The amount of cholesterol esters (CE) was calculated using the following formula: CE = (CHOL − FC) × 1.67. Lipoprotein concentrations have been calculated by addition of the concentrations of individual proteins and lipid components.

Statistical Analysis

Analyses of variance among treatment groups (trial 1: male chickens and capons; trial 2: sham, capons implanted with cholesterol, and low, medium, and high doses of testosterone) were calculated using the GLM procedure of the SAS (1985). Duncan's new multiple range test was used to compare the means according to Steel and Torrie (1960).

RESULTS AND DISCUSSION

Trial 1

Growth Performance. Table 2 presents the caponization effect on growth performance and sex steroid concentration in the blood of male chickens. Caponization of male chickens decreased serum testosterone ($P < 0.05$) but not estradiol ($P > 0.05$) and increased abdominal fat and relative abdominal fat weight ($P < 0.05$).

Caponization of cockerels decreased testosterone and increased abdominal fat as expected, which was in agreement with previous reports (Cason et al., 1988; Fennell and Scanes, 1992; Chen et al., 2000a). Body weights did not differ between the caponized and intact SCWL cockerels in this study, which differed from the results from similar studies using broiler chickens (Welter, 1976; Mast et al., 1981; Chen et al., 2000a) but agreed with those using SCWL cockerels (Fennell et al., 1990, 1996; Fennell and Scanes, 1992). The differences between broilers and layer chicken strains may be attributed to the slower growth rate of the latter. This reflects the trend toward heavier mean body weight in capons compared with intact male chickens ($P < 0.05$). The different growth response of capons is probably due to differences in strains, caponization age, caponization method, slaughtering age, and feeding regimen (Cason et al., 1988).

Caponization did not increase the estradiol concentration ($P > 0.05$), hence the increase in lipogenesis capability and lipid accumulation in the body may not be attributed to estradiol but may be due to decreased testosterone after caponization.

Lipid Metabolism

Table 3 presents the caponization effect on blood lipid constituents and lipoprotein proportion in intact male chickens. Caponization did not influence ($P > 0.05$) blood lipid constituents or lipoprotein proportion compared with in intact male chickens.

Although caponization did not influence the blood lipid constituents ($P > 0.05$), it did tend ($P < 0.08$) to increase the TG concentration by 19%. Hsieh et al. (2001) observed a significant increase in the blood TG content of caponized cockerel Taiwan country chickens (meat type) from caponization to 26 wk of age. The positive correlation between blood TG content and abdominal lipid accumulation differed from the current trial, which exhibited

TABLE 4. Caponization effects on lipoprotein profiles in chickens (trial 1)

Parameter[1]	Male	Capon	SEM
LDL			
Total content, μg/mL	235[b]	397[a]	24.3
Triacylglycerol, %	15.5	15.4	1.11
Phospholipids, %	16.8	16.8	1.56
Free cholesterol, %	32.8[a]	25.5[b]	2.05
Cholesterol ester, %	22.1	21.8	4.10
Protein, %	12.5[b]	20.3[a]	1.34
HDL			
Total content, μg/mL	1,881[b]	2,132[a]	101
Triacylglycerol, %	9.00	9.32	0.56
Phospholipids, %	28.0[a]	12.6[b]	1.32
Free cholesterol, %	11.0[a]	8.98[b]	0.56
Cholesterol ester, %	25.2[b]	30.8[a]	1.76
Protein, %	26.6[b]	38.1[a]	3.19

[a,b]Means within the same row with different superscripts are significantly different ($P < 0.05$).
[1]LDL = low density lipoprotein; HDL = high density lipoprotein.

[8]Kit was provided by Wako Chemicals, Düsseldorf, Germany.

TABLE 5. Effects of testosterone implantation on growth performance and blood sex steroid concentration in caponized chickens (trial 2)

Parameter	Sham	Implantation				SEM
		CHOL	Low	Medium	High	
Body weight, g	1,694	1,679	1,696	1,722	1,563	52.8
Feed intake, g/day per bird	72.2	73.0	75.4	74.9	75.6	3.05
Abdominal fat weight, g	3.62^{c}	39.5^{a}	37.9^{a}	26.8^{ab}	16.2^{bc}	5.61
Relative abdominal fat weight, g/100 g of BW	0.21^{c}	2.37^{a}	2.16^{a}	1.51^{ab}	1.11^{bc}	0.32
Testosterone, pg/mL	817^{a}	139^{c}	266^{bc}	342^{bc}	405^{b}	75.7

$^{a–c}$ Means within the same row with different superscripts are significantly different ($P < 0.05$).

only a trend in increased weight gain. This result may be attributed to the smaller males in layer strains with lower lipogenesis capability and blood TG concentrations, thus making differences harder to detect.

The liver is the major lipogenesis site in chickens (Donaldson, 1990; Griffin et al., 1992). Hepatic lipid transport in supplying fuel for body tissues depends on binding polar lipids with proteins and forming hydrophylic lipoproteins in the blood (Scriver et al., 1989; Hermier, 1997). In this trial, the lipoproteins contained the largest HDL and LDL ratios. VLDL was lowest with less than 5% observed in the blood. This lipoprotein ratio differed from that observed in laying hens with VLDL as the major lipoprotein supplied for egg production (Walzem et al., 1994). This result means that large amounts of fat are transported out of liver through VLDL and hydrolyzed by lipoprotein lipase, in which TG is hydrolyzed into NEFA and glycerol to provide the energy required or for body storage. The remnant of the hydrolyzed VLDL is found in the LDL form (Hermier, 1997).

Lipoprotein Profile

Table 4 presents the caponization effect on the lipoprotein profile in male chickens. Caponization increased ($P < 0.05$) the LDL concentration and LDL protein and decreased the LDL-free cholesterol (LDL-FC) percentage. Caponization increased ($P < 0.05$) the HDL concentration, HDL protein percentage, and HDL-cholesterol ester (HDL-CE) percentage and decreased ($P < 0.05$) HDL-PL and HDL-FC compared with in intact male chickens.

Serum VLDL ratios were as low as 4 to 5% in this trial, and the collected amount was not adequate for lipoprotein composition analysis. In this trial, capons were observed to contain higher LDL and HDL than intact males. This result showed that caponization increased ($P < 0.05$) the protein percentage and decreased the FC percentage when lipoprotein composition was converted into a percentage. Thus, caponization increased lipoprotein density. Caponization decreased the FC percentage in LDL and the FC and PL percentages in HDL ($P < 0.05$). FC and PL were mainly located in the outer layers of the particles in direct contact with blood. The FC and PL percentages decreased without change or increased the TG and CE percentages in the inner nucleus, which might result in transport and metabolic changes.

Trial 2

Growth Performance. Table 5 presents testosterone implantation effects on growth performance and blood sex steroid concentrations in capons. Capons implanted with testosterone up to the high dose had increased ($P < 0.05$) serum testosterone concentration compared with capons implanted with CHOL. This value was still lower ($P < 0.05$) than found with the sham. Abdominal fat and relative abdominal fat declined as the dose of implanted testosterone was increased and reached significance ($P <$

TABLE 6. Testosterone implantation effect on the blood lipid constituents and lipoprotein proportion in caponized chickens (trial 2)

Parameter	Sham	Implantation				SEM
		CHOL	Low	Medium	High	
Glucose, mg/dL	230^{ab}	179^{c}	201^{bc}	207^{bc}	247^{a}	10.3
Triacylglycerol, mg/dL	30.3^{b}	50.6^{a}	60.3^{a}	45.4^{ab}	49.6^{a}	7.71
Cholesterol, mg/dL	129^{b}	151^{ab}	132^{b}	142^{b}	171^{a}	8.44
Phospholipids, mg/dL	53.6^{b}	57.0^{ab}	66.8^{a}	56.7^{ab}	67.2^{a}	3.82
Nonesterified fatty acid, μM	142^{a}	127^{bc}	133^{b}	142^{a}	119^{c}	2.72
Glycerol, mg/dL	8.19^{b}	8.61^{b}	10.6^{a}	9.28^{ab}	10.5^{a}	0.52
VLDL,[1] %	2.94^{b}	5.49^{ab}	3.41^{ab}	6.06^{a}	4.95^{ab}	0.92
LDL,[1] %	38.2^{a}	31.6^{ab}	31.8^{ab}	28.0^{b}	34.2^{ab}	2.25
VLDL + LDL, %	41.2	37.1	35.2	34.0	39.1	2.32
HDL,[1] %	58.7	62.8	64.7	65.9	59.6	2.45

$^{a–c}$Means within the same row with different superscripts are significantly different ($P < 0.05$).

[1]Percentage of total lipoprotein. VLDL = very low density lipoprotein; LDL = low density lipoprotein; HDL = high density lipoprotein.

TABLE 7. Effects of testosterone implantation on lipoprotein profile in caponized chickens (trial 2)

		Implantation				
Parameter[1]	Sham	CHOL	Low	Medium	High	SEM
LDL						
Total content, μg/mL	291	303	265	267	316	25.3
Triacylglycerol, %	15.0	15.5	19.4	19.8	20.1	1.63
Phospholipids, %	16.5[a]	12.4[b]	12.2[b]	10.2[b]	12.0[b]	1.13
Free cholesterol, %	34.7	33.9	31.9	33.3	38.2	3.14
Cholesterol ester, %	20.7	22.0	14.2	18.6	13.6	5.01
Protein, %	12.9[b]	16.4[ab]	22.0[a]	17.7[ab]	15.7[b]	1.96
HDL						
Total content, μg/mL	1,622	1,710	1,578	1,811	1,750	90.8
Triacylglycerol, %	9.23[ab]	8.57[b]	10.1[ab]	10.6[a]	9.42[ab]	0.56
Phospholipids, %	19.7[a]	15.6[b]	15.6[b]	13.7[b]	14.8[b]	1.34
Free cholesterol, %	10.6[b]	11.8[ab]	12.7[a]	11.6[ab]	12.8[a]	0.60
Cholesterol ester, %	31.4[a]	26.3[ab]	20.7[b]	25.7[ab]	28.2[a]	2.03
Protein, %	28.9[b]	38.9[a]	40.5[a]	38.1[a]	34.7[ab]	2.08

[a,b]Means within the same row with different superscripts are significantly different ($P < 0.05$).

[1]LDL = low density lipoprotein; HDL = high density lipoprotein.

0.05) for the high implantation dose compared with CHOL implantation. This value did not differ ($P > 0.05$) compared with the sham.

By implantation of different androgen compound substitute levels [i.e., testosterone, 5α-dihydrotestosterone (5α-DHT), or 19-nortestosterone (19-NorT)] in SCWL capons, Fennell and Scanes (1992) demonstrated decreased BW and abdominal fat and concluded that androgen suppresses growth in capons. The growth suppression effectiveness was in the order 19-NorT > 5α-DHT > testosterone. Only the high testosterone dose decreased abdominal fat. This result agreed with the observation that only the high testosterone implantation dose decreased abdominal fat weight in this trial. The decline in abdominal fat weight was closely related to the testosterone concentration in the blood. The high testosterone dose concentration was more than 3 times that found in the CHOL implanted capons. This result was only half the concentration found in the sham operated birds.

Lipid Metabolism

Table 6 presents the effects of testosterone implantation on blood lipid constituents and lipoprotein proportions in capons. The high testosterone implantation dose increased ($P < 0.05$) glucose and glycerol contents compared with the sham group. The medium dose increased the NEFA content compared with CHOL implantation. Blood TG was higher and glucose and NEFA contents were lower ($P < 0.05$) in the group implanted with CHOL implanted than the sham group.

The higher glucose concentration in the sham group and high dose testosterone capons over the CHOL implanted capon group reflected the effect of testosterone on glucose concentration. The reason for this effect is still unclear. The glucose and NEFA concentrations are dynamic, maintaining a balance to provide fuel for the energy required through a homeostatic control mechanism within the body (Caprio et al., 1989). During regular feeding, blood NEFA and glycerol are hydrolyzed mainly by lipoprotein lipase from portomicrons and VLDL. The increase in the blood NEFA concentration feeds back into lipid synthesis (Tanaka et al., 1979; Donaldson, 1985). Increased NEFA under normal feeding conditions reflects that more energy is required to fuel the body. This result agreed with the decreased abdominal fat in the sham and increased ($P < 0.05$) NEFA concentration in the sham or medium dose testosterone implanted capons. The role of NEFA is to fuel the energy needed in the body, and therefore a rapid dynamic state with a fast turn over rate and short half-life is required. This therefore creates the large concentration variation in the blood (Caprio et al., 1989).

The TG concentration was significantly lower in the sham birds than in CHOL implanted capons in this and the previous trials, which indicated that testosterone implantation did not decrease the TG content after caponization. This finding could be attributed to the lower testosterone implantation amount.

Lipoprotein Profile

Table 7 presents the effects of testosterone implantation on the lipoprotein profile of capons. The medium testosterone implantation dose increased ($P < 0.05$) the HDL-TG percentage compared with the different testosterone dose in CHOL implanted capons. The PL percentages in LDL and HDL were lower ($P < 0.05$) and the HDL protein percentage was higher ($P < 0.05$) in the CHOL implanted capons compared with the sham operated birds.

Testosterone implantation increased the HDL-TG percentage compared with CHOL implantation in capons. The blood TG content in both groups did not differ ($P > 0.05$) (Table 6), whereas the TG percentages for LDL and HDL increased, representing decreased TG content in VLDL. This pattern reflected that testosterone increased the TG percentage in HDL and, conversely, decreased it in VLDL. From the analyses in trials 1 and 2, we determined that TG in VLDL were significantly higher in the capons or the CHOL implanted capons compared with

male chickens or the sham group. The TG could be determined from VLDL, and the other contents, including PL, CHOL, and protein, could not be determined from VLDL due to the trace amount available (data not shown).

The VLDL contains mainly TG. The increase in blood VLDL reflected more fat accumulation in the body and, therefore, was positively correlated to the amount of abdominal fat. This result could be used as an indicator in chicken breeding (Whitehead et al., 1984). This result was also proven from the higher abdominal fat in capons and CHOL implanted capons observed in these trials. The PL and protein percentage values in the CHOL implanted capons and the sham group observed in this trial also agreed with the findings of our first trial and again proved that androgen depressed the protein percentage and increased the PL percentage in HDL. Implantation of testosterone was unable to reach the level detected in the sham group.

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