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BIOCHEMICAL EFFECTS OF CHOLINE AND CARNITINE ON LIPID PROFILE OF LAYING HENS

By

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ABSTRACT

The study was conducted with sixty 36 weeks old Lohman brown hens. According to the type of lipotropic factors supplemented to the basal ration the birds were divided into three main groups, each one consisting of 20 laying hens placed in separated rooms and classified as follows:

Group I: (control group), comprised 20 laying hens, they were fed on a basal diet only, used as control for all experimental groups.

Group II: (Choline supplemented group) including 20 laying hens, they were fed on a basal diet supplemented with Choline, where Choline was added as double amount to the basal ration (1500 mg/kg ration).

Group III: (L-Carnitine supplemented group) consisted of 20 laying hens, they were fed on a basal diet supplemented with L-carnitine, where L-carnitine was added to the basal ration at the concentration of (500 mg/kg).

Random blood samples were collected six times, from each group, at biweekly intervals, from the onset of lipotropic factors supplemented to the basal ration.

Blood samples for serum separation were obtained from the wing vien from all groups (control and experimental groups).

We can be concluded that there was mostly, an increase in all serum lipid fractions and some lipoprotein profiles with decrease in serum NEFA and increase in serum lipase activity during supplementation of laying hens with choline, and L-carnitine. Which did increase all serum lipid and lipoprotein profiles except serum NEFA during egg production period.

It can therefore, be argued that they are potential agents for reducing the incidence of metabolic disease like sudden death syndrome in laying chickens obtain a maximum rate of egg production, treatment and prevention of fatty liver.

INTRODUCTION

Lipid metabolism in avian species has attracted the attention of biologists to elucidate biochemical mechanisms and to increase economic

productivity of domestic birds. The liver is the major site of the lipid synthesis. Lipoproteins serve to transport these lipids to visceral and subcutaneous adipose tissue or other extrahepatic tissues. In the sexually active female, estrogen stimulates the hepatic production of specialized lipoproteins which blood transports to the ovary where they are taken up and deposited in the ova as the yolk of the avian egg (Garlich, 1979).

Fatty liver syndrome or fatty liver hemorrhagic syndrome (FLHS) is a metabolic disorder of laying hens in which lipid accumulation in the hepatic parenchyma is accompanied by marked hemorrhage (Butler, 1976). This disease generally results in decreased egg production and increased mortality, and causes considerable economic loss to egg producers. It has been well known that the reduction in FLHS is associated with reduction in liver fat.

Choline is a major lipotropic factor in both young growing birds and mammals, has been investigated to some extent with respect to liver lipid accumulation in mature avian females (Jensen, 1979).

On the other hand, L-carnitine, a betaine derivative of beta-hydroxybutyrate, is found in virtually all cells of higher animals and also in some microorganisms and plants. It acts as carrier of activated acyl groups, L-carnitine functions as a buffer for acetyl groups which may be present in excess in different tissues during ketosis and hypoxic muscular activity for protection of membrane structures, stabilizing of a physiologic CoA-SH/acetyl-CoA ratio and reduction of lactate production (Zeyner and Harmeyer, 1999).

Moreover, it has been shown that L-carnitine has a marked hypo-cholesterolemic effect when used in conjunction with lipid-rich diets (Diaz *et al.*, 2000).

Accordingly, the objective of the present study was to investigate the effects of supplementary of choline and L-carnitine on serum lipids and lipoproteins in laying hens.

MATERIALS AND METHODS

Materials:

Animals:

Sixty Lohman brown hens, 36 weeks old, were used in the experimental investigation of this study. They were kept and housed in separated rooms at a constant environmental and nutritional condition throughout the period of the experiment. The birds were grown up on a formulated balanced ration closely meet the recommendation of National Research Council (1994). Experimental diets and water were provided for

ad libitum consumption and the birds were exposed to 16 hours of light per day.

Experimental Design:

According to the type of lipotropic factors supplemented to the basal ration birds were divided into three main groups, each one consisting of 20 laying hens placed in separated rooms and classified as follows:

Group I: (Control group), comprised 20 laying hens used as control for all experimental groups.

Group II: (Choline supplemented group) where Choline chloride 50% dry supplements was added as double amount to the basal ration (1500 mg/kg ration) according to National Research Council (1994). Each Kilogram of choline chloride 50% dry contains minimum 500.00 gm of choline chloride (equivalent to minimum 433.95 gm of choline) in addition to corn cob and rice husk 50%. Choline chloride 50% dry (feed grade on cereal carrier) manufactured in Tachnical Collaboration with DuCoa, U.S.A. by VAM Organic Chemicals LTD.

Group III: (L-carnitine suppleneted group) consisted of 20 laying hens, where L-Carnitine was added to the basal ration at the concentration of (500 mg/kg ration) according to Buyse *et al.* (2001). It is manufactured by Arab Co. For Pharmaceuticals & Medical Plants-MEPACO-Egypt.

Blood samples:

Random blood samples were collected six times, from each group, at biweekly intervals, from the onset of lipotropic factors added to the rations. From the wing vein, in clean dry tubes, then allowed to coagulate at room temperature, then centrifuged at 3000 r.p.m. for 10 minutes. The clear serum was separated and used freshly for quantitative determination of:

1- Total lipids (Zollner and Kirsch, 1962).
2- Total cholesterol (Meiattini *et al.*, 1978).
3- Triacylglycerols (Bucolo and David, 1973).
4- Phospholipids (Zilversmit and Davis, 1950).
5- Non esterified fatty acids (NEFA) (Duncombe, 1964).
6- High-density lipoproteins cholesterol (HDL-Cholesterol) (Grove, 1979).
7- Low-density lipoproteins cholesterol (LDL-Cholesterol) (Friedewald *et al.*, 1972).
8- Very low-density lipoproteins cholesterol (VDL-Cholesterol) (Bauer, 1982).
9- Lipoprotein lipase (Neumann *et al.*, 1984).

RESULTS

A very highly significant increase in serum total lipids concentration was observed in both groups during the first two weeks of the experiment. This increase became non-significant after 6 and 10 weeks in choline and after 4 week in L-carnitine and highly significant after 12 weeks in both. However, there is a non significant decrease after 4 weeks, this decrease became very highly significant after 8 weeks of dietary choline supplementation and a very highly significant decrease in serum total lipids concentration after 6 weeks of the experiment. This decrease became non-significant after 8 weeks and significant after 10 weeks of carnitine supplementation.

Serum TC concentration in the choline-supplemented group was very highly significant increase after 2, 6, 8 and 12 weeks of the experiment. However, after 4 weeks the value showed a very highly significant decrease. This decrease became highly significant after 10 weeks. While, in the L-carnitine group was very highly significant increase after 2 and 8 weeks. This increase became non-significant after 4 and 12 weeks. However, it caused a highly significant decrease after 6 and 10 weeks of the experiment.

Serum Tg in the choline-supplemented group was very highly significant increase after 2, 6, 10 and 12 weeks. This increase was not significant after 4 weeks, however, after 8 weeks the value of serum triacylglycerols concentration showed a very highly significant decrease.

Supplemental L-carnitine increased serum Tg concentration very highly significant during the first 2 weeks. However, there was a highly significant decrease after 4 weeks of the experiment. This decrease became very highly significant after 6, 10 and 12 weeks and non-significant after 8 weeks of supplementation.

Supplemental choline caused a very highly significant increase in serum PHL during the first 2 weeks of the experiment. This increase was not significant after 4 weeks. A significant decrease was observed after 8 weeks, however, a non significant decrease after 6, 10 and 12 weeks. Also, L-carnitine increased serum PHL very highly significant during the first 2 weeks of the experiment. This increase became non-significant after 4, 8 and 10 weeks. However, caused a non significant decrease after 6 weeks, this decrease became highly significant after 12 weeks of the experiment in comparison with the control group. Choline caused a significant decrease NEFA during the first 2 and 6 weeks. This decrease was very highly significant after 4, 8 and 10 weeks. Moreover, a non significant decrease was observed after 12 weeks. Wherever, a highly significant decrease was observed after 2, 6 and 10 weeks in L-carnitine supplemented group. This

decrease became significant after 4 weeks. However, a very highly significant decrease after 8 weeks, this decrease became non significant after 12 weeks in comparison with the control group.

A non-significant increase in serum HDLC was observed in choline after 2 and 12 weeks. This increase became significant after 4 and 8 weeks and very highly significant after 6 weeks. However, serum HDL-cholesterol level showed a non-significant increase after 12 weeks. While, in the L-carnitine group was significantly decrease during the first 2 weeks. This decrease became highly significant after 10 weeks and non-significant after 4, 8 and 12 weeks. However, a significant increase in serum HDL-cholesterol concentration after 6 week of the experiment than in the control.

Choline supplementation did not significantly affect the value of serum LDLC during the first 2 weeks whereas at the fourth week a non-significant increase. This increase became highly significant after 6 and 12 weeks and very highly significant after 8 and 10 weeks. While, in the L-carnitine group showed a significant decrease after 10 weeks of study. This decrease was very highly significant after 2, 6 and 12 weeks. However, a significant increase after 4 weeks. This increase was non-significant after 8 weeks of supplementation of the diet.

A very highly significant increase in serum VLDLC in the choline group was after 2, 6, 10 and 12 weeks. This increase became non-significant during the fourth week. However, a very highly significant decrease after 8 weeks. While, in the L-carnitine group was very highly significantly increase than in the control after 2 weeks. However, a highly significant decrease at fourth week, this decrease became very highly significant after 6, 10 and 12 weeks and non-significant after 8 weeks in comparison with the control group.

A highly significant increase in serum lipase activity in the choline group was observed after 2, 4 and 12 weeks. This increase became very highly significant 8 and 10 weeks of study. However, a significant decrease in serum lipase activity after 6 weeks. While, in the L-carnitine group showed a non-significant increase after 2, 10 and 12 weeks of the study. This increase became significant after 8 weeks. However, a non significant decrease after 4 weeks. This decrease became very highly significant after 6 weeks of supplementation in comparison with the control group.

DISCUSSION

The marked increase in serum total lipids concentration observed in laying hens fed on choline supplemented diet may be due primary to an increased ability of liver to synthesized triacylglycerols or secondary to an

increased concentration of serum free fatty acids and the hyperlipidemic effect of choline could be related to the decreased hepatic fat due to its lipotropic effect El-Ghannam *et al.* (1995) who stated that, liver fat was significantly reduced by the addition of choline to the diet. Also, An *et al.* (1997) suggested that, dietary safflower phospholipids might be a valuable ingredient to layers for reducing liver triglycerides and serum cholesterol without any adverse effects.

The marked decrease in serum total lipids after 8 weeks. These results are similar to those reported by Iwata *et al.* (1992) who demonstrated that the feeding of safflower phospholipids or crude safflower phospholipids in laying hens resulted in some desirable alterations, as the reduction of plasma and liver lipid levels, an increase in HDLC and the excretion of fecal neutral steroid. This decrease may be due to decrease level of serum Tg and VLDL observed after 8 weeks of the experiment. Furthermore, chicken adipocyte has a limited capacity to synthesize de Novo fatty acids and most of the fat deposited within the adipose tissue must be derived from the plasma. It would seem reasonable to suppose that the rate of fat deposition might be influenced by the concentration of available triglyceride in the plasma.

The increase in serum total lipids of laying hens supplemented with L-carnitine may be attributed to an increased rate of fatty acid oxidation within the cell (in mitochondria) induced by L-carnitine as confirmed by Bremer (1983) who revealed that, L-carnitine play a role in reducing the undesirable fat in carcasses of broiler chickens. Also, has a key role in facilitating the transport of long-chain fatty acids across the inner mitochondrial membrane before β-oxidation. Thus, under its insufficiency the transport of long-chain fatty acids could be impaired (Rabie and Szilagyi, 1998). It has been also found that, L-carnitine supplementation resulted in lowered muscle and liver lipid contents (Szilagyi, 1998). Also, Buyse *et al.* (2001) observed that, dietary L-carnitine had no significant effects on any of these production parameters, except for a reduction in the abdominal fat content of female chickens.

On the other hand, the decrease in serum total lipids at 6 and 10 weeks may be due to decrease level of serum triglycerides, total cholesterol and VLDL-cholesterol observed at the same periods in the present study. As confirmed by Lien and Horng (2001) who demonstrated that carnitine did increase the activity of hepatic carnitine palmitoyl transferase and decrease serum triglycerols and NEFA concentrations. It is possible that carnitine may increase the rate of fatty acid transportation in broilers and thus the reduction in serum non esterified fatty acid and triacylglycerol contents.

The obtained data showed a marked decrease in serum total cholesterol concentration in laying hens kept on choline and L-carnitine rations during different periods of the experiments. These results are nearly similar to the results reported by Diaz *et al.* (2000) who revealed that L-carnitine plays an important role in the mitochondrial uptake of long-chain fatty acids in mammals. Who showed that this compound has a marked hypo-cholesterolemic effect when used in conjunction with lipid-rich diets in rabbits.

Furthermore it may be attributed to the decreased cholesterol and apo-A-I- secretion from the liver, or increased uptake of HDL into the liver (An *et al.*, 1997).

The marked hypertriglyceridaemia observed in laying hens fed on choline, and L-carnitine may be attributed to a consequence of either over production of VLDL by the liver or defective removal of triglyceride rich lipoproteins from the circulation, or both. The later possibility can be explained through lipoprotein lipase, an insulin dependent enzyme involved in triglyceride removal (Yost *et al.*, 1995). Moreover, the increased serum triacylglycerol synthesis and very low density lipoprotein (VLDL) secretion (Hussein and Azab, 1998).

The recorded low plasma triglyceride concentration might also reflect the low rate of hepatic lipogensis or the use of plasma triglycerides by tissue other than adipose tissues and the carnitine did increase the activity of hepatic carnitine palmitoyl transferase and decrease serum triacylglycerol and non esterified fatty acid concentrations. It is possible that supplementary carnitine may increase the rate of fatty acid transportation in broilers and thus the reduction in serum non esterified fatty acid and triacylglycerol contents (Lien and Horng, 2001).

The marked increase in serum phospholipids on supplemented with choline, and L-Carnitine may be attributed to increased activity of choline phosphotransferase enzymes involved in phospholipids synthesis the activities of all enzymes involved in lipid synthesis were significantly increased (acyl-CoA synthetase, acyl-CoA : cholesterol acyltransferase and choline phosphotransferase). Furthermore it was may possibly be due to the supplementation of the lipotropic factors which increased the synthesis of phospholipids by the liver. Since, there is evidence that synthesis of phospholipids in birds occurs mainly if not entirely in the liver or could be due to increased synthesis of lipoproteins by the liver (Diaz, 2000).

Furthermore, the dramatic decrease in seum phospholipids concentration observed in this study was may possibly be due to increased uptake of plasma very low density lipoprotein cholesterol and their transport from the liver into the ovarian follicle and the oocyte, probably

caused by L-carnitine (Rabie *et al.*, 1997). It has been reported that 95% of yolk total lipids is derived from triaglycerol-rich lipoprotein, which is synthesized in the liver and transferred into rapidly developing yolks from the plasma over a period of several days before ovulation. The remaining yolk lipid is derived from the lipovitellin component of plasma vitellogenin (Griffin *et al.*, 1984).

The decrease in serum NEFA concentration of laying hens observed in the present study in response to L-carnitine supplementation may be attributed, at least partly, to an increased rate of fatty acid oxidation within the cell (in mitochondria) induced by L-carnitine (Owen *et al.*, 1996).

It is possible that, supplementary carnitine may increase and facilitated the rate of fatty acid transportation in broiler and thus the reduction in serum NEFA concentration (Lien and Horng, 2001). Moreover, the increased fatty acid oxidation induced by L-carnitine may result in decreased availability of long-chain fatty acids for esterification to triacylglycerols, and at same time can raise the mitochondrial level of acetyl-CoA (Cyr *et al.*, 1991). Also, it was may possibly be due to decrease of the lipolytic effect of lipase activity reported in the present experiment which decrease the lipolysis of triacylglycerols into glycerol and free fatty acids (Hussein *et al.*, 2001).

The decrease in serum VLDL-cholesterol concentration of laying hens fed diet supplemented with choline and L-carnitine in the present study may be attributed to the massive uptake of hepatically synthesized yolk precursor proteins from the plasma during the final rapid growth phase of the chicken oocyte. This suggestion was confirmed by Nimpf and Schneider (1991) who reported that in laying hens, VLDL and vitellogenin (VTG) are secreted by the liver and eventually taken up by the growing oocyte via receptor-mediated endocytosis. Once taken up by the growing zygote, apolipoprotein B, the major protein constituent of VLDL, is proteolytically cleaved by a chicken-specific cathepsin-D.

On the other hand, the increase in serum VLDL-cholesterol concentration in laying hens given diets supplemented with lipotropic factors may possibly be due to the secretion of estrogen from the active ovaries of mature egg laying hens. This female sex hormone is being implicated or responsible for the elevation of serum VLDL-cholesterol concentration due to estrogenic modulation of lipid metabolism in laying hens (Jaccoby *et al.*, 1996).

Also, egg production is an energy intensive process, but energy excess nrgatively affects egg production (Walzem *et al.*, 1999), who added that overfed-involuted hens showed significant elevations of plasma LDL, VLDL cholesteryl ester, and HDL triacylglycerol content. Moreover,

overfeeding generally increases VLDL size and plasma HDL levels suggesting that increased peripheral metabolism and perhaps mechanical exclusion of enlarged LDL by the granulose basal lamina, contribute to the obesity and impaired yolk deposition observed in hens with excessive energy intakes.

The increase and decrease in serum lipase activity in laying hens supplemented with choline and L-carnitine may be due to the secretion of estrogen from the active ovaries of mature egg laying hens. This female sex hormone is being implicated or responsible for the elevation of serum lipase activity due to estrogenic modulation of lipid metabolism in laying hens (Jaccoby *et al.*, 1996).

Table (1): Effects of choline and L-carnitine on serum total lipids, triacylglycerols, phospholipids and non esterified fatty acids concentration in laying hens (mg/dl).

Weeks after supplementation	Total lipids			Triacylglycerols			Phospholipids			Non esterified fatty acids		
	G I	GII	GIII	G I	GII	GIII	G I	GII	GIII	G I	GII	GIII
2 weeks	651.50 ±54.96	1606.67 ±74.95***	199.44 ±46.58***	312.00 ±13.42	749.50 ±15.42***	752.83 ±3.11***	190.32 ±14.43	281.52 ±13.48***	336.71 ±13.89***	67.46 ±0.48	45.13 ±6.14*	40.61 ±6.64**
4 weeks	1135.00 ±42.95	1038.33 ±91.01	1203.33 ±37.48	504.17 ±21.73	511.33 ±21.29	422.67 ±9.71**	215.08 ±12.62	249.99 ±12.21	224.09 ±13.22	77.78 ±1.62	36.69 ±3.33***	50.51 ±8.61*
6 weeks	1350.00 ±106.77	1540.00 ±66.12	961.67 ±57.41***	563.50 ±9.19	672.33 ±14.24***	416.33 ±6.38**	300.68 ±8.85	289.42 ±5.89	284.91 ±5.35	44.76 ±4.70	25.47 ±2.04*	17.41 ±3.42**
8 weeks	1264.83 ±29.01	526.67 ±26.57***	1171.67 ±35.54	556.50 ±5.53	153.67 ±5.34***	538.00 ±25.43	248.87 ±15.15	207.77 ±6.96*	251.69 ±14.69	39.21 ±1.28	19.66 ±0.92***	24.01 ±1.84***
10 weeks	1745.00 ±37.53	1815.00 ±82.82	1540.00 ±61.10*	658.40 ±1.30	860.60 ±1.99***	598.70 ±1.42***	225.23 ±15.37	198.23 ±14.96	233.11 ±10.99	70.36 ±5.43	24.61 ±2.98***	31.30 ±3.11**
12 weeks	1713.33 ±8.82	2573.33 ±177.04**	2060.00 ±134.29**	785.23 ±2.86	976.43 ±4.66***	726.30 ±4.35***	257.89 ±5.06	248.87 ±4.75	218.92 ±10.70**	44.98 ±7.09	30.15 ±6.86	32.53 ±9.13

G I: Control group. G II: Choline supplanted group G III: L-carnitin supplemented group

Data are presented as Mean ± S.E. S.E.: Standard error.

*: Significant at (P<0.05) **: Highly significant at (P<0.01) ***: Very highly significant at (P<0.001)

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Table (2): Effects of choline and L-carnitine on serum total cholesterol, HDL, LDL, VLDL(mg/dl), and Lipase activities(IUL) concentration in laying hens .

Weeks after supplementation	total cholesterol			HDL			LDL			Lipase		
	G I	GII	GIII	G I	GII	GIII	G I	GII	GIII	G I	GII	GIII
2 weeks	131.33 ± 2.73	146.63 ±2.03*	173.17 ±1.98**	31.52 ±1.61	32.57 ±2.10	25.78 ± 1.05*	37.42 ±4.61	37.00 ±4.25	14.52 ± 1.65***	23.90 ±0.69	29.39 ±0.77*	26.30 ±0.73
4 weeks	138.00 ±1.93	126.67 ±0.99*	147.50 ±6.65	35.10 ±0.50	36.78 ±0.25*	33.70 ±0.69	10.07 ±1.64	14.45 ±3.45	29.27 ±6.25**	23.35 ±0.72	31.50 ±0.90*	21.57 ±0.47*
6 weeks	131.00 ±2.96	150.50 ±1.89*	119.83 ±1.44**	32.12 ±0.37	39.68 ±0.60*	35.83 ±1.38*	13.82 ±1.47	23.65 ±1.84**	6.82 ±1.41***	29.90 ±0.82	25.30 ±0.87*	19.90 ±0.57**
8 weeks	139.33 ±1.33	148.37 ±1.53	160.60 ±3.71**	37.67 ±1.62	45.33 ±1.92*	34.60 ±0.81	9.63 ±1.69	27.30 ±2.39***	18.30 ±1.66**	23.90 ±0.90	37.86 ±0.90**	27.91 ±1.14*
10 weeks	159.17 ±0.95	146.63 ±2.30*	147.30 ±1.54*	34.37 ±0.98	33.80 ±1.67	25.20 ±1.50**	6.88 ±0.39	19.29 ±1.05***	3.63 ±0.89*	23.80 ±0.75	31.90 ±0.90**	24.90 ±0.58
12 weeks	144.58 ±1.06	174.50 ±1.35**	146.03 ±0.69	27.53 ±0.27	29.10 ±1.15	26.47 ±1.20	33.99 ±1.14	47.87 ±1.49**	25.69 ±0.74**	21.90 ±0.82	30.10 ±0.90*	25.49 ±0.87

G I: Control group.
Data are presented as Mean ± S.E.
*: Significant at (P<0.05)

G II: Choline supplanted group
S.E.: Standard error.
**: Highly significant at (P<0.01)

G III: L-carnitin supplemented group
***: Very highly significant at (P<0.001)

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التأثيرات الكيميائية الحيوية للكولين والكارنيتين على نمط الدهون فى الدجاج البياض

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أجريت الدراسة على عدد ستون دجاجة بياض من نوع لوهمان بنى فى عمر 36 أسبوع، وقد تم تقسيمهم إلى ثلاثة مجموعات رئيسية اشتملت كل مجموعة على عدد 20 دجاجة كالآتى:

١ –المجموعة الأولى (المجموعة الضابطة): اشتملت على عدد 20 دجاجة تمت تغذيتها على العليقة الأساسية بدون أية إضافات واستخدمت كمجموعة ضابطة للمجموعات الأخرى.

٢ –المجموعة الثانية (الدجاج المضاف لعليقته الكولين): اشتملت أيضا على عدد 20 دجاجة تمت تغذيتها على العليقة الرئيسية مضاف إليها الكولين بكمية تماثل ضعف الكمية الموجود فى العليقة الرئيسية وهى 1500 مجم/كجم عليقة.

٣ –المجموعة الثالثة (الدجاج المضاف لعليقته إل–كارنيتن): اشتملت أيضا على عدد 20 دجاجة تمت تغذيتها على العليقة الرئيسية مضاف إليها إل–كارنيتين بمعدل 500 مجم/كجم من العليقة.

هذا وقد استمرت فترة الدراسة لمدة 12 أسبوع. تم فيها تجميع عينات الدم 6 مرات بصورة عشوائية وعلى فترات كل أسبوعين من بداية إضافة موانع ترسيب الدهون (الكولين والمثيونين وإل–كارنيتين).

من خلال النتائج التى تم التوصل إليها نستخلص أن الكارنيتين عامل هام لأيض الدهون فى الدجاج البياض، حيث أن إضافته للعليقة أحدثت تغيرات فى أيض الدهون والبروتينات بالمصل، مثل انخفاض مستوى الأحماض الدهنية الحرة وأنماط البروتينات الدهنية وزيادة فى الدهون الكلية والكوليسترول الكلى والجليسريدات الثلاثية، والدهون الفوسفورية فى الدجاج البياض. لذلك يجب إضافة إل–كارنيتين إلى علائق الدجاج البياض كعامل أساسى أو غضافة عنصرى تخليقه من الأحماض الأمينية الأساسية وهما الليسين والمثيونين، كما أن إضافة الكولين لعليقة الدجاج البياض أحدثت زيادة فى مستوى الدهون والبروتينات بالمصل ما عدا الأحماض الدهنية الحرة خلال فترات التجربة وهذا يؤكد تأثيرها الواضح على التمثيل الغذائى للدهون وعدم تراكمها فى خلايا الكبد مما يقلل من حدوث اضطرابات أيض الدهون التى ينتج عنها تشحم الكبد أو ظاهرة النزيف الكبدى الدهنى فى الدجاج البياض.

ولذلك فإنه من أجل الحصول على أعلى معدلات لإنتاج البيض ينصح بإضافة كميات مناسبة من الكولين وإل–كارنيتين إلى علائق الدجاج البياض لمنع ترسيب الدهون فى الكبد والوقاية من ظاهرة التشحم الكبدى وعلاجه.