

Regulation of Lipid Metabolism in Chicken by Feeding Orotic Acid

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ABSTRACT

This study was conducted to investigate the potential effects of different concentrations of dietary orotic acid (OA) on lipids metabolism in chicken meat.

Administration of 0.3% OA increased significantly ($P < 0.05$) body gain and improved feed efficiency by 10%. However, addition of OA to chicken diets resulted in a reduction of total cholesterol at all OA levels. Reduction was 25 to 41% in liver, 12 to 25% in both blood and breast meat, and 25 to 35% in leg meat. This reduction of total cholesterol in all tested tissues was accompanied by elevation of tissue total neutral lipid (NL). This elevation was between 4 to 16% in liver 8 to 24% in leg and 10 to 25% in breast meat. There was no concomitant change in tissue total phospholipid (PL), in all tested tissues.

INTRODUCTION

It is well recognized that diet composition influence lipid biosynthesis in the chicken. It has been also reported that hepatic lipogenesis is significantly reduced by feeding diets high in fat to the chicken (14, 25). Feeding a diet composed mainly of corn and soybean meal resulted in greater lipid accumulation in caged hens than those fed isocaloric and isonitrogenic products, wheat, or yeast, (12, 15).

Fatty infiltration of the liver can be induced by the administration of various chemicals. OA is one of those chemicals (10, 13). The development of fatty liver induced by OA administration unlike the one induced by a choline deficient diet does not seem to be accompanied by other serious pathological disturbances. Handschumcher, *et al.*, (10) reported that the addition of OA to the diets of young rats resulted in fatty liver formation and the effect of OA was not counteracted by lipotropic factors such as folic acid, cobalamine or choline. It was also noted that neither uracil nor thymine induced fatty livers in the rat.

Creasey *et al* (7), found that liver triacylglycerol has increased fifteen folds in rats

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ingesting 1% OA in their diet. The fatty liver produced in rats by OA administration appears to result from an inhibition of synthesis or release of hepatic very low density lipoprotein. This is supported by the observation of Windmueller (24) that very low density lipoprotein is important in the normal transport of triacylglycerol from the liver. Roheim, *et al.*, (20) illustrated that OA specifically depressed the formation of very low density lipoprotein without an over-all inhibition of protein synthesis. They also indicated that synthesis of the apoprotein portion of the lipoprotein was unaffected by OA feeding. They have suggested that one or more steps in lipoprotein formation subsequent to the synthesis of the protein may be inhibited by administration of OA. It was also noted that the incorporation of methionine into serum lipoprotein was decreased. This effect could be reversed by the addition of phosphatidylcholine (PC), (20). OA was found to inhibit liver cholesterologenesis in rats (5).

MATERIALS AND METHODS

Chicken and Diet:

Sixty-day old, «Hypro» chicken were wing-banded, equally divided into six groups and maintained in batteries at the poultry farm of Al-Fateh University Research Station. The chicks were subjected to standard environmental conditions. They were raised on diet and water was added ad libitum. The control group chicks were fed a laying hen starter diet, which was composed of: 17% crude protein, 4% ether extract, 5% crude fiber, 61% nitrogen free extract, 5% ash and 9% moisture. The other five groups were fed the same laying hen starter diet plus 0.1%, 0.3%, 0.5%, 0.7% or 0.9% W/W OA. After 8 weeks, feed consumption, body weight and feed efficiency were determined.

The birds were slaughtered, their livers, breasts, and legs were removed and weighed. The bone was removed and flesh meat was ground with a meat-grinder, homogenized in an Omni homogenizer for 2 minutes and stored in sealed polyethylene bags at -20°C. The samples were thawed at room temperature and total lipids were extracted according to Floch *et al.*, (8).

Cholesterol Determination:

Cholesterol was determined according to a modified colorimetric method of Bhandaru *et al.*, (4). Five to ten microliters of fat extract were placed in a microcentrifuge tube and one ml of uranyl acetate was added. The tubes were mixed thoroughly, left to settle for 10 min. The clear supernatant was transferred to a test tube. One ml of Ophthaldialdehyde reagent was added immediately and left for 10 min. Then one ml concentrated H₂SO₄ was added. The contents were mixed thoroughly in a Vortex mixer, cooled, and the absorbance was measured within 15 min. at 566 nm using model 25 spectrophotometer (Beckman Instrument). For each set of samples, appropriate blanks were measured.

Separation of phospholipids (PL), neutral lipids (NL) and free fatty acids (FFA):

PL were isolated first using a modified Hirsh and Ahrens, silicic acid chromatography column (11). NL and FFA were fractionated using the McCarthy and Duthie alkaline silicic acid column. (16).

Separation of phospholipids by thin layer chromatography:

The various PL, phosphatidylethanol amine (PE), Phosphatidyl serine (PS) Sphingomyelin (SPH) and PC were separated using a glass plate coated with a 0.5 mm layer of silica gel (H), (E-Merck, DARMSTAD) followed by activation for 2 hrs at 110°C. The plates were developed with a modified system of Skipski et al. (22), using chloroform: methanol: acetic acid: water (25: 15:2:1.5 V/V). The chromatograms were visualized under ultraviolet light after being sprayed with a solution of dichloroforecin in methanol: water (1:1 V/V), and exposed to ammonia vapor. The FA composition of individual PL and NL were determined by gas liquid chromatography (GLC) (Perkin Elmer 23420) using a glass column packed with 10% SE 30 or 100-120 mesh (Deatomic DMCS). The samples were transmethylated in the presence of the gel scraps and incubated for 14 hrs at 80-85°C in 6% (V/V) H₂ SO₄ in methanol. The samples were concentrated to 0.5 ml and 0.1-0.5 microliters, and were injected on to the GLC system. The peaks were identified by comparing their retention time with standards.

The same procedure was used for the analysis of the FFA except that a stainless steel column (200 cm × 4m) was used.

Fatty acids analysis by gas liquid chromatography:

Nitrogen was used as a carrier at a pressure of 35 PSI. The analysis was performed between 70-190°C column temperature with elevation of 16°C/min and detector temperature of 230°C.

Statistical analysis:

All statistical analyses were conducted using the split plot technique described by Steel and Torrie (23). Differences in body gain, feed conversion, total Lipids, total NL, total PL, total FFA, and Cholesterol concentrations were subjected to analysis of F-test.

Relationships between FA composition of different Lipid fractions (NL, PL and FFA) were examined by correlations coefficient described by Geoffrey, (9).

RESULTS AND DISCUSSION**Effect of orotic acid on****Body gain and feed conversion:**

The effect of OA administration on body gain and feed conversion in the chicken broilers is summarized in Fig. 1. The data indicate that the addition of the OA at all levels did not show significant mortality or abnormal symptoms in the living birds. The results also show that chickens on the low level of OA (0.3%) exhibited better feed conversion than chicken fed no OA ($p < 0.05$). This conversion was 0.40, 0.32 respectively. Also according to the data presented in Fig. 1, OA affected the body gain significantly at low levels of 0.3% OA.

The increase in body gain was 20% as compared to chickens consuming only the basal diet. However, the higher levels of OA, (0.5-0.9%) did not increase or decrease feed conversion or body gain significantly. Similar observations were reported by Ahmed (2) who tested different levels of OA (0.05-2%) on two breeds of laying hens

(High Sex Brown and High Sex White). The high levels of 1 and 2% had no effect on egg production. This may be due to the fact that OA works as a growth factor (19).

Effect of orotic acid on lipid biosynthesis:

The data obtained in Fig. 2 show that the total lipid content was affected by OA consumption. An elevation occurred in total tissue lipids. It was between 2-15% in liver tissue, 24-28% in legs tissue and 3-27% in breast tissue. This result is in agreement with Sabesin *et al.* (21) who reported that consumption of OA caused a fatty liver in rats. Present data show that the ratio of total lipid in white meat and dark meat is 1: 2. This has been used to explain the rancidity development in dark meat that occurs more readily than in white meat.

Effect of orotic acid on cholesterol biosynthesis:

The observed effects of OA on cholesterol biosynthesis are summarized in Table 1. The data indicate that all concentrations of OA caused a significant reduction in the cholesterol level in the different tested tissues. The average reduction in liver was 25-40%, in leg meat was 25-35%, and 12-25% in both breast meat and blood plasma. Similar effects of OA on cholesterol biosynthesis and serum cholesterol are reported in other studies (1, 2, 3, 5). The mechanism by which dietary OA depresses hepatic cholesterogenesis is not clear. In 1978, Ahmed *et al.*, (1) reported that OA affected the incorporation of acetate into cholesterol biosynthesis in rats. The prime focus for cholesterol biosynthesis is the conversion of hydroxymethyl glutaryl CoA (HMG-CoA) to mevalonate by HMG-CoA reductase (6, 14).

The reduction in tissue cholesterol content may be explained by the inhibition of synthesis or release of hepatic very low density lipoprotein. This is important in the normal transport of cholesterol and triacylglycerol from the liver to the different tissues. Also it might be explained by inhibition of endogenous cholesterol synthesis which is reflected in the transfer of hepatic cholesterol to the tissues (20, 24).

Effect of orotic acid on neutral lipids content:

The effect of OA on NL biosynthesis in tested tissues is indicated in Table 2. The results show a significant ($P < 0.05$) increase in the total NL. The increase was between 4-16% in liver tissue, and 6-40% in both dark and white meat. These results are in agreement with those reported by Ahmed (2) who found that consumption of OA at 0.05- 2% by two breeds of laying hens caused an accumulation of 42% in egg NL's. Table 3 shows the FA distribution in NL. It is clear that OA caused an elevation of oleic and linoleic acids content, and caused a reduction in palmitic and stearic acids content in all tested tissues. According to present data the reduction in palmitic and stearic acids was equal to the increase in oleic and linoleic acids.

Effect of orotic acid on free fatty acid

Tables 2 and 4 show the FFA's composition and distribution of three tested tissues of the eight weeks old chickens. Rations containing OA at levels of 0.9 and 0.7% caused a significant elevation of total FFA ($P < 0.05$) than the corresponding unsupplemented ration. These elevations were 8-12% in liver, 8-24% in legs, and 10-25% in breast meat at the levels of 0.5, 0.7 and 0.9% OA, respectively. The supplementation of the diets with 0.3% OA or less showed no significant differences in the amount of

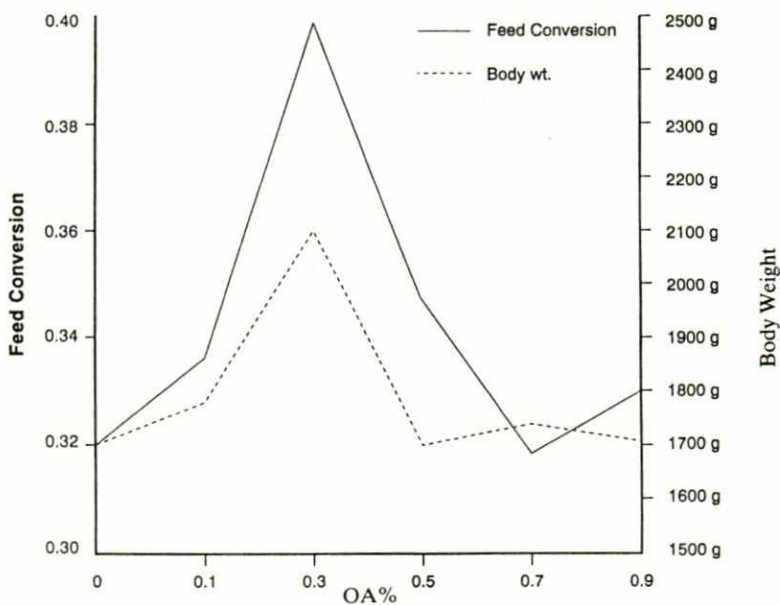


Fig. 1: Effect of dietary orotic acid on feed conversion and body weight of Hypro chicken.

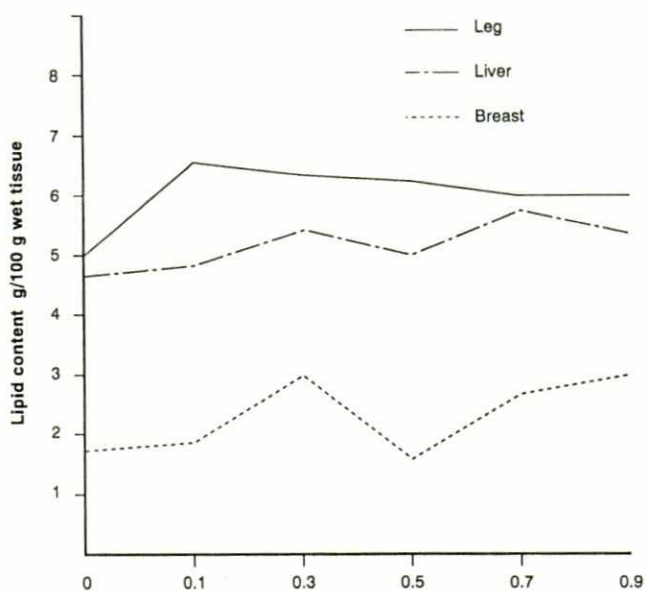


Fig. 2: Effect of dietary orotic acid on lipid content of different tissues of Hypro chicken.

Table 1 — Effect of dietary orotic acid on cholesterol content of different tissues of Hypro chicken.

Cholesterol mg/ 100g. wet tissue					
	Plasma	Breast	Legs	Liver	OA%
	42.66 ± 3.8	90.0 ± 1.73	186.64 ± 6.50	219.8 ± 11.18	0
	37.5 ± 2.75	89.66 ± 2.84	35.0 ± 3.70*	140.0 ± 5.20*	0.1
	36.5 ± 2.36	72.66 ± 75	138.64 ± 5.90*	160.6 ± 10.90*	0.3
	31.0 ± 3.2*	68.33 ± 5.17	136.6 ± 6.66*	179.0 ± 7.60*	0.5
	31.66 ± 1.2 *	73.66 ± 2.42	138.6 ± 5.92*	170.0 ± 6.90*	0.7
	33.0 ± 2.08*	70.0 ± 2.88	142.0 ± 4.16*	177.33 ± 1.76*	0.9

Each value is the mean ± SE of three replicates

* Means within the same column are significantly different at 5% level.

Table 2 — Effect of dietary orotic acid on neutral lipids, phospholipids⁽¹⁾, and free fatty acids content of different tissue of Hypro chickens.

	Breast			Leg			Liver			OA%
	FFA	PL	NL	FFA	PL	NL	FFA	PL	NL	
20.00	0.80	1.40	42.00	1.90	3.90	186.60	1.60	3.20	0	
20.60	0.77	1.56	42.60	2.20	4.13	184.60	1.60	3.30	0.1	
22.00	0.80	1.93	45.30	2.20	4.37	202.60	1.80	3.60	0.3	
23.00	0.90	2.04	48.00	2.00	4.42	205.00	1.70	3.70	0.5	
24.30	0.80	2.05	54.60	1.90	4.46	209.30	1.70	3.70	0.7	
27.00	0.90	2.10	56.00	2.00	4.61	208.00	1.70	3.90	0.9	

Each value is the mean ± SE of three replicates.

(1) 1 g/ 100 gm on wet base (2) mg/ 100gm on wet base

total FFA among the control and experimental chickens. The supplementation of the diet with 0.5-0.9% OA increased the relative concentration of total FFA to 25% in both leg and breast meat which may be considered a disadvantage.

When the FFA fraction was identified by GLC, the results showed an elevation of oleic, and linoleic acids by 7-25% at 0.5, 0.7 and 0.9% OA levels almost in all tested tissues. At the same time 8-20% reduction occurred in palmitic acid in the liver tissue, and 7-15% reduction in both dark and light meat. According to data presented in Table 4, one expects that the consumption of OA by chicken may cause activation of unsaturated fatty acids (UFA) biosynthesis, and a deactivation of the biosynthesis of saturated fatty acids (SFA).

Effect of orotic acid on total phospholipids

Table 2 presents the Total PL in different tissues of experimental and control chickens. It was determined by weighing the total PL which was fractionated by silicic acid chromatography column (see experimental procedure). The supplementation of OA at any level to the chicken broilers for eight weeks caused no change in PL content in all tested tissues. The results are in excellent agreement with those reported by Ahmed (2) who noted that the variations in dietary OA consumed by two breeds of laying hens had no apparent affect on total egg PL in either breeds.

Four classes of PL's were identified from the treated and control chicken tissues. These were PC, PE, PS, and SPH which is the predominant PL of muscle and liver tissues. All liberated FFA of PL fractions were converted to methyl esters and determined by GLC. Table 5. presents the FFA obtained from liver PL. Table 6 presents the FFA obtained from breast meat PL, and Table 7 presents, the FFA obtained from leg meat PL. Both tables show that the consumption of OA caused an elevation of UFA content especially oleic and linoleic acids.

In general, PL fractions contained mainly oleic, linoleic, palmitic and stearic acids. In 1965, Peng (18) reported that the SFA especially palmitic, stearic, tricosanoic and lignoceric acids were found in the α - position. While the unsaturated oleic, linoleic and arachidonic acids are found in the B-position.

Since the distribution pattern of FA in PL was the same from either sources. The similarity in FA composition of all PL fractions, suggest either direct deposition or little modification due to biosynthesis in the liver tissue (14, 17).

In conclusion the results indicate that the synthesis of cholesterol in chicken can be reduced by adding a small amount of OA to chicken ration. This can be used to produce chicken meat with a lower level of cholesterol since providing chicken meat with a lower cholesterol content can be of great value. The findings also indicate that adding a proper amount of OA (0.3%) in the chicken diet can improve the rate of growth and feed conversion. More studies are needed to investigate the mechanisms of action of OA in chicken metabolism.

Table 3— Effect of dietary orotic acid on the fatty acid composition of neutral lipid of different tissues of Hypro chickens (Area %)

Breast				Leg				Liver				OA%
18:1 18:2	18:0	16:0	14:0	18:1 18:2	18:0	16:0	14:0	18:1 18:2	18:0	16:0	14:0	
63.50	10.80	25.00	0.67	62.30	10.80	23.60	1.17	68.20	10.60	19.60	1.50	00
61.70	9.40	24.00	1.16	61.70	10.70	27.00	1.18	63.70	13.10	21.40	1.80	0.1
66.30	9.30	23.40	0.97	64.10	10.80	23.80	1.17	61.70	11.70	25.50	1.00	0.3
64.60	9.30	23.90	0.80	64.50	9.40	25.40	0.90	65.90	8.40	24.20	1.40	0.5
67.50	8.60	23.00	0.87	67.50	10.44	21.50	0.60	68.40	9.60	20.17	1.80	0.7
66.10	7.20	23.00	0.86	64.70	9.56	24.60	1.00	68.40	8.90	20.50	2.00	0.9

Each value is the mean of three replicates.

Table 4— Effect of dietary orotic acid on total free fatty acid obtained from different tissues of Hypro chickens (Area %).

Fatty acids																Orotic acid		
18:1		18:0			16:0			14:0			12:0		10:0		(%)			
(3)	(2)	(1)	(3)	(2)	(1)	(3)	(2)	(1)	(3)	(2)	(1)	(3)	(2)	(1)	(3)	(2)	(1)	
25.8	26.5	22.2	28.1	26.5	37.7	40.7	40.9	34.2	2.5	3.1	1.7	1.7	1.9	1.1	1.9	0.9	3.0	0.0
27.4	26.5	18.2	27.4	26.5	38.4	39.7	40.9	37.9	2.5	3.1	1.8	1.7	1.9	1.1	2.1	0.9	2.6	0.1
27.6	26.5	23.0	27.6	28.5	37.7	38.5	40.7	30.3	2.2	2.2	2.6	2.0	1.9	2.1	2.4	1.4	3.0	0.3
28.8	30.1	25.2	27.6	26.1	37.8	38.7	38.6	35.2	2.5	2.3	2.4	1.5	2.1	2.1	2.0	1.4	3.1	0.5
29.6	30.3	26.2	29.6	27.0	33.6	36.7	37.2	26.9	2.0	2.4	3.4	1.9	2.2	3.1	2.0	0.9	4.4	0.7
30.4	33.1	28.9	25.4	26.4	35.2	37.6	37.2	27.1	3.8	2.6	3.3	2.0	1.9	2.8	2.0	1.1	4.5	0.9

(1) Liver tissue

(2) leg tissue

(3) breast tissue

Each value is the mean of three replicates.

Table 5—Effect of dietary orotic acid on fatty acids composition of phospholipids of liver tissue of Hypro chickens (Area %).

Sphingomyeline				Phosphatidyl serine				Phosphatidyl choline				Phosphatidyl ethanolamine				OA%
18:1	18:0	16:0	14:0	18:1	18:0	16:0	14:0	18:1	18:0	16:0	14:0	18:1	18:0	16:0	14:0	
18:2				18:2				18:2				18:2				
46.6	14.3	34.0	5.0	41.3	18.7	37.4	2.6	40.8	20.9	36.4	1.7	41.5	19.5	36.16	2.3	0
45.8	14.9	34.8	4.5	40.7	18.3	37.6	2.3	39.9	20.7	37.0	2.2	41.3	19.4	36.2	2.7	0.1
46.5	13.8	35.1	4.4	47.3	21.7	28.7	1.9	41.8	20.5	35.1	2.5	41.2	23.2	32.8	3.5	0.3
45.8	14.5	32.3	3.4	43.7	23.0	30.8	1.7	40.0	23.1	33.3	3.1	45.6	21.0	29.3	3.9	0.5
46.1	14.0	34.8	5.0	42.0	16.5	39.6	1.7	42.4	21.4	33.0	3.1	41.9	23.9	31.2	2.9	0.7
45.4	15.6	33.7	5.3	39.7	18.0	40.8	1.6	43.8	19.9	32.9	3.3	41.2	24.8	30.4	3.5	0.9

Each value is the mean of three replicates

Table 6—Effect of dietary orotic acid on fatty acid composition of phospholipids of breast tissue of Hypro chickens (Area %)

Sphingomyeline				Phosphatidyl serine				Phosphatidyl choline				Phosphatidyl ethanolamine				OA%
18:1	18:0	16:0	14:0	18:1	18:0	16:0	14:0	18:1	18:0	16:0	14:0	18:1	18:0	16:0	14:0	
18:2				18:2				18:2				18:2				
30.0	20.7	43.3	5.9	27.3	12.3	54.7	4.9	37.2	22.0	37.3	3.3	27.9	10.4	52.8	8.8	00
29.9	19.7	44.5	5.7	31.9	15.1	48.6	4.4	37.8	21.2	38.1	3.0	26.3	11.6	55.0	6.9	0.1
32.2	19.5	40.4	7.2	29.6	14.8	50.5	5.0	39.4	16.9	39.2	4.0	28.7	12.6	50.9	7.7	0.3
32.5	18.2	42.4	6.8	30.6	14.3	49.1	5.0	39.4	20.9	34.1	4.7	30.8	14.2	49.8	7.3	0.5
32.5	18.0	42.2	7.3	26.0	17.0	51.9	4.4	41.6	20.9	33.2	4.4	33.8	16.9	47.3	7.0	0.7
34.2	17.9	40.6	7.1	31.8	15.0	48.6	4.5	42.6	19.4	32.7	4.5	35.6	19.8	38.0	6.4	0.9

Each value is the mean of three replicates.

Table 7 — Effect of dietary orotic acid on fatty acid composition of phospholipids of leg tissue of Hypro chickens (Area %).

Sphingomyeline			Phosphatidyl serine			Phosphatidyl choline			Phosphatidyl ethanolamine			OA%
18:1 18:2	18:0	16:0	18:1 18:2	18:0	16:0	18:1 18:2	18:0	16:0	18:1 18:2	18:0	16:0	
43.1	15.1	40.7	36.9	17.0	45.9	46.2	25.8	28.0	38.0	27.4	34.0	00
42.9	17.9	39.1	40.8	16.3	42.8	45.8	24.2	30.0	35.7	25.6	38.6	0.1
42.5	17.1	40.4	43.0	14.1	41.9	43.7	21.7	34.5	41.1	23.0	35.0	0.3
44.2	16.1	39.7	38.9	16.4	44.6	45.2	25.6	29.2	38.9	22.7	38.0	0.5
44.3	20.0	34.8	40.7	15.3	43.9	45.7	27.1	25.8	41.9	20.6	37.5	0.7
40.8	17.9	40.4	39.9	16.6	43.5	46.2	23.8	29.9	41.2	22.8	36.0	0.9

Each value is the mean of three replicates.

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تنظيم أفضة الدهون في الدجاج بتغذيتها حامض الأوروتيك

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المستخلص

كان الغرض من إجراء هذا البحث هو دراسة مدى تأثير التركيزات المختلفة لحامض الأوروتيك المقدم مخلوطاً بالعلف على تمثيل الدهون المختلفة في دجاج اللحم. وجد أن تركيز حامض الأوروتيك بنسبة 0.3% أدى الى زيادة هامة ($P < 0.05$) في وزن الدجاج الحي، كما أن الكفاءة التحويلية للعلف ازدادت بنسبة 10%.

كذلك كان استهلاك حامض الأوروتيك من قبل دجاج اللحم سبباً في انخفاض مستوى الكولسترول الكلي على جميع تركيبات حامض الأوروتيك المقدم، ويتراوح هذا الانخفاض بين 25-41% في كبد الدجاج، 12-25% في لحم الصدر والدم، 25-35% في لحم الأرجل. ولقد قوبل هذا الانخفاض في الكولسترول بارتفاع في الدهون المتعادلة الكلية بنسبة 4-16% في كبد الدجاج، و 8-24% في لحم الأرجل و 10-25% في لحم الصدر. ولم تسجل أي تغيرات على الدهون المفسفرة بسبب تناول حامض الأوروتيك.