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Effects of Maternal Dietary Treatment with Dehydroepiandrosterone on Lipid Metabolism Parameters in Offspring Broilers

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Abstract: To explore the effects of maternal dietary treatments with Dehydroepiandrosterone (DHEA) on lipid metabolism this study investigated lipid parameters and related hormones in the offspring broilers. Seventy five female broilers were allocated into 3 groups and they were provided with a commercial diet supplemented with DHEA at 0, 25 or 50 mg kg^{-1} diet. Eggs were collected after DHEA treatment and incubated at 37.5°C and a relative humidity of 60%. Broiler offspring were fed the same basal diets from 1-42 days. The results showed that the body weight was decreased ($p<0.05$) at 21 day in all DHEA treatment group while a decrease was observed with 25 mg kg^{-1} DHEA treatment in male offspring broilers at 42 day ($p<0.05$). The daily gain and feed/gain were decreased with DHEA treatment during 1-3 weeks in offspring broilers ($p<0.05$). The Percent of Liver (PL) and Percent of Abdominal Fat (PAF) were decreased ($p<0.05$) in male offspring broilers in all DHEA treatment groups and the Percent of Thigh Muscle (PTM) and Percent of Breast Muscle (PBM) were increased in male offspring broilers with 50 mg kg^{-1} DHEA treatment. The content of hepatic Triglycerides (TG) was decreased with 50 mg kg^{-1} DHEA in male offspring broilers while the level of serum TG was increased ($p<0.05$). The concentration of Non-Esterified Fatty Acid (NEFA) was higher with 25 mg kg^{-1} DHEA treatment in both male and female offspring broilers as compared to the control group ($p<0.05$). An increase in serum Triiodothyronine (T3) and a decrease in serum Thyroxin (T4) were observed in all experimental groups with supplement maternal DHEA ($p<0.05$), except in female offspring broilers from 50 mg kg^{-1} DHEA maternal-fed birds. Also, 25 mg kg^{-1} DHEA supplement enhanced the serum Glucagon (GLU) concentration in male offspring broilers and 50 mg kg^{-1} DHEA could increased the leptin level in female offspring broilers. Overall, the results of this study indicated that maternal Dietary DHEA would be beneficial in decreasing abdominal lipid deposition in offspring broilers.

Key words: Lipid metabolism, Dehydroepiandrosterone (DHEA), maternal effect, offspring broilers, TG

INTRODUCTION

Modern bird strains often exhibit excessive body fat deposition (Xu *et al*., 2003). This tendency has proven to be one of the main problems encountered within the broiler industry today. Not only does it have a negative effect on feed efficiency but it also results in economic losses at poultry processing plants (Wu *et al*., 2006). Considerable research efforts have been expended to study the factors that are associated with fat deposition and the methods needed to reduce it and several beneficial solutions to address this complex problem have been identified (Wang *et al*., 2006; Xu *et al*., 2003).

Maternal effect refers to parental phenotypes having a direct influence on their offspring phenotype (Arnold, 1994). It is a phenotypic response of the maternal offspring to different environmental conditions and an important source of evolutionary dynamics (Bernardo, 1996). Much evidence has emerged to suggest that alterations in maternal nutrition may irreversibly affect aspects of physiological and biochemical functions in the offspring (Jarocka-Cyrta *et al*., 1998). Aurelie *et al*. (2008) earlier reported that benefits from maternal dietary carotenoids were transferred to chicks and they also found that chick growth was enhanced when carotenoids were assimilated both before and after hatching. This highlights the importance of the maternal effect in offspring development.

Dehydroepiandrosterone (DHEA), the most abundant steroid hormone in circulation is a major secreted

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product of the human adrenal gland (Labrie *et al*., 2005). It has been shown to exert numerous beneficial effects including anti-obesity, anti-diabetes and anti-carcinogenesis actions in various animal models and in human (Schwartz and Pashko, 2004). A number of studies have demonstrated that DHEA decreases food intake and body weight in rats (Richards *et al*., 2000) serum triglyceride levels in hyperlipidemic rats (Han *et al*., 1998) and directly affects the peroxisomal β-oxidation pathway in mouse hepatocytes (Waxman, 1996). The earlier study confirmed that the administration of DHEA to broiler chickens results in decreased abdominal lipid deposition and an increased rate of lipid catabolism through the regulation of metabolic hormones and parameters with no adverse effects on growth performance or carcass composition (Ma *et al*., 2008). In contrast to the large number of research has conducted on the regulation of DHEA on lipid metabolism in mice, rats and humans little is known about the effect of DHEA on lipid metabolism in poultry, especially in broiler offspring. Therefore, the present study was designed to study the effect of maternal diets supplemented with DHEA on lipid metabolism hormones and parameters in offspring broilers.

MATERIALS AND METHODS

Animal experiment: Seventy five female and six male broilers (Aconred, 16 weeks old) were obtained from Jiangsu Wuxi chicken breeding company (Wuxi, China). All birds were randomly divided into three equal groups (25 female and 2 males per group) and offered the same basal diets. Diets were fed from week 16-40 including starter (grower) and finisher (layer) rations that were formulated according to NRC recommendations (Table 1). The animal care and use protocol was approved by the Institutional Animal Care and Use Committee of Nanjing Agricultural University. All broiler chickens were kept on the ground with natural lighting and free access to drinking water.

Hens started the laying period at week 23 and reached a stationary phase of production at week 35 (lay crest-time is at week 25-44). At that point, the maternal diets were supplied with DHEA (Changzhou Jiaerke Pharmaceuticals Group Corp.) at levels of 0, 25 or 50 mg kg^{-1} feed for 5 weeks before collecting weight similar eggs for incubation. Eggs from the different groups were naturally inseminated and they were placed into an electric forced-draft incubator at 37.5±0.5°C and 60% relative humidity. One hundred and eighty off-spring broilers were allocated to 3 groups according the content of DHEA that added to the maternal diets with three replications per group and each replication consisting of twenty birds which were individually housed. The offspring were fed from days 1-42 during the starter (days 1-21) and finisher (days 21-42) phases of the growth cycle, nutrient levels of the diets (Table 1) were based on the NRC recommendations. Animal care and use were approved by the Institutional Animal Care and Use Committee of Nanjing Agricultural University. The broilers were floor-reared under natural lighting during the finisher phase and the chickens were weighed at 1, 21 and 42 days of age to determine Average Daily Gain (ADG). Everyday, feed consumption per replicate was recorded, the uneaten feed was discarded and fresh feed was replenished. Average Daily Feed Intake (ADFI) and the feed conversion ratio were then determined. At the end of experiment, birds were randomly selected from each replication, deprived of feed for 12 h. Then, the birds were slaughtered, gender was identified and the abdominal fat pad, liver, pectoral and leg muscles were subsequently removed and weighed and stored at −80°C before analysis.

Table 1: Ingredient composition and nutrient content of diets

Composition	Grower	Laying	Starter	Finisher
Ingredient (%)				
Corn	64.30	67.30	52.60	57.40
Soybean meal	18.00	16.00	31.10	27.00
Wheat bran	10.00	0.00	2.00	4.00
Fish meal	3.00	4.00	6.00	3.00
Rapeseed oil	0.00	0.00	5.00	5.00
Salt	0.40	0.40	0.30	0.30
Calcium phosphate	1.50	2.30	1.00	1.50
Limestone	1.60	6.80	1.20	1.20
DL-Methionine	0.10	0.10	0.30	0.10
Lysine	0.10	0.10	0.00	0.00
Vitamin-mineral premix[1]	1.00	1.00	0.50	0.50
Barm	0.00	2.00	0.00	0.00
Nutrition composition calculated				
ME (Mcal kg^{-1})	2.78	2.75	3.10	3.14
CP (%)	15.98	15.20	22.52	19.74
Lysine (%)	0.10	0.10	1.19	1.08
Methionine+cystine (%)	0.71	0.93	0.93	0.71
Ca (%)	1.50	3.00	1.00	0.90
Total P (%)	0.80	0.88	0.80	0.76
Available P (%)	0.45	0.53	0.47	0.39

ME = Metabolizable Energy. Nutrient level of the diets was based on National Research Council recommendations. [1]Vitamin-mineral premix supplied the after per kilogram of diet: vitamin A, 1,500 IU, vitamin D3, 200 IU, vitamin E, 10 mg, vitamin K3, 0.5 mg, thiamine, 1.8 mg, riboflavin, 3.6 mg, D-pantothenic acid, 10 mg, folic acid, 0.55 mg, pyridoxine, 3.5 mg, niacin, 35 mg, cobalamin, 0.01 mg, biotin, 0.15 mg, Fe, 80 mg, Cu, 8 mg, Mn, 60 mg, Zn, 40 mg, I, 0.35 mg and Se, 0.15 mg

Sample preparation: Blood samples were collected from each bird and allowed to clot at 4°C and centrifuged at 1,520×g for 20 min prior to the harvesting of the serum. Serum samples were stored at −40°C until assayed. Liver, left breast and thigh muscle were collected, blotted dry and weighed. Samples of liver tissue were weighed and stored at −40°C prior to homogenization.

Lipid metabolic hormone detection and lipid parameters assay: The plasma concentrations of Triiodothyronine (T3), Thyroxine (T4), Glucagon (GLU) and Leptin (LEP) were determined using commercial RIA kits (Beijing Biotechnology Corp., Beijing, P.R. China). The same blood samples and commercial kits (Nanjing Jianchen Biotechnology Institution, China) were used to detect Glucose (BG), Triglycerides (TG) Total serum Cholesterol (TC), High Density Lipoprotein-Cholesterol (HDL-C), Low Density Lipoprotein-Cholesterol (LDL-C) and Non-Esterified Fatty Acids (NEFA). Total liver TG content was determined on homogenized liver samples using a mixture of chloroform and methanol (2:1 v/v), according to the method of Folch *et al*. (1957). Total liver TC content was determined on homogenized liver samples using physiological saline. An aliquot of these homogenates was used for protein determination according to the method described by Markwell *et al*. (1978) and the remainder of the homogenate was centrifuged at 1,000×g for 10 min at 4°C. Clear supernatant was retained and used for the assay.

Statistical analyses: The experimental data were expressed as the mean+SEM and differences were considered significant when $p<0.05$ as tested by multiple comparisons of variance (one-way ANOVA, to compare interactions among control and DHEA-treatment groups) using the statistical packages for social science 16.0 and Excel 2003 in Microsoft. Replicate was considered as the experimental unit for the entire index determined.

RESULTS

DHEA effect on growth performance: The effect of DHEA on the body weight is shown in Table 2. Researchers found that the initial body weights were lower for both male and female off-spring broilers fed with 25 mg DHEA/kg as compared to their respective control groups ($p<0.05$). The male offspring broilers weight was lower with 25 and 50 mg kg^{-1} ($p<0.05$) DHEA treatment than the control group at 21 day but only 25 mg kg^{-1} DHEA treatment decreased ($p<0.05$) the body weight of male offspring broilers at 42 day. Also, the female offspring broilers were significantly lower body weight with 25 or 50 mg kg^{-1} DHEA treatment at 21 day ($p<0.05$) while the body weight were no different with DHEA-treat at 42 day ($p>0.05$).

Due to initially body weigh has significantly different between control and DHEA-treat group. So, researchers analyzed the effect on daily gain in offspring broilers. As Table 3 shown, the daily gain were significantly decreased with 25 or 50 mg kg^{-1} ($p<0.05$) DHEA treatment during 1-3 weeks but the daily gain was significantly decreased only with 50 mg kg^{-1} DHEA treatment during 4-6 weeks. Also, DHEA-treatment could decrease the daily gain in female offspring broilers during 1-3 weeks ($p<0.05$) while there was no different during 4-6 weeks ($p>0.05$). The DHEA-treatment had decreased the feed/gain ratio both in male and female offspring broilers during 1-3 weeks ($p<0.05$), while there was no different during 4-6 weeks except a declined in the female offspring broilers with 50 mg kg^{-1} DHEA treatment ($p<0.05$) (Table 4).

The effect of DHEA on Percent Breast Muscle (PBM), Percent Thigh Muscle (PTM), Percent Liver (PL) and Percent Abdominal Fat (PAF) in offspring broilers were shown in Table 4. Researchers could found that the PBM and PTM were no different between DHEA treatment and control group in male offspring broilers ($p>0.05$) while 50 mg kg^{-1} DHEA treatment could increase

Table 2: Effect of maternal DHEA on body weight in offspring broilers[1]

	Male broiler chicken maternal DHEA (mg kg^{-1})			Female broiler chicken maternal DHEA (mg kg^{-1})		
Items	0	25	50	0	25	50
IBW (g)	42.6±0.80[a]	40.4±0.40[b]	41.6±0.60[a]	41.3±0.60[a]	39.9±0.50[b]	40.7±0.60[a]
21 day BW (g)	518.5±22.0[a]	449.6±20.1[b]	441.3±12.2[b]	524.1±17.1[a]	451.9±10.1[b]	446.6±11.2[b]
42 day BW (g)	1919.3±58.7[a]	1679.7±36.8[b]	1788.2±50.1[a]	1733.0±25.7	1676.5±42.2	1772.1±32.5

IBW = Initial Body Weight; BW = Body Weight. [1]Data in each group represent mean values of three replicates, one replicate indicating the average of 10 chickens in one cage. [a,b]Values indicate the results of significance testing for difference between treatments. Unshared letters indicate significant difference among treatment groups $p<0.05$ while shared letters indicate no significant difference

Table 3: Effect of maternal DHEA on offspring broilers performance[1]

		Male broilers maternal DHEA (mg kg^{-1})			Female broilers maternal DHEA (mg kg^{-1})		
Time	Items	0	25	50	0	25	50
1-3 weeks	Daily gain (g)	21.10±0.70[a]	18.80±0.60[b]	19.20±0.40[b]	21.40±0.70[a]	19.60±0.50[b]	19.80±0.40[b]
	Daily feed intake (g)	40.50±5.40	30.20±3.30	31.30±3.30	39.30±3.20	29.10±4.30	30.80±2.70
	DFI/DG (g:g)	1.81±0.06[a]	1.44±0.05[b]	1.49±0.03[b]	1.77±0.05[a]	1.41±0.04[b]	1.44±0.03[b]
4-6 weeks	Daily gain (g)	76.70±2.40[a]	62.70±1.40[b]	71.00±3.00[a]	66.80±1.10	63.20±1.80	68.20±2.30
	Daily feed intake (g)	195.50±22.6	176.70±18.8	180.50±19.5	190.10±19.4	172.30±15.6	175.10±16.3
	DFI/DG (g:g)	2.34±0.08	2.53±0.06	2.37±0.11	2.66±0.04[a]	2.55±0.07[a]	2.43±0.08[b]

DG = Daily Gain; DFI = Daily Feed Intake; DFI/DG = Daily Feed Intake/Daily Gain. [1]Data in each group represent mean values of three replicates, one replicate indicating the average of 10 chickens in one cage. [a,b]Values indicate the results of significance testing for difference between treatments. Unshared letters indicate significant difference among treatment groups $p<0.05$ while shared letters indicate no significant difference

Table 4: Effect of maternal DHEA on the body composition in offspring broilers[1]

	Male broilers maternal DHEA (mg kg^{-1})			Female broilers maternal DHEA (mg kg^{-1})		
Items	0	25	50	0	25	50
PBM	14.65±0.39	14.55±0.28	14.81±0.28	15.46±0.34^{a}	15.86±0.26^{a}	16.49±0.31^{b}
PTM	15.24±0.25	15.50±0.28	15.37±0.15	14.30±0.25^{a}	14.90±0.24^{a}	15.06±0.13^{b}
PL	2.34±0.04^{a}	2.21±0.03^{b}	2.17±0.03^{b}	2.25±0.05	2.19±0.05	2.19±0.04
PAF	1.87±0.13^{a}	1.45±0.08^{b}	1.38±0.10^{b}	2.00±0.11	2.00±0.15	1.79±0.10

PBM = Percent Breast Muscle; PTM = Percent Thigh Muscle; PL = Percent Liver; PAF = Percent Abdominal Fat. [1]Values are mean+SEM. Each treated group represents 12 chickens at the age of 42 days (12 chickens per treatment are representative of 3 birds per replicate). $^{a, b}$Values indicate the results of significance testing for difference between treatments. Unshared letters indicate significant difference among treatment groups $p<0.05$ while shared letters indicate no significant difference

Table 5: Effects of maternal DHEA on the lipid metabolism parameters in offspring broilers[1]

		Male broilers maternal DHEA (mg kg^{-1})			Female broilers maternal DHEA (mg kg^{-1})		
Organs	Item	0	25	50	0	25	50
Serum	BG (mmol L^{-1})	11.900±0.440^{a}	10.270±0.270^{b}	11.050±0.230^{a}	11.130±0.070	10.670±0.1000	10.87±0.0800
	TC (mmol L^{-1})	3.530±0.100	3.680±0.200	3.590±0.150	3.000±0.160	3.400±0.1500	3.12±0.1800
	TG (mmol L^{-1})	0.310±0.040^{ab}	0.260±0.030^{b}	0.400±0.060^{a}	0.410±0.030	0.370±0.0300	0.36±0.2000
	HDL-C(mmol L^{-1})	1.940±0.100	1.690±0.090	1.920±0.130	1.590±0.080	1.800±0.0800	1.80±0.0800
	LDL-C(mmol L^{-1})	0.850±0.090	0.630±0.120	0.600±0.100	0.950±0.030	0.720±0.1000	0.67±0.1000
	NEFA (imol L^{-1})	576.460±37.74^{b}	759.730±42.09^{a}	562.190±28.12^{b}	797.680±95.69^{a}	1402.710±104.21^{b}	843.32±115.29^{a}
Liver	TC/TP(mmol mg^{-1} protein)	0.023±0.002^{a}	0.035±0.002^{b}	0.039±0.003^{b}	0.028±0.003^{a}	0.034±0.0030^{a}	0.04±0.0040^{b}
	TG (mmol L^{-1})	2.640±0.120^{ab}	2.810±0.200^{a}	2.020±0.290^{b}	2.650±0.150	2.440±0.2100	2.54±0.1200

BG = Blood Glucose; TG = Triglycerides; TC = Total Cholesterol; HDL-C = High Density Lipoprotein-Cholesterol; LDL-C = Low Density Lipoprotein-Cholesterol; NEFA = Non-Esterified Fatty Acid; TC/TP = Total Cholesterol/Total Protein. [1]Values are mean+SEM. Each treated group represents 12 chickens at the age of 42 days (12 chickens per treatment are representative of 3 birds per replicate). $^{a, b}$Values indicate the results of significance testing for difference between treatments. Unshared letters indicate significant difference among treatment groups $p<0.05$ while shared letters indicate no significant difference

Table 6: Effect of maternal DHEA on the content of serum hormone in offspring broilers[1]

	Male broilers maternal DHEA (mg kg^{-1})			Female broilers maternal DHEA (mg kg^{-1})		
Items	0	25	50	0	25	50
T3 (ng mL^{-1})	0.78±0.020^{a}	1.12±0.060^{b}	1.02±0.060^{b}	0.91±0.030^{a}	1.22±0.070^{b}	0.93±0.050^{a}
T4 (ng mL^{-1})	2.52±0.400^{a}	1.23±0.080^{b}	1.26±0.090^{b}	2.00±0.260^{a}	1.36±0.110^{b}	0.92±0.040^{c}
GLU (pg mL^{-1})	599.22±35.47^{a}	686.03±30.21^{b}	601.84±19.12^{a}	722.62±31.01	631.29±33.89	670.47±32.04
LEP (ng mL^{-1})	0.37±0.060	0.27±0.070	0.46±0.070	0.47±0.090^{a}	0.42±0.030^{a}	0.66±0.050^{b}

T3 = Triiodothyronine; T4 = Thyroxine; GLU = Glucagons; LEP = Leptin. [1]Values are mean+SEM. Each treated group represents 12 chickens at the age of 42 days (12 chickens per treatment are representative of 3 birds per replicate). $^{a-c}$Values indicate the results of significance testing for difference between treatments. Unshared letters indicate significant difference among treatment groups $p<0.05$ while shared letters indicate no significant difference

the PTM and PBM in female offspring broilers ($p<0.05$). Compared to the control group, the PL and PAF were decreased with 25 or 50 mg kg^{-1} DHEA treatment ($p<0.05$) in male offspring broilers. Interesting, DHEA did not any effect on PL and PAF in female offspring broilers ($p>0.05$).

Lipid metabolic parameters: The effects of maternal DHEA on serum concentrations of Blood Glucose (BG), Triglycerides (TG), Total Cholesterol (TC), High Density Lipoprotein-Cholesterol (HDL-C), Low Density Lipoprotein-Cholesterol (LDL-C), Non-Esterified Fatty Acid (NEFA) and hepatic Total Cholesterol (TC), Triglycerides (TG) in off-spring broilers were shown in Table 5. The blood BG level was significantly lower in male offspring broilers fed 25 mg kg^{-1} DHEA than in the control animals ($p<0.05$). The content of hepatic TG was decreased in the group with 50 mg kg^{-1} DHEA treatment group in male offspring broilers ($p<0.05$) while the level of serum TG was increased ($p<0.05$). The concentration of NEFA was higher with 25 mg kg^{-1} DHEA treatment in both male and female offspring broilers as compared to the control group ($p<0.05$). No significant differences were observed in serum TC between DHEA and control group in both male and female offspring broilers but the concentration of hepatic TC/TP was higher in male offspring broilers with DHEA-treatment than those fed no DHEA (control) ($p<0.05$) while it was higher in female broilers with 50 mg kg^{-1} DHEA maternal-fed birds only ($p<0.05$). During the entire experimental period, no differences were observed in serum HDL-C, LDL-C in offspring broilers.

Lipid metabolic hormone: The effect of DHEA on the concentrations of plasma Triiodothy-ronine (T3), Thyroxine (T4), Glucagon (GLU) and Leptin (LEP) were shown in Table 6. During the experimental period, maternal dietary treatment with DHEA significantly increased the serum concentration of T3 ($p<0.05$) in male offspring broilers while significantly increased the serum T3 ($p<0.05$) level with 25 mg kg^{-1} DHEA treatment group

in female offspring broilers. Also, the concentration of T4 were decreased with DHEA treatment groups than to control group broilers in all treatment group ($p<0.05$). No different were observed in the serum GLU concentration in all group ($p>0.05$) except it was higher in male offspring broilers in the 25 mg kg^{-1} DHEA group than the control group ($p<0.05$). Similar, there were no different were observed in the serum LEP in all group ($p>0.05$) except the LEP level was higher in female offspring broilers in the 50 mg kg^{-1} DHEA group ($p<0.05$).

DISCUSSION

The results of this study demonstrate for the first time that maternal diets supplemented with DHEA affect to the regulation of lipid metabolism in offspring broiler chickens. The data in this study shown that the initially body weight of offspring broilers were lower with DHEA treatment than control group although, researchers had selected the similar weight fertilized eggs to hatching. No comparative data are available from the literature relative to the maternal effects of DHEA on body weight and lipid metabolism in commercial broilers. However, the earlier study confirmed that maternal diets supplemented with DHEA reduce body weight in both the hen and her embryos (Chen *et al*., 2010). Also, researchers had observed that the embryos weight had decrease following *in ovo* administration of DHEA in broiler chickens during embryonic development (Zhao *et al*., 2007).

The present results shown that maternal diets supplemented with DHEA could decreased the body weight in both male and female offspring broilers at 21 day while the body weight was decreased in male offspring broilers only with 25 mg kg^{-1} DHEA-treatment group and no different were observed in all group in female offspring broilers at 42 day. These results are in agreement with reports (Cleary, 1991) which show that long term DHEA treatment results in suppressed body weight gain in rodents and also similar to the results of Gansler *et al*. (1985) who have reported that DHEA, administered chronically to lean or obese Zucker rats results in decreased body weight. Due to initially body weigh has significantly different between control and DHEA-treat group thus researchers analyzed the effect of DHEA on daily gain in offspring broilers and feed/gain in offspring broilers. The results showed that maternal diets supplemented with DHEA decreased the daily gain in both male and female offspring broilers during 1-3 weeks but the daily gain was decreased in male offspring broilers only with 50 mg kg^{-1} DHEA-treatment group and no different were observed in all group in female offspring broilers during 4-6 weeks. Similar, DHEA-treatment had decreased the feed/gain ratio both in male and female offspring broilers during 1-3 weeks and there was no different during 4-6 weeks except a declined in the female offspring broilers with 50 mg kg^{-1} DHEA treatment. Those results were consistent with the earlier studies (Zhao *et al*., 2007; Tang *et al*., 2007) reported a significant decrease in the body weight of DHEA-treated chickens.

Many reports have demonstrated that early maternal effects can result in substantial reorganization of offspring phenotypes and formation of novel developmental pathways (Badyaev *et al*., 2006; Karadas *et al*., 2005). In this study, researchers found that the PTM and PBM were increased with 50 mg kg^{-1} DHEA treatment in female offspring broilers, while PL and PAF were decreased with 25 mg kg^{-1} DHEA treatment in male offspring broilers. As a steroid hormone precursor, DHEA can be transferred to several steroid hormones by the action of steroidogenic enzymes in the peripheral tissue and exerts its action either indirectly in the peripheral target tissues of the sex steroid hormones (following conversion to the androgens, estrogens or both) or directly, as a neuro-steroid (via interaction with neurotransmitter receptors in the brain) (Labrie *et al*., 1997). The earlier study demonstrated that DHEA affects steroid hormone metabolism and steroidogenic enzyme mRNA expression during a 24 h treatment period in rat (Song *et al*., 2010). From the above findings, researchers speculate that the frequently documented temporal gradient in allocation of steroid hormones from hen into eggs can produce a corresponding directional shift in offspring broilers phenotypes including changes in growth and morphology. While the precise mechanism should be investigated further.

It has been demonstrated that DHEA has a fat-reducing effect and that this effect may be exerted by different mechanisms (De Pergola, 2000). The earlier studies proved that DHEA reduces adipose tissue by reducing its TG and TC content and increasing the rate of lipid catabolism by regulating various serum metabolic parameters (Tang *et al*., 2007). Zhao *et al*. (2007) reported that *in ovo* administration of DHEA to fertilized eggs before incubation reduced plasma and liver TG content. The present study showed that the content of hepatic TG was decreased in the group with 50 mg kg^{-1} DHEA treatment group in male offspring broilers while the level of serum TG was increased. No significant differences were observed in serum TC, HDL-C and LDL-C between DHEA treatment and control group in both male and female offspring broilers but the concentration of hepatic TC/TP was higher with 50 mg kg^{-1} DHEA than those fed

no DHEA (control) group. Those results was similar with those of Chen *et al.* (2010) which showed that 20 mg DHEA kg^{-1} decreased serum TG, TC and LDL in embryonic chickens while 100 mg DHEA kg^{-1} caused a significant increase in serum TC (Chen *et al.*, 2010).

Another interesting observation was that the concentration of NEFA was significantly higher with 25 mg kg^{-1} DHEA treatment in both male and female offspring broilers as compared to the control group. This was similar to a result study which revealed that DHEA increased the concentration of blood NEFA in broiler chickens (Tang *et al.*, 2007). Also, the earlier study had confirmed that DHEA could directly stimulated the β-oxidation in the hepatocyte, catalyzed the conversation of TG to glycerol and fatty acids and increased the hepatic uptake the released NEFA (Tang *et al.*, 2007). Under normal condition, poultry blood NEFA is mainly hydrolyzed by lipoprotein lipase from portomicrons and VLDL when the available energy is sufficient to meet the body's requirements, NEFA bind to albumin and then transported into the liver where they can be re-synthesized into TG to later meet the embryos' energy requirements (Rodoman *et al.*, 2001). Based on the above data it is reasonable to speculate that the maternal supplement with DHEA leads to higher concentration of NEFA in serum by acceleration the hydrolysis of TG to glycerol and fatty acids so that energy storage via fat deposition was reduced.

Of those thyroid hormones that we study, no reports were available regarding the effect of DHEA on circulation thyroid hormones levels in poultry. In the present study, an increase in serum T3 and a decrease in serum T4 were observed in all experimental groups with supplement maternal DHEA except in female off-spring broilers from 50 mg kg^{-1} DHEA maternal-fed birds. The results for serum T3 and T4 confirm a shift in the conversion of T4 to T3 but rather suggest a specific alternation in metabolism of T3 and T4 following treatment with DHEA. As observed previously (Buyse *et al.*, 2002), reduced circulating thyroid hormone levels lower the metabolic rate as e protective mechanism for the body's energy reserves. Therefore, these results are quite useful in that they indicate a similar effect of DHEA on T3 and T4 in offspring broilers. That is to say, DHEA lead to less fat accumulation by enhancing the metabolic rate.

In the present study, 25 mg kg^{-1} DHEA supplement enhanced the serum GLU concentration in male offspring broilers and 50 mg kg^{-1} DHEA could increased the LEP level in female offspring broilers which is in agreement with earlier study on GLU-stimulated, enhanced Leptin expression in broiler chicken adipose tissue (Ashwell *et al.*, 1999). Leptin is secreted by adipose tissue and has been shown to play an important role in feed intake regulation, energy metabolism and reproduction in mammals (Sun *et al.*, 2006). Although, the chicken Leptin promoter gene has not been cloned, chicken Leptin is highly conserved and therefore is similar to the mammalian gene. Taouis *et al.* (2001) reported that the impact of GLU on liver Leptin may be attributed to an elevation in intracellular cyclic AMP (cAMP). However, the relationship between GLU and Leptin in poultry, especially the serum levels is still unclear. The results of the present study suggest that DHEA up-regulates the serum Leptin level and that inhibit the accumulation of fat in offspring broilers, resulting in reduced abdominal fat deposition.

A rapidly increasing number of studies demonstrated the wide array of the effects of maternal hormones on the offspring such as on hatching time, early muscular growth, early postnatal growth of body mass and structural size and precluding survival. In addition, evidence is beginning to emerge that prenatal exposure to maternal hormones exerts long-lasting effects on morphology and behavior (Groothuis and von Engelhardt, 2005). The data confirm that maternal dietary treatment with DHEA accelerates lipid catabolic processes in 42 days offspring broiler chickens through the regulation of the lipid metabolic pathways. These findings are of general importance and demonstrate that hens treated with DHEA can influence the development and lipid metabolism of their offspring by nongenetic inheritance. This advantage can provide the parents with a relatively flexible tool to adjust offspring development to against fat deposition. As a steroidal compound, DHEA may be a powerful tool for such a parental or maternal effect. Thus, the study of maternal DHEA and its effects provides an excellent possibility to study reducing excessive carcass fat of nongenomic maternal effects.

CONCLUSION

Researchers maybe conclude that decreasing the fat deposition of broiler chickens can occur through supplementation of DHEA to the maternal diets and that maternal dietary DHEA would be beneficial in decreasing abdominal lipid deposition.

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