Effects of dehydroepiandrosterone on growth performance, lipid metabolic hormones and parameters in broilers

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ABSTRACT: One hundred and eighty (180) day-old broiler chickens were used to investigate the effects of dehydroepiandrosterone (DHEA) on growth performance, carcass composition, and the serum concentrations of several lipid metabolic hormones and metabolic parameters (indicators). The broilers received the same basal diets, with DHEA added at 0 (control), 5 and 20 mg/kg feed. During the experimental period, broilers fed DHEA exhibited lower levels of triglycerides (TG), total serum cholesterol (TC), high density lipoprotein-cholesterol (HDL-C) and non-esterified fatty acids (NEFA) as compared to the control animals, but a marked increase in lipoprotein lipase (LPL) activity. Adding DHEA to the diet significantly decreased serum concentrations of thyroxine (T_4), serum free triiodothyronine (FT_3), and serum free thyroxine (FT_4), but significantly increased the serum leptin (LEP) and glucogon (GLU) levels in male broiler chickens. However, female broiler chickens showed pronounced differences in LEP, FT_3 and FT_4 only, while there were no differences in the other three metabolic hormones (T_3, T_4 and GLU). Overall, these results indicate that DHEA improves lipid metabolism through the regulation of metabolic hormones and metabolic parameters, while not adversely affecting growth performance in broiler chickens.

Keywords: dehydroepiandrosterone (DHEA); growth performance; carcass composition; lipid metabolic hormones and parameters; broilers

Dehydroepiandrosterone (DHEA, 3β-hydroxy-5-androsten-17-one) is a steroid hormone that is secreted by the adrenal cortex in mammals (Orentreich et al., 1992; Parker, 1999; Aoki et al., 2004). It is known to have several physiological effects, including antiobesity, antidiabetes and anticarcinogenisis, when administered to rats and mice (Schwartz and Pashko, 2004). A number of studies have demonstrated that DHEA decreases fat intake and body weight in rats (Tagliaferro et al., 1986; Yamada et al., 1991; Richards et al., 2000), decreases serum triglyceride levels in hyperlipidemic rats (Han et al., 1998), and directly affects the peroxisomal β-oxidation pathway in mouse hepatocytes (Sakuma et al., 1993; Suga et al., 1996; Waxman, 1996).

Although some research has been conducted in the area of DHEA regulation and lipid metabolism in rats and mice, there is little information available on the effect of DHEA on the activities of lipid metabolic hormones and parameters in poultry, especially in broiler chickens. Currently, the production of broiler chickens with excessive body fat is a significant economic problem in the poultry industry. Several factors, such as nutrient availability and genetics, contribute to the tendency for broilers to accumulate excess body fat (Weltzien, 2002). The accumulation of abdominal fat in chickens results in increased poultry feed cost and decreased final product quality (Elkin, 1998; Assaf et al., 2004). Although hormone addition to

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animal feeds is currently not allowed in Europe, the administration of DHEA to avian species may be a practical way to modify carcass lipid deposition and metabolism where regulations allow; therefore, investigation in this area is warranted.

Therefore, a study was carried out to examine the effects of DHEA on lipid metabolic hormones and parameters in male and female broiler chicks.

MATERIALS AND METHODS

Animals and housing

One-hundred and eighty (180) commercial broiler chickens (1-day old, 38 g, Nanjing Jiachang commercial broiler chicken company, China) were housed from days 1 to 42 during the starter- (Days 1 to 21) and finisher- (Days 21 to 42) phases of the growth cycle. Starter-phase broiler chickens were housed in lighted coops (24-h day), at constant temperature (20 ± 3°C) and humidity (50 ± 3%); finisher-phase broilers were kept on the ground under natural lighting. Nutrient levels within each diet (Table 1) were based on NRC (1994) recommendations. Animal care and use were approved by the Institutional Animal Care and Use Committee of Nanjing Agricultural University.

Treatment and diets

All birds were randomly divided into three equal groups (60 birds per group), and each group was assigned to 1 of 3 treatments. Birds in each group were kept in three pens (20 broilers per pen). All birds were offered the same basal diet with added DHEA (Changzhou Jiaerke Pharmaceuticals Group Corp) at levels of 0 (control), 5 and 20 mg/kg. All birds had free access to feed and drinking water. At the conclusion of the experiment, the birds were fasted for 12 h prior to slaughter. The birds were then slaughtered, the gender was identified, and blood samples were taken and retained for subsequent analysis.

Table 1. Ingredients and nutrient composition of diets[1]

Ingredient (%)	Starter (Days to 21)	Finishing (Days 21 to 42)
Corn	52.6	57.4
Soybean meal	31.1	27
Wheat bran	2.0	4
Fish meal[2]	6.0	3
Rapeseed oil[3]	5.0	5
NaCl	0.3	0.3
Calcium phosphate	1.0	1.5
Limestone	1.2	1.2
DL-Methionine	0.3	0.1
Vitamin-mineral premix[4]	0.5	0.5
Nutrition composition		
Calculated		
ME (Mcal/kg)	3.10	3.14
CP (%)	22.52	19.74
Lys (%)	1.19	1.08
Met + cysteine (%)	0.93	0.71
Ca (%)	1.00	0.90
Total P (%)	0.80	0.76
Available P (%)	0.47	0.39
Analyzed		
CP (%)	22.44	19.66
Ca (%)	1.03	0.94
Total P (%)	0.84	0.79

[1]nutrient level of the diets was based on NRC recommendations

[2]crude protein content is 62.5% and ME is 2.79 Mcal/kg

[3]metabolizable energy is 8.8 Mcal/kg

[4]supplied per kilogram of diet: vitamin A (retinyl acetate), 1 500 IU; cholecalciferol, 200 IU; vitamin E (DL-α-tocopheryl acetate), 10 IU; riboflavin, 3.5 mg; pantothenic acid, 10 mg; niacin, 30 mg; cobalamin, 10 μg; choline chloride, 1 000 mg; biotin, 0.15 mg; folic acid, 0.5 mg; thiamine, 1.5 mg; pyridoxine, 3.0 mg; Fe, 80 mg; Zn, 40 mg; Mn, 60 mg; I, 0.18 mg; Cu, 8 mg; Se, 0.15 mg

Preparation of samples and lipid metabolic hormone detection

Blood samples were allowed to clot at 4°C and centrifuged at 1 520 × g for 20 min before harvesting the serum. Serum samples were stored at –20°C until assayed. Concentrations of plasma triiodothyronine (T_3), thyroxine (T_4), serum free triiodothyronine (FT_3), serum free thyroxine (FT_4), glucagon (GLU), and leptin (LEP) were determined using the

commercial RIA kit (Beifang Biotechnology Corp., Beijing, P.R. China).

Lipid parameters assay

The aforementioned blood samples were also used to detect triglycerides (TG), total serum cholesterol (TC), high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C), non-esterified fatty acids (NEFA), and lipoprotein lipase (LPL), using commercial kits (Nanjing Jianchen Biotechnology Institution, China).

Statistical analysis

The results were expressed as the mean ± SE, and differences were considered significant when $P < 0.05$, as tested by one-way analysis of variance (ANOVA), using the GLM procedure with SAS software 9.0 (SAS Institute Inc., 2006).

RESULTS

Growth performance

There was a marked response to DHEA supplementation in terms of weight gain and the feed conversion ratio. Feed intake was not affected by DHEA treatment (Table 2).

Lipid metabolic parameters

The levels of TC and HDL-C were significantly ($P < 0.05$) lower in female broiler chickens fed 5 or 20 mg DHEA/kg than in the control animals, while only the male broiler chickens fed 20 mg DHEA/kg produced a similar result. In contrast, a marked ($P < 0.05$) increase in LPL activity was found in both male and female birds treated with 20 mg DHEA/kg. Female broiler chickens fed 5 mg DHEA/kg, as well as male birds fed 20 mg DHEA/kg, had markedly ($P < 0.05$) lower levels of TG compared to the control birds. During the entire experimental period, the addition of 5 or 20 mg DHEA/kg resulted in a significant ($P < 0.05$) decrease in the level of NEFA in female birds, whereas the addition of 5 mg of DHEA/kg significantly ($P < 0.05$) increased the concentration of NEFA in male birds. No significant differences were observed in LDL-C among birds fed 0, 5 or 20 mg DHEA/kg (Table 3).

Lipid metabolic hormones

In the present study, adding 20 mg DHEA/kg significantly ($P < 0.05$) decreased serum concentrations of T_4, FT_3, FT_4, but significantly ($P < 0.05$) increased the serum level of GLU and LEP in male broiler chickens. No significant differences were observed in T_3 in males fed 0, 5 or 20 mg DHEA/kg. However, female broiler chickens fed 5 or 20 mg DHEA/kg had significantly ($P < 0.05$) lower levels of FT_3, FT_4, and higher concentrations of LEP as

Table 2. Effect of DHEA on growth performance of broilers[1]

	Male broiler chickens DHEA (mg/kg)			Female broiler chickens DHEA (mg/kg)		
	0	5	20	0	5	20
Weight gain (g/bird/day)	48.63 ± 1.39	44.80 ± 1.21*	44.73 ± 1.29*	44.34 ± 1.47	38.93 ± 0.89*	37.48 ± 1.30*
Feed intake (g/bird/day)	173.40 ± 1.2	171.44 ± 0.92	176.15 ± 1.08	168.67 ± 1.15	166.99 ± 1.34	167.00 ± 0.83
Feed:Gain (g:g)	3.52 ± 0.05	3.83 ± 0.06*	3.94 ± 0.04*	3.80 ± 0.06	4.29 ± 0.03*	4.46 ± 0.02*

Data are means ± SE

[1]data in each group represented mean values of three replicates, one replicate indicating the average of 20 chickens in one cage

*means significant difference $P < 0.05$

Table 3. Effects of dehydroepiandrosterone (DHEA) on serum concentrations of triglycerides (TG), total serum cholesterol (TC), high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C), non-esterified fatty acid (NEFA) and lipoprotein lipase (LPL) in broilers[1]

	Male broiler chickens DHEA (mg/kg)			Female broiler chickens DHEA (mg/kg)		
	0	5	20	0	5	20
TG (mM)	0.43 ± 0.02	0.42 ± 0.02	0.38 ± 0.01*	0.47 ± 0.04	0.39 ± 0.02*	0.43 ± 0.01
TC (mM)	3.99 ± 0.12	3.89 ± 0.12	3.68 ± 0.07*	3.73 ± 0.11	3.24 ± 0.11*	3.37 ± 0.11*
HDL-C (mM)	1.99 ± 0.08	1.86 ± 0.06	1.79 ± 0.04*	2.01 ± 0.11	1.56 ± 0.05*	1.60 ± 0.06*
LDL-C (mM)	1.76 ± 0.08	1.84 ± 0.08	1.72 ± 0.06	1.51 ± 0.09	1.50 ± 0.09	1.69 ± 0.08
NEFA (μM)	704.92 ± 27.58	810.30 ± 36.75*	750.08 ± 35.59	792.05 ± 54.63	677.55 ± 36.46*	633.82 ± 23.09*
LPL (U/ml)	1.24 ± 0.07	1.44 ± 0.10	1.64 ± 0.08*	1.30 ± 0.09	1.57 ± 0.14	1.58 ± 0.09*

Data are means ± SE

*means significant difference $P < 0.05$

[1]means represent 12 chickens at the age of 42 days (four chickens from each of three pens) per treatment

Table 4. Effects of dehydroepiandrosterone (DHEA) on serum concentrations of thyroxine (T_4), triiodothyronine (T_3), serum free triiodothyronine (FT_3), serum free thyroxine (FT_4), Leptin (LEP), and glucogan (GLU) in broilers[1]

	Male broiler chickens DHEA (mg/kg)			Female broiler chickens DHEA (mg/kg)		
	0	5	20	0	5	20
T_3 (ng/ml)	1.71 ± 0.13	1.74 ± 0.08	1.49 ± 0.09	1.53 ± 0.10	1.34 ± 0.12	1.41 ± 0.16
T_4 (ng/ml)	49.00 ± 2.78	46.05 ± 1.50	38.97 ± 2.28*	46.70 ± 1.86	43.52 ± 2.55	46.74 ± 2.76
FT_3 (fmol/ml)	3.14 ± 0.32	2.29 ± 0.20*	1.75 ± 0.20*	2.50 ± 2.08	2.08 ± 0.16	1.68 ± 0.22*
FT_4 (fmol/ml)	8.04 ± 0.56	7.66 ± 0.40	6.09 ± 0.91*	8.67 ± 0.70	7.127 ± 0.47*	6.60 ± 0.34*
LEP (ng/ml)	0.23 ± 0.05	0.24 ± 0.05	0.44 ± 0.07*	0.13 ± 0.013	0.55 ± 0.03*	0.45 ± 0.09*
GLU (pg/ml)	272.35 ± 9.10	282.44 ± 15.65	308.18 ± 9.32*	303.23 ± 10.82	319.19 ± 12.48	315.81 ± 12.76

Data are means ± SE

*means significant difference $P < 0.05$

[1]means represent 12 chickens at the age of 42 days (four chickens from each of three pens) per treatment

compared to the control birds, but there was no significant effect on the other three metabolic hormones (T_3, T_4 and GLU; Table 4).

DISCUSSION

The current study clearly demonstrated the effect of DHEA on growth performance, lipid metabolic hormones and parameters in broiler chickens. To our knowledge, this is the first report relative to the effect of 5 or 20 mg/kg of natural, orally-administrated DHEA on subsequent hormone levels, growth parameters, and lipid metabolism in poultry. These results showed that supplementation of DHEA decreased body weight gain in the absence of any reduction in feed intake. Furthermore, in our early research, we found that chickens fed DHEA exhibited decreased body weight and abdominal fat weight with no significant difference in breast and leg muscle weight (Tang et al., 2007). This was in accordance with previous reports that found that DHEA administration reduced abdominal fat accumulation in both rats (Cleary and Zisk, 1986) and elderly women and men (Villareal and Holloszy, 2004). Few studies have reported the effects of DHEA on poultry growth performance or carcass composition. Therefore, it is important to

indicate that DHEA may decrease overall abdominal fat content without influencing feed intake or carcass composition. A possible explanation for these results is as follows: DHEA is an activator of peroxisome proliferator activated receptor α (PPARα) (Poynter and Daynes, 1998). Activation of PPARα induces transcriptional up-regulation of fatty acid transport proteins that facilitate fatty acid entry into cells and the enzymes involved in the β-oxidation of fatty acids, resulting in the decreased expression of fatty acid synthesis (Schoonjans et al., 1996; Tang et al., 2007).

The levels of NEFA and TG in serum are important indicators of fat metabolism (Zhan et al., 2006). A higher concentration of NEFA in serum may enhance muscle fatty acid deposition (Xu et al., 2003). Generally, TG are broken down into fatty acids and glycerol as a source of metabolic energy (Sato et al., 2006). In this experiment, an increase in serum NEFA was observed with 5 mg DHEA/kg treatment in male broiler chickens and a decreased TG level was evident in both sexes fed 5 or 20 mg DHEA/kg. Based on the above data, it is reasonable to speculate that the addition of DHEA leads to higher concentration of NEFA in serum by accelerating the hydrolysis of TG to glycerol and fatty acids, so that energy storage via fat deposition was reduced (Leighton and Tagliaferro, 1987). Another interesting observation was the decrease in total and HDL cholesterol levels following DHEA administration. Reductions in total and HDL cholesterol levels have been reported by some authors following human treatment with DHEA (Nestler et al., 1988; Arlt et al., 1999; Barnhart et al., 1999). Although the mechanism by which this occurs is not fully understood, these reductions are thought to be mediated by the effect of androgens in increasing hepatic lipase activity, thus impairing hepatic cholesterol formation (Tan et al., 1998). The same results were demonstrated in female broiler chickens supplemented with 5 or 20 mg DHEA/kg. Therefore whether these reductions are also mediated by the effects of estrogen, is still unclear. In contrast to the results for TG, TC and HDL-C, 20 mg DHEA/kg markedly increased the serum LPL level, which is consistent with results described using the rat as the model (Rebuffe-Scrive, 1987; Deshaies et al., 1994; Maurieqe et al., 2003). As activity increases, LPL accelerates the hydrolysis of circulating TG-rich lipoproteins, which has been suggested to play a major role in regulating the deposition of fat in the animal's body and, thus, reducing the deposition of fatty acids in adipose tissue (Maurieqe et al., 2003). Unfortunately, the mechanisms by which DHEA influences the LPL activity are still not clearly understood. The results of this study imply that DHEA reduces the deposition of broiler abdominal fat by decreasing *de novo* fatty acid synthesis and fat deposition.

Of those thyroid hormones that we studied, no reports were available regarding the effect of DHEA on circulating thyroid hormone levels in poultry. In the present study, metabolic hormonal changes, as evidenced by the circulating levels of T_3, T_4, FT_3 and FT_4, were observed, whereas no significant differences were found between T_3 and T_4, except the T_4 level in the 20 mg DHEA/kg treated male broiler chickens. The results for T_3 and T_4 argue against a shift in the conversion of T_4 to T_3 (Caerw et al., 1997), but rather suggest a specific alteration in the metabolism of T_3 and T_4 following treatment with DHEA. However, the specific metabolic alteration is unknown. Furthermore, DHEA significantly decreased FT_3 and FT_4 in both male and female birds. As observed previously (Geris et al., 1999; Buyse et al., 2002), reduced circulating thyroid hormone levels lower the metabolic rate as a protective mechanism for the body's energy reserves. Therefore, these results are quite useful in that they indicate a similar effect of DHEA on FT_3 and FT_4 in broilers, whereby the homrone prevents the accumulation of excess abdominal fat.

In the present study, DHEA addition enhanced the concentrations of serum LEP and GLU, which is in agreement with previous work on GLU-stimulated enhanced LEP expression in broiler chicken adipose tissue (Ashwell et al., 1999). LEP 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 LEP promoter gene has not been cloned, chicken LEP is highly conserved and, therefore, is similar to the mammalian gene (Taouis et al., 2001). Taouis et al. (2001) reported that the impact of GLU on liver LEP may be attributed to an elevation in intracellular cyclic AMP (cAMP). However, the relationship between GLU and LEP in poultry, especially in serum levels, is still unclear. Taouis et al. (2001) also indicated that chicken LEP expression is regulated by nutritional status. From the results of the present study, it is possible that DHEA up-regulates the serum LEP level, and that the enhanced concentration of LEP might inhibit the accumulation of

fat in broilers, resulting in reduced abdominal fat deposition.

In conclusion, the administration of DHEA to poultry improved lipid metabolism by influencing metabolic hormones and their physiological parameters, while not adversely affecting growth performance or carcass composition.

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