L-Carnitine and its use as a feed additive in poultry feeding a review

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SUMMARY

L-carnitine exists in animal and plant cells and microorganisms. In animals, it is synthesised almost exclusively in the liver. Two essential amino acids, i.e., lysine and methionine serve as primary substrates for its biosynthesis, and vitamin B<sub>6</sub>, nicotinic acid, vitamin C and folates are also required as cofactors. L-carnitine plays a key role in energy metabolism of cells, mainly, by transferring acyl groups from cytoplasm to mitochondrial matrix for β-oxidation. In addition, L-carnitine regulates coenzyme A concentrations in cytosol and mitochondria, glucose and lipid metabolisms. Endogenous biosynthesis together with dietary L-carnitine supply are sufficient to cover normal requirements in poultry breeding. However, supplementation with L-carnitine or precursors and cofactors may be required when metabolic rate and energy demands are increased (growth, laying periods, environmental stress, fat enriched diets...). Although dietary supplementation does not generally improve bird performance (feed intake, growth), L-carnitine promotes the lipid redistribution in organism, mainly in broilers, by increasing intramural fat and by decreasing subcutaneous and abdominal fat deposits and by lowering plasma cholesterol and triglyceride concentrations. But, L-carnitine does not modify the N utilization. In laying hens and quails, L-carnitine supplementation slightly improves egg production and hatchability, but it mainly modifies egg composition. Albumen quantity and quality is improved and also albumen and yolk carnitine concentrations were enhanced, promising embryo development. Nevertheless, many factors (stress condition in breeding and environment, fat supply, species, intestinal degradation) would affect dietary L-carnitine utilisation and metabolic capacity, and would explain the discrepancies evidenced in experimental studies.

Keywords : L-carnitine - poultry - biosynthesis - lipid metabolism - egg.

RÉSUMÉ

Utilisation de la L-carnitine en nutrition aviaire. Une revue. Par C. ARSLAN.

La L-carnitine existe dans les cellules animales, végétales et dans les micro-organismes. Chez les animaux, elle est synthétisée en quasi-totalité dans le foie. Deux acides α-aminés essentiels, la lysine et la méthionine, interviennent comme substrats primaires durant la biosynthèse et la vitamine B<sub>6</sub>, l’acide nicotinique, la vitamine C et les folates sont également requis en tant que cofacteurs. La L-carnitine joue un rôle central dans le métabolisme énergétique principalement en transférant les groupements acyles du cytoplasme à la matrice mitochondriale en vue de la β-oxidation. De plus, la L-carnitine intervient dans la régulation des concentrations de coenzyme A dans le cytosol et les mitochondries, et dans la régulation des métabolismes glucidique et lipidique. La voie de biosynthèse couplée aux apports alimentaires est suffisante pour couvrir les besoins normaux en élevage aviaire. Cependant, une supplémentation en L-carnitine ou en précurseurs et cofacteurs devient nécessaire en cas de demandes énergétiques accrues (croissance, période de ponte, stress environnementaux, alimentation enrichie en graisses). Bien qu’en général un apport alimentaire supplémentaire n’améliore pas les performances des oiseaux (prise alimentaire, croissance), la L-carnitine promeut une redistribution des lipides dans l’organisme, principalement chez les poulets, en augmentant la graisse intramusculaire et en diminuant les dépôts adipeux sous-cutanés et abdominaux, et en diminuant les concentrations plasmatiques de cholestérol et de triglycérides. Mais la L-carnitine ne modifie pas l’utilisation de l’azote. Chez les poules et les cailles pondueuses, la L-carnitine améliore faiblement la production en œufs et l’élosabilité des œufs, mais elle modifie surtout la composition de l’œuf : la quantité et la qualité de l’albumen sont améliorées et les concentrations de carnitine dans l’albumen et le jaune sont augmentées favorisant le développement embryonnaire. Néanmoins, de nombreux facteurs (conditions stressantes de l’élevage et de l’environnement, apport en graisses, espèces, dégradation intestinale…) compromettent l’utilisation de la carnitine et ses capacités métaboliques et pourraient rendre compte des divergences entre les différentes études expérimentales.


Introduction

Vitamins are defined as a group of complex organic compounds present in small amounts in natural foodstuffs that are essential to normal metabolism and lack of vitamins in the diet causes deficiency disease. Because of the possibility of specific biosynthesis pathways in some species, some “vitamins”, such as carnitine, could be considered only as essential metabolites in these species and dietary sources are not needed.

In 1905, L-carnitine was discovered in great quantities in muscle tissue by GULEWITSCH and KRIMBERG [32]. As shown in Table I, numerous studies on carnitine were conducted and revealed its implication in the transfer of fatty acids into mitochondria. Moreover, the occurrence of endogenous synthesis (from lysine and butyrobetaine) was also evidenced.

The amino acid derivative L-carnitine has gained interest in recent years as a potential feed additive for improving domestic animal production because of its metabolic functions (Part I, Table I). In this context, experiments have been carried out with various domestic birds including broilers, hens, quails, ducks and geese in order to study the effects of carnitine on energy metabolism and on improvement of growth performance (Part II).

PART I : Chemical structure, metabolism, sources and requirements

Carnitine or β-hydroxy-γ-trimethylaminobutyrate is a quaternary amine (Figure 1). It is a very hygroscopic compound easily hydrosoluble and has a molecular weight of 161.2 D. Methods of analysis first utilised the bioassay technique using Tenebrio molitor. Other methods developed for carnitine determination include chemical, enzymatic, gas chromatography and radioisotopic procedures [46].

Carnitine was synthesized in vivo from lysine and methionine in the kidney (feline, man), testes (rat), skeletal muscle (sheep), brain (man) and liver in all mammals [11, 24, 26, 58]. During the synthesis, L-lysine provides the carbon chain and nitrogen atom of carnitine, and L-methionine provides the methyl groups (Figure 2) [59].

The conversion of TrimethylLysine to carnitine requires 2 hydroxylations catalysed by 2 specific monooxygenases that use α-ketoglutarate as an electron donor to activate dioxygen. The α-amino acids is cleared into CO₂ and succinate. The both enzymes require Fe (II) and are activated by ascorbate [9, 10, 39, 59, 63, 73]. Endogenous biosynthesis may be sufficient to cover normal requirements in all mammals and bird species, when precursors and cofactors of the L-carnitine is sufficient in the diet. However, this is not the case in neonates (in which, the biosynthesis is not fully developed), under the stress, in animals with the higher performance and when diets are rich in fat [56, 59]. Carnitine biosynthesis is regulated by the diet, age, and hormonal status of the animal. L-carnitine concentrations vary according to species, tissue types and nutritional status [53, 54].

Dietary carnitine appears to cross rapidly the mucosal intestinal membrane by both passive and active transport mechanisms [33]. Then carnitine is extracted from the portal circulation by the liver and subsequently released into the systemic circulation [16]. Carnitine is not carried in blood in any tightly bound forms, in contrast to other water-soluble vitamins. Free carnitine is excreted in urine, and the principal excretory product is trimethylamine oxide [46].

The crucial event referred by FRITZ [28] is that carnitine stimulates fatty acid oxidation. In the following years, the carnitine acyltransferases were detected in mitochondria and carnitine esters as intermediates in the oxidation of fatty acids were evidenced [67]. Cytoplasmic fatty acyl-CoA esters can not directly cross the membranes of mitochondria whereas the β-oxidation and the consequent release of energy with high yield are typically mitochondrial events.

### TABLE I. — Main historical developmental process of carnitine [52].

<table>
<thead>
<tr>
<th>Year</th>
<th>Discovery</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>1905</td>
<td>Carnitine was isolated from muscle</td>
<td>GULEWITSCH and KRAMBERG [32]</td>
</tr>
<tr>
<td>1927</td>
<td>The chemical structure of carnitine was established</td>
<td>TOMITA and SENDJU [69]</td>
</tr>
<tr>
<td>1952</td>
<td>Carnitine was shown to be a vitamin (B7) for a meal worm (Tenebrio molitor)</td>
<td>CARTER et al. [19]</td>
</tr>
<tr>
<td>1955</td>
<td>Carnitine was shown to stimulate fatty acid oxidation in liver homogenates</td>
<td>FRITX [28]</td>
</tr>
<tr>
<td>1961</td>
<td>Butyrobetaine was shown to be a carnitine precursor</td>
<td>LINDSTEDT and LINDSTEDT [42], BREMER [12]</td>
</tr>
<tr>
<td>1962-63</td>
<td>Fatty acid esters of carnitine were shown to be intermediates in fatty acid oxidation</td>
<td>BREMER [13], BREMER [14], FRITZ and YUE [29]</td>
</tr>
<tr>
<td>1965</td>
<td>High concentration of carnitine were found in epididymis and sperm</td>
<td>MARQUIS and FRITZ [44]</td>
</tr>
<tr>
<td>1966</td>
<td>Carnitine palmitoyltransferase (CPT) was localised in the inner mitochondrial membrane and acyl-CoA synthetase in the outer mitochondrial membrane</td>
<td>NORUM et al [49], YATES et al [72]</td>
</tr>
<tr>
<td>1971</td>
<td>Lysine was shown to be a precursor of carnitine</td>
<td>HORNE et al. [35]</td>
</tr>
<tr>
<td>1973</td>
<td>Carnitine acyltransferases were detected in peroxisomes</td>
<td>MARKWELL et al. [43]</td>
</tr>
<tr>
<td>1975</td>
<td>Carnitine translocase was demonstrated in mitochondria</td>
<td>RAMSAY and TUBBS [57], PANDE [52]</td>
</tr>
<tr>
<td>1987</td>
<td>CPT-I was shown to be localised in the outer membrane of the mitochondria</td>
<td>MURTY and PANDE [48]</td>
</tr>
<tr>
<td>1995</td>
<td>Direct evidence was obtained for the function of carnitine in the transfer of acyl groups from peroxisomes to mitochondria</td>
<td>JACOBS and WANDERS [37]</td>
</tr>
</tbody>
</table>

### Figure 1. — Chemical structure of carnitine.
Carnitine is required for the transport of long-chain fatty acids from the cytoplasm to the matrix compartment of mitochondria. The Carnitine acyltransferase is the enzyme responsible for this shuttle mechanism and it exists in two forms, the carnitine acyltransferase I (CAT I) and the carnitine acyltransferase II (CAT II). After activation of the long chain fatty acids into acylCoA in cytosol, acyl groups are transferred from coenzyme A to carnitine to form O.acylcar- nitine by the CAT I on the outer surface of the mitochondrial membrane. The carnitine esters move to the inner surface by exchange with free carnitine using an antiport mechanism. The CAT II, located on the inner surface, catalyses the reverse reaction by transferring acyl groups from carnitine to coenzyme A to form O-acetylcar- nitine by the CAT I on the outer surface of the mitochondrial membrane. The carnitine esters move to the inner surface by exchange with free carnitine using an antiport mechanism. The CAT II, located on the inner surface, catalyses the reverse reaction by transferring acyl groups from carnitine to coenzyme A to form O-acetylcar- nitine by the CAT I on the outer surface of the mitochondrial membrane. The carnitine esters move to the inner surface by exchange with free carnitine using an antiport mechanism.

Other functions of Carnitine are 1) the buffering and removing of the potentially toxic acyl groups from cells, 2) to regulate the ratios free CoA / acylCoA in 2 separate cellular compartments (cytosol and mitochondria), 3) to regulate glu- coneogenesis, fatty acid synthesis and ketone bodies, branched chain amino acid, triglyceride and cholesterol metabolism [50]. In addition, it can exhibit immunomodulatory effects, while growth performance is not improved [45]. The mechanism(s) accounting for the positive effect of L-carni- tine on antibody production is not yet fully understood. Restoration of the cellular L-carnitine content may enhance the lipid metabolism and improve the cellular energy balance [45]. Furthermore, L-carnitine (esters) are known to have free radical scavenging properties [51]. This hypothesis is currently under investigation. L-carnitine has been reported as a hypolipidemic drug, able to reduce the circulating concentrations of cholesterol, triglycerides, free fatty acids, phospholipids, and very low density lipoproteins (VLDL) and to increase the concentrations of high, intermediate, and low density lipoproteins (HDL, IDL, LDL, respectively) in murine [25]. The plasma lipid-lowering effects of L-carnitine would be associated with several possible processes, including increase of cholesterol turnover due to increased

![Figure 2. — Endogenous synthesis pathway of L-carnitine in mammals. (SAM : S adenosyl-methionine, SAHC : S adenosyl-homocysteine).](image-url)
conversion of cholesterol to bile acids and biliary excretion or due to a modified repartition of whole body cholesterol [8]. However, very little is known on the molecular basis of such processes.

Carnitine occurs naturally in microorganisms, plants and animals. The D-form does not occur in nature but may be obtained by chemical synthesis [73]. The D isomer of carnitine is biologically inactive and it also hinders activities of L-carnitine. At high dosages, D carnitine shows detrimental affects [47]. The major dietary sources are red meat, poultry, fish and dairy products [59]. Animal meals contain 10 to 20 times more L-carnitine than plant-based feedstuffs (Table II). However, little L-carnitine is found in cereal grains and their by-products [5, 10, 59].

In normal physiological and nutritive conditions, L-carnitine requirements are, however, largely covered by dietary sources. Dietary L-carnitine supplementation could improve fatty acid and energy utilisation and therefore gain and feed efficiency, especially in young animals where synthesis is insufficient to cover endogenous requirement [31]. Feeding studies have shown that the performance of animals sharply drops when the L-carnitine content falls below 15-20 mg per kg of food. Because of the cereal grains usually represent the major component of poultry diets, it may be useful to incorporate this compound into diets. To ensure that poultry receive adequate amounts of L-carnitine, feed recipes should be contained the dosages reported in Table III.

L-carnitine oral supplementation can be realised either in the drinking water or directly in the diet. Since this compound is easily dissolved in water, the drinking water is often preferred to achieve a homogeneous mixture.

### PART II : L-carnitine usage in poultry feeding

#### DIETARY SUPPLEMENTATION WITH L CARNITINE PRECURSORS AND COFACTORS

Although plant products are low in carnitine, poultry diets are composed mainly of maize and soybean. Nevertheless, it is generally accepted that endogenous L-carnitine synthesis

<table>
<thead>
<tr>
<th>Feedstuffs</th>
<th>Content</th>
<th>Feedstuffs</th>
<th>Content</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cereals</td>
<td>By-products of the oil processing</td>
<td></td>
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<tr>
<td>Barley</td>
<td>7</td>
<td>Coconut oil meal</td>
<td>10</td>
</tr>
<tr>
<td>Corn</td>
<td>5</td>
<td>Linseed meal</td>
<td>15</td>
</tr>
<tr>
<td>Milo</td>
<td>5</td>
<td>Palm-kernel meal</td>
<td>5</td>
</tr>
<tr>
<td>Oat</td>
<td>5</td>
<td>Peanut meal</td>
<td>10</td>
</tr>
<tr>
<td>Rye</td>
<td>5</td>
<td>Rapeseed meal</td>
<td>5</td>
</tr>
<tr>
<td>Sorghum</td>
<td>5</td>
<td>Safflower seed meal</td>
<td>10</td>
</tr>
<tr>
<td>Triticale</td>
<td>5</td>
<td>Soybean meal</td>
<td>12</td>
</tr>
<tr>
<td>Wheat</td>
<td>5</td>
<td>Sunflower meal</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>By-products</td>
<td>By-products of the starch and sugar industry</td>
<td></td>
</tr>
<tr>
<td>barley bran</td>
<td>15</td>
<td>Beet molasses</td>
<td>10</td>
</tr>
<tr>
<td>Brewers grain (dried)</td>
<td>5</td>
<td>Beet pulp (dried)</td>
<td>2</td>
</tr>
<tr>
<td>Brewers yeast (dry)</td>
<td>15</td>
<td>Corn sugar</td>
<td>0</td>
</tr>
<tr>
<td>Corn bran</td>
<td>12</td>
<td>Feeding sugar</td>
<td>10</td>
</tr>
<tr>
<td>Corn germ</td>
<td>15</td>
<td>Manioc meal</td>
<td>5</td>
</tr>
<tr>
<td>Corn gluten</td>
<td>5</td>
<td>Potato pulp (dried)</td>
<td>5</td>
</tr>
<tr>
<td>Malt sprouts</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oat (rolled)</td>
<td>5</td>
<td>Animal meal</td>
<td>150</td>
</tr>
<tr>
<td>Oat hulls</td>
<td>3</td>
<td>Blood meal</td>
<td>10-15</td>
</tr>
<tr>
<td>Rice bran</td>
<td>25</td>
<td>Feather meal</td>
<td>120</td>
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<tr>
<td>Rye bran</td>
<td>15</td>
<td>Fish meal</td>
<td>120-150</td>
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<tr>
<td>Rye meal</td>
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<td>Meat bone meal (40% CP)</td>
<td>100</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>15</td>
<td>Meat meal (62 % CP)</td>
<td>150</td>
</tr>
<tr>
<td>Wheat meal</td>
<td>5</td>
<td>Poultry by-product meal</td>
<td>120</td>
</tr>
<tr>
<td></td>
<td>Grain legumes</td>
<td>Milk derivates</td>
<td></td>
</tr>
<tr>
<td>Field beans</td>
<td>10</td>
<td>Butter milk</td>
<td>130</td>
</tr>
<tr>
<td>Lupines</td>
<td>10</td>
<td>Cows milk</td>
<td>20</td>
</tr>
<tr>
<td>Protein peas</td>
<td>10</td>
<td>Cows milk (dried)</td>
<td>140</td>
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<tr>
<td>Oil seed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cotton seed</td>
<td>20</td>
<td>Skim milk powder</td>
<td>130</td>
</tr>
<tr>
<td>Linseed meal</td>
<td>15</td>
<td>Skim milk</td>
<td>15</td>
</tr>
<tr>
<td>Soybean meal (cooked)</td>
<td>15</td>
<td>Whey (wat)</td>
<td>450</td>
</tr>
<tr>
<td>Sunflower meal</td>
<td>5</td>
<td>Whey powder (lactose extracted)</td>
<td>800</td>
</tr>
<tr>
<td></td>
<td>Oils and fats</td>
<td>Green forage</td>
<td></td>
</tr>
<tr>
<td>Animal fats</td>
<td>0</td>
<td>Alfalfa meal (dried)</td>
<td>10</td>
</tr>
<tr>
<td>Vegetable fats</td>
<td>0</td>
<td>Grass meal (dried)</td>
<td>5</td>
</tr>
</tbody>
</table>

TABLE II. — Natural L-carnitine contents in some feedstuffs, mg/kg [5].

*Revue Méd. Vét.*, 2006, **157**, 3, 134-142
Together with its dietary intake should be sufficient for normal functions. Since carnitine can be biosynthesised endogenously from methionine and lysine, these two amino acids are usually the more important limiting amino acids in poultry nutrition. Methionine and lysine are frequently supplemented in the formulated diets. When diets are not supplemented with these two amino acids, the chicken may synthesise an inadequate amount of carnitine. In a study conducted on fish, piglets and quail, SCHUHMACHER et al. [66] concluded that carnitine seemed effective for improving body weight gain and feed conversion, mainly in groups with diets marginally deficient in lysine and methionine plus cysteine respectively. The influence of low and high dietary contents of lysine and methionine requirements (20% below or above optimum requirements, respectively) on body weight gain, abdominal fat and carnitine content in some tissues was examined by IBEN et al. [36] in broiler chickens. The highest body weight was achieved in groups with optimum dietary contents of the 2 amino acids. In groups with low lysine and methionine supplementation, the abdominal fat content has higher than in the other groups. By contrast, the different supplies of carnitine precursors did not affect the tissue carnitine contents, whereas a fat dietary supplementation decreased the tissular carnitine concentrations except in the kidney. In another study, LEIBETSEDER [39] investigated the effectiveness of carnitine and its precursors (lysine and methionine) for reducing the formation of abdominal fat in broilers fed with diets supplemented with 0 or 50 g fat/kg. He found that body weight gain, feed conversion, and abdominal fat content of broilers were not influenced by dietary carnitine (L or DL form) at a dosage of 200 mg/kg diet. He also reported that carnitine concentrations in the liver, kidney, heart and some skeletal muscles significantly increased in response to supplemental dietary L-carnitine.

Because of the ascorbic acid, niacin, folate and vitamin B6 are required as cofactors in the L-carnitine biosynthesis pathway, supplementation of L-carnitine alone or in combination with one of these vitamins is also examined. CELIK and OZTURKCAN [21] have analysed the effects of a dietary supplementation by L-carnitine alone (50 mg/kg in drinking water) by ascorbic acid alone (500 mg/kg in drinking water) or by the combination (L-carnitine + Ascorbic acid) on growth performance and plasma L-carnitine concentrations in broilers submitted or not to a thermal stress (34-36 °C for 8h, 20-22 °C for 16h and 20-22 °C for 24h, respectively). The body weight gain was significantly enhanced in vitamin supplemented (L-carnitine, ascorbic acid or both) animals under high temperature conditions, whereas L-carnitine or L-carnitine+ascorbic acid supplementation significantly reduced the growth performance in broilers under normal ambient temperature. Plasma L-carnitine concentrations were also significantly affected by the cosupplementation. Marked increases were observed in supplemented stressed broilers, while this parameter was lowered in supplemented birds under normal conditions. It was also reported [22] that growth performance was improved by L-carnitine supplementation alone (50 mg/l) or in combination with niacin (50 mg/l) in drinking water, during the early stages of chicken growth. Moreover, plasma L-carnitine concentrations were also increased by the dietary treatment.

LEIBETSEDER [39] produced evidence that the supplementation of a standard layer’s ration with either 500 mg L-carnitine or 500 mg nicotinic acid, or a combination of the two compounds, had no effect on egg production, feed intake, body weight or cholesterol concentration in serum and in yolk during the early laying period in thermoneutral conditions. However, yolk L-carnitine content was significantly higher in the supplemented groups. It has been suggested that the enhancement of carnitine in yolk would be beneficial for the development of the chick embryo [61].

### EFFECTS OF DIETARY L-CARNITINE SUPPLEMENTATION ON FAT METABOLISM

Body fat accumulation results from dietary absorbed fat, endogenous fat synthesis (lipogenesis) and fat catabolism via β-oxidation (lipolysis). Production of broiler chickens with excessive body fat is a problem in the poultry industry. Several factors, such as nutrients and genetics, contribute to the tendency of broilers to accumulate body fat in excess. Therefore, improving carcass composition with additives has become a focus on nutrition research. For example, the dietary fat type affects the metabolism and deposition in broiler chickens [64]. Birds were fed with diets containing either dietary saturated (beef tallow) or polyunsaturated fat (sunflower oil) for 32d. The abdominal fat deposition was significantly lower in chickens receiving the sunflower oil-enriched diet compared the birds fed with the tallow-enriched diet. Furthermore, the specific activities of heart carnitine palmitoyltransferase I and L-3-hydroxyacylCoA dehydrogenase were increased in chicks fed with the polyunsaturated acid enriched diets, indicating a greater rate of β-oxidation, whereas liver fatty acid synthetase activity was depressed, suggesting hepatic lipogenesis reduction. Postprandial plasma triglyceride concentrations were reduced too, showing that the dietary lipid clearance from the bloodstream to tissues was amplified. In conclusion, SANZ et al. [64] suggested that the lower fat deposition in broilers fed with sunflower oil-enriched diets resulted from the increase of lipid catabolism and the decrease of fatty acid synthesis despite higher dietary fat absorption.

In case of increased metabolic demand the dietary low carnitine supply would become insufficient for metabolic requirements, and animals would become less productive and fatter. Theoretically, supplementing the broiler diet with an adequate content of carnitine would facilitate the fatty acids oxidation, and decrease esterification reactions and triacylglycerol storage in the adipose tissue. Reports on the effects of dietary L-carnitine supplementation on the growth performance and body composition of broiler chickens are conflic-

### Table III. — Recommended L-carnitine dosage for poultry, mg/kg of diet, [6].

<table>
<thead>
<tr>
<th>Animal Type</th>
<th>Dosage (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laying hens</td>
<td>50</td>
</tr>
<tr>
<td>Breeder hens</td>
<td>50</td>
</tr>
<tr>
<td>Broiler</td>
<td>50</td>
</tr>
<tr>
<td>Turkeys</td>
<td>60</td>
</tr>
<tr>
<td>Pigeons (Permanent administration)</td>
<td>25</td>
</tr>
<tr>
<td>Breeding pigeons</td>
<td>80</td>
</tr>
</tbody>
</table>
L-CARNITINE AND ITS USE IN POULTRY FEEDING

Some studies have shown that supplemental L-carnitine improved body weight gain and reduced the abdominal fat content of broilers [40, 53, 54]. In the same way, RABIBE and SZILAGYI [56] reported that L-carnitine supplementation (50 mg / kg) of broiler diets increased breast muscle yield, thigh meat yield and fat content of breast muscle, whereas quantity and percentage of abdominal fat was reduced. Therefore, lower body fat deposition may be attributed to increased fat catabolism or diminished endogenous fatty acid synthesis or both processes. SAYED et al. [65] have demonstrated that addition of L-carnitine (50 mg / kg) to diet containing 2 and 4 % of sunflower oil increased feed intake, weight gain and feed conversion and decreased serum cholesterol and lipid concentrations compared to the control group. Moreover, the association of carnitine and 2 % dietary fat has significantly decreased abdominal fat deposits.

In a study conducted by LIEN and HORNG [41] the effects of diets supplemented with 0 and 160 mg/kg L-carnitine on growth performance, serum components, carcass traits and enzyme activities were investigated on broiler chickens. The supplementary carnitine did not significantly influence the performance and carcass characteristic’s of the broilers, but the serum triacylglycerol and nonesterified fatty acid concentrations in the carnitine supplemented group were significantly lowered compared to the control group. In addition, in the supplemented group, carnitine palmitoyl transferase activity was increased, but not the β-oxidation enzyme activities (β-hydroxy acyl-CoA dehydrogenase, Acyl-CoA dehydrogenase, Enoyl-CoA hydratase). Furthermore, LETTNER et al. [40] have found that addition of L-carnitine (20 to 60 mg/kg) to soybean and corn diets has decreased the linoleic acid contents of abdominal fat.

The relationships between L carnitine dietary content (0 to 100 mg / kg) and growth performance and fat metabolism in broilers were investigated by XU et al. [71]. Whatever the L-carnitine dosages, no significant effect was observed on feed intake, daily weight gain or feed conversion. By contrast, decreases of abdominal fat associated with increase of breast muscle yield and muscle fat contents were obtained when carnitine (≥ 25 mg / kg) was added to diets. Higher doses (≥ 50 mg / kg) of dietary L-carnitine induced partial inhibition of the glucose-6-phosphate dehydrogenase, malic dehydrogenase, isocitrate dehydrogenase and lipoprotein lipase in the subcutaneous fat and also of carnitine palmitoyltransferase I in breast muscles. Consequently, L carnitine would reduce subcutaneous fat deposit and enhance intramuscular fat. In contrast, some investigators failed to observe any favourable response to added carnitine [4, 18, 20, 21]. Supplementation of carnitine neither improved growth supported by a low energy diet nor reduced excessive fat deposition elicited by high energy feeding [20]. Similarly, BAR-KER and SELL [4] observed no influence of dietary L-carnitine supplementation (0, 50 and 100 mg/kg) on performance and carcass composition of broilers and young turkeys fed with low- and high fat diets.

Ducks and geese are genetically predisposed to the fatness, but excessive fat ducks and geese are unattractive for consumers who are sensitive to the negative effects of saturated fat. Therefore, ARSLAN et al. [1, 2] have investigated the potential effects of L-carnitine on abdominal fat quality and quantity in these bird species. Unfortunately, addition of L-carnitine to drinking water (100 mg/l in geese and 200 mg/l in ducks) did not significantly improve growth performance (body weight gain and feed intake) and carcass traits (abdominal and mesenterial fat weight) and did not significantly affect biochemical markers of lipid and energy metabolisms (serum cholesterol, lipid, triglyceride and glucose concentrations). Nevertheless, the abdominal fat composition was slightly altered by L-carnitine supplementation in both species (Table IV).

Dietary vitamin supplementations (L-carnitine and vitamin C) has lowered the lipid metabolism (decrease of serum cholesterol and triglyceride concentrations) in male Japanese quails [70]. ARSLAN et al. [3] also confirmed the metabolic effects of L-carnitine in quails. After an experimental period of 6 weeks, they observed increases of glycaemia in treated birds (100 mg/l L-carnitine in drinking water), whereas the other biochemical markers (serum cholesterol, lipid, triglyceride, total protein and albumin concentrations) were not significantly altered.

Carnitine could also increase fatty acid oxidation rates in animals under thermal stress. In a study conducted by BUYSE et al. [18] on broiler chickens reared under normal temperature or submitted to thermal stress (rapid decrease of temperature from 28° C to 20° C when birds were 14 day old), the L-carnitine supplementation (100 mg / kg) induced marked increases of heart weights, plasma triiodothyronine concentrations and transient elevation of growth hormone, glucose and triglyceride concentrations, particularly in the stressed birds. In a more recent study [30] a cosupplementation with L-carnitine and CoQ 10 (a vitamin-like compound which plays a crucial role for producing energy in cells) reduced acites and mortality of broilers reared under low ambient temperature (15-18° C) and these protective effects were probably in relation with their antioxidant properties.

The positive effects of L-carnitine supplementation were also evidenced when birds were orally exposed to aflatoxin [68]. In deed, the percentage of abdominal fat and the plasma lipid concentrations were significantly reduced by L carnitine (50 mg / kg), particularly in birds exposed to the higher aflatoxin dose (5 mg / kg).

**EFFECT OF DIETARY SUPPLEMENTATION ON PROTEIN UTILISATION**

RODEHUTSCORD et al. [62] investigated the effects of L-carnitine supplementation (80 mg / kg diet) in diets containing 4 or 8 % of fat and differently distributed (ad libitum in a growth trial, 95 and 85 % of ad libitum in a balance trial). The amino acid concentrations of diets were adequate growth and feed conversion tended to increase by about 5 % in the supplemented diets distributed ad libitum, and feed conversion was simultaneously improved by carnitine and dietary fat content. However, L-carnitine supplementation has not positively affected the yield of energy utilisation, neither the N accretion and the dietary protein utilisation efficiency. These authors concluded that endogenous carnitine synthesis is not the limiting factor for energy utilisation in broiler chicken, even at high dietary fat concentration and L-carnitine must not be expected to increase protein utilisation under condition of adequate amino acid contents.
In many animal species body weight gain in growing animals is very sensitive to changes in dietary protein intake. HEO et al. [34] showed a greater efficiency of N utilisation, in response to L-carnitine supplementation in young pigs receiving diets with restricted metabolisable energy. However, this positive effect of L-carnitine was not confirmed by RODEHUTSCORD et al. [62] in broiler chickens. KITA et al. [38] found that L-carnitine supplementation at 200, 500 and 1000 mg/kg diet increased IGF I concentrations and growth of chickens when dietary protein supply was adequate (200 g/kg diet), but not when it was excessive (400 g/kg diet) or deficient (50 mg/kg). In the same way, RABIE et al. [53, 54] have demonstrated that L-carnitine supplementation (50, 100 and 150 mg/kg) has increased feed intake, weight gain and feed conversion and has decreased abdominal fat in broiler chickens fed with isoenergetic diets, containing variable crude protein contents (18, 20 and 22 %).

EFFECTS OF DIETARY L-CARNITINE SUPPLEMENTATION ON EGG PRODUCTION AND EGG QUALITY

There are several factors influencing the quality and quantity of eggs. Among these, the most important factors are nutrition, disease, age, breed or strain of bird and environment. Many important egg-quality components were deteriorated and become more variable with the age of the bird. Nutritional factors can modify egg quality by inducing metabolic changes. In fact, reports on the effects of dietary L-carnitine on egg production and egg quality of laying hens are limited. LEIBETSEDER [39] has reported that egg hatchability increased from 83 % to 87 % and from 82.4 % to 85.3 % in broiler breeders supplemented with L-carnitine at 50 and 100 mg/kg diet respectively.

RABIA et al. [55] conducted a study for the determination of the effects of L-carnitine supplementation (50, 100 or 500 mg/kg diet) on 65-week-old hens kept in cages. Dietary L-carnitine supplementation did not affect the laying performance (egg production rate, mean egg weight, daily feed intake, egg mass and feed conversion) or external egg quality but modified the egg composition. Albumen quality (relative albumen weight) was improved, in supplemented hens, probably because of a higher metabolic rate in the magnum and/or a higher activity of the shell gland. The increase of albumen could be beneficial for the nutritional point of view and also for improving storage time. Yolk index and yolk colour score did not significantly vary, whereas absolute or relative yolk weights were significantly lowered. L-carnitine has probably induced reduction of hepatic biosynthesis of yolk precursors, or an alteration of their transport from the liver to the ovarian follicle and the oocyte.

The egg production tended to increase, but not significantly, in 44 to 72 weeks old hens dietary supplemented by high L-carnitine doses (50 and 100 mg/kg). Moreover, a positive correlation between supplemental L-carnitine amounts in diets and the carnitine concentrations in egg albumen and yolk [60]. Additionally, these authors should show that the yolk contained 6-7 times more carnitine than the albumen. In Japanese quails, [7] supplementation with L-carnitine (500 mg/kg diet) alone or combined with vitamin C (500 mg/kg diet) in diet did not improve growth and carcass yield. However, egg production was significantly enhanced by L-carnitine.

The optimum performance of birds is obtained in thermoneutral zone, and high and low environmental temperatures depress bird productivity and also affect the quality of products in laying hens exposed to high ambient temperature (35-37 °C 8h and 20-22 °C 16h). The relative albumen weight and height were significantly increased by supplemental L-carnitine (50 mg/kg) in drinking water [23]. However, L-carnitine did not affect the growth performance (body weight gain, feed consumption), the laying performance (egg mass and egg weight) the shell quality (weight, thickness, and egg shape index) and the yolk quality (weight and colour score).

\[ \begin{array}{llllll}
\text{Fatty acids} & \text{Goose} & \text{P} & \text{Duck} & \text{P} \\
\hline
\text{Control} & \text{L-carnitine} & \text{Control} & \text{L-carnitine} & \text{P} \\
C14:0 & 0.44 \pm 0.02 & 0.50 \pm 0.03 & \text{NS} & 0.57 \pm 0.01 & 0.53 \pm 0.04 & \text{NS} \\
C16:0 & 21.85 \pm 0.56 & 27.57 \pm 0.46 & \text{NS} & 23.77 \pm 0.88a & 22.65 \pm 0.19b & 0.05 \\
C18:0 & 7.39 \pm 0.34 & 6.07 \pm 0.26 & \text{NS} & 4.48 \pm 0.32 & 4.03 \pm 0.17 & \text{NS} \\
\Sigma \text{SFA} & 29.98 \pm 0.23 & 34.40 \pm 0.63 & 0.05 & 29.22 \pm 1.19 & 27.57 \pm 0.20 & 0.05 \\
C16:1 & 2.30 \pm 0.11 & 4.10 \pm 0.15 & \text{NS} & 5.16 \pm 0.26 & 5.33 \pm 0.27 & \text{NS} \\
C18:1 & 52.99 \pm 0.63 & 47.35 \pm 0.83 & \text{NS} & 49.62 \pm 1.27 & 51.43 \pm 0.72 & \text{NS} \\
\Sigma \text{MUFA} & 55.35 \pm 0.58 & 51.77 \pm 0.72 & \text{NS} & 55.28 \pm 1.48 & 57.34 \pm 0.55 & \text{NS} \\
C18:2 & 11.77 \pm 0.51 & 11.13 \pm 0.22 & \text{NS} & 11.24 \pm 0.37 & 11.95 \pm 0.48 & \text{NS} \\
C18:3 & 0.54 \pm 0.02 & 0.50 \pm 0.01 & \text{NS} & 0.52 \pm 0.01 & 0.58 \pm 0.02 & \text{NS} \\
\Sigma \text{PUFA} & 12.44 \pm 0.53 & 11.72 \pm 0.22 & 0.05 & 12.09 \pm 0.38 & 12.80 \pm 0.51 & \text{NS} \\
\end{array} \]

SFA: Saturated fatty acid, MUFA: Monounsaturated fatty acid, PUFA: Polyunsaturated fatty acid.

Table IV.—Fatty acid composition (%) of abdominal fat in geese and ducks treated by L-carnitine in drinking water (100 mg/l and 200 mg/l, respectively). Results are expressed as means ± standard errors.

L-CARNITINE AND ITS USE IN POULTRY FEEDING

Conclusions

Contradictory results were reported from studies on the L-carnitine usage in poultry feeding. Differences in doses of L-carnitine or of its precursors used in the diet, the duration of the supplementation period, supplies of metabolisable energy, fat and glucide cereals of the diet, the sex, the genotype and physiological status of the animals, the breeding and environmental conditions may be responsible for these discrepancies.

Exogenous L-carnitine supplementation could be useful in case of metabolic burdens (such as exercise, heat or cold exposure) or when energy demands are elevated (growth in young animals, high zootechnical performance or fat-enriched diet).

Moreover, the limited intestinal absorption capacity and its considerable microbial degradation would lead to increase dietary L-carnitine dosages in future investigations.

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